VOLUME XXXVIII JANUARY 1940 NUMBER 1

United States Naval Medical Bulletin

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

Issued Quarterly by the Bureau of Medicine and Surgery

Navy Department

Washington, D. C.

VOL. XXXVIII JANUARY 1940 No. 1

UNITED STATES NAVAL MEDICAL BULLETIN

FOR THE INFORMATION OF
THE MEDICAL DEPARTMENT OF THE NAVY

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DIVISION OF PUBLICATIONS
THE BUREAU OF MEDICINE AND SURGERY

THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY

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

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Compiled and published under the authority of Naval Appropriation
Act for fiscal year 1940, approved May 25, 1939

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UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON: 1940

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NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary,

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Volume XXXV, 1937, No. 1.
Volume XXXVI, 1938, No. 1.

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

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means 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 Medical Department personnel, and reports from various sources, notes, and comments on topics of professional 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 professional interest.

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

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

ROSS T. MCINTIRE,
Surgeon General, United States Navy.

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EBEN E. SMITH, *Editor*,
Commander, Medical Corps, United States Navy.

U. S. NAVAL MEDICAL BULLETIN

VOL. XXXVIII JANUARY 1940 No. 1

SPECIAL ARTICLES

ADVANCES IN CLINICAL CHEMISTRY

By Commander W. W. Hall, Medical Corps, United States Navy, and Chief Pharmacist P. S. Gault, United States Navy

In a world tuned to the clash of nations, the practice of medicine has often been termed a war on disease. In this war, clinical pathology and its increasingly important branch, clinical chemistry, assume the rôle of a service combining espionage, intelligence, and a modern mechanized highly mobile scouting force. This service supplies to the alert clinician or surgeon that commodity, indispensable to any successful campaign, information. As new methods of gathering information come upon the scene, medicine, always eager for new and better tools, puts them to the test and if they prove effective, adopts them. Thus, each year we advance by developing, here a more efficient and accurate method, there an entirely new technic, to gather the old information in a better way or to follow the advance of chemistry and physiology into new fields and discover new facts.

It is not our intention to attempt a review of recent literature, but to present some methods recently introduced which have proven of value, to discuss briefly the application and interpretation of each method and to give the technic.

To those not interested in the technic of methods, may we suggest, that they read only the preliminary discussion and the range of normal values, referring to application and interpretation in each section.

Under the heading, then, of Advances in Clinical Chemistry, we present seven methods in that field. Five of the seven are quite new, the others, though not quite so new, may not have gained the wide recognition in the service which their value and usefulness deserve.

The use of these methods in the Naval Medical School laboratories has in some cases entailed minor change or modification of the technic. In two cases (hippuric acid and formaldehyde in milk) such modification has so improved the delicacy or accuracy of the test in question that special notation of this change may properly be made and priority claimed.

SULFOSALICYLIC TEST FOR PROTEIN

FOR ALBUMIN IN URINE.[1]—This method is not new; in fact, it has been in use for a period of some 10 years. It is introduced with these

[1] Kingsbury, F. B., Clark, C. P., Williams, G., and Post, A. L., J. Lab. Clin. Med., 11: 981, July 1926.

newer methods so that wider use may be made of it. Its simplicity, accuracy, speed, and economy justify substitution of it for other methods heretofore employed in the service.

The need for rapid, accurate, and delicate methods of detecting albumin in urine is quite apparent to every one dealing with the subject. Heller's ring test which produced a contact ring precipitation, using nitric acid, was, for reasons of speed, economy, and accuracy, replaced by such tests as the heat and acetic, or heat, salt, and acetic.

While these tests represented great improvements over the nitric acid method, they did not provide reliable accurate detection of the smaller amounts of albumin, that is, from 10 to 100 mg. percent. Quantities were roughly estimated and then reported as light trace, trace, heavy trace, and large amount or, on the scale of 1 to 4 plus.

Folin and Dennis [2] in 1914 described a method of precipitating albumin from urine by means of sulfosalicylic acid. In 1922 Folin [3] devised a method of quantitative estimation of albumin in urine by comparison with albumin solutions of known strength, precipitated by the same method.

To avoid the difficulty of obtaining and analyzing sheep's serum and the preparation of standards for each day's work, Kingsbury et al.[1] developed permanent turbidity standards. These standards were made by mixing quantities of a turbidity-standardized "formazin" suspension in clarified gelatin. Formazin is the name given to the precipitate produced by the reaction of methenamine (urotropin) and hydrazine sulphate. By this method, standards are produced which correspond in turbidity with albumin solutions of known strength which have been precipitated with sulfosalicylic acid. These standards are prepared in 10 mg. percent strength, 20 mg. percent, 30 mg. percent, and so forth up to 100 mg. percent. In estimating quantities of albumin greater than 100 mg. percent the unknown specimen is diluted. The comparison obtained after precipitating 2.5 cc. of the dilution, as noted below, is multiplied accordingly.

In this method 2.5 cc. of urine or albumin solution are mixed with 7.5 cc. of 3 percent sulfosalicylic acid and allowed to stand for 10 minutes. Tubes used should correspond in dimensions with those used in the standards, which are 150 by 15 mm. These tubes are then placed between the previously prepared formazin gelatin standards and viewed in a black backed test tube rack (fig. 1), which is striped with two horizontal white lines across the black back, at the viewing level. This black backing and white striping, combined with a proper angle of top illumination, greatly facilitates comparison of turbidities.

The sulfosalicylic acid method of albumin detection and estimation has been in use in the Naval Medical School for some years. Its delicacy, accuracy, and dependability make it a very valuable test.

[2] Folin, O., and Dennis, W., J. of Biol. Chem., 18: 273, 1914.

[3] Folin, O.: Folin's Clinical Quantitative Test for Albumin in Urine. Hawk, P. B.: Practical Physiological Chemistry, 8th ed., Addendum, p. 665, P. Blakiston's Sons & Co., Phila. Pa.

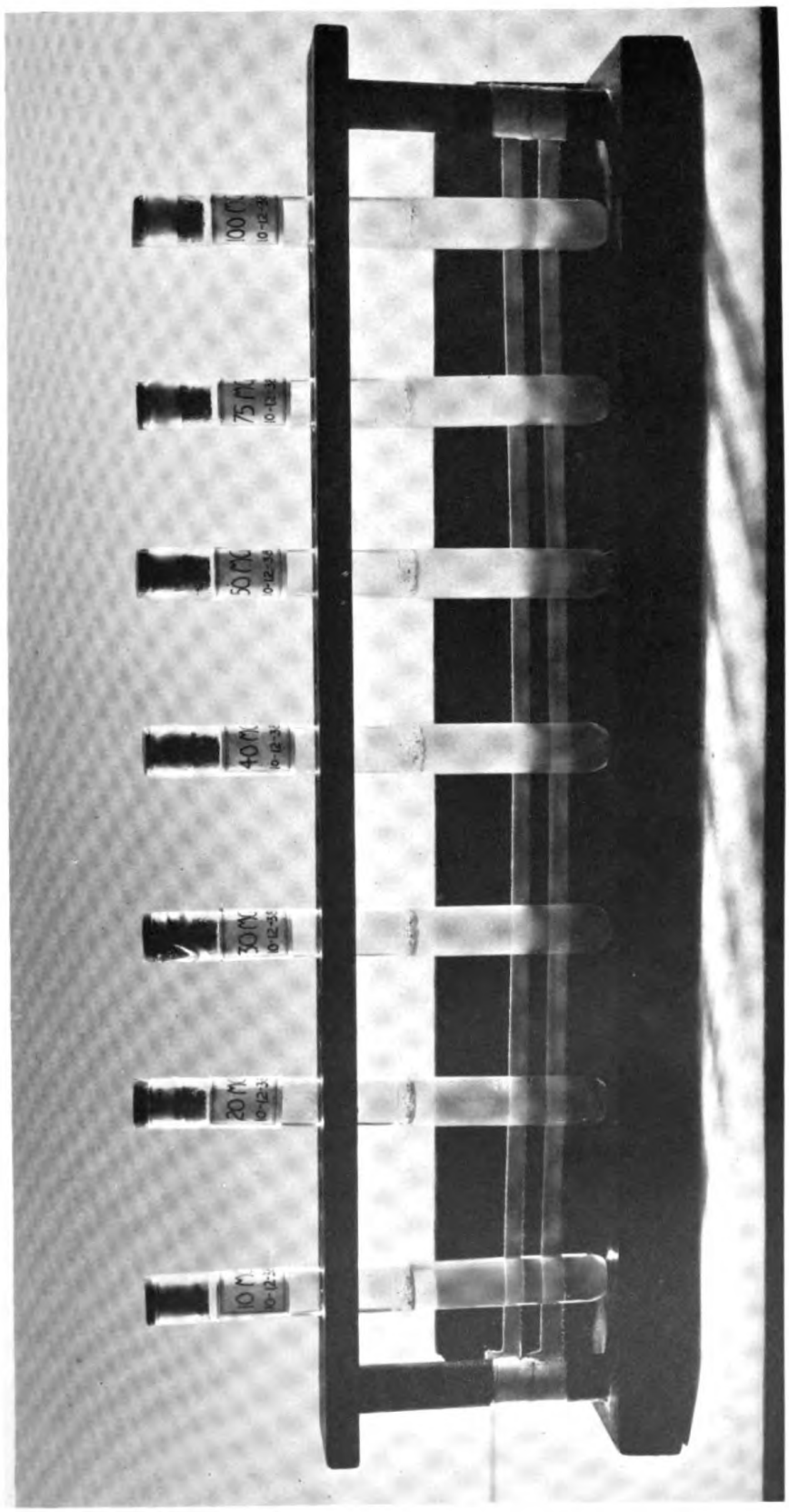


FIGURE 1.—TEST TUBE RACK WITH STANDARDS FOR SULFASALICYLIC TEST FOR PROTEIN.

Its economy and speed recommend it for general use in the service. It has been adopted as the official method for the determination of albumin in urine by the Committee on Urinary Impairment of the Association of Life Insurance Medical Directors of America.

FOR PROTEIN IN SPINAL FLUID.—This method may also be used for the quantitative estimation of protein in spinal fluid, using the same formazin gelatin standards and the following technic: [4]

Into a standard (150 by 15 mm.) sized tube, pipette 2 cc. cerebrospinal fluid and 6 cc. of the sulfosalicylic acid reagent (3%).

Allow to stand for 5 minutes and compare in standard comparator rack as for urine. If protein content is more than 100 mg. per 100 cc., dilute the spinal fluid with distilled water and recstimate. This method checks well with other standard methods and is very much more rapid and less laborious. Normal spinal fluid proteins usually run from 10 to 30 mg. percent. The high limit of normal may be set at 45 mg. percent.

Formazin gelatin albumin standards are prepared for the service by the Naval Medical School. Notes and standards are supplied to approved activities upon request to the Commanding Officer of the Naval Medical School. The technic for preparation of albumin standards is as follows: [5]

ARTIFICIAL STANDARD

Gelatine.—Digest 50 grams of sheet gelatine (Pfanstiehl Gold Label grade) overnight at 38° C. in 350 cc. of water. Complete solution by heating at 45–50° C. and dilute to 500 cc. Add the white of an egg and mix well. Heat on a boiling water bath for 1 hour and filter while hot. Do not stir so as to break up the coagulated egg white before filtering. Let cool and store the perfectly clear gelatine solution in a refrigerator. No preservative is permissible. It must therefore be used within a few days.

Formazin suspension.—Dissolve 2.5 grams of hexamethylene tetramine in 25 cc. of water. Dissolve 0.25 grams of hydrazine sulfate (Eastman Kodak Co.) in 25 cc. of water. Pour the hexamethylene solution slowly into the hydrazine sulfate solution with stirring. Stopper and let stand. In 2 to 3 hours turbidity appears and in 15 to 18 hours is completely developed. Some amorphous white precipitate can be redispersed for use. The turbidity is constant for months if protected from evaporation.

Standard dilutions.—Warm the 10 percent gelatine solution to 40° C. and add 0.3 cc. of 40 percent formaldehyde solution to each 100 cc. Excess will cause the standards to change in strength. Mix 14.5 cc. of the freshly agitated formazin suspension with 100 cc. of the 10 percent gelatine solution. Both should be at 40 to 45° C. This mixture corresponds to the turbidity obtained from 0.1 percent albumin by the procedure. Produce the series of standard mixtures as shown in the following table:

[4] Todd & Sanford: Clinical Diagnosis by Laboratory Methods, 8 ed. Saunders, p. 544.

[5] Snell, F. D., and Snell, C. T.: Colorimetric Methods of Analysis, D. Van Nostrand Co., Inc., New York City, 1937, p. 317.

TABLE 1.—*Dilution standards and protein equivalents*

Formazin-gelatine standard	10 percent gelatine	Protein equivalent	
		Mgm. per 100 c. c.	Percent
c.c.	c.c.		
50	0	100	0.10
37.5	12.5	75	.075
25	25	50	.05
20	30	40	.04
15	35	30	.03
10	40	20	.02
5	45	10	.01

Pour each standard into a suitable tube and seal with a waxed stopper. Chill well to set the gelatine, after which it will not melt at room temperature. The usual attention must be given to uniformity of dimensions of sample and standard tubes. Check against natural standards prepared from serum protein. These artificial standards are stable for at least 6 months. The particle size is similar to that of the natural standard.

NATURAL STANDARDS

Should it be desired to make natural standards for check with the artificial standards described they can be made as follows:

Allow sheep blood to coagulate and centrifuge to free from cells. Determine nitrogen by the Kjeldahl method and multiply the nitrogen value by 6.25 to get the globulin and albumin value. Dilute with 15 per cent sodium chloride solution to give a 1 percent solution of protein. Dilute with distilled water to give a 0.1 percent solution. This solution keeps indefinitely. For use dilute 1, 2, 3, 4, and 5 cc. of the solution to 10 cc., corresponding to 0.01, 0.02, 0.03, 0.04, and 0.05 per cent solutions. The diluted standards will keep for a few days in a refrigerator.

PHOSPHATASE TEST FOR PASTEURIZATION

Those interested in the newer developments in public health work and dairy technology will know this test and may have been using it for a year or two. To those who have not yet met with the test, may we say, it is the most useful thing that has come into public health control of the dairy industry in modern times. It is the long sought clue of the public health officer. It enables him to tell whether the milk has been properly or completely pasteurized, whether raw milk has been subsequently added (accidentally or intentionally), in fact, it seems too good to be true, and yet it is true.

This work, appearing first in the Journal of Dairy Science [6] was called to our attention by Captain George F. Cottle, (MC), U. S. N.

Briefly the test depends upon the following points:

(1) The enzyme phosphatase is present in all raw milk as well as in blood, urine, saliva, and other body fluids.

(2) Proper pasteurization causes its complete disappearance or inactivation.

[6] Scharer, H.: Jour. of Dairy Sci., 21: 21, January 1938.

(3) The test for the presence of the enzyme phosphatase is, in its essentials, relatively simple and depends upon the liberation of phenol from a phenolphosphoric acid ester and the development of a blue color roughly proportionate to the amount of the enzyme present. This blue color, in turn, indicates under-pasteurization and according to the elaborateness of the test, it indicates very accurately (laboratory test) or roughly (field test) the degree of under-pasteurization.

It is so very valuable because it will detect as little as 0.5 percent raw milk contamination of pasteurized milk, or it will detect under heating of only a few degrees or of shortening of the "holding" time of only a few minutes. [7] [8]

Failure in proper pasteurization is more often due to accidents in the dairy plant or carelessness on the part of handlers than to evil intent, but whether the failure is due to inaccurate thermometers, foaming or incomplete mixing or stirring in the vat, or whether raw cream is added to bring the pasteurized milk up to proper fat percentage, the phosphatase test is the third degree. It answers the question, which has heretofore been unanswerable: Has this milk been properly pasteurized?

Low bacterial counts are not *per se* evidence of pasteurization. Added formaldehyde does not interfere with the reaction. Only phenol or cresol could cause a false positive reaction.

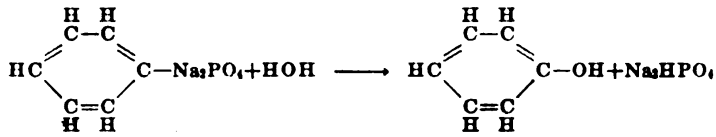
PRINCIPLES OF TEST.—Unpasteurized milk contains an enzyme known as phosphatase, or more correctly termed phosphomonoesterase which enzyme is readily destroyed by heat treatment at 142°–143° F. for 30 minutes. This enzyme destruction is, more or less, directly proportional to the time as well as the temperature of pasteurization. The holding method of pasteurization is the one most widely used in this country; it requires that milk or cream be subjected to a temperature of 142°–143° F. for at least 30 minutes. The ability of the milk enzyme (phosphomonoesterase), present in raw or improperly pasteurized milk, to split phosphoric acid esters is applied here in testing milk or cream to determine if it has been properly pasteurized. In this method the phenol which is liberated quantitatively from the ester disodiumphenylphosphate by the enzyme, is estimated. In other methods (blood) for the estimation of phosphatase, the inorganic phosphate liberated is estimated. In either case, the phosphatase is thus measured.

CHEMICAL REACTIONS OF TEST: The disodium-phenyl-phosphate ($C_6H_5Na_2PO_4$) is hydrolysed by the enzyme phosphatase liberating phenol from the former according to reaction (1):

[7] Gilcreas, F. W.: Am. Jr. Pub. Health, 29: 158, February 1939.
[8] Tiedeman, W. Von D.: Am. Jr. Pub. Health, 28: 316, March 1938.

(1) $C_6H_5Na_2PO_4+HOH \longrightarrow C_6H_5OH+Na_2HPO_4$

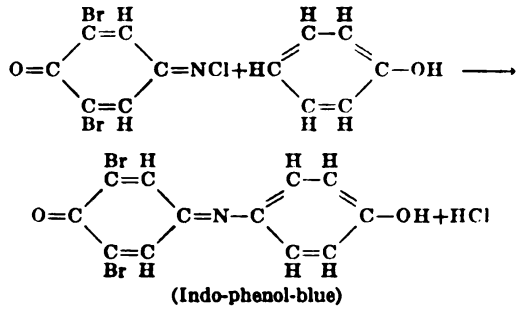
or



The BQC reagent (2.6 dibromo-quinone-chloroimide—$C_6H_2OBr_2NCl$) then reacts with the liberated phenol producing the blue colored compound indophenol-blue according to reaction (2):

(2) $C_6H_2OBr_2NCl+C_6H_5OH \longrightarrow C_6H_2OBr_2N.C_6H_4OH+HCl$
(Indo-phenol-blue)

or



(Indo-phenol-blue)

PRACTICAL VALUE OF TEST.—The FIELD test can be completed in 30 minutes or less and the appearance of any blue color in this test is indicative of improper pasteurization. The LABORATORY (quantitative) test is employed where one wishes to know the amount of enzyme present in the milk or cream. The following table will serve to illustrate. The temperature at which all specimens of milk were held was 143° F.

TABLE 2.—*Showing effect of shortening holding time in pasteurization*

Time held (minutes)	Laboratory test		Field test
	Enzyme units	Milk quality	
0	500	(Raw milk)	Strongly positive.
5	175	(Improperly pasteurized)	Strongly positive.
10	35	(Improperly pasteurized)	Positive.
15	17.5	(Improperly pasteurized)	Positive.
20	10	(Improperly pasteurized)	Weakly positive.
25	5	(Improperly pasteurized)	Weakly positive.
30	2.5	(Properly pasteurized)	Negative.

REAGENTS

Borate buffer solution.—Dissolve 28.427 grams of sodium borate, reagent grade ($Na_2B_4O_7.10\ H_2O$) in 900 cc. of distilled water. Stir vigorously while powder is being added to prevent lumping. Add 3.27 grams of sodium hydroxide in the form of a strong solution (2 to 5 normal), cool and make up to 1,000 cc. with distilled water.

Buffered substrate solution.—Dissolve 1.09 grams of disodium-phenyl-phosphate in 900 cc. of distilled water which has been previously saturated with chloroform. Add 50 cc. of borate buffer solution and dilute to 1,000 cc. with distilled water. Add a few drops of chloroform. Store in refrigerator. The pH of this buffer

solution should be about 9.6 and should be checked using thymol-phthalein as an indicator.

Should this reagent produce a blue color with the BQC reagent described below, it should be discarded and fresh reagent prepared. It has been found that some samples of disodium-phenyl-phosphate contain some free phenol. Should this occur the salt should be washed with ethyl ether (USP) until the washings give a negative test for phenol. In making this test for phenol add 10 cc. of distilled water to 100 cc. of the ether washings, evaporate off the ether, add 0.5 cc. of the borate buffer solution and 0.08 cc. of the BQC reagent. The development of a blue color indicates the presence of phenol. The washed, phenol free, salt should be briefly air dried, then dried in a desiccator and stored in refrigerator.

Basic lead acetate solution.—Boil 280 grams of dry basic lead acetate with 500 cc. of distilled water for 5 to 10 minutes. Cool, allow to settle, filter and dilute filtrate to 500 cc. with distilled water. (Use basic lead acetate prepared by the Horne method which meets American Chemical Society specifications.)

BQC reagent.—Dissolve 0.04 gram of 2,6-dibromo-quinone-chloroimide in 10 cc. of 95% ethyl alcohol. This solution is stable for several days when kept tightly stoppered in a dark bottle. It is safer, however, to prepare this reagent fresh at least once a week.

Note.—The above reagent solutions, with the exception of the basic lead acetate solution, are employed in both the FIELD and LABORATORY tests. The chemicals for their preparation may be purchased from the following firms:

Sodium Borate	Merck & Co.
Disodium-phenyl-phosphate	Eimer & Amend.
Basic Lead Acetate, Horne Method	Merck & Co.
2,6-Dibromo-quinone-chloroimide	Eastman Kodak Co.

Compressed tablets for preparing the buffered substrate solution and the BQC reagent may be purchased from R. P. Cargille, 118 Liberty St., New York City, N. Y. These tablets cannot be employed in the LABORATORY test, but are intended only for use in the FIELD test.

METHODS

Field test:

1. Place 5 cc. of buffered substrate solution in a pyrex test tube (15 x 125 mm.).
2. Add 0.5 cc. of milk. Stopper and shake well.
3. Incubate for 10 minutes in water bath at about 100° F.
4. Remove from water bath and add 0.12 cc. of BQC reagent solution. Stopper and shake well.
5. No blue color should develop after standing 5 minutes. The appearance of any blue color is indicative of improper pasteurization.

Properly pasteurized milk may show a gray to light brown color with this test. Raw milk will show a very dark blue, the intensity of the blue color is directly proportional to the degree of improper pasteurization or the percentage of raw milk present. This test will be positive wherever the LABORATORY (quantitative) test shows 5 units or more of phosphomonoesterase, as indicated in the table.

6. A blank test should be run daily on the reagents alone. If the blank test produces any blue color, the buffered substrate solution should be discarded.

Laboratory test (quantitative):

Place 1 cc. of milk in a pyrex test tube (15 x 125 mm.) by means of 1 cc. pipette. The pipette should be plugged with cotton to prevent contamination with saliva. (Saliva contains phosphatase as do other body fluids, including blood).

2. Add 10 cc. of buffered substrate solution. Shake well.

3. Incubate for 1 hour in water bath at 37.5° C.

4. Place tube in boiling water bath for 5 minutes. Cool in ice water.

5. Add 0.1 cc. of basic lead acetate solution. Shake well and allow to stand for 1 to 2 minutes. (The proteins will coagulate and separate sharply). In some instances it may be necessary to add an additional 0.05 cc. of basic lead acetate solution to completely precipitate the protein.

6. Filter through a fine filter paper (Whatman No. 40 or 42) collecting filtrate which should be water clear.

7. Place 5 cc. of the clear filtrate in a pyrex test tube (15 x 125 mm.).

8. Add 0.25 cc. of the borate buffer solution; mix gently.

9. Add 0.04 cc. of BQC reagent solution; mix gently. (Employ a 0.2 cc. serological pipette for measuring this reagent.)

10. Allow to stand 15 minutes for development of color and compare with the set of phenol color standards.

11. Report as phosphatase units. A unit being the amount of phosphatase which under conditions of the test would produce the color equivalent of 1 gamma (0.001 mg.) of phenol in 5 cc.

Phenol Standards

Stock Phenol Standard.—Prepare a standard solution of phenol in distilled water such that 1 cc. contains 1 milligram of phenol. Standardize with N/10 bromine as outlined in U. S. P. XI.

Dilute Phenol Standards.—Dilute stock phenol standard as illustrated in table 3 to prepare 100 cc. each of standards containing 0.5, 1, 1.5, 2, 3, 4, 5, 10, 20, 50 and 100 parts of phenol per million. The first column shows quantity of stock phenol standard required to prepare 100 cc. of the dilute standards.

Table 3.—*For preparation of phenol standard dilutions*

Stock phenol standard	Dilution. Parts per million	Actual quantity phenol in 100 c.c. of dilute standard	Stock phenol standard	Dilution: Parts per million	Actual quantity phenol in 100 c.c. of dilute standard
	Parts phenol	*Milligrams*		*Parts phenol*	*Milligrams*
0.05 cc	0.5	0.05	0.50 cc	5.0	0.50
0.1 cc	1.0	0.10	1.0 cc	10.0	1.00
0.15 cc	1.5	0.15	2.0 cc	20.0	2.00
0.20 cc	2.0	0.20	5.0 cc	50.0	5.00
0.30 cc	3.0	0.30	10.0 cc	100.0	10.00
0.40 cc	4.0	0.40			

Phenol color standards.—Take 5 cc. portions of each of the dilute phenol standards, add 0.25 cc. of borate buffer solution and 0.04 cc. of BQC reagent solution. Shake gently and stopper tightly. These color standards are suitable for several weeks if stored under refrigeration and kept out of direct sunlight when not in use. Label these standards in terms of phosphomonoesterase units as illustrated in table 4. A phosphatase or phosphomonoesterase unit is the amount of enzyme which under conditions of the test will produce the color equivalent of 1 gamma (0.001 milligram) of phenol in 5 cc.

TABLE 4.—*Phenol color standard dilutions*

Dilution. Parts phenol per million	Phosphomonoesterase. Units and gamma equivalents	Dilution. Parts phenol per million	Phosphomonoesterase. Units and gamma equivalents
0.5	2.5	5.0	25.0
1.0	5.0	10.0	50.0
1.5	7.5	20.0	100.0
2.0	10.0	50.0	250.0
3.0	15.0	100.0	500.0
4.0	20.0		

Note.—Buffered substrate and BQC reagent are available in tablet form for field test purposes (Cargille Co.). One tablet of buffered substrate dissolved in 50 cc. water is sufficient for 10 tests. The activity of this solution is destroyed by heat. One tablet of BQC reagent dissolved in 5 cc. ethyl alcohol is sufficient for 30 tests or more. The activity of this reagent is destroyed by heat and light. Both solutions as made from tablets are good for only 1 to 2 days.

WELTMANN SERUM COAGULATION REACTION

The Weltmann Serum Coagulation Reaction is a nonspecific laboratory test applied to blood serum. It has been known since 1930 in Europe and there it has grown steadily in favor. In 1935 Kraemer [9] reported on its use in diseases of the liver. Recently Levinson and Klein [10] reported on the use of the test, as a laboratory diagnostic aid, and compared it with the sedimentation rate. It is from their article that the technic and discussion, with the chart here presented, are taken. For a full discussion of the application of the test their article should be consulted.

Briefly the test depends upon Weltmann's [11] observation that normal serum diluted 1 in 50 contained too low an electrolyte concentration to coagulate when exposed to boiling temperatures. When the electrolyte concentration of the serum was raised by the addition of sufficient calcium chloride, it then coagulated when properly heated. Serum taken from patients suffering from certain illnesses was found to require greater concentrations of the electrolyte, than normal serum required, to produce heat coagulation. In other types of cases, sera required less electrolyte than normal.

TECHNIQUE.—The test as devised by Weltmann [11] and given by Levinson [10] is as follows:

A stock solution of 10 percent $CaCl_2 \cdot 6H_2O$ is prepared and from this, 10 dilutions are made up consisting of 0.1 percent, 0.09 percent, 0.08 percent, 0.07 percent, 0.06 percent, 0.05 percent, 0.04 percent, 0.03 percent, 0.02 percent, and 0.01 percent calcium chloride. Ten small test tubes are placed in a rack and numbered in order from 1 to 10. Into each tube is pipetted 5 cc. of the similarly

[9] Kraemer, M.: Am. Jr. Digest, Dis. and Nutr., 2: 14, 1939.
[10] Levinson, S. A., and Klein, R. I.: Ann. Int. Med., 12: 1948, June 1939.
[11] Weltmann, O.: Med. Klin., 26: 240. 1930.

numbered $CaCl_2$ solution and 0.1 cc. of hemoglobin-free serum. The tubes are shaken and placed in a boiling water bath for 15 minutes. They are then removed and the tubes which show coagulation or flocculation are noted.

INTERPRETATION: Coagulation always begins in the tubes with the higher concentrations of calcium chloride and stops toward the lower concentrations. For example, if there is coagulation in the fifth tube, there will necessarily be coagulation in the first four tubes. The number of tubes in which coagulation occurs gives us the "coagulation band" of the serum. There is usually a sharp unmistakable differentiation between the last tube which shows coagulation and the

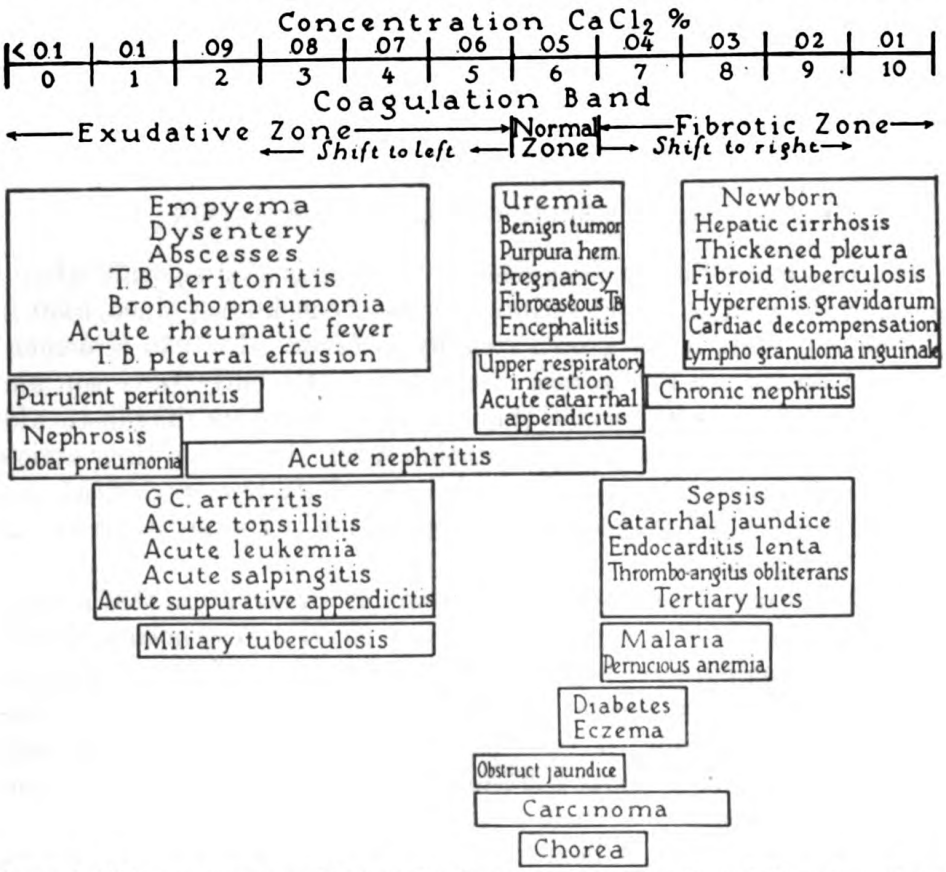


FIGURE 2.—Weltmann serum coagulation reaction illustrating three zones of coagulation and reactions in certain diseases. (From Levinson & Klein, Annals of Internal Medicine, 12: 12, 1948, June 1939) Reproduced by permission of the publisher.

next tube which may be only turbid or cloudy. Sometimes, however, there is a slight or doubtful coagulation in the intermediate tube and in such cases the coagulation band is said to be between that tube and the one before it. For example, if tube 6 has a definite coagulation, tube 8 has none, and tube 7 has a slight coagulation, the coagulation band is 6½. In normal serum the first six tubes usually show coagulation so that the normal coagulation band is 6. If the coagulation band is less than 6 the reaction shows a shift to the left, whereas if the coagulation band is greater than 6, there is a shift to the right.

As figure 2 indicates, inflammatory and exudative conditions cause a "shift to the left." That is, serum in such conditions requires a higher concentration of $CaCl_2$ before heat coagulation occurs. As noted above, the "coagulation band" is the number of tubes in which coagulation takes place. In pathological conditions it may be normal (coagulation in tubes 1 to 6), shortened (shift to the left, in which less than 6 tubes show coagulation) or lengthened (shift to the right, in which more than 6 tubes show coagulation).

Weltmann demonstrated that, in general, inflammatory or exudative diseases cause a shift to the left or shortening of the coagulation band; whereas fibrotic diseases and conditions with parenchymal liver damage cause a lengthening of the coagulation band, or a shift to the right.

It will be seen by referring to figure 2 that, in a number of conditions, in which the sedimentation rate is increased, fibrosis and exudation appear to be so balanced that they give a normal Weltmann serum coagulation reaction. While in many conditions the Weltmann reaction "shift to the left" corresponds to increased sedimentation (the exudative group) there are important and notable exceptions.

Comparison of the results of the sedimentation test and the Weltmann serum coagulation test are most informative in some instances of which a few will be cited. Acute suppurative appendicitis does not increase the sedimentation rate; it causes a definite shortening of the Weltmann coagulation band. Malaria markedly accelerates sedimentation while the Weltmann coagulation band is lengthened. Normal pregnancy increases sedimentation but the Weltmann remains normal. Subacute bacterial endocarditis and rheumatic valvulitis or carditis both cause increased blood sedimentation. In subacute bacterial endocarditis there is a Weltmann band of six or more but rheumatic carditis causes a definite shift to the left, and should in this connection prove a sign of great value. Lastly the Weltmann shift to the right, which occurs in parenchymal liver damage, should be emphasized.

Levinson and Klein emphasize the fact that the Weltmann reaction is, like the sedimentation test, a nonspecific phenomenon of unknown causation. They suggest that alteration in the amount and character of the blood proteins, the blood pH, and the blood electrolyte balance are factors of greatest importance in alteration of the Weltmann reaction. The authors stress the statement that this test is an easy and simple one, which distinguishes exudative conditions, on the one hand, from fibrotic or productive anatomic change on the other hand, and therefore should become a valuable diagnostic criterion as well as an aid in prognosis.

190162—40——2

HIPPURIC ACID LIVER FUNCTION TEST

One of the important functions of the liver is the detoxication of noxious substances that are either ingested or arise in the gastro-intestinal tract. Of the detoxication processes, the conjugation mechanisms are of particular interest because not only does the organism apply these to a wide variety of substances, but also because these mechanisms seem to be definitely interrelated with various normal physiologic functions. Thus, since any impairment of a detoxication mechanism is apt to bring about a disturbance in normal physiologic processes, the importance of determining the efficiency of the conjugation mechanisms becomes apparent.

The conjugation of benzoic acid with glycin to form hippuric acid is undoubtedly the best known and the most thoroughly studied of all the detoxication mechanisms; nevertheless, it has, until recently, received comparatively little attention in clinical medicine. The reason for this can probably be found in the fact that, due to the discovery of Bunge and Schmiedeberg that in the dog hippuric acid is synthesized only in the kidney, almost all subsequent workers tacitly assumed that in man likewise the formation of hippuric acid depended primarily on the kidney.

The results of several years' investigation indicate, however, that it is quite probable that in man, as in the rabbit, the synthesis of hippuric acid also takes place in the liver. Furthermore, Quick has demonstrated that **the rate of the synthesis of hippuric acid is dependent upon the speed with which the organism furnishes glycin.** Irrespective of the amount of benzoic acid administered, **the quantity of hippuric acid excreted per hour** (in the absence of exogenous glycin) is **relatively constant**, indicating a **definite maximum capacity of the organism to synthesize** glycin. Since it is commonly accepted that glycin is formed in the liver, it seemed probable that certain types of **liver damage might produce impairment of this synthesis**, which should result in a diminished output of hippuric acid. On this basis a new test of liver function was developed by Quick.[12] [13] [14]

THE TEST.—Six grams of sodium benzoate dissolved in 30 cc. of water is administered one hour after a breakfast consisting of coffee and toast. The patient is then given one-half glass of water. Immediately after taking the drug the patient voids, and then collects complete hourly specimens for four hours. These are preserved separately with toluene, and hippuric acid determined in each specimen.

DISCUSSION OF THE METHOD: The method of Weichselbaum and Probstein ([15]) for the determination of hippuric acid, which is a

[12] Quick, A. J.: Am. Jr. Med. Sci. 185: 630, May 1933.
[13] Quick, A. J.: Arch. Int. Med. 57: 544, 1936.
[14] Yardumian, K., and Rosenthal, P. J.: Lab. & Clin. Med., 22: 1046, July 1937.

modification of Quick's method, was tried in this laboratory and we wish to offer the following suggestions for improvement of the method.

Their modification of Quick's method consists of adding NaCl (solid) to make the urine saturated with this salt, thereby more completely precipitating the hippuric acid and leaving a constant amount of hippuric acid in solution unprecipitated. (0.123 gm. per 100 ml.). They collect the hippuric acid precipitated from the urine, wash with a saturated NaCl solution, until free of the precipitating acid, (sulfuric acid), put the crystals of hippuric acid into solution with hot water, and titrate while still hot with standard alkali (0.5 normal NaOH) using phenolphthalein as indicator. The hippuric acid is calculated as sodium benzoate and suitable correction is applied for the amount remaining in solution in the original volume of urine.

Our first suggestion for improvement of the Weichselbaum and Probstein[15] modification is as follows:

The authors offer no method for testing the washing solution for sulfuric acid, although they do advise washing the precipitated hippuric acid free of sulfuric acid. Although the failure to designate a method of testing for sulfate in the washing solution is a minor point it may be emphasized that it is highly important to remove all sulfuric acid from the precipitated hippuric acid, as the final step in the method is titration of hippuric acid with an alkali. For this reason we believe it important to call attention to the method of testing for the sulfate ion.

Test the effluent washing solution (saturated solution of NaCl and hippuric acid) as follows: Place 5 cc. of the washing solution in a test tube, add 5 cc. distilled water, 0.5 c.c. concentrated hydrochloric acid (reagent grade) and 2 cc. of 5 percent barium chloride solution. If sulfuric acid is present a white cloud of barium sulfate results. Dilution with distilled water as suggested above avoids the possible precipitation of barium chloride from solution, which precipitation is apt to occur when barium chloride is added to a saturated salt solution.

Our second suggestion: The amount of hippuric acid dissolved and lost in the washing solution is not considered by Weichselbaum and Probstein.[15] Using 1 gm. of the hippuric acid per 100 ml. and varying the wash solution amount, we find the following results:

1. One gm. hippuric acid dissolved in distilled water and precipitated was washed with 250 cc. saturated salt solution as recommended by Weichselbaum, and Probstein—recovered 0.734 gm. To this is added 0.123 gm. of hippuric acid, which, according to Weichselbaum and Probstein, is unprecipitable from the water. This results in a figure of 0.857 gms. of hippuric acid recovered.

2. One gm. hippuric acid similarly dissolved in urine, precipitated and washed with 100 cc. of saturated NaCl solution—recovered 0.842 gm. hippuric acid. To this is added 0.123 gm. as above; equals 0.965 gm. of hippuric acid recovered.

3. Repeated, using urine as in No. 2 but using 500 cc. of washing solution—recovered only 0.620 gm. of hippuric acid. Add 0.123 gm. of acid as above equals 0.743 gms. of hippuric acid as the total recovered.

From these data, it is evident that the amount of hippuric acid recovered is in an inverse proportion to the amount of wash solution used. Either a correction must be applied for the amount of acid

[15] Weichselbaum, T. E., and Probstein, J. G., Jr.: Lab. & Clin. Med. 24: 636, March 1939.

going into solution from the wash solution, which would have to be a constant amount, or a wash solution should be used that was saturated with both NaCl and hippuric acid. We recommend that the latter be used, as the former would be another empirical factor, and too, the time of contact would vary with each washing. Thus the factor would be incorrect. With the latter method, we find the following data:

1. Using water with 1 gm. hippuric acid, precipitated, etc., as before, washing solution, a saturated solution of NaCl and hippuric acid. Washed with 250 cc. of this solution—recovered 0.913 gm. of acid. Applying correction for amount remaining unprecipitable (0.123 gm. as given by Weichselbaum and Probstein) gives a figure of 1.036 gms. of hippuric acid recovered.

2. Using urine as above, washed with 100 cc. saturated solution of NaCl and hippuric acid—recovered 0.910 gm. of acid plus Weichselbaum and Probstein correction figure equals 1.033 gm. of acid recovered.

3. Using urine as above—washing solution 65 cc.—recovered 0.893 gm. of acid plus correction figure (Weichselbaum and Probstein) equals 1.016 gm. of acid recovered.

From these data it is evident that the empirical factor (0.123 gm.) in Weichselbaum and Probstein's method is much too high, and was arrived at, by the failure of the authors to account for the amount taken into solution by their wash solution. By using the above recommended procedure (saturated NaCl and hippuric acid washing solution), any amount of washing solution may be used with impunity and the empirical correction is much lowered. The average for all determinations gave us a correction figure of 0.090 gm. hippuric acid or 0.061 calculated as benzoic acid.

Procedure for the Determination of Hippuric Acid in Urine [16]

1. Measure volume of urine and record. (If above 150 cc., add a few drops of glacial acetic acid, and evaporate to volume not in excess of 150 cc.)

2. Add 30 gms. of NaCl (reagent grade) per 100 cc. of urine and heat with shaking until dissolved. (cc. x 0.3 equals amount of NaCl to be added.)

3. Cool solution to 15–20° C. in ice cold water bath, add 1.2 cc. of 10 times normal H_2SO_4. (10/N)

4. Scratch the sides of the flask with a glass stirring rod to initiate proper crystallization and stir thoroughly.

5. Allow to stand for 15 minutes in cold water, then filter.[17]

6. Wash precipitate with prepared washing solution (paragraph under reagents).

7. When free of sulfuric acid (test 5 cc. portion of washings for SO_4 by adding 5 cc. of distilled water, 0.5 cc. concentrated HCl and 2 cc. of 5% $BaCl_2$) dissolve precipitated hippuric acid in hot water quantitatively and titrate while still hot with N/5NaOH, using 2–3 drops of 1% alcoholic phenolphthalein.

[16] We wish to give credit to Ozburn, E. E., pharmacist's mate second class, for especially valuable work in connection with the development of this modification.

[17] In filtering hippuric acid, a Jena Crucible (obtained from Fisher Scientific Co., A. H. Thomas, or other standard laboratory supply houses) size G-3 is recommended, as it speeds up the work considerably. Only one is needed for any number of determinations, although three or four will save time needed for filtering. A clogged filter may be cleaned by washing with alcohol, hot water, aqua regia, and finally with distilled water until no aqua regia remains.

CALCULATIONS: Hippuric acid equals cc. of alkali used in the titration multiplied by the factor 0.03584. To this figure add for each 100 cc. of urine, 0.09 gm. (Amount of hippuric acid remaining unprecipitable in each 100 cc. of urine). Report as amount excreted in each hourly specimen, and also the total excreted.

1. To convert hippuric acid to sodium benzoate multiply amount of the hippuric acid by the factor 0.8043.

2. To convert hippuric acid to benzoic acid multiply amount of the hippuric acid by the factor 0.6816.

REAGENTS—*N/5 NaOH.*—(8 gms. of pure NaOH per liter). Place 21 cc. of carbonate free concentrated NaOH (Stitt [18]) in a 2 liter volumetric flask. Add distilled water to mark and mix. This is standardized against an exact strength potassium acid phthalate (reagent grade) using phenolphthalein as an indicator.

NaCl–Solid.—Reagent grade should be employed as U. S. P. grade may contain traces of sulfate.

Washing Solution.—Dissolve 300 gms. of NaCl in 1 liter of water and heat until dissolved. Add hippuric acid crystals, 1.0 gm. (Pfanstiehl) while heating. Allow to cool and filter through a Jena filter before use.

Sulfuric Acid, 10/N, 30% by Volume.—(30 cc. concentrated H_2SO_4 added to 70 cc. distilled water).

NORMALS.—The excretion of hippuric acid in normal subjects is given as one or more grams for the second and third hours with a total of 3 or more grams for 4 hours. (Most normals fall between 3.0 and 3.5 grams for the 4-hour period.)

TOXIC SYMPTOMS due to sodium benzoate are of two kinds: (1) Those due to local irritation of the gastro-intestinal mucosa, with nausea, vomiting, diarrhea, anorexia; and (2) central nervous system with headache, tinnitus, vertigo, giddiness, and reflex vomiting.

Vomiting is one of the difficulties encountered when the test is applied to toxic patients. In other patients it occurs only rarely and it is greatly minimized by allowing a light breakfast prior to the test. The test has no value in patients with advanced cardiac decompensation, for in chronic passive congestion of the liver the elimination is very low. The other disadvantage of the test is that it cannot be applied to cases of nephritis with nitrogen retention, since hippuric acid behaves like other nitrogenous excretory products. Furthermore, it is contraindicated in cases of nephritis with nitrogen retention. Fortunately this complication is not frequently encountered in the ordinary liver cases.

Quick [13] reports regarding this test:

While the test measures only one mechanism of the liver, it must be realized that the various functions of this organ are not distinct and independent, but are closely interrelated, so that an injury to any one mechanism is apt to affect several others as well. Many examples of this interlocking of functions can be cited. Thus, the synthesis of amino acids such as glycin, on the one hand, depends on a precursor which is presumably derived from the metabolism of carbohydrates, while the formation of bile acids, on the other hand, requires an ample supply of glycin and taurin. Furthermore, it is quite probable that the so-called detoxication mechanisms are in reality mainly concerned with normal metabolic processes, and the conjugation of abnormal substances is only an inci-

[18] Stitt, Clough and Clough, Blakiston: Practical Bacteriology, Haemotology and Parasitology, 9th ed., p. 874.

dental function. For example, the mechanism that synthesizes glycocholic acid, i. e., brings about the conjunction of cholic acid with glycin, is probably the same which effects the union of benzoic acid with glycin. It is significant that obstructive jaundice, which brings about a decrease in the formation of bile acids, as has been noted by Greene, Walters and Fredrickson, Ravdin, and others, also causes a reduction in the output of hippuric acid. As further parallelism, the production of bile acids and hippuric acid does not immediately return to normal after the relief from the obstruction but is definitely delayed.

This test is valuable to differentiate intrahepatic and extrahepatic jaundice. In liver damage the elimination is very low, while in acute obstruction, the elimination is within normal limits. However, in chronic obstruction where there is liver damage, the differential diagnosis will be impossible with this test.

FORMALDEHYDE IN MILK AND CREAM [19] [20]

The use of formaldehyde as a preservative for milk is of course illegal in all jurisdictions. It is legislated against not so much because it is harmful in itself to the consumer, for in the concentrations used it probably is not, but because the use of formaldehyde allows a dirty and probably dangerous milk to meet low bacterial count standards and thus masquerade as a good safe product. In spite of the illegality of the practice, ignorant or unscrupulous farmers and dairymen still occasionally resort to the use of formaldehyde or to one of the milk preservatives, essentially formaldehyde, which are sold by unscrupulous houses for this purpose. The farmers have often been deluded by the statements that the preservative is harmless, and that its presence cannot be detected.

Repeatedly it has been shown that formaldehyde is the most efficient of all milk preservatives. The strength employed is usually one part of formaldehyde gas in 20,000 parts of milk, to one part in 50,000. The cheapness, effectiveness, and availability of formaldehyde make it necessary to constantly be on guard against its use.

Formaldehyde added to milk gradually disappears owing to the formation of condensation products with the proteins.[21] For this reason it is desirable to have a very sensitive test for the detection of this preservative. In the original Hehner test the purple contact ring formed in the presence of formaldehyde is very hard if not impossible to detect, in high dilutions of formaldehyde. This difficulty is due to the interferring deep brown color resulting from charring of the lactose by the concentrated sulfuric acid.

Fulton[19] showed that the Hehner test could be made much more sensitive by the use of bromine as an oxidizing agent. He employed a saturated bromine solution in sulfuric acid or a crystal of potassium

[19] Fulton, C. G.: The Hehner Test for Formaldehyde, Ind. & Eng. Chem., Analy. Ed., 3:2, p. 199, 1931.
[20] Leach and Winton: Food Inspection and Analysis, 4th ed. John Wiley & Sons, p. 165, 1920.
[21] Williams, R. H., and Sherman, H. C.: J. A. Chem. Soc. 27: 1503, 1905.

bromide in diluted sulfuric acid as the oxidizing agent. The strength of sulfuric acid which he employed ranged from 1.6 cc. sulfuric acid to 1 cc. of water up to 5 cc. sulfuric acid to 1 cc. of water.

We have found Fulton's test to be very sensitive when employing his saturated bromine solution in sulfuric acid as the oxidizing agent. The strength of sulfuric acid which he employs, however, produces a faint pink tinge in the negative control. In order to overcome this interferring color and to eliminate the hazards associated with the handling of liquid bromine as well as the delayed color reaction when employing potassium bromide, the modification here reported has been developed.

AUTHORS' MODIFICATION OF THE HEHNER TEST

It was found in these laboratories that an aqueous bromide-bromate solution serves better than the sulfuric acid-bromine reagent, is easier to prepare, remains stable over a longer period, and particularly, it eliminates the hazards associated with the handling of liquid bromine. The bromide-bromate solution in the presence of sulfuric acid gives an immediate color formation due to the rapid release of bromine by the oxidizing action of the bromate; whereas, the reaction in the presence of potassium bromide alone is greatly delayed due to the very slow release of bromine by the sulfuric acid. As to the strength of the sulfuric acid it was found that a 50 percent (by volume) aqueous solution gave excellent results. With this strength acid no interfering color was produced in the negative control, the curd first formed was completely redissolved, and the sensitivity of the test was not decreased. The color, though perhaps slightly less intense, is easily recognized even in a milk which contains one part of formaldehyde in one million.

Method

1. Place 5 cc. of the sulfuric acid reagent in a test tube (15 x 150 mm.).
2. Add 1 cc. of milk or cream to be tested, mix well to be sure that all curd is redissolved. A colorless, slightly turbid solution is formed.
3. Add 0.5 cc. of the bromide-bromate solution and mix well.

Reagents

Sulfuric acid reagent.—Pour slowly 50 cc. of reagent sulfuric acid into 50 cc. of distilled water. Allow to cool and keep in a glass stoppered bottle.

Bromide-bromate solution.—Dissolve 3.0 grams of reagent potassium bromide and 0.3 grams of reagent potassium bromate in sufficient distilled water to make 100 cc. Store in a glass stoppered bottle away from direct sun light.

Reaction

If formaldehyde is present a violet color develops at once. The color for very small proportions of formaldehyde, is purplish-pink, after standing about 5 minutes. If formaldehyde is present in greater proportion than 1 to 5,000, which

would practically never occur, (and in which circumstances, probably, your nose knows) no purple color will be formed, but instead a fairly deep yellow color develops. In such case the milk can be diluted with distilled water, or with a known negative milk, and the test repeated. If no formaldehyde is present the original colorless, slightly turbid solution remains unchanged.

Formic acid, acetaldehyde, benzaldehyde, glycerin, maltose and dextrose have been found not to interfere with this test. It has been in use at this school for 1 year and has proven very satisfactory.

DETERMINATION OF SULFANILAMIDE IN BLOOD

The introduction of sulfanilamide into the field of chemotherapy has been without doubt the greatest thing that has occurred in medicine in the past hundred years. It may startle one to think that such a statement means that sulfanilamide should rank ahead of salvarsan; yet salvarsan revolutionized the treatment of only one disease, while sulfanilamide and the drugs in its class, which already have begun to follow it upon the scene, have revolutionized the treatment of probably half a dozen or more. We have just seen the beginning of the parade of progress that the introduction of sulfanilamide began.

In the use of sulfanilamide it is unquestionably established that therapeutic effectiveness is a direct function of the concentration of the drug in the body tissues and fluids. Quantitative methods for the determination of the concentration of the drug in the blood and urine therefore, early became an essential in this new departure in chemotherapy. It is well recognized that most, if not all, of the toxic reactions of sulfanilamide may result from concentrations too low to produce the desired therapeutic effect. It appears then that the dosage of sulfanilamide should be held to the desired level of concentration, or the drug withheld entirely.

Data so far available [22] do not indicate clearly that the same correlation of dosage, blood concentration, and therapeutic results hold with sulfapyridine as with sulfanilamide. Further experience with sulfapyridine is necessary and perhaps we must await the advent of more readily soluble and absorbable compounds of sulfapyridine.

Quantitative Determination of Sulfanilamide in Blood [22]

Procedure:

1. Place one volume (4 cc.) of oxalated blood in a 125 cc. Erlenmeyer flask.
2. Add 8 volumes (32 cc.) of distilled water, mix and allow 2 minutes to complete laking of blood.
3. Add slowly, while gently rotating the flask, 1 volume (4 cc.) of 20% trichloracetic acid. Continue rotation of flask until well mixed and allow to stand for 5 minutes.

[22] Pepper, S. D., Flippin, H. F., Schwartz, L., and Lockwood, J. S.: Am. Jr. Med. Sci., **198**: 22, July 1939.

[22] Marshall, E. K., and Litchfield, J. T.: Science **88**: 85, July 1938.

4. Filter through fine, dry filter paper (Whatman no. 42). Filtrate should be clear and colorless.

5. Place 10 cc. of the filtrate in a test tube (25×150 mm.) having graduation marks at 12.5 and 25 cc.

6. Prepare a standard in a similar tube as follows:

	cc.
Sulfanilamide (working standard)	1.0
Distilled water	3.5
20% trichloracetic acid solution	1.0

7. Add to all tubes (unknown and standard) 1 cc. of the 0.1% sodium nitrite solution and allow to stand for 3 minutes.

8. Add 1 cc. of the ammonium sulfamate solution. Mix and allow to stand for 2 minutes.

9. Add to all tubes 5 cc. of the dimethyl-alpha-naphthylamine solution. Mix and allow to stand for 5 minutes for development of color.

10. Dilute the UNKNOWN to 25 cc. and compare grossly with the standard. If standard appears too strong dilute to 25 cc. with distilled water, making a WEAK standard. Compare in a colorimeter using all-glass cups.

CALCULATIONS:

Strong standard (12.5 cc.):

$$\frac{S}{U}\times\frac{0.05}{1}\times\frac{25}{12.5}\times\frac{100}{1} \text{ or}$$

$$\frac{S}{U}\times 10 = \text{mgms. of sulfanilamide in 100 cc. of blood.}$$

Weak standard (25 cc.):

$$\frac{S}{U}\times\frac{0.05}{1}\times\frac{100}{1} \text{ or}$$

$$\frac{S}{U}\times 5.0 = \text{mgms. of sulfanilamide in 100 cc. of blood.}$$

REAGENTS: *Trichloracetic acid, 20% aqueous solution.*—This solution should be titrated and adjusted so that 100 cc. contains 20 grams of the acid. (U. S. P. assay method may be employed.)

Sodium nitrite ($NaNO_2$) 0.1% aqueous solution.—If kept in ice box, this solution will remain satisfactory for about 1 month.

Ammonium sulfamate solution.—1 molar dihydrogen sodium phosphate containing 0.5% ammonium sulfamate ($NaH_2PO_4.H_2O$, 13.8 grams; ammonium sulfamate, 0.5 gram; water 100 cc.). This solution is used to destroy the nitrous acid which causes the color to fade rapidly.

Dimethyl-alpha-naphthylamine solution.—1 cc. of dimethyl-alpha-naphthylamine in 250 cc. of 95% ethyl alcohol. Keep this solution in ice box. This chemical may be obtained from the Eastman Kodak Co.

Sulfanilamide standard solution.—Prepare a 0.1% solution in distilled water. *Working standard:* place 5 cc. of the 0.1% solution in a 100 volumetric flask, make up to the mark with distilled water. (1 cc. of this working standard contains 0.05 mg. of sulfanilamide.)

Note.—A buffered solution of ammonium sulfamate is used to destroy the excess nitrous acid derived from the sodium nitrite. This results in a more rapid color development as well as a more permanent color.

Ammonium sulfamate can be obtained from Le Mott Chemical Products Company, Baltimore, Md.

Determination of Sulfapyridine in Blood

The procedure for the determination of sulfanilamide in blood as given above may be adapted to the determination of sulfapyridine, using a sulfapyridine standard.

Reagents:

A *stock standard* of sulfapyridine containing 200 mgms. per liter is prepared as follows:

Warm 700 cc. of distilled water to 50° C., place in a liter volumetric flask, add 200 mgms. sulfapyridine and shake vigorously. Cool and add water to mark. This solution contains 20 mgms. per 100 cc. (20 mg%).

The sulfapyridine *working standard* is prepared as follows:

Place 25 c.c. of above stock standard in a 100 cc. volumetric flask. Dilute to mark with distilled water and mix well. One cc. of this working standard contains 0.05 mg. of sulfapyridine.

PROTHROMBIN AND VITAMIN K

Surgery on jaundiced patients has always been attended with added risk, due to the danger of so called cholemic bleeding. In the past two decades much has been done to reduce surgical mortality in such cases by the use of glucose and perhaps also by the use of calcium. Toxic liver damage was much reduced by this method of protecting the liver and increasing its functional reserve. However, current figures [24] still indicate that cholemic bleeding accounts for about 50 percent of the surgical mortality in such cases, and that cholemic bleeding, of itself, carries a surgical risk of about 5 percent.

It is now generally recognized that this hemorrhagic tendency in the presence of jaundice is not occasioned by the presence of bile salts, by bilirubin, or by any alteration in the amount of calcium, platelets, fibrinogen or thromboplastin.[24] Quick's original observation[25] that hemorrhage in jaundice was due to the lack of one substance, prothrombin, not previously studied, has now been amply confirmed. It also appears well proven that a certain fat soluble substance, normally present in leafy green foods, notably in alfalfa, and also formed by bacterial action, that is, vitamin K, is necessary to maintain proper prothrombin level. This substance is absorbed, as the fats are absorbed, with the aid of bile, through the intestinal wall and carried to the liver, where it is used in the formation of prothrombin.

The nature, composition or chemistry of prothrombin is not known, though its function as a physiological complex which has the capacity to form thrombin is well known.

As we are herewith burying some of the previously held concepts in connection with the mechanisms of coagulation defects in blood, it is only proper that some appropriate words be said over the bier. For this purpose we quote from the discussion of Quick, Brown and Bancroft,[25] on the subject of fibrinogen and calcium as follows.

[24] Butt, H. R., Snell, A. M. and Osterberg, H. E.: J. A. M. A. **113**: 383, July 1939.

[25] Quick, A. J., Stanley-Brown, M., and Bancroft, F. W.: Am. Jr. Med. Sci. **190**: 501, October 1935.

FIBRINOGEN.—The concentration of fibrinogen in plasma varies normally from 0.3 to 0.75%, but this wide range has no demonstrable effect on clotting time. We have even found that adding purified fibrinogen to plasma does not alter the speed of clotting. It seems fairly certain that fibrinogen plays an entirely passive rôle, and that the rate of clotting depends primarily on the concentration of thrombin. Nevertheless, it is obvious that in the total absence of fibrinogen, no clotting can occur, and interestingly, 2 such cases have been reported in which fatal hemorrhage had to be attributed to a lack of fibrinogen in the blood. This condition Wohlisch has named pseudohemophilia. It can be reproduced in animals as illustrated by the following experiment:

A freshly prepared and filtered saline extract of rabbit brain (20 cc.) was injected intravenously into a dog. A severe reaction occurred immediately, respirations became shallow, and a nystagmus developed. The fibrinogen which was 0.44% before the injection dropped to 0.09 in 5 minutes and to a mere trace 30 minutes after the injection. The dog was killed 1 hour later and autopsied. No definite intravascular clotting could be found, but there was a relatively small pulmonary infarct and also a small mesenteric infarct.

While fibrinogen does not influence the clotting time, it is conceivable that if its concentration is too low, a fragile and poorly adhering clot will form. In hemophilia, however, a normal fibrinogen concentration has been found by all investigators. In obstructive jaundice the literature is more conflicting. The work of Doyon, Whipple and Hurwitz, Foster and Whipple and others show that liver damage can markedly reduce the blood fibrinogen. Recently, however, Linton studied a series of obstructive jaundice cases, and found an elevation of fibrinogen even in those patients that died from hemorrhage. We have obtained essentially similar results. It seems fairly improbable, therefore, that fibrinogen deficiency can be the causative factor for the hemorrhagic diathesis.

CALCIUM.—Contrary to the widely accepted view that ionized calcium of the blood influences the rate of clotting, it seems more likely that it is only the organically combined calcium which is directly essential for the formation of thrombin. Scott and Chamberlain have demonstrated that the quantitative removal of ionized blood calcium with oxalate will not prevent clotting; and they as well as Vines, and Steward and Percival have found that it requires 3 times the theoretical amount of oxalate before clotting is completely inhibited, which suggests that the organically bound calcium must be precipitated before the clotting process is stopped. Eagle in a recent paper discussed the possibilities that thrombin may be a calcium-containing compound. Since these same considerations may be applied to prothrombin, it is likewise conceivable that in the latter, calcium may be an essential part of the molecule and that therefore in the conversion of prothrombin to thrombin, inorganic calcium perhaps plays no direct rôle. Ionized calcium is however a dominant factor in platelet lysis, as Ferguson has convincingly demonstrated in a recent work, thus confirming the observations of the older investigators. The question why calcium only becomes effective in shed blood still remains unanswered. It is doubtful whether clinically even marked changes in the concentration of inorganic blood calcium have any significant influence on the clotting time. No hemorrhagic diathesis has been observed in the extreme hypocalcemia of parathyroid deficiency, nor has intravascular clotting been reported in cases of hyperparathyroidism in which hypercalcemia of long duration often was present. Experimentally, Ravdin, Riegel, and Morrison could demonstrate no change in the coagulation time of blood after inducing a hypocalcemia in dogs by parathyroidectomy, nor by bringing about an elevation of blood calcium with parathormone. In hemophilia the concentration of calcium in blood is normal, and in obstructive jaundice, even in severe hemorrhagic cases, no significant change in blood calcium is found. Wangensteen who has carefully reviewed

the literature concluded that a deficiency of calcium has not been adequately demonstrated in any hemorrhagic disease. Ravdin and his coworkers were unable to reduce the clotting time of normal and of jaundiced dogs by intravenous calcium injections. It is probable that any beneficial effect obtained by calcium administration may be due to its therapeutic action on liver function rather than on any direct influence on clotting.

Eagle has demonstrated that the amount of thrombin formed is independent of calcium and platelets but varies directly with the plasma factor (prothrombin).

Discovery of the existence of vitamin K, study of its physiology and function, concentration, isolation and purification, and now at least partial chemical identification of the substance, have followed one another with almost bewildering rapidity.

Only 9 years ago Dam [26] of Copenhagen noted that when newly hatched chicks were placed upon a fat free diet they developed spontaneous and traumatic hemorrhages into the skin mucous membranes and other parts of the body. Subsequent studies by Dam and his colleagues and others, notably Almquist of California, established the following facts as given by Smith and associates.[27]

1. Vitamin K is present in large amounts in certain green vegetables such as alfalfa, spinach, and kale. The vitamin is a colorless compound, however.

2. The vitamin can be produced by bacterial action and is therefore present in the lower portion of the intestine, even when the animal is maintained on a diet free of the vitamin. Absorption from the lower part of the intestine is minimal in the chick, but in mammals absorption does occur and hence mammals rarely show vitamin K deficiency except in cases of faulty absorption.

3. By rather elaborate chemical technic it has been possible to isolate the vitamin in the form of a highly potent oil.

4. In chicks maintained on a diet free of vitamin K it was found that the prothrombin level was low, and this explained the bleeding. It has not yet been determined whether the vitamin K enters chemically into the formation of prothrombin or whether the vitamin merely keeps certain tissues in a normal healthy state of activity essential for the formation of prothrombin. The Danish word for coagulation is spelt with a K (koagulation), therefore the name, vitamin K.

As previously noted, man and other mammals, obtain vitamin K from their diet and from the substance produced *de novo* by bacterial action in the intestines. In this connection, birth or neonatal hemorrhages in children may be mentioned. Prothrombin has been reported as being very low in the blood of the child shortly after birth. It appears to be about normal just at birth, to drop sharply soon after, and then to rise later. This rise perhaps corresponds to the development of a luxuriant bacterial garden, in the child's intestinal tract, for the manufacture of vitamin K. Incidentally, one of the original sources of vitamin K was decayed fish. It was later found that the bacteria, not the fish, produced the vitamin. Fowls do not absorb from the lower gut, as do mammals, therefore all their vitamin K

[26] Dam, H.: Ueber die Cholesterinsynthese in Tierkörper, Biochem. Zeitschr., **220**: 158, April 1930.
[27] Smith, H. P., Siffren, S. E., Owen, C. A., and Hoffman, J. R.: J. A. M. A., **113**: 380, July 1939.

requirements must be supplied from the dietary. Vitamin K is a compound having the solubility properties of fats. Bile salts are therefore necessary for its intestinal absorption.

It may be noted that this is also true of vitamins A, D, and E, yet deficiencies of these vitamins are not noted in jaundice. This apparent contradiction is explained by the fact that these vitamins are stored in the body in considerable amounts, while vitamin K is not stored to any great degree.

In conditions in which no bile reaches the intestine, obstructive jaundice and biliary fistulae, the absorption of the vitamin is interrupted. However, the utilization by a functionally active liver of enough vitamin K to produce an adequate supply of prothrombin being the essential point, the control of a deficiency of prothrombin depends upon several factors [24]: (*a*) a normal diet containing the antihemorrhagic vitamin, (*b*) the presence of adequate amounts of bile salts in the intestine, (*c*) a normal intestinal absorptive surface and (*d*) a physiologically active normal liver.

Butt, Snell, and Osterberg [24] note that:

> In a majority of patients suffering from jaundice, the second and fourth factors may be impaired singly, or more usually, in combination. The routine methods of treatment suggested in this report may become obsolete when vitamin K is available in pure form or when it is possible to administer this vitamin by the parenteral route. No matter which method eventually may be employed, the importance of anticipating the danger of prothrombin deficiency and detecting it by means of laboratory studies will remain a paramount consideration in successful treatment.

In treating patients, bile or bile salt must be fed along with the vitamin to permit the absorption from the intestine of the latter. It is customary to feed daily at least an ounce of bile or its equivalent in the form of dried bile or bile salt.[27] Smith and associates [27] report two cases which failed to respond on this dosage when given along with the usual dose of vitamin K. They did respond, however, when the dose of bile was increased 5 fold, the dose of vitamin K being unchanged. It is probable that some patients require more bile than others; also that some require more vitamin K than others. Evidence now available indicates that bile salt may be as effective as whole bile, but further work is needed in regard to this.

The rate at which the plasma prothrombin level rises with vitamin K therapy varies in different cases. As a rule a definite rise occurs in 24 hours, but from 3 to 8 days of treatment are usually needed to obtain the maximal response.

These authors [27] further strongly emphasize that the vitamin treatment should be continued during the postoperative period, especially if bile continues to drain from the wound. In addition to the lack of vitamin, such patients no doubt consume prothrombin in large amounts

in forming fibrinous exudate in the margins of the wound. It is not an uncommon experience to see wounds which in the first few postoperative days seem to be healing by first intention but shortly the margins separate and the wound gapes with absolutely no further tendency to agglutinate or to heal. In all cases the clotting test, herewith described, should be performed every few days during the postoperative period in order to guard against a disastrous fall in prothrombin.

Finally, it need hardly be mentioned that vitamin K is of no value in the treatment of hemophilia or thrombocytopenic purpura. In these diseases there is no deficiency in vitamin K and no deficiency in prothrombin; *i. e.*, plasma from a case of hemophilia clots in the same time as normal plasma, when an excess of thromboplastin is added. Nor can one expect a response to the low prothrombin levels often present in cirrhosis of the liver or in cases of severe hepatitis. In these cases the "factory" that forms prothrombin is disordered, and the disease is not essentially one of vitamin K deficiency.

Vitamin concentrates have been prepared and are marketed commercially by at least two laboratories (Abbott Laboratories, North Chicago, Ill., under the name of Klotogen; Cerophyl Laboratories, Kansas City, Mo., under the name of Cerophyl). Probably other equally satisfactory products will, by the time of publication, be available.

Following the discovery that the capsule of the tubercle bacillus contained a substance which was effective as vitamin K, a number of laboratories quickly and almost simultaneously announced the identification of this substance (phthiocol) as a naphthoquinone. It seems now to be generally agreed that the vitamin contains a quinone structure. Thus Almquist and Klose [28] declare that the antihemorrhagic activity of phthiocol lies between that of the methyl naphthoquinone and the hydroxynaphthoquinone.

Ansboches and Fernholz [29] found, in confirmation, that 2 methyl, 1, 4 naphthoquinone is practically as active as vitamin K. Though this work is too recent for any amount of clinical work to have been done with phthiocol or synthetically prepared quinones, Smith, et al.[27] state in a footnote to their previously cited article:

> Since this paper was written, Almquist and Klose have shown in chicks that phthiocol, a yellow compound prepared synthetically by R. J. Anderson, has vitamin K activity. Immediately thereafter work from that and other laboratories showed that vitamin K activity was possessed by a number of other naphthoquinones.
>
> With the aid of Dr. Joseph E. Flynn we have recently had success in the use of phthiocol intravenously in cases of obstructive jaundice. In one case, 45 cc. of 0.2 percent solution in isotonic phosphate buffer (pH 7.4), given in divided

[28] Editorial, J. A. M. A. 113: 5, July 1939.

[29] Ansboches, S., and Fernholz, E.: J. Am. Chem. Soc. 61: 1924, July 1939.

doses, raised the prothrombin from the 39 to the 75 percent level in 24 hours. No toxic effect was observed.

Accepting the formula for the formation of fibrin as (*a*) prothrombin plus thromboplastin plus calcium equals thrombin and (*b*) fibrinogen plus thrombin equals fibrin, it is apparent that, if thromboplastin and calcium are furnished in adequate amounts, the rate of clotting is proportionate to the amount and utilization of prothrombin present. On the basis of this reasoning Quick developed a method for the estimation of prothrombin coagulation time.[25] Quick pointed out that we have bleeding in jaundice when the level of prothrombin reaches a point as low as 20 percent of the normal and that, conversely, as long as the level remains above 20 percent, no prolonged clotting time occurs. This would explain why a jaundice patient, who has an apparently normal clotting time, determined by the usual methods, suddenly and unpredictably begins to bleed following an operation, although the amount of blood lost at operation has not been great. It would indicate that at the beginning there was a reduced amount of prothrombin which fell to 20 percent or less of the amount normally present, possibly as a result of even a small amount of blood loss.

Quick's Method for Determining Prothrombin Time

1. The blood is oxalated and the plasma obtained.
2. Plasma is mixed with an emulsion of dried rabbit brain to supply an excess of thromboplastin.
3. Calcium chloride is then added to the mixture.

The number of seconds it takes to form a clot in a 37.5° C. water bath is recorded as the prothrombin time.

Magath [30] found that the temperature of the bath was a critical factor and that the brain emulsion varied in its effect, therefore, a normal standard must be run with each batch of unknowns. Normal values are about 20 seconds. Bleeding tendencies in the presence of jaundice are not apparent when values below 40 seconds are obtained. Conversely, above 40 seconds, hemorrhagic tendencies are constantly observed. A quick, simple, direct method of quantitative estimation of prothrombin, though needed, is not at hand at present.

Magath [30] suggested that, lacking a quantitative method for prothrombin, the test also be performed with the plasma diluted with equal amounts of normal sodium chloride whenever the clotting time was less than 40 seconds. If the coagulation time was then prolonged beyond twice the clotting time of undiluted plasma, one should then conclude that there was actually a prothrombin deficiency, which, after operation or loss of blood, might cause hemorrhage.

The method of Quick is quite satisfactory as an indirect test for prothrombin, but it has the disadvantage of requiring a centrifuge and a titration procedure.

[30] Magath, T. B.: Proc. Staff Meet. Mayo Clin., 13: 67, February 1938.

The test devised by Smith, et al.[27] is similar in principle to Quick's test but is carried out on the whole blood at the bedside and is so simple that it can be mastered with very little practice. As an excess of thromboplastin is added and as calcium has been found not to be sufficiently deficient at any time, due to disease, to interfere with proper coagulation, prothrombin then becomes the variable which determines the rate of coagulation in relation to normal. The technic of the Smith test for vitamin K deficiency is given by the authors as follows: [27]

SIMPLIFIED TEST FOR VITAMIN K DEFICIENCY

With a serologic pipet, 0.1 cc. of thromboplastin, described in the next paragraph, is placed in a small serologic tube (75 x 10 mm. outside diameter). In the tube is then placed blood, freshly drawn from the patient, up to a 1 cc. mark previously made on the side of the tube. The tube is at once inverted over the finger to obtain complete mixing of the blood and thromboplastin. The tube is tilted every second or two in order to observe clotting. As a control, the test is also carried out on the blood of a normal subject. The calculation is as follows:

$$\frac{\text{Clotting time of normal control}}{\text{Clotting time of patient's blood}} \times 100 = \text{Clotting activity (In percentage of normal)}$$

Thus, if the patient's blood clotted in 48 seconds and the normal person's blood in 24 seconds, the clotting activity is calculated to be 50 percent of normal.

To prepare thromboplastin, fresh lung of ox or rabbit is ground, and to each 10 gm. portion is added 10 cc. of physiologic solution (0.9 percent) of sodium chloride. This is stirred at intervals for several hours. The fluid then obtained by straining through gauze is the "thromboplastin" employed in the test described. This thromboplastin keeps well in the ice box.

Warner, Brinkhous, and Smith [31] have devised an elaborate and accurate method for prothrombin to be used in research, but too intricate for routine clinical work. The simple test, given above, like that of Quick is admittedly a presumptive measure of the amount of prothrombin present in plasma, and actually measures the prothrombin plus its degree of convertibility, the latter having been found to vary somewhat in certain cases. Rather than an exact measure of prothrombin, one thus obtains from this test a measure of the patient's tendency to bleed, which is the single finding of a final and primary importance. Bleeding is found to occur in cases with biliary fistulae or obstructive jaundice when the test gives values of 40 percent or less. In extreme cases, the level falls to as low as 15 percent of normal.[27] Values from 40 to 70 percent are definitely in the danger zone.

SUMMARY

In the presentation of new methods in the field of clinical chemistry, the following have been briefly discussed and the technic presented:

[31] Warner, E. D., Brinkhous, K. M. and Smith, H. P.: Am. J. Physiol. **114**: 667, February 1936.

1. Sulfosalicylic acid method for quantitative determination of, (*a*) albumin in urine, (*b*) protein in spinal fluid.
2. Phosphatase test for pasteurization.
3. Weltmann serum coagulation test.
4. Hippuric acid liver function test.
5. Hehner test for formaldehyde in milk and cream.
6. Quantitative determination of sulfanilamide and sulfapyridine in blood and body fluids.
7. Prothrombin coagulation as a test for vitamin K deficiency in jaundice and liver disease.
8. The authors present modifications of the hippuric acid test and of the Hehner formaldehyde test.

CURRENT PROBLEMS IN DIABETES MELLITUS [1]

By Elliott P. Joslin, M. D., Howard F. Root, M. D., Priscilla White, M. D., and Alexander Marble, M. D.

This article emphasizes certain features, old and new, of diabetes mellitus which may be of especial interest to readers of the Bulletin. It does not represent a complete review of all the important contributions in the last year. The reader is referred to the comprehensive summary of Wilder, Rutledge, and Wilbur [2] and to the authors' brief review of literature published recently in the New England Journal of Medicine.[3]

PITFALLS IN THE DIAGNOSIS OF DIABETES

The diagnosis of diabetes is usually not difficult, but mention is made here of two situations in which errors may easily be made. They are important in that the innocent may be made to suffer.

Pentosuria.—Cases of pentosuria are more common than generally thought. Pentose reacts promptly, indeed very promptly, with Benedict's solution and the unwary may attribute a positive Benedict's test to glucose when in reality it is due to pentose. A simple method of differentiation, useful in all undiagnosed cases of melituria, is to set up the Benedict test not only in the usual manner but also in the cold, leaving the tube at room temperature overnight. With pentose, and indeed with levulose, a reduction of the Benedict solution takes place. Incidentally, the reduction of Benedict's solution by pentose with heat occurs in less than a minute after the urine and solution are placed in bubbling, boiling water. Confirmation of the suspicion of pentosuria should be established with the Bial's test and even by a determination of the melting point of the pentose itself. It is unfortunate for a patient to be given insulin for months or years

[1] From the George F. Baker Clinic, New England Deaconess Hospital, Boston.
[2] Wilder, R. M., Rutledge, D. I., and Wilbur, D. L.: Arch. Int. Med. **63**: 356–427, Feb. 1939.
[3] Joslin, E. P., Root, H. F., White, P., and Marble, A.: New Eng. J. Med., **221**: 173–178, 1939.

upon the supposition of diabetes when the diagnosis in reality is pentosuria, a harmless condition.

The Glucose tolerance test.—A positive glucose tolerance test is by no means decisive for the presence of diabetes unless one knows the previous diet of the patient. Sweeney and others [4] years ago pointed out the susceptibility to hyperglycemia of those on a low carbohydrate diet or after fasting, when fed 100 grams of glucose. The results of the first of the two tests shown in table I definitely suggest diabetes, but those of the second quite as definitely its absence and yet they were obtained in the same individual, a man 55.7 years old, within an interval of 10 days. Before the first test the patient had been living for some time on a partially restricted diet; during the interim between the two tests the diet had been unlimited. No individual should be diagnosed as a diabetic upon the results of a glucose tolerance test unless definite facts regarding the previous diet are available and, in addition, no sign of infection is present. It is well to include in the routine of the test a record of the body temperature at its beginning and end.

TABLE 1.—*Glucose tolerance tests of case 18,115*

Time	July 25		August 4	
	Blood sugar percent	Urine sugar percent	Blood sugar percent	Urine sugar percent
Fasting	0. 06	0	0. 08	0
½ hour after glucose	0. 16	0	0. 12	0
1 hour after glucose	0. 22	0. 9	0. 15	trace
2 hours after glucose	0. 22	1. 9	0. 09	0
3 hours after glucose	0. 12	1. 7	0. 09	0

NOTE.—Tests were carried out 10 days apart. Prior to the test of July 25, the patient had been living on a partially restricted diet. In the interim between the two tests the diet was unrestricted. One hundred grams of glucose were given by mouth. Venous blood samples were used and the sugar content determined by Folin's methods.

HEREDITY

Heredity is the underlying feature in the etiology of diabetes; statistics confirming its importance are steadily accumulating. The incidence of heredity is greatest among those groups in which a knowledge of the relatives, living and dead, of the patient is most complete. Questioning of the patient alone may disclose no heredity, but this often becomes positive when relatives are present. No casual query about heredity is apt to lead to reliable data. Through

[4] Sweeney, J. S., and others, cited by Joslin, E. P.: The Treatment of Diabetes Mellitus, Phila., Lea & Febiger, 6 ed., 1937, p. 123.

the courtesy of the Veterans' Administration we learn that among 704 deceased veterans the incidence of heredity is only 7 percent, but this is easily explained because histories of these patients are taken by a multitude of doctors who are not at the moment especially concerned with the thought of diabetes, and therefore contributory facts may not be elicited. Furthermore, groups of veterans may not have as intimate a knowledge of their own families as groups of men, all of whom are living in their own homes, and, finally, a great many diabetics may at the onset of their disease have no diabetic relatives and yet such appear in subsequent years and escape notation. It is a good rule whenever a diabetic is seen always to ask if a case of diabetes has developed in the family since the last visit. In this way one comes much nearer the true incidence of heredity in diabetes.

As an illustration of the difference in the incidences of heredity of diabetes when taken by a group of doctors in a large clinic and later by an individual doctor of the group are the following facts: A certain physician reported to us the heredity in his group of diabetics in 1928 was 9.9 percent, but this year for 630 diabetics, whose history had been taken by himself, the heredity was 36 percent. In another instance the heredity in 1926 was 19.4 percent, but now for a series of 1,030 diabetics seen in 1938 was 44.5 percent.

THE PITUITARY AND DIABETES

Of great interest and importance was the announcement in August 1938 by F. G. Young [5] of England that permanent diabetes could be produced in normal dogs by the intraperitoneal injection of crude, saline extracts of the anterior lobe of beef pituitaries. Young and his associates have continued this work and in a recent article [6] the results to date are summarized. Young states that to produce a lasting diabetes the amount of extract injected must be regularly increased, beginning with that made from 5 to 10 grams of anterior pituitary substance and increasing the amount every 3 or 4 days until finally extracts from 20 to 25 grams are administered daily. One reason for Young's success is the fact that he has kept all glands and extracts at as nearly 0° C. as possible from the time of removal from the oxen until injection into the dogs. Permanent diabetes results in from 11 to 26 days despite discontinuance of the daily injections. Although the diabetes of these animals resembles closely that of depancreatized dogs, there are differences, among which is the fact that although the amount of insulin needed for control of the diabetes may often be a little larger than that required for depancreatized animals, if insulin is gradually withdrawn from the pituitary diabetic dogs, they will survive for long periods; with gradual withdrawal of

[5] Young, F. G.: Lancet, 2: 374, 1937, Biochem. Jr., 32: 513–523, 1938.
[6] Young, F. G.: New Eng. J. Med., 221: 635, 1939.

insulin from depancreatized animals there soon comes a time when acidosis and coma intervene and death occurs.

Young's work has been confirmed by Campbell, Keenan, and Best [7] in Toronto and by Dohan and Lukens [8] in Philadelphia. Highly significant was the finding of the Toronto investigators that in two of three dogs with pituitary diabetes, extirpation of the entire pancreas led to little or no increase in the severity of the diabetes. Moreover, when the pancreases were assayed for insulin content it was found that instead of a normal amount of about 60 units per pancreas, no measurable quantity of insulin was found in one pancreas and less than 2 units in each of the other two. These results were explained when histological studies were made. Both the Toronto workers and Richardson and Young [9] found extensive degeneration of islet tissue. It is instructive that Young's investigation originally undertaken, one assumes, to elucidate extra-insular influences in diabetes, in the end redirected attention to the pancreas as the primary seat of the disorder.

DIET

Standard Diabetic Diet.—The standard diet of the diabetic has changed gradually and there are now few, if any, clinics in which less than 120 grams carbohydrate are given daily. Probably the average amount of carbohydrate prescribed in this country is now 150 grams daily and in most clinics the view is held that if the patient cannot maintain the urine free from sugar and a nearly normal blood sugar upon this diet, it is wiser to administer protamine zinc insulin hoping thereby to prevent complications in later years which might be incident to a lowered carbohydrate intake.

Vitamins.—The diabetic diet is apt to be richer in vitamins than the diet of individuals in the general population. It includes the protein of meat, fish, eggs and dairy products thus insuring an abundance of animal, in addition to vegetable, protein. The colored vegetables containing 3 and 6 percent carbohydrate are particularly in evidence, although one should emphasize the importance of strictly green vegetables, e.g., the outer leaves of lettuce in contradistinction to iceberg lettuce. Fruits are generally included at each meal. All acknowledge that additional vitamins are needed in the presence of infections and of certain other states and, therefore, it may be advisable under certain circumstances to protect the diabetic with extra quantities of thiamin, riboflavin, nicotinic acid, ascorbic acid, vitamin D and carotene. On the other hand, the evidence that any of these vitamins, important as they are in the internal metabolism of the

[7] Campbell, J., and Best, C. H.: Lancet **1**: 1444, 1938. Campbell, J., Keenan, H. C., and Best, C. H., Am. J. Physiol., **126**: 455, 1939.

[8] Dohan, F. C. and Lukens, F. D. W.: Am. J. Physiol., **126**: 478, 1939, Ibid., **125**: 188–195, 1939.

[9] Richardson, K. C., and Young, F. G.: Lancet, **1**: 1098–1101—1938.

body particularly of carbohydrate, have a specific value and that their administration may cause a reduction in the requirement of insulin, is not conclusive. Perhaps as carefully prepared an article as any is that by Martin [10] who demonstrated that in depancreatized dogs the insulin requirement must be greatly increased if vitamin B is eliminated, but again falls upon return to normal nutrition.

Treatment of Obese Diabetics by Diet Alone.—The possibility of the treatment of mild cases of diabetes, characterized by middle or advanced age and overweight, with diet alone has been emphasized by Newburgh and co-workers.[11] Their careful studies show that the diabetes can be well controlled in such patients by reduction in weight and they even go so far as to suggest or even state that "cures" have resulted. Any work reported by Newburgh can be considered reliable, but the reviewers disagree with him in the interpretation of these data.

Before "cures" are reported in a diabetic, it is suggested that a 5-year limit be demanded just as in cancer and that patients shall be proven positively diabetic at the beginning of the period and at the end of the period shall be shown to be free from glycosuria and to have normal blood sugar tests not only while living on a normal diet but also following the administration of glucose in a tolerance test. It is well that Newburgh has again called attention to the importance of obesity in diabetics and the desirability of undernutrition in such individuals as emphasized by F. M. Allen in his contributions between the years of 1914 and 1922.

INSULIN

Crystalline Insulin.—Although insulin was first prepared in crystalline form in 1926 by Abel and his associates [12] until recently no great practical application of this achievement was made, due in large part to the difficulty of preparation. Large-scale production was furthered by Scott's [13] observation that crystals are formed only in the presence of certain heavy metals, such as zinc, nickel, cobalt or cadmium. With the use of small amounts of zinc, crystals are obtained from crude pancreatic extracts without difficulty. Beginning in 1926 crystalline insulin was used in certain clinics experimentally and in August 1938 it was released for general sale under the name of "Insulin, specially prepared as solution of zinc-insulin crystals." Although some of the preparations used prior to August 1938 contained 0.9 mg. or more zinc per 1,000 units, those now offered for sale must conform to certain standards among which is the requirement that the

[10] Martin, R. W.: Ztsch. f. Phys. Chem., 248: 242-255, 1937.
[11] Newburgh, L. H., Conn, J. W., Johnston, M. W., and Conn, E. S.: Trans. Assoc. Am. Phys., 53: 245-257, 1938.
[12] Abel, J. J.: Proc. Nat. Acad. Sc., 12: 132, Feb. 1926.
[13] Scott, D. A.: Biochem, J., 28: 1592, Aug. 1934.

amount of zinc be not less than 0.2 mg. and not more than 0.4 mg. per 1,000 units.

In some of the earlier reports, workers stated that crystalline insulin was a preparation having a prolonged action, approaching that of protamine zinc insulin. Recent studies,[14] [15] however, have shown conclusively that the solution of zinc-insulin crystals now on general sale (containing 0.2–0.4 mg. per 1,000 units) acts promptly and for a relatively short time. Its hypoglycemic effect seems to be slightly greater and its action slightly prolonged as compared to "regular" insulin, but the differences are not great. However, because it has these definite though minor advantages and because it is the purest type of insulin available, the reviewers have used it almost exclusively since June 1939 in place of regular insulin in situations in which prompt action of short duration has been desired. The experience to date with over 250 patients has been uniformly satisfactory.

Protamine Zinc Insulin.—Protamine zinc insulin continues to be the slowly-acting, long-lasting insulin of choice. The diabetes of almost all patients can be satisfactorily controlled with either a single dose of protamine zinc insulin before breakfast or with this plus an accompanying dose of clear insulin (regular or crystalline). The use of protamine zinc insulin makes possible not only more convenient treatment but more adequate control of diabetes. In the reviewers' experience, complications, including hypoglycemic reactions, have been definitely less than under the former regimen which included regular insulin only.

HYPOGLYCEMIA

Insulin Reactions.—As coma recedes and diabetic treatment improves, its place is taken more and more by the necessity to prevent and treat insulin reactions. It is recognized that with protamine zinc insulin these are more apt to occur during the night or before eating in the morning. The dosage of this type of insulin is regulated, first, by the level of the fasting blood sugar and, second, by the amount of sugar in the specimen of urine voided before breakfast (not the overnight specimen). Reactions in the late forenoon and early afternoon are more apt to be explained by accessory doses of quick-acting insulin, either regular or crystalline, administered likewise before breakfast at the same time as the protamine zinc insulin. The prevention of reactions from either kind of insulin is aided by making it a rule that patients should take 5 or 10 grams of carbohydrate regularly in the late forenoon and afternoon and upon retiring even though then the urine may sometimes contain a trace of sugar.

[14] Marble, A., and Vartiainen, I.: J. A. M. A., **113**: 1303–1309, Sept. 1939.
[15] Ricketts, H. T., and Wilder, R. M., J. A. M. A., **113**: 1310–1312, Sept. 1939.

In order to prevent reactions from protamine zinc insulin during the night, Pollack[16] has emphasized the desirability of making the evening meal the heaviest in protein for the day and the retiring lunch rich in protein as well.

Insulin reactions must be prevented because an insulin reaction in a public place handicaps not only the individual but also all other diabetics. If it were not for insulin reactions a great obstacle to the diabetic securing a job would be removed. The confusion between the symptoms of an insulin reaction and early stages of alcoholism is so easy that diabetics must forego alcohol in any place where a possibility of such a mistake might occur. Like airplane pilots they had best eliminate it altogether.

Hyperinsulinism.—Although hyperinsulinism is related to diabetes mellitus chiefly by contrast, it is of much interest to those concerned with the problems of diabetes and carbohydrate metabolism. At the New England Deaconess Hospital the reviewers have seen three patients in 1939 in whom the presence of an islet cell tumor was demonstrated at operation by Drs. L. S. McKittrick and T. C. Pratt. In the first two, a girl of 16 and a woman of 55, complete cure resulted following removal of the adenomata. In the third patient, a woman of 60, the growth was found to be invasive and metastatic nodules were seen in the liver.

Our experience, admittedly limited, leads us to these conclusions: (1) The most valuable points in the diagnosis of islet cell tumors are (*a*) a history of attacks of weakness, mental confusion, drowsiness and finally unconsciousness, brought on by fasting, hastened by physical exercise and relieved or prevented by the taking of food; (*b*) a blood sugar curve (following a test meal of glucose) in which the initial value is subnormal, the ½- and 1-hour values are normal or slightly above normal, and values at the fourth, fifth and sixth hours descend to definitely hypoglycemic levels with little or no tendency toward spontaneous recovery.

(2) In patients in whom the history and laboratory findings suggest definite hyperinsulinism and particularly in those individuals in whom symptoms appear after an overnight fast, operation with careful exploration of the pancreas should be recommended. "Functional" hyperinsulinism undoubtedly occurs, but one should be extremely careful not to overlook an islet cell adenoma. The possibility of transition of a benign to a malignant growth must be kept in mind.

DIABETIC COMA

Despite the fact that adequate means exist for the prevention and treatment of diabetic acidosis, all too many patients still acquire and

[16] Pollack, H., and Dolger, H.: Proc. Soc. Exp. Biol. and Med., 39: 242-244, Nov. 1938.

succumb to this dreaded complication. In most instances the onset is slow enough—over a period of hours or days—to allow time for the institution of treatment before serious stages are reached. Consequently, except for those patients in whom the condition is the first definite indication of a hitherto unrecognized diabetes, the presence of diabetic coma implies a neglect on the part of someone—the patient, his family, his friends, or the doctor. To be sure, diabetic coma may be precipitated by a complicating disease such as an acute infection with fever, but if the diabetic condition is kept in mind, ample opportunity is afforded to keep this constantly under control with insulin.

Once the patient with diabetic coma comes under the care of the physician, then it becomes his responsibility to give prompt, energetic treatment including adequate, often large, amounts of insulin and electrolyte-containing fluid. Deaths are tragic and excusable, if ever, only in certain situations: (1) when a complicating disease is present which in itself bears a fatal prognosis; in such cases every attempt should be made to confirm the second diagnosis by autopsy; (2) when the duration of the acidosis, and particularly of the unconsciousness, is so long as to produce irreversible changes in the body. Such changes are more readily produced in older patients and in those with cardiovascular disease. However, no case of coma, regardless of the duration of unconsciousness, should be regarded as hopeless but should receive from the moment of diagnosis constant personal attention and energetic treatment.

It is generally agreed that no one fact from the history or no one physical or laboratory finding may be used as a guide to prognosis in an individual case. In an attempt to facilitate the estimation of prognosis, Rabinowitch[17] of Montreal has proposed a numerical "severity index" arrived at from a consideration of nine features, viz., age, duration of coma, degree of unconsciousness, coffee ground vomitus, infection, blood pressure, plasma CO_2, blood urea nitrogen and associated complication.

A still more recent contribution to the subject of the prognosis in diabetic coma is that of Owens and Rockwern[18] of the Cincinnati General Hospital. They emphasize the fact that the duration of ketosis and especially of unconsciousness is of primary importance in the outlook for the patient. However, the reviewers believe that the statement of Owens and Rockwern that "the usually accepted methods of treatment are inadequate in the majority of the unconscious diabetic coma patients" is not justified. Among the 447 cases of diabetic acidosis with a plasma CO_2 of 20 volumes percent or below treated by the reviewers at the New England Deaconess Hospital from May 1923 to July 1939 there have been 52 deaths, a case mortality of 11.6

[17] Rabinowitch, I. M., Fowler, A. F., and Bensley, E. H.: Ann. Int. Med., 12: 1403-1428, Mar. 1939.
[18] Owens, L. B. and Rockwern, S. S.: Am. Jour. Med. Sci., 198: 252-260, Aug. 1939.

percent. In 84 of the 447 cases, the patients were totally unconscious; despite this only 28, or one third, of the patients died. This figure leaves much to be desired, but it is low enough to allow hope and assurance that even in the group of the totally unconscious, the patient has twice as great a chance of living as of dying.

JUVENILE DIABETES

Juvenile diabetes is uncommon. Only one child in 8,000 contracts this disease under 15 years of age and there are probably not more than 20,000 juvenile patients in the entire country. The onset in the child is often acute and the early course so virulent that the first recognition of diabetes may be made in semicoma. Benign glycosuria, common in childhood, must be differentiated from mild diabetes by standard sugar tolerance tests.

The dietary regime must include calories adequate for growth and development. Our rule is to allow 1,000 calories at age one, adding 100 calories for each year of age until age 13 for girls and 19 for boys, with carbohydrate, protein and fat prescribed in gram ratios of 2:0.9:1, respectively. Insulin is administered to all juvenile patients. Only 10 percent do well with protamine zinc insulin alone; 90 percent require both protamine zinc and clear insulin given simultaneously before breakfast. All adjustments are made upon the basis of tests of pre-meal and retiring specimens of blood and urine.

Coma, lowered resistance to infection, necrobiosis lipoidica diabetica, xanthomata and hepatomegaly appear to be related to the control of diabetes and are relieved by adequate control of the disease. Dwarfism, possibly arteriosclerosis and cataracts appeared to be more inherently associated with the disease rather than its control. Dwarfism may be corrected by the administration of anterior pituitary extract and thyroid extract. The control of arteriosclerosis is an unsolved problem. Longer duration of life and improvement in their chances of attaining economic independence are now assured to juvenile diabetic patients.

PREGNANCY IN DIABETES

Until recently it was nearly as true of the insulin as of the pre-insulin era, that only every other diabetic pregnancy terminated successfully. The accidents concerned did not involve the welfare of the mother, for maternal mortality was low, but did concern the welfare of the child destroyed by spontaneous abortion, miscarriage, premature delivery, stillbirth or neonatal death.

Lack of control of diabetes and the specific complications of the disease—coma and hypoglycemia—do not appear to be specifically responsible for the accidents of pregnancy, because a normal outcome

may be seen in patients with poorly controlled diabetes and an abnormal outcome in those with the disease under good control. In large part, the accidents of late pregnancy in diabetes appear to be associated with an abnormal hormonal balance indicated by the rise of blood prolan and fall of estrin and progestin. The most common accident, stillbirth, is associated with clinically mild pre-eclamptic toxemia, the signs and symptoms of which may appear and disappear in 1 week with the death of the fetus, but are predictable from 2 to 6 weeks usually by the rise of the serum prolan.

The value of quantitative prolan B examinations in predicting the outcome and the value of substitutional hormone therapy is shown by the following: Since 1935, 44 completed pregnancy cases have been studied in part by Smith and Smith [19] and in part by White et al.[20] The results were as follows:

(*a*) Sixteen patients with normal prolan values in late pregnancy had no toxemia, no premature deliveries and the pregnancies resulted in 15 live births and 1 neonatal death, a fetal mortality of 6 percent.

(*b*) Eleven patients whose values for prolan were high and who received no hormone therapy developed pre-eclamptic toxemia in 8 instances. Premature delivery occurred in 4. There were 2 intra-uterine and 4 neonatal deaths, the fetal mortality being 55 percent. Three patients with hyperprolanemia, self-corrected, had mild transient toxemia with no fetal deaths.

(*c*) Thirteen cases with high prolan values were treated continuously with massive doses of estrin and progestin. There was only one fetal death. This occurred in the neonatal period. The fetal mortality was 8 percent. In another patient who was treated in the early part of this work, therapy was omitted after the prolan had fallen to normal values. This we considered wise because of the cost of therapy and the fact that we did not know whether or not therapy was replacement or stimulating in type. This patient was discharged from the hospital and returned for delivery 3 days after intra-uterine fetal death.

This type of therapy was inaugurated in January, 1938. From January, 1938 to December, 1939, there have been 28 pregnant diabetic women in succession delivered with 26 living babies. The cost of the therapy has been great, but was supplied to us through the courtesy of the Schering Corporation. Actually we used from $30 to $60 worth of therapy daily. New inexpensive, synthetic preparations are now being tried. It is true the number of cases above reported is few, but if the results are confirmed it opens the possibility for a new epoch in the treatment of toxemia, not only for diabetics but for non-diabetics.

The type of delivery depends upon the clinical course and obstetrical complications.

Diabetic management throughout pregnancy consists of a diet providing 30 calories per kilo body weight. The usual diet includes

[19] Smith, O. W., Smith, G. van S., Joslin, E. P., and White, P.: Am. Jour. Obs. and Gyn., 33: 365—Mar. 1937.

[20] White, P., Titus, R., Joslin, E. P., and Hunt, H.: Am. Jour Med. Sci. 198: 482, Oct. 1939.

about 200 grams of carbohydrate, 2 grams of protein per kilogram of body weight and enough fat to make up the caloric prescription. Protamine zinc and clear insulin are usually used before breakfast and frequently supplementary doses of regular insulin are necessary at noon and before the evening meal. The management of the second trimester is difficult because of the low renal threshold.

The care of the infant is that of any premature infant. Five percent glucose, 50 cubic centimeters at a time, may be given subcutaneously at birth and in 8 hours if no blood sugar studies can be made, or if blood sugars are made and the values obtained at 4-hourly intervals fall to 50 mgs. percent or below. In our last 26 consecutive deliveries there have been 2 fetal deaths.

TUBERCULOSIS

The extraordinary susceptibility of the diabetic patient to the development of pulmonary tuberculosis persists in spite of the great improvement in diabetic treatment by means of insulin and the special advantage of insulin itself even in nondiabetic patients with tuberculosis. As brought out by Root and Bloor [21] it is still young diabetics, particularly with onset in childhood or in the second decade of life, who show a rate of development of tuberculosis more than 12 times in excess of nondiabetic children in the same period. The incidence of pulmonary tuberculosis in American series of diabetics is low in comparison with the 19 percent mortality of tuberculosis among diabetic patients in Finland.[22] The explanation for the increase in susceptibility is not clear, although Root and Bloor's analyses of the cholesterol and lipoid composition of the lungs of diabetic patients at autopsy indicate chemical differences between diabetic and nondiabetic lungs. The low values obtained for phospholipid are of interest in connection with the fact pointed out by Himsworth [23] that pulmonary tuberculosis and true diabetic cataract are particularly prone to occur in young diabetic patients who are not well controlled.

The onset of tuberculosis in diabetic patients is no more insidious than in other patients, but the striking fact remains that, up until the present, the discovery of tuberculosis in diabetic patients in a truly incipient stage where the prognosis is favorable, has been extremely rare. The other outstanding fact is that when such a discovery is made by routine x-ray treatment, diabetic patients, even those with more advanced processes, are capable of demonstrating extraordinary improvement when properly controlled under sanatorium conditions with adequate diet and insulin. Indeed, it may almost be said that the diabetic patient with pulmonary tuberculosis

[21] Root, H. F., and Bloor, W. R.,: Am. Rev. Tub., 39: 714-737, June 1939.
[22] Ponteva, E.: Acta med. Scandinav., Supp. 88: 1-108, 1938.
[23] Himsworth, H. P.: Quart. J. Med., 7: 373-395, July 1938.

has a somewhat better prognosis than the nondiabetic patient whose pulmonary tuberculosis is of apparently similar character, since the diabetic may have reached a certain stage partly under the influence of a remedial metabolic disturbance.

Uncontrolled diabetes best illustrated by diabetic coma is particularly prone to lead to pulmonary tuberculosis. Root and Bloor state that of 73 patients recovering from diabetic coma between February 1929 and November 1932, 13 developed pulmonary tuberculosis within 5 years. Only the more liberal use of routine x-ray of the lungs in diabetic patients can hope to improve the care and prevention of tuberculosis. Diabetics make excellent subjects for pneumothorax or thoracoplasty, provided the diabetes is well controlled.

SURGERY

Increasing emphasis is placed upon the importance of cooperation by the medical and surgical members of a team continuously interested in the diabetic problem. Such cooperation must be founded not only upon an understanding of the surgical problem itself but also upon the effects of diabetes particularly of long duration upon the patient as well as of its effects upon the course of treatment of the surgical complications. Two different surgical series, one in Boston and the other in Cleveland, make these points clear. McKittrick [24] analyzed 1,276 diabetic operations at the New England Deaconess Hospital performed during the last 10 years illustrating the great variety of diabetic surgery. The mortality was 8 percent for the total series. John's [25] series included 983 major operations and 290 minor operations with an average mortality of 5.8 percent.

Often too little attention is given to the history of the diabetic patient and particularly to the duration of his diabetes. Coronary thrombosis after the age of 40 years is at least 4 times as frequent in diabetic as in nondiabetic patients of the same age as the autopsy figures of Root, Gordon, Bland and White [26] clearly indicate.

Although this arteriosclerotic background is of chief importance in relation to the possibility of cardiac complications and in relation to the problem of healing, at least in the extremities, it is also true that careless or inexperienced care of the diabetes may result in glycosuria and lack of control of the diabetes with not only the danger of acidosis, but more important still its deleterious effect upon healing and resistance to infection. Inexperienced diabetic treatment may on the other hand, result in the use of excessive doses of insulin in relation to the diet and to correspondingly severe accidents with insulin hypoglycemia.

[24] McKittrick, L. W.: Surg., Gyn. and Obs., 68: 508-518, Feb. 1939.
[25] John, H. J.: Ann. Surg., 108: 1052-1075. Dec. 1938.
[26] Root, H. F., Bland, E. F., Gordon, W. H., and White, P. D.: J. A. M. A., 113: 27-30, July 1939.

In pre-operative preparation for surgery the glycogen stores particularly in the liver must be considered. It is desirable that patients shall have had 100 to 200 grams of carbohydrate during the days preceding operation and that it shall have been normally utilized by means of insulin properly prescribed.

All diets should be accurately weighed. It is not necessary to delay operation in order to make the urine sugar free or to bring the blood sugar to normal. Indeed, recently at the New England Deaconess Hospital a diabetic boy of 21 years had an acute appendix removed even in the presence of diabetic coma as indicated by a blood CO_2 of 18 volumes percent, a blood sugar of 600 mgs. and hyperpnea. If the patient has been adequately prepared during the days preceding operation there is no necessity for giving carbohydrate by mouth on the morning of operation. The use of protamine zinc insulin is ideally suited to surgical diabetics because it can be given before the operation with the assurance that its slow action will not lead to hypoglycemia, and yet that acidosis can be controlled and glycogen stores protected during the anesthesia. The liberal use of physiologic solution of sodium chloride for patients who have been dehydrated or in obvious need of water is important since the water metabolism in diabetes is so easily disturbed. Anesthetics should be chosen with regard, first, for the surgical need, second, the age and severity of the diabetes and, third, the experience and skill of the anesthetist. If ether is used, its effect in reducing the glycogen of the liver must not be forgotten. An elderly patient, aglycosuric, scheduled to have a gall bladder operation under ether anesthesia, may well need glucose solution given intravenously on the day before and even on the morning of operation as well as after operation. Spinal anesthesia avoids nausea and vomiting and has been ideally suited for operations upon the extremities except for one complication and that is the danger of bladder paralysis following operation particularly in elderly patients. Pre-operative medication should be used with caution in elderly diabetics since such drugs as morphine, scopolamine, and even coal-tar products may have deleterious effects. McKittrick [24] recommends 1½ grains of nembutal or phenobarbital orally the night before operation and ⅛ to ¼ grain of morphine with $\frac{1}{150}$ grain of atropin subcutaneously 30 minutes before an inhalation anesthetic.

The technical indications for various types of operations upon the extremities and the abdomen are discussed by McKittrick. Whether diabetic gangrene is increasing or diminishing will require analysis of case records which will take into account the strikingly increased duration of life in diabetes brought about by the more general and increasing use of insulin, and particularly protamine zinc insulin.

GASTRO-ENTEROLOGY

A REVIEW OF PEPTIC ULCER, DIET AND CONSTIPATION

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

This review is limited to a brief consideration of modern trends in the treatment of peptic ulcer and its complications, the digestibility of various foods in relation to the healthy and diseased gastro-intestinal tract, and the therapeutic management of constipation. These closely related subjects include most of the problems commonly encountered in the field of gastro-enterology.

PEPTIC ULCER

In order to understand how to treat a case of peptic ulcer, one must realize the underlying principles in its management which determine the successful accomplishment—either in healing the ulcer, preparing the patient for surgical treatment, or preventing complications which may follow a stomach wound unsatisfactorily medicated.

The first and most important principle is the quieting and readjustment of the patient's psychic and emotional life; second, his nutritional condition; third, the alleviation of the patient's symptoms; and fourth, prevention of possible complications.

The patients of the Services, because of the perforce discipline of the personnel, present a much easier task in the treatment of ulcers than civilian cases. In civilian life handling of a patient is closely related to his economic status and the scientific procedures underlying the treatment of an ulcer are modified by his economic ability to meet the medical expense, many times to his detriment. In the Service hospital, on the other hand, he is able to receive all of the advantages of medical management without consideration of the cost.

A patient with a peptic ulcer, upon admission to the hospital should be weighed, measured, given a thorough examination and placed in cheerful surroundings. Special emphasis should be placed on carious gums and teeth, cloudy sinuses (paranasal sinuses) post-nasal drips and infected tonsils. These should be corrected promptly.

A sociological-psychiatric approach should be made by the gastro-enterologist. He should discuss at length the problem of emotional, economic, or other disturbances of the psycho-emotional field and try, if possible, to readjust those difficulties, and encourage the patient. Sedatives should be administered at bedtime, preferably Carbromal (U. S. P.), grains 10 to 20, and continued until definite physical and mental improvement ensues.

It is easily overlooked by many clinicians that a patient when treated for a considerable time on milk, cream, and alkalies, will reach a state of deficiency, first because of the lack of a balanced

diet and second, because of an impaired digestive assimilability. It is, therefore, imperative that the patient receive a full balanced meal which contains proteins, carbohydrates, fats, minerals, and vitamins. He should be given finely pureed foods for a period of 6 months. Only lean meats should be allowed such as lamb, fresh tongue, and chicken. Beef should be omitted from the peptic ulcer diet because of its tendency to stimulate excessive secretion of hydrochloric acid. Use of gelatine is very well adapted in this treatment because it is very bland and its high protein content absorbs the excessive secretion without added stimulation.

Sweet breads, liver, tripe, stomach, oysters, and lean fish should be given at least twice a week as these proteins are easily digestible and contain highly nutritive principles. The meats may be boiled, broiled, or baked, but it is important to remind the patient that in the preparation of these meats fat must not be added and when present should be removed. As already mentioned, the meat must be ground up before served.

Vegetables and fruits should be chosen for their low cellulose content. Any with rough fibres and skins should be excluded from the diet. Vegetables should be cooked with little water, so as to preserve all minerals and vitamins, and then run through a grinder. Raw fruits are not permitted except over-ripe bananas and scraped apples. Stewed fruits are allowed, provided they are prepared without sugar. Fruit juices should be given liberally and for variety may be incorporated in gelatine.

Sugars, concentrated sweets, pastries, and candy are not permitted. Honey, pure maple, or corn syrup in moderate amounts may be used to sweeten foods to make it more palatable.

Stale, white bread and zwieback are allowed. Other breads, especially hot breads or toast, should be avoided.

All condiments should be eliminated from the diet; salt may be used very sparingly.

The patient may include in his diet well-cooked cereals such as cream of wheat and rice. In addition to these the patient should take a certain amount of a commercially prepared cereal such as Pablum or Cerevim that possess supplemental mineral constituents lacking in the ordinary cereal.

Soft boiled, poached, and scrambled eggs are allowed. Milk should be given preferably in evaporated form as it produces a much finer curd than raw or pasteurized milk and less tendency to constipation. Cream, salt-free butter, and olive oil should be given liberally, but in small amounts at a time, otherwise they may produce atony of the stomach and diarrhea. Cottage and cream cheese only are allowed.

Alcohol is absolutely not permitted, nor is tobacco as they increase hydrochloric acid and inhibit the proteolytic activity of the gastric enzymes. Extreme hot or cold foods are prohibited. This includes ice water, ice cream, and ices.

Vitamin B complex should be given in large doses. Two Cerelexin tablets after principal meals, 3 to 4 times a day for at least a month and four to six Super D Upjohn Cod Liver Oil perles at bedtime (in order to prevent regurgitation) are recommended. For vitamin C one quart a day of unsweetened citrus fruit juice, preferably freshly strained orange juice, is satisfactory. Three regular small meals a day with supplemental feedings of fruit juice, fruit juice with gelatine or warm evaporated milk diluted with lime or vichy water provide a satisfactory routine.

In addition to the outlined dietary regime and sedatives for the relief of symptoms, application of continuous infrared heating to the abdomen will bring relief within 24 hours in the majority of cases. The patient should be instructed how to correct his constipation and in the event he has not moved his bowels in 48 hours and has an extreme distention of gas in his lower abdomen, he is allowed to use two or three ounces of olive oil per rectum to be retained, at bedtime. He may also receive for meteorism in his lower abdomen hot, moist Turkish towel fomentations. This usually relieves the gas distress. If the pains do not disappear under the procedure outlined, he may then be given antispasmodics such as Trasentin (Ciba) one tablet or Syntropan, one tablet, repeated in intervals of 10 minutes for three or four doses. These antispasmodics are preferable to Belladonna because they have less side effect on the secretion of hydrochloric acid in the stomach. On the other hand, if all of these procedures fail and the patient continues to have distress calcium carbonate, grains 30 to 60 in a powdered form mixed with warm evaporated milk may be necessary. Should not the desired results be obtained and pain is not alleviated a final resort is to lavage the stomach with a warm solution of 10% Cremalin. The ideal regime is to keep the patient under strict dietary and therapeutic surveillance for a period of a month after which time he should continue on a pureed, bland diet for 6 months.

In chronic peptic ulcers the therapeutic procedure is similar to the management of a case of acute ulcer with the exception that if there is edema or retention due to fibrosis over the scar, it is usually advisable to evacuate the stomach contents around bed time and wash the stomach with a warm solution of 10% Cremalin (Colloidal, aluminum hydroxide).

In cases of hemorrhage from peptic ulcer, morphine is absolutely contraindicated as it produces an increased pylorospasm which is undesired. The best procedure is to give veronal rectal suppositories,

10 grains each in intervals of 3 hours; placing heat to the upper abdomen and giving a mixture of warm evaporated milk and gelatine sipped through a straw every 10 minutes to quiet down the peristaltic motions of the stomach, followed in 4 hours with feedings of freshly strained orange juice and gelatine. Twelve hours later a full balanced meal as outlined for an acute ulcer is given. If hemoglobin falls under 60%, daily transfusion of 500 cc. of blood should be given for 3 days. A patient who has once had a hemorrhage should be matched for a blood donor and the type of his blood made known to him. In case of recurrence prompt transfusion can then be administered.

In conclusion, the patient with a peptic ulcer should be aware of his condition and educated to consider it just as serious as if he had diabetes or tuberculosis, and a proper readjustment of his life be made accordingly. A roentgenological recheck should be made at least every 6 months.

DIET

MECHANICAL FACTORS.—1. *Liquids* are more quickly available for absorption in that they leave the stomach rapidly. Because of this fact fluids are not acted on well by the gastric enzymes and juices and tend to pass into the intestines poorly prepared for the action of the intestinal secretions. Therefore, liquids high in carbohydrate content are utilized well, those high in protein content poorly. Liquids high in fat content slow up the emptying time of the stomach. This principle may be utilized in conditions in which there is gastric hypermotility such as in gastritis or peptic ulcer.

2. *Solids* when finely ground up, admixed with saliva, receive the best reception in the stomach. Rapid eating allowing large chunks of food to be swallowed without proper admixture of saliva may mechanically traumatize the walls of the stomach. Eating large quantity of food at one sitting may distend the stomach to a point interfering with proper churning of the ingested food. Roughage such as nuts, bran, fruits or vegetables with tough skins does not interfere with the proper digestion in a healthy stomach provided such food is not eaten excessively and exclusively at one sitting. In a traumatized or edematous stomach roughage produces additional injury and a reflex pylorospasm.

3. *Gaseous beverages* have no effect on a healthy stomach. In an edematous or traumatized stomach gaseous beverages should not be used as they distend the stomach causing pylorospasm and pain.

PSYCHIC AND BODY EXERCISE FACTORS.—No food can be well digested if patient is not well relaxed mentally and physically. Happiness, laughter, non-sexually stimulating literature or cinema, pleasant environment promote good digestion. Anger, anxiety, unhappiness, social or sexual maladjustments and pain interfere with good digestion.

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Short exercises such as, walking, golf, swimming, Swedish gymnastics, help digestion. Overwork, or too vigorous exercise, such as tennis, handball, hockey, and horseback riding, interfere with digestion if performed within 2 hours of utilization of foods. This is in reference to a normal stomach. In gastritis or active peptic ulcer it actually produces harm by provoking pylorospasm and pain.

Tepid baths from 15 to 30 minutes before partaking food promote digestion. Hot or cold baths interfere with digestion. Attractively prepared food with pleasant odors stimulate gastric juices and hasten the process of digestion.

THERMAL FACTORS.—Extreme cold or hot foods interfere with proper digestion. Warm foods have the optimum chance of being utilized. It is a fallacy to use chilled or iced drinks in peptic ulcer as the cold increases the hypermotility and antral spasm. In gastric disturbances it is better to sin on the warm side than on the cold.

CHEMICAL FACTORS.—1. *Carbohydrates:* Simple sugars, such as glucose found in fruit juices, fructose found in greatest abundance in honey, or galactose found in milk sugar, require no preparation in digestion. They are the easiest assimilable sugars and should be given preference in malnutrition and disturbed gastro-intestinal tract. Double sugars, such as table sugar or malt sugar, require one step in digestion to become simple sugars, therefore they are to be used less freely in cases of disturbed gastro-intestinal function than the simple sugars. Complex sugars, such as starch, glycogen, or cellulose, require two steps in digestion and, therefore, are still less easily digestible than the simple or double sugars. Excess intake of starchy foods leads to constipation. In the form of concentrated sweets it may lead to gastro-intestinal disturbances because they favor intestinal fermentation and promote gas formation causing irritation of the intestinal tract.

2. *Fats* are divided into hard fats and soft. The hard fats, such as animal fats, have a higher melting point than the soft fats, such as vegetable oils, cream, butter, or egg yolk. The readiness with which fats are absorbed depends largely upon their melting point. Hard fats are absorbed slowly and sometimes not at all, whereas soft fats are absorbed almost completely. Fat depresses the motility of the stomach and secretion of the HCl. Excess of fat intake, because of the slowing up of the gastric motility may permit a certain amount of decomposition to take place and these products of decomposition may cause irritation to the stomach lining, resulting at times in diarrhea. Also, an excess of fat produces a sense of fullness and nausea in the stomach.

3. *Proteins* are classified as complete proteins and incomplete. The complete proteins contain all the essential amino-acids necessary for optimum health. The incomplete proteins lack some of these amino-

acids. Lean meats, fish, eggs, cheese, and some nuts are foods which furnish complete proteins. Cereals, legumes, and gelatine are foods which contain incomplete amino-acids. Of all the proteins stimulating the secretion of hydrochloric acid, beef and beef extract are most potent, gelatine and milk least potent. This principle is well utilized in dietotherapy of peptic ulcer or gastritis with hyperacidity. Although beef absorbs the acid hypersecreted in peptic ulcer, it also stimulates acid production and should therefore not be used. On the other hand, gelatine mixed with milk absorbs all of the extra hydrochloric acid without provoking added secretion. In trying to aid digestion of proteins in a diseased gastro-intestinal tract there are several ways to do so. Pepsin can be added to milk and thus predigests it. Papain added to different meats for 48 hours makes them pronouncedly more tender. Cooked eggs are slightly easier to digest than raw eggs. If eggs are cooked at a temperature under the boiling point about 30 minutes, the time of digestion is lessened. Ground up meats, fish, or eggs are easier to digest. Meat or fish low in fat content are easier to digest. Cooking at low to moderate heat and plenty of moisture makes the proteins more digestible.

CONSTIPATION

CLASSIFICATION.—In order to facilitate the therapeutic management of constipation, a simplified classification is presented.

Primary constipation.—Under this classification are listed all the congenital anomalies which produce constipating tendencies.

1. Congenital intestinal atony or hypoplasia which is frequently seen in children and which is due to under-development of peristalsis stimulating nerve fibres.
2. Redundant colon.
3. Low fixed cecum seen often in asthenic women who have a vomiting tendency.
4. Hyposthenic or asthenic individuals with enteroptosis.

Secondary constipation.—Under this classification the following are embraced:

1. All conditions due to laxative habit whether resulting in spastic or atonic constipation.
2. Postoperative or postperitonitis constipation due to constricting action of adhesions.
3. Dyschezia or rectal fecal impaction.

DIAGNOSIS: In order to determine whether a patient is constipated, a barium meal is essential. The normal emptying time for the colon is from 48 to 72 hours after the administration of a barium meal. If barium is visible in the rectum or colon at the end of 72 hours the diagnosis of constipation is established with the exception of the

dyschezia when at the end of 48 hours the rectal impaction can be determined by finger examination of the rectum. There is no more potent psychotherapy than showing to a patient, who for many years has not had a normal stool, a roentgen plate presenting the satisfactory progress of a barium meal which the patient felt never reaches the rectum.

In congenital intestinal atony, one gets a history of constipation with vomiting tendency, pains in the right lower quadrant and intractable constipation. Most of these patients are children. In people with redundant colon, we get a history of from 3 to 7 day intervals between spontaneous bowel movements. The passage of flatus is difficult but when it does occur it affords great relief. These patients are also great followers of colonic irrigation therapy. Not all of the cases enumerated as having congenital anomaly must necessarily be constipated, but most of them are.

Females with low fixed cecum will complain of the ease with which they vomit and of pains in the right lower quadrant. They can be easily spotted, having an anxious facies, being asthenic and in many cases having had an appendix removed because of the right lower quadrant complaint. In the primary constipation group we have a visceroptotic, asthenic individual with the face of a martyr.

TREATMENT.—The bulk of constipated patients are victims of laziness, bad habits, and laxative or enema addiction. No laxative is good and even the mildest, such as mineral oil or agar, has no place in the treatment of constipation. The treatment of constipation, whether primary or secondary, should have the following objectives:

1. Complete withdrawal of all laxatives, colonic irrigations and enemas.

2. Demonstration to the patient of the progress of barium meal and the time element at which this patient should expect a normal stool as shown by the roentgen 24- or 48-hour plate.

3. Allay anxiety by giving sedatives at bedtime, such as Bromural gr. x.

4. Teach the patient bowel movement technique:

(*a*) He must always go to the toilet at the same time, not differing 5 minutes from day to day.

(*b*) He must never read, look at pictures, hold conversation, be chilled, inconveniently clothed while sitting on the bowl.

(*c*) He must sit there 10 to 15 minutes and if no movement ensues, get up and try again several times during the day.

(*d*) He must always obey a defecation impulse and go at that time to the toilet.

(*e*) If the patient tries to move his bowels several times within 48 hours without results, that evening a 4-ounce retention enema of olive

oil should be given. Next morning patient cannot fail to have a normal stool. This procedure may have to be repeated several times.

(*f*) Under no circumstance is the patient to be allowed to strain while sitting on the bowl. It should be explained to him that straining contracts the sphincter preventing fecal passage and produces hemorrhoids.

5. He must take an adequate amount of fluids, at least 10 to 12 glasses daily. Especially is it important for him to take 2 glasses of tap water on arising.

6. The diet of constipated persons should be devoid of condiments. It should be of bland variety whether in atonic or spastic constipation. It should contain at least 8 tablespoons of vegetables and fruits a day, sufficient amount of butter or olive oil. In the spastic type, the food at first should be pureed, but in a few days may be modified to a normal well-cooked variety. Addition of bran or other bulk-producing substances is to be condemned since the colon soon adjusts itself to a particular bulk and responds to its stimulation. An adequate amount of stewed fruits prepared without syrup, fruit juices, or buttermilk is helpful.

7. In cases of atony of the bowel, large amounts of vitamin B complex are helpful. Cerelexin compound, 6–10 tablets per day, is very satisfactory.

8. The cases of constipation due to adhesions should have 3 ounces of olive oil between meals twice a day.

9. Cases of dyschezia should be instructed not to get up from the toilet at the first passage of the stool, but wait long enough until they have a satisfactory movement. These cases should be told to try to move their bowels in the morning and at bedtime.

10. In spastic constipation antispasmodics may also be given.

The poet de Sylva informs us that "Not all of the feeling of anxiety, spiritual depression, mental martyrdom, is due to love." Some, doubtless, is due to constipation.

NEWER ASPECTS OF ABDOMINAL ROENTGENOLOGY [1]

By Commander Charles F. Behrens, Medical Corps, United States Navy

The gastro-intestinal tract is practically on a 24-hour working basis from the cradle to the grave. It is perhaps trite to state this inasmuch as so many of the physiological activities of the body are continuous. However, the alimentary tract in carrying on its work is obliged to encounter exceptional difficulties not to say insults. Regardless of its needs, it must take what it gets, and what it gets only too often amounts to grave abuse. Proper food is not always avail-

[1] From U. S. Naval Hospital, Washington, D. C.

able. Eating habits and the appetite are often perverse. A large amount of mankind's pleasures center about the festive board, and every celebration is accompanied by feasting; thus over-eating is common. Over-indulgence in alcoholic and other beverages is widespread. In addition, the functions of this portion of the anatomy, delicately balanced and intricate as they are, are to no little extent at the mercy of psychogenic factors. That these factors are considerable, especially in times of marked stress such as the present, needs no arguing. So, in general, it is not to be wondered at that the abdomen presents to the medical profession a veritable Pandora's box of miscellaneous and grevious miseries, the diagnostics of which are only too often well nigh insoluble.

Recent advances in the field of abdominal roentgenology have been in the nature of refinements of technique. The fundamental basis for the study of the stomach and intestines was laid in the early years of roentgenology by the introduction of the radiopaque meal and enema. The basis for gall bladder studies was laid more than 15 years ago. However, the refinements are of great importance and worthy of account.

STOMACH

Some years ago it was quite common to start the roentgenoligical examination of the stomach by having the patient promptly fill the stomach with several glasses of a barium suspension. This, of course, precluded careful study of the mucosal folds. Most workers now have the patient take a minimal amount of barium and manipulate this so as to bring out mucosal relief. This type of study has been of great benefit. For one thing, it has improved our concepts of gastritis. For many years, this was a popular dumping ground diagnosis and, as such, early in this century came into deserved disrepute, being regarded with scepticism and even derision. A patient had ulcer or cancer or nothing, unless perchance neurosis; and neurosis then, as now, was a dangerous diagnosis. We all know of instances of neurosis which turned out to be based on real organic lesions. I recall a patient, thought to be a neuropath, suspected of exaggeration and even malingering who, after 20 years of suffering, came to the hospital almost maribund from the effects of a chronic, partially obstructive, pyloric ulcer.

Gastritis and duodenitis are on a firmer footing and are regarded with more interest and less suspicion. Roentgenologically, something has been accomplished, although it must be admitted that the criteria are still under debate, and that the diagnosis often has to be tentative. Recently, correlation of x-ray and gastroscopic studies has been attempted. A study by Templeton and Schindler,[2] how-

[2] Templeton, F. E., and Schindler, R.: Am. J. Roentgenol. 41: 354, March 1939.

ever, gave very poor agreement in results. The fault here, however, may not lie entirely with roentgenological errors. Submucosal inflammation and changes in the muscularis, such as may produce coarsened rugal folds, will escape the gastroscope. The most convincing picture of gastritis includes the following combination of signs:

1. Coarse rugal folds.
2. Disordered peristalsis, especially peristalsis of hectic fibrillary type.
3. Undue clinging of barium to the walls together with granular mottling.
4. Increased tonicity.

In duodenitis we tend to find:

1. Increased tone.
2. Coarsened mucosal pattern and tendency to ragged outlines.
3. Disordered motility of the duodenal contents.

Closely related to and often associated with gastritis and duodenitis is peptic ulcer. We are all more or less familiar with the roentgen picture of this disease. The roentgenologist is at present making greater efforts to visualize the crater and to rely less on the indirect findings, most of which are quite undependable. Thus the incisura, supposed to be characteristically found opposite an ulcer on the lesser curvature of the stomach, is frequently conspicuous by its absence. On the other hand, a very tiny gastric ulcer near the pylorus may produce marked spastic deformities of the duodenal cap.

When a crater or penetrating lesion of the stomach is found, careful study will sometimes help determine if malignancy be present. In benign ulcers, rugal folds extend to the ulcer. In malignancy, we usually find a clear zone about the ulcer in which mucosal markings are obliterated.

Careful study of the duodenum, aside from bringing out indications of inflammation, at times will reveal infiltrative pathology such as might result from carcinoma of the head of the pancreas. A number of such cases are on record, and they occur sufficiently often to make it decidedly worth while to investigate the duodenum from this standpoint.

Recently studies have been made of the intestinal mucosa in cases of nontropical sprue syndrome or idiopathic steatorrhea. Kantor,[3] in a recent article, described some cases in which marked changes were present in the upper intestinal tract. Typically there was coarsening and often ironing out of the valvulae conniventes even to the point of resemblance to a wax filled tube for which appearance he coined the term "moulage sign". These findings were most marked in the proximal coils of the jejunum. Aside from this sign, dilatation, spasm, and sausage formation sometimes dominate or complicate the picture.

[3] Kantor, J. L.: Am. J. Roentgenol., 41: 758, May 1939.

Accompanying these upper intestinal findings, according to Kantor, pronounced cases are apt to show:

1. Dilatation and redundancy of the colon.
2. Faint visualization of the gall bladder.
3. Osteoporosis from loss of calcium.

In youthful subjects, dwarfism and tendency to tetany have been noted.

Regional enteritis has been a subject of great interest lately and many studies have been published. Its exact nature and etiology are still to be determined, chiefly because we usually see only the latter stages of the disease. At present it seems that the best we can do is to pronounce it a nonspecific granulomatous disease. Roentgenologically we look for a narrowed, irregular lumen in the affected part with absence of mucosal markings. Retention beyond 9 hours is usually noted proximal to this portion. However, instead of a narrowed lumen we may be unable to visualize the affected part due to irritability. Thus a careful study and good judgment may be especially necessary for correct diagnosis. In this connection it is well to point out that the routine barium enema and gastro-intestinal series are not apt to uncover this disease. Serial studies at 30- to 60-minute intervals are necessary and perhaps a barium enema with special effort to obtain an ileal leak. Thus if regional enteritis is suspected in a patient it is well to request an examination specifically with that diagnostic possibility in view, rather than to put in a routine request for a G. I. series. In general, a statement as to the condition suspected is always a help to the roentgenologist and may prevent pathology being overlooked. It is impractical to carry on all manner of extensive special studies routinely in every case that is sent in for study of the G. I. tract.

GALL BLADDER

The Graham-Cole test is based on the ability of the liver to excrete foreign substances, one of the liver's most important functions. The liver excretes substances of a colloidal nature, and also those substances soluble only in alkaline solutions, or as alkaline salts. Crystalloids and acid substances are excreted by the kidneys. In the category of alkaline soluble chemicals are the halogen derivatives of the phthalic dyes; these are accordingly excreted by the liver. In their great pioneering work, Graham and Cole found these dyes the best for roentgen diagnosis, and, of these, tetraiodophenolphtalein has proved most satisfactory. Various systems of administering the dye have been tried including the intravenous route. At present commercial preparations of one sort or another are largely used as a matter of convenience and to give the patient a more palatable concoction. However, the pure dye mixed, for instance, with grape juice is not

objectionable. Various systems are still in vogue but there is an increasing tendency to adopt what is called the double dose method. One dose (usually 4 gms.) is given after the noon meal and a second dose after the evening meal which must be fat free. Paregoric is also given by many to obviate or minimize unpleasant effects. Pictures are made the following day with follow-up views after a fatty meal. This system gives more consistent results than the single dose method and one can be more certain that failure of the gall bladder to visualize means pathology. A recheck series, however, is advisable, particularly if surgery hinges on the result. There is no need here to speak of the value of the Graham-Cole test in the general run of cases. In cases of jaundice, however, there is considerable uncertainty both as to the permissability and the value of cholecystography. This matter is well reviewed by Ottenberg.[4] Here it is pointed out that the toxicity of the dye is low and that reactions from intravenous use, chiefly in earlier days, were probably due to impure chemicals or faulty technique. Since 1930, numerous cases of jaundice have been given this test both by oral and intravenous route with no ill effect. In general it appears safe unless the liver is severely damaged. Ottenberg suggests that a very low value for the cholesteral esters in the blood (40 mgms. per 100 cc. or less) or a galactose excretion of 3 or more gms. contraindicate the test. The presence of less than 3 gms. of hippuric acid in Quick's test or the presence of tyrosine in the urine are also contraindications.

A gall bladder series is not often of much value in jaundice cases. This is because in so many instances the gall bladder fails to visualize and because in these instances, one cannot be sure whether the jaundice is due to obstructive pathology or to parenchymal failure to excrete. If the gall bladder does visualize and functions well, obstruction is, of course, ruled out.

A number of interesting studies of the liver and spleen have been made with thorotrast, a colloidal thorium dioxide preparation. However, it has not as yet been sufficiently well proven that this preparation is innocuous. Until such time its use had best be held in abeyance, certainly as a routine procedure.

COLON

In the search for possible neoplasm, it is decidedly advisable to supplement a barium enema by proctoscopy whenever a lesion in the rectum or lower sigmoid is suspected. The barium enema is least reliable here and naturally this region is readily investigated by the protoscope.

In modern practice the barium enema is carried out with great attention to detail during the flow of barium and postevacuation

[4] Ottenberg, R.: Am. J. Roentgenol., 38: 859, Dec. 1937.

films are usually routine practice. In addition, air injections are used occasionally to supplement the barium and provide better mucosal detail by double contract. Personally, I like to study the colon by ingested barium whenever the functional state of the colon is in question. In general, ingested barium enema is indispensable for demonstrating neoplasm.

This by no means exhausts the possibilities of our subject. There are many other matters of importance not only pertaining to the G. I. tract but also to such things as gynecological and obstetrical roentgenology. Much interesting work is being done in pelvimentry and uterosalpingography. Also studies are being made concerning the diagnosis of placenta praevia. Much of this work has proven of great value.

MENINGOCOCCUS ANTITOXIN IN CEREBROSPINAL FEVER

WITH A REVIEW OF THE LITERATURE AND CASE REPORTS

By Lieutenant Frederick R. Lang, Medical Corps, United States Navy

Until comparatively recently, cerebrospinal fever was one of the most dreaded of all diseases. Its mortality rate, while varying considerably according to epidemiological conditions, was always relatively high. As a result of the intense irritation of the brain and spinal cord, death usually occurred with the patient either moribund or delirious and was frightful to witness. Until the development of antiserum, the attending physician had to stand by treating the case symptomatically, waiting for death or recovery, with no specific form of therapy in his armamentarium.

During the past few years, with the advent of the Ferry antitoxin, therapy, progress, and prognosis of this disease have shown further progress. One has but to witness the dramatic effect of antitoxin on a series of cases to become enthusiastic regarding its use. To see patients admitted, in extremis, in coma or wild threshing delirium, with pronounced opisthotonos, a very high temperature, and every evidence of advanced toxicity, and then after the administration of meningococcus antitoxin, to see the signs and symptoms of meningitis vanish in a few hours, is an impressive experience. In this series of cases every patient who was admitted comatose or in delirium was restored to consciousness and was rational within 8 to 15 hours after antitoxin therapy was instituted.

Between October 1936 and October 1937, a series of 13 cases of cerebrospinal fever was treated in the contagious wards of the U. S. S. *Relief*. Twelve of these were treated entirely with Ferry antitoxin, plus supportive measures. One case, discussed more fully later, because of its unusual severity, complications, and prolongation, was treated by a variety of measures. Among the 13 cases, there was

no fatality. Two were invalided from the service because of disabilities resulting from complications.

In 1931, Ferry[1] and his co-workers discovered that the meningococcus developed a soluble exotoxin which when injected into laboratory animals resulted in the production of signs and symptoms of meningitis. These appeared to be of the same degree of severity as those produced when meningococci were introduced into the spinal canal of animals. Ferry demonstrated that animals injected with the exotoxin developed a true antitoxin specific for the meningococcus which had produced the exotoxin. He further discovered that this antitoxin was to a lesser degree effective for all strains. In pushing his research farther, he learned that antitoxin, when injected intraspinally into laboratory animals, protected them against lethal doses of the live organisms when these were injected intraperitoneally.

It is generally agreed that the disease progresses through three more or less sharply definable stages which are a pharyngitis, a bacteremia, and a meningitis. However, there are some among the medical profession today who still believe in the theory of direct extension of the disease from pharynx to meninges, either by way of the lymphatics of the nasopharynx or of the ethmoid and sphenoid sinuses, or by way of the nerve filaments of the olfactory nerve through the cribiform plate of the ethmoid.[2] The majority of leading authorities believe in the three-stage theory which has more evidence to substantiate it.

There is evidence accumulating which seems to indicate that the usual progress of the disease may become arrested in either the first or second stages and that it may remain arrested in either of these stages, becoming subacute or chronic in its course. Repeated reference is made in the medical literature to the increase in so-called carriers during an epidemic. It is believed that for the most part, these are in reality mild subacute cases of the disease in its first stage rather than true carriers.

Silverthorne has done some interesting work in determining the carrier rate in a noncontact population over a 2-year period. A total of 1,227 swabs were taken from the pharynges of 63 normal healthy individuals at monthly intervals. He reported an incidence of 19.8 percent of these positive for meningococcus. On the monthly examinations, the number of positive individuals varied from 16 to 28 percent. The astounding fact brought out by this investigation was that 41 percent were positive at one time or another. Of these, 11 were persistent, 13 intermittent and 2 were transient carriers. The 11 persistent carrier strains were investigated by bacteriocidal and mouse-

[1] Ferry, N. S., Norton, J. F., and Steele, H. L.: Jour. of Immun., 21: 293, Oct., 1931.

[2] Leake, J. P.: Cerebrospinal Fever, in Practice of Medicine, by Tice, Vol. III, Chap. 6, p. 83, Publ. by W. F. Prior Co., Hagerstown, Md.

mucin tests. Three of these strains were reported as virulent, and 8 as avirulent or saprophytic. Silverthorne discovered that the carriers possessed bacteriocidal properties in their blood for the respective strains, irrespective of whether these strains were virulent or avirulent and that blood samples from carriers possessed bacteriocidal properties to certain cerebrospinal fluid strains.[3]

From the knowledge recently acquired, it is conceivable that many of the cases reported as carriers during an epidemic are not carriers but are actually mild cases in which progress of the disease has become arrested in the first stage. However, there is no real distinction between the epidemiological importance of a true carrier and a transient carrier. Each constitutes a menace in a community. In fact, as Stevens [4] points out, the healthy carrier who has picked up pathogenic meningococci from others is a greater menace than the patient with meningitis.

Leake has demonstrated that, while cases of cerebrospinal fever occur in all large communities throughout the year, a characteristic seasonal prevalence takes place and that this is in the late winter and early spring. He has shown from seasonal analysis that 76.9 percent of cases occurred between January and April inclusive. Endemic and epidemic peaks usually occur during this period.[2]

Although cerebrospinal fever often prevails simultaneously in different parts of the world, it never actually becomes pandemic. The majority of outbreaks are confined to a small area of a country, to a single city or even to a certain part of a city, and epidemics rarely last longer than 6 months in a locality. In the wake of these epidemics, sporadic cases continue to occur and under favorable conditions these may so increase in number as to form another epidemic. Epidemics of the disease are prone to occur in cycles every 9 to 10 years.[4]

Cerebrospinal fever is found in both tropical and arctic countries. There have been more epidemics and a heavier sporadic incidence in the United States than in any other nation. This is especially true if the World War European epidemics be omitted. Although the sharpest outbreaks are not in the large cities, the heaviest incidence, both epidemic and sporadic, is in the urban rather than the rural population. The nonspecific incidence in American localities may be expected to run less than 25 cases per 100,000 population per annum. The disease appears to fluctuate in waves of increasing and decreasing incidence. The periods during which the disease has been most prevalent are 1805–15, 1837–50, 1854–75, and from 1904 until the present.[2]

The case-fatality rate appears to vary according to year, section of the country, and general type of treatment. Recent reports of treatment with antitoxin therapy indicate that a marked reduction in

[3] Silverthorne, N.: Toronto Journ. of Ped., St. Louis, Mo., 9: 328, Sept. 1936.
[4] Stevens, A. A.: The Practice of Medicine, Pub. by W. B. Saunders Co., Phila., Pa., 1932.

case-fatality rate is to be expected following a more universal use of this form of therapy. Craster and Simon recently analyzed the cases occurring in Newark, N. J. during the past 19 years. They found a case-fatality rate of 75 percent in 1923, 25 percent in 1926, 40 percent in 1932, and 46.8 percent in 1936. It averaged 44.4 percent over the entire period [5]

Walsh made a recent survey of the incidence of the disease in 22 large American cities during the period 1920 to 1936 inclusive. The lowest fatality ratio for the period was 38 percent in Seattle, and the highest was 68 percent in San Francisco.[6] During the calendar year 1936, there were 51 original admissions for cerebrospinal fever with 13 deaths in the Navy. This constituted an admission rate of 41 per 100,000 and a case-fatality rate of 25.54 percent.[7]

During the period October 1936 to October 1937, covered by this analysis, a total of 30 cases were reported throughout the Navy, with 4 deaths and a case-fatality rate of 13.3 percent. It must be remembered that antitoxin therapy was in general use throughout the entire Navy during only the latter part of this period. In the 12 months preceding the period herein reported, 11 cases were treated aboard the hospital ship with 1 death and 1 invaliding from the service. The earlier cases of this group were treated with antiserum alone. Later a combination of antiserum and antitoxin was used, and as the antitoxin became more readily available, its use superseded other forms of therapy.

Table 1 reveals some general information concerning the 13 cases included in this report. Three cases admitted with the diagnosis of glandular fever were subsequently proven to be cases of meningococcemia without meningitis. The average number of days on the sick list before the diagnosis of cerebrospinal fever was established varied from 0 days (cases admitted with diagnosis already established) to 67 days, (case 4, with a subacute intermittent course of meningococcemia). The duration of acute symptomatology varied from 9 to 67 days, with an average of 21.8 days. The two extremes of total number of days on the sick list were 17 and 361 days. The latter case was one of the two invalided from the service because of complications. The average total days on the sick list was 43.2 for the 13 cases. This was increased considerably by the 2 cases invalided from the service, because of disability resulting from complications. No deaths occurred, 11 men were returned to a full duty status and the remaining 2 were invalided from the service. Four cases, or 30.7 percent, suffered exacerbations of the disease; 3 had 1 relapse, and 1 had a total of 5 exacerbations which emphasize the importance of a close follow-up

[5] Craster, Chas. V., and Simon, Henry: J. A. M. A., 110: 1069, April 1938.
[6] Walsh, G., J. A. M. A. 110: 1894.
[7] Statistics of Diseases and Injuries in U. S. Navy for Calendar Year 1936, Annual Report of the Surgeon General of United States Navy.

of each patient for several weeks after the acute signs and symptoms have disappeared

TABLE 1.—*General Course of the Disease*

Case No.	Rate	From USS	Admission date	Diagnosis on admission	Number of days			Day of exacerbation	Final disposition
					Before diagnosis established	Acute or subacute	On sick list		
1	F 1/c	*Nevada*	Sept. 28, 1936	Tons. chr	2	14	34	0	Duty.
2	S 2/c	*Texas*	Jan. 9, 1937	Cereb. sp. f	0	14	17	0	Do.
3	S 2/c	*Nevada*	Jan. 10, 1937	DU Cer. sp. f	0	13	361	19	Surveyed.
4	S 2/c	*Nevada*	Jan. 23, 1937	Gland. fev	67	67	85	0	Duty.
5	Cox.	*Vestal*	Feb. 9, 1937	Cereb. sp. f	0	14	38	0	Do.
6	BM2/c	*Minneap*	Feb. 15, 1937	DU Infl	1	20	52	0	Do.
7	S 2/c	*Texas*	Mar. 1, 1937	DU Cer. sp. f	1	16	39	14	Do.
8	WT1/c	*Pinola*	Mar. 19, 1937	Cat. fever	1	9	26	0	Do.
9	S 1/c	*Nevada*	Apr. 13, 1937	Gland. fev	25	39	70	11 13 22 26 31	Do.
10	S 1/c	*Nevada*	Apr. 27, 1937	Cereb. sp. f	0	13	39	0	Do.
11	S 2/c	*Nevada*	Apr. 28, 1937	Gland. fev	15	16	55	0	Do.
12	S 2/c	*Trenton*	May 12, 1937	DU Cer. sp. f	6	44	126	11	Surveyed.
13	F 3/c	*Calif*	Oct. 8, 1937	Cereb. sp. f	0	5	21	0	Duty.
		Average				21.8	43.2		

Signs of a recrudescence are: (*a*) Return of symptoms, especially headache, anorexia, nausea, arthralgia, myalgia or stiffness of the neck musculature; (*b*) increase in TPR after a period of normalcy; (*c*) return of leucocytosis; (*d*) positive spinal fluid findings, especially the reappearance of a meningitic type of colloidal gold curve, or a lowering of the spinal fluid sugar or chloride content, for these findings will often reappear before leucocytosis or the meningococci. Recrudescences or relapses occur in from 20 to 30 percent of cases. The quantitative estimation of the spinal fluid sugar as a matter of routine is of definite clinical importance. It forms a basis for being forewarned of a recrudescence and this finding presages a return of haziness or cloudiness.[8]

One must be on guard in order not to confuse signs, symptoms and findings of serum sickness with those of a relapse. Serum sickness may manifest itself with the appearance of headache, myalgia, arthralgia, swelling of joints, and increased temperature. In several of these cases the spinal fluid became hazy and the cell count increased concomitant with the appearance of serum sickness. However, it is to be emphasized that in these instances, the cells were predominantly lymphocytes and their presence is the guiding criterion.

This series illustrates the difficulty of correctly diagnosing a case of cerebrospinal fever in the early stage. Only four had the diagnosis

[8] Herrick, W. W., and Kennedy, Foster: Cerebrospinal Fever, in A Textbook of Medicine, by Cecil, R. L., Pub. by W. B. Saunders Co., Phila., Pa., 1938.

of cerebrospinal fever definitely established on admission. Difficulties in making the proper diagnosis occur particularly when: (*a*) The case is seen in the first or second stage; (*b*) the disease process becomes arrested in the first stage, constituting what might be termed a "false carrier;" or (*c*) in the second stage in which a temporarily or prolonged meningococcemia results. A case which has become arrested in the first stage may simulate catarrhal fever, simple pharyngitis, early influenza, acute or subacute tonsillitis, coryza or the early stages of some of the acute exanthemata. A case becoming arrested in the second stage becomes a meningococcemia, either subacute or chronic. It may be confused with staphylococcus bacteremia, undulent fever, relapsing fever, malaria, subacute bacterial endocarditis, or rheumatic fever, in its symptomatic or clinical course. The patient may have symptoms and a temperature chart resembling early pulmonary tuberculosis or pyelitis and the toxemia often present may resemble that resulting from such conditions as chronic sinusitis, chronic prostatitis, chronic cholecystitis, chronic middle ear disease or of chronic salpingitis.

The diagnosis of glandular fever, with which three cases were admitted, apparently was based on a positive Paul's test. The histories and clinical courses of these were very similar. With each, the symptoms, onset, and early course of their infection were suggestive of a mild subacute or hidden chronic infection, and presented no indication suggestive of cerebrospinal fever. All three later proved to be cases of meningococcemia. Their onset was gradual with common complaints of loss of appetite, malaise, occasional periods of feverishness and chills, and frequent mild headache. Each complained of generalized myalgia, occurring periodically with the above. Cases 4 and 9 complained of arthralgia. Case 4 also complained of muscle tenderness and occasional nausea and vomiting. Case 9 had hypertensive deep reflexes and had painful and tender posterior cervical lymph nodes. This case also exhibited transitory subcutaneous nodules at one time during its course. Cases 9 and 11 had a few petechiae at variable intervals. All three cases were characterized by having the above symptoms and findings occurring in definite exacerbations, variable in intensity, indefinite in intervening time intervals, and inconstant in duration.

Case 4 was the most baffling, inasmuch as it was the first of these unusual cases and there were no leads suggestive of any definite diagnosis. The patient underwent study and observation, receiving symptomatic treatment for a period of 72 days before the true diagnosis was determined and specific treatment instituted. During this period, the patient was considered as a suspect for a variety of diseases, included among which were undulant fever, relapsing fever, syphilis, and malaria. Agglutination tests, frequent blood smears

and frequent blood cultures were made with negative results. The various foci of chronic infection were eliminated as possible etiological factors. The irregularity of the temperature chart which was shown during each exacerbation and the nature of the clinical course of the individual exacerbations strongly suggested the cause as being intermittent showers of bacteria entering the blood stream. Therefore, a blood culture was ordered to be taken each time that the patient's temperature reached 101°. After several negative blood cultures, one showed an organism which on morphological and cultural study proved to be meningococcus. Institution of specific antitoxin therapy was promptly efficacious. The exacerbations stopped and the patient felt improved for the first time in months. As a result of this experience, cases 9 and 11 which were similar in nature and progress, underwent a shorter period of hospitalization before the diagnosis of meningococcemia was made.

A thorough search of the literature reveals a comparative paucity of articles and case reports on meningococcemia. One of the most comprehensive articles on cerebrospinal fever was written 16 years ago by Bloedorn. In it he recognized and repeatedly stressed the fact that a meningococcemia may exist as a disease entity without meningitis ever developing. His impressions were that the cases of meningococcemia, if untreated, would eventually develop localizing signs either in the meninges, in the joints or in the endocardium. It is highly significant that 16 years ago he stated, "Nevertheless, a primary meningococcemia may exist for weeks or even months either with or without localizing symptoms." [9]

Leake states that cerebrospinal fever may occur without a meningitis.[2] Stevens calls attention to the fact that occasionally meningococcemia may occur as a primary condition and may persist without the occurrence of meningitis, and that this condition may or may not terminate in meningitis, after existing for a long period extending for weeks or even months.[4] Conklin reported two cases of meningococcemia. For one of these cases, consultants suggested everything from typhus to drug dermatitis and diagnosis was finally established on the finding of a positive blood culture.[10] Grundy and Phalen reported a case of meningococcemia without meningitis. They state that the condition is undoubtedly more common than is generally appreciated and that the triad of chills, arthralgia, and skin eruption should prompt one to secure an early blood culture.[11]

It has been shown that the rash is caused by bacterial emboli becoming lodged in the arterioles and capillaries in the stage of bacteremia. In fulminating cases petechiae may be found anywhere

[9] Bloedorn, W. A.: Meningococcus Septicaemia, Naval Med. Bull., May 1922.
[10] Conklin, C. B.: Med. Annals of Dist. of Columbia, Dec. 1935.
[11] Grundy, F. W.: and Phalen, T. H.: New York State Jour. Med., March 1937.

on the body particularly on the extremities. They have a particularly "flea bite" appearance and it is from this fact that the expression "spotted fever" has arisen. An important point is that they do not fade on pressure and may vary from a pin point to one-half inch in diameter.

Boone and Hall reported a case of cerebrospinal fever septicemia in which the meningococci were discovered in a direct blood smear. The patient succumbed to the disease 2½ hours after admission and only 5 hours from the time he was first seen by a medical officer. At autopsy, this proved to be a case of the Waterhouse-Fredericksen syndrome, in which the rapid collapse and termination with death are due to hemorrhage into the adrenals. In this type of case, the life of the patient rests upon prompt diagnosis and the immediate administration of specific antitoxin.[12]

The usual symptoms and laboratory and physical findings of a prolonged meningococcemia are: (*a*) Persistent or intermittent periods of malaise and lassitude; (*b*) irregular or intermittent fever; (*c*) intermittent chills; (*d*) periods of moderate or profuse perspiration; (*e*) joint pains; (*f*) recurrent macular or maculopapular rash; (*g*) recurrent showers of petechiae; (*h*) leucocytosis during febrile periods; (*i*) marked drop in sedimentation rate; and, (*j*) loss of appetite and weight during exacerbation. Frequent blood cultures should be taken during the febrile periods. In a case of meningococcemia, the meningococcus usually can be isolated from the blood by persistent repeated culturing, especially if ascitic broth is used as a culture medium. It must be borne in mind that failure to secure a positive blood culture does not rule out the possibility of meningococcemia.

Hence, meningococcemia may present a very perplexing diagnositic problem. It may be looked upon as a chronic low-grade intermittent form of an acute contagious disease which may be mild or may be severe in its course. If untreated, it may either clear up spontaneously or result fatally. Apparently, owing to a low degree of virulence of the organism, or to a high degree of resistance of the patient, or to a combination of both, the disease progresses to the second or bacteremic stage where further progress becomes arrested.

Among the three cases admitted with the diagnosis of glandular fever, and which subsequently were proven to have meningococcemia, one had enlarged painful posterior cervical lymph glands. Another had painful and tender enlargement of the inguinal nodes as well as enlargement of the posterior cervical chains. The third had generalized painless lymphatic adenopathy. All three had negative Kahn tests. The diagnosis of glandular fever apparently had been based

[12] Boone, J. T., and Hall, W. W.: Naval Med. Bull., Oct. 1935.

upon these findings plus the fact that all three had a positive Paul's test. Specimens of blood had been sent to a naval hospital, and all three were reported as containing heterophile antibodies in sufficient titer to constitute a positive Paul's test. However, it must be emphasized at this point that not one of these cases either prior to or after admission showed any indication of a mononucleosis.

Paul's test is a nonspecific heterophile antibody test. The term "heterophile antibodies" refers to the hemolysins and agglutinins found in the blood serum which react with a nonspecific antigen. In the Paul's test, the antigen is sheep erythrocytes, something which seemingly has nothing to do with the formation of the antibodies. Davidsohn is generally recognized as being the leading investigator of the Paul's test. In a recent article [13] he emphasizes the fact that the heterophile antibodies, which are responsible for the agglutination of the sheep erythrocytes, and the presence of which constitutes a positive Paul's test, are found in low titers in the blood stream of most normal individuals, and that 1 percent of normal individuals show a titer as high as 1:56. Among a large group of individuals studied by him the average heterophile antibody titer was 20. In a series of three cases studied by Davidsohn, suffering from serum sickness, the average titer was found to be 91. In contrast with these he reported on a group of nine cases of glandular fever in which the average titer for these antibodies was 342. Davidsohn mentions eight conditions, including meningitis, which may give a positive Paul's test.

A comparison of the symptomatology in this series of cases is presented in table 2. It will be seen that only one symptom, namely headache, was present in all cases. Stiffness of the cervical musculature appeared in 10 cases, and muscle tenderness in 9 cases. Sweating and mental apathy were present in only four instances.

TABLE 2.—*A Comparison of Symptoms*

Case No.	Sore throat	Head-ache	My-algia	Arth-ralgia	Mental apathy	Irrita-bility	Stiff-ness of neck	Mus-scle tender-ness	Nau-sea	Vom-iting	Sweat-ing	Chills
1	+	+	0	0	0	0	+	0	+	0	0	0
2	0	+	+	0	+	0	0	0	+	+	0	+
3	+	+	+	+	0	0	+	+	+	+	+	+
4	0	+	+	+	0	0	+	+	+	0	+	+
5	+	+	0	0	+	0	+	+	+	+	0	+
6	0	+	+	0	0	0	+	+	+	+	0	0
7	0	+	0	0	0	0	+	+	+	+	0	0
8	+	+	0	0	0	0	+	+	+	+	0	0
9	0	+	0	+	0	0	0	0	0	0	+	+
10	0	+	0	0	+	0	+	+	0	0	0	+
11	0	+	0	0	0	0	0	0	0	0	+	+
12	0	+	+	0	+	0	+	+	0	0	0	0
13	+	+	+	+	0	0	+	+	0	0	0	0

[13] Davidsohn, Israel: J. A. M. A., **108**: 289, January 1937.

A comparison of the physical findings in this series of cases is presented in table 3 which illustrates the commoner signs encountered and shows the wide variance in combinations. Rigidity of the neck, the most frequent finding, was present in 11 cases. Next in order of frequency were Kernig's sign, general hypertonia, and hyperactive deep reflexes. The highest individual case temperatures ranged from 101.0° to 104.4° F.

TABLE 3.—*A Comparison of Physical Findings*

Case No.	Neck rigidity	General hypertonia	Kernig's sign	Ankle clonus	Babinski sign	Opisthotonos	Brudzinski sign	Hyperactive deep reflexes	Absent superficial reflexes	Dissociated deep reflexes	Delirium	Convulsions	Coma	Rash	Herpes	Painful cervical lymph-nodes	Subcutaneous nodules	Petechiae	Highest temperature
1	+	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	101.0
2	+	+	+	+	+	+	+	0	0	0	0	0	0	0	0	0	0	0	101.4
3	+	+	+	+	+	+	+	0	0	0	0	0	0	0	0	0	0	+	103.4
4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	+	0	103.4
5	+	+	+	+	+	+	+	0	0	0	0	0	0	0	0	0	0	0	101.6
6	+	+	+	+	+	+	+	+	+	0	+	0	0	0	0	0	0	0	102.4
7	+	+	+	+	+	+	+	0	0	0	0	0	0	0	0	0	0	0	101.4
8	+	+	+	+	+	+	+	+	+	0	+	0	0	0	0	0	0	0	101.0
9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	102.0
10	+	+	+	+	+	+	+	+	+	0	+	0	0	+	+	0	0	0	104.4
11	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	+	103.8
12	+	+	+	+	+	+	+	+	+	+	+	0	0	0	0	0	0	0	103.8
13	+	+	+	0	0	0	0	+	+	0	0	0	0	0	0	+	0	0	103.4

TABLE 4.—*Laboratory Findings*

Case No.	Blood						Spinal fluid									
	White blood count 1,000 cells				Sedimentation index	Culture	Cell count 1,000 cells			Meningococci		Sugar	Chlorides	Meningitic gold curve	First normal day	Precipitation test
	On admission	24 hours after admission	Highest	First normal day			On admission	24 hours after admission	Highest	Smear	Culture					
1	25.4	13.8	25.4	5	20	+	6.2	2.5	6.2	+	+	42	660	----	----	0
2	35.0	33.4	35.0	16	----	0	12.6	12.6	29.0	0	0	12	680	----	20	----
3	28.6	11.8	28.6	3	57	----	9.8	------	9.8	+	------	50	------	----	----	----
4	30.6	31.1	30.6	67	54	+	.004	------	------	0	+	----	------	+	----	----
5	18.4	21.2	21.2	5	----	----	22.4	29.0	29.0	+	0	----	------	----	----	+
6	10.6	------	22.1	21	----	0	5.5	7.8	7.8	0	0	57	346	----	----	+
7	15.2	19.9	23.4	16	----	0	5.2	11.4	11.4	0	0	29	709	----	17	----
8	12.0	------	14.2	10	----	----	.002	.2	.2	0	+	32	660	+	13	----
9	9.9	------	14.1	21	47	+	9.0	------	9.0	0	+	----	------	+	27	----
10	19.0	------	19.0	----	----	----	13.0	2.5	13.0	+	+	----	------	+	9	----
11	12.6	------	14.5	37	11	+	.002	.1	.1	0	0	----	------	+	----	----
12	15.2	24.6	24.6	22	----	----	19.6	8.0	31.8	+	+	----	------	+	----	----
13	15.9	------	15.9	3	----	----	4.4	------	4.4	+	------	----	------	----	8	----

The principal laboratory findings pertaining to these cases are listed in table 4 for comparative study. It will be seen that the highest white blood count of each case varied between 14,100 and 35,000 and that 8 exceeded 20,000. Spinal fluid cell counts varied tremendously,

the range being between 4 and 29,000. Six cases had meningococci present in the initial spinal fluid smear and in four additional cases meningococci were demonstrated by spinal fluid culture. A blood culture was done on seven of the cases, chiefly because the organisms were found either in the spinal fluid smear or culture.

A marked lowering of the spinal fluid sugar content is almost specific for cerebrospinal meningitis.[8] [14] Neal states that early in meningococcus meningitis, the spinal fluid sugar may be normal, but as the disease progresses, it becomes decreased or even absent. She further points out that the spinal fluid sugar level parallels the improvement in the patient's condition, and that a fall in sugar content usually precedes an exacerbation.[15] Nissen ran spinal fluid sugar determinations on 529 patients. Some of these were normal, some had meningitis and some had evidence of meningeal irritation. He found that among 282 patients with benign disturbances of the meninges, only 3 showed values below 40 milligrams per 100 cc. and 28 patients with malignant disorders of the meninges had values under 40 mgms. He considered a spinal fluid sugar content of 40 mgms. as the boundary between benign and malignant disturbances of the meninges and concluded that determination of sugar in the spinal fluid is of greater diagnostic value than the cytological examination.[16]

This writer is not in complete accord with such a conclusion, but is of the opinion that spinal fluid sugar content is of definite value in cases in which it is impossible to find the causative organism, but which clinically are suffering from cerebrospinal fever. Spinal fluid sugar determinations were made in six of these cases. Values varied between 12 and 57 mgms. Cases 2 and 7 had a lowering of the spinal fluid sugar content to 12 and 29 mgms. respectively.

The spinal fluid chloride content is extremely constant at 720 to 750 mgms. per 100 cc.[14] [17] All agree that lower values occur in all types of acute generalized meningitis except the syphilitic and that the chloride values are not lowered in brain abscess, brain tumor, encephalitis or hydrocephalis. A lowering of the chlorides is much more significant than an increase. Among the 5 cases of this series in which chloride determination was done, the values varied between 346 and 709 mgms.

For the most part sugar and chloride determinations are not considered essential except in those cases which are clinically cerebrospinal fever but in which the meningococcus cannot be found.

Throat cultures were taken in only 2 of the 13 cases, both were negative.

[14] Greenfield, J., and Carmichael, E. A.: Cerebrospinal fluid in clinical diagnosis. Publ. by MacMillan and Co., London, England, 1935.

[15] Neal, Josephine, M. D.: Meningococcus meningitis in children, J. A. M. A., 105: 568, Aug. 24, 1935.

[16] Nissen, N. I.: Valuation of determination of sugar in spinal fluid in the diagnosis of meningitis. Ugeskrift for Laeger, Copenhagen, 98: 1309, Jan. 31, 1936.

[17] Osgood, E. E.: A Textbook of Laboratory Diagnosis, 2d ed., 1935, p. 1309, P. Blakiston and Son Co., Inc., Phila., Pa.

Sedimentation rates were made on five of the cases. These ranged from 11 to 57 mm. drop in 60 minutes.

The spinal fluid precipitation test, done on cases 5 and 6, proved positive in both cases. This test is specific and is of great material value in diagnosing cases in which the meningococci cannot be found.

Reduction of colloidal gold occurred in the six cases in which this test was tried. All had a meningitic type of curve. It is significant that the three meningococcemia cases (cases, 4, 9, and 11), had a meningitic type of colloidal gold curve before positive blood cultures were obtained. In all three, the spinal fluid was otherwise negative.

The therapy of these 13 cases is outlined in table 5. Based on the theory that the general symptomatology is a direct result of the dissemination of meningococcic exotoxins throughout the body by way of the blood stream it was concluded that intravenous therapy is by far the most important means of introducing the specific antitoxin. This method results in a prompt dispersion of the antitoxin throughout the fluid media of the body where it can more quickly come in contact with and neutralize the greatest amount of meningococcus toxin. For this reason, practically all cases received large initial intravenous doses.

TABLE 5.—*Antitoxin Therapy*

Case No.	Antitoxin administered during the first, second, and third 24-hour periods after diagnosis was established and total amount received during entire course of treatment							
	1,000 units intravenously				1,000 units intraspinally			
	First	Second	Third	Total	First	Second	Third	Total
1	50	30	20	120	30	----	----	30
2	100	70	50	270	0	0	0	0
3	100	60	50	300	0	0	0	0
4	0	0	0	0	10	0	0	10
5	60	60	60	240	0	10	10	30
6	60	60	60	180	0	0	0	0
7	40	60	60	160	0	0	0	20
8	0	0	0	0	10	0	20	30
9	0	0	0	115	0	0	0	10
10	30	40	60	130	10	10	0	30
11	50	0	0	90	0	0	0	0
12*	50	55	50	295	10	15	10	95
13	60	60	0	120	0	0	0	0

* Case 12 also received 48 cc. of antiserum intravenously and 20 grams of Prontosil by mouth and 375 cc. of 2.5 percent solution Prontosil intramuscularly over a period of 9 days.

As soon as the diagnosis of cerebrospinal fever was established, the usual technique in treatment of these 13 cases was as follows:

(*a*) For a mild or moderately severe case, an initial dose of 50-60,000 units of meningococcus antitoxin was administered intravenously.

(*b*) For severe cases the initial dose was 100,000 units.

(*c*) Intravenous antitoxin therapy was repeated daily or at 48-hour intervals until first, meningeal irritation had decreased or disappeared, second, the spinal fluid was free of meningococci and had approached normal, and third, the TPR had reached normal and remained there. For intravenous use the antitoxin was diluted one part to three parts of 10 percent glucose in saline. When the patient

was found to be sensitive to horse serum, he was desensitized, and the dilution was increased. The dilution was also increased in those cases in which the patient showed evidence of marked dehydration, or of extreme toxicity. All intravenous therapy was administered by the gravity method and with the mixture at body temperature. The antitoxin was not diluted when given intrathecally.

(*d*) Intraspinal antitoxin was given only in cases in which there was evidence of severe meningeal irritation.

(*e*) Spinal taps were performed frequently for the purpose of relieving intracranial pressure and to remove the organisms.

(*f*) Fluids were forced and the patient placed on liquid or soft diet.

(*g*) Narcotics and analgesics were given to relieve delirium, opisthotonus, arthralgia, and myalgia.

(*h*) Soporifics were administered to induce sleep and rest.

(*i*) Other symptomatic and supportive measures were taken as indicated in the individual case.

Cases 2, 3, 6, 11, and 13 were treated by the intravenous method alone. Cases 7 and 9 received small doses of antitoxin intraspinally only after a recrudescence had occurred in which meningeal symptoms and findings predominated. All cases received intravenous antitoxin on the day the correct diagnosis was established but only five cases received intraspinal antitoxin immediately. Of these five, three were of marked severity and had a preponderance of meningeal symptoms and findings. Case 4 was one of mild meningococcemia. Because of the mildness and relapsing character of the case plus the predominance of cerebrospinal findings and despite the fact that both blood and spinal fluid cultures were ultimately positive, it was decided, purely as a matter of scientific interest to limit treatment to the intrathecal route alone and to observe the results of administering a minimal dose. Since the antitoxin comes in 10,000 unit ampules this was the dose used. Only one injection of this dosage was given intrathecally and this proved sufficient to arrest and overcome the disease. Case 8 also received treatment by the intraspinal route alone. Meningococci were found present after repeated blood culture. This case was a mild one. Again, for scientific comparison, it was decided to restrict treatment to intraspinal therapy only. This case received 10,000 units on the day diagnosis was established, and remained permanently cured after an additional dose of 20,000 units on the third day. The comparatively small amounts used in these cases attest their mildness and possibly the avirulence of the strain of organism.

The initial intravenous dosage of the moderately severe, and severe cases varied from 30,000 to 100,000 units. The average initial dose was 60,000 units. Total intravenous dosage of antitoxin varied between 115,000 and 300,000 units. For all cases treated in this fashion, the average total intravenous dosage was 183,636 units. The total amount of antitoxin given intraspinally per case varied between 10,000 and 95,000 units. In the latter instance (case 12), the case appeared

obstinate and intractable and did not respond well to treatment. The average total intraspinal dosage was 3,187 units. This average was raised considerably by case 12.

In theory, it would seem that the use of antitoxin is the treatment of choice because it is a specific antibody which, according to the researches of Ferry,[1] is capable of neutralizing meningococcus toxins contained anywhere in the body fluids, irrespective of strain. Antiserum, on the other hand, is to a marked degree specific according to strain. The antibody contained in antiserum functions by bacteriolysis. The rationale and specificity of this form of treatment is borne out by the results obtained in this series of cases. It is fully appreciated that 13 cases is too small a number from which to derive conclusions regarding a comparison of effectiveness of antitoxin and antiserum. However, these findings and results are substantiated by an increasing number of articles appearing in the medical literature.

A thorough perusal of the recent literature on therapy of meningococcus infection reveals a wide diversity of forms of nonspecific and specific treatment being practiced throughout the medical world. On the question of the effectiveness of intraspinal as compared with intravenous forms of treatment, there appear to be three definite schools. One group, apparently in the minority, advocates spinal drainage plus the intrathecal use of serotherapy alone. Another group clings to the intravenous method alone. The third group believes in the use of a combination of the two methods.

The third method appears to be the most efficacious. If the case is one of mild meningitis, or of meningococcemia either with or without symptoms and findings of severe toxemia, or if it is a case in the stage of relapse, then it is believed that intravenous therapy alone will suffice. If, however, there are symptoms and physical findings indicating that the disease is severe and in the meningitic stage, then a combination of both methods of treatment is indicated. Specific treatment should be supplemented by supportive measures and by the treatment of individual complications. The question of placing sole reliance on the intravenous method of treatment with specific antibodies remains debatable. It can be settled definitely only by a comparative study of a long series of cases. Reports in the current medical literature indicate that this is the preferable method.

The effectiveness of the new type of antitoxin is brought out very forcibly in the light of recent articles which report a marked decrease in mortality rate that is directly attributed to the Ferry antitoxin. Levy states that the use of antitoxin has reduced the mortality rate from 53 to 35 percent for all ages.[18]

Hoyne states that, since the introduction of meningococcus antitoxin in the Cook County Hospital, the mortality rate has been reduced 50

[18] Levy, G. H.: Journal of Pediatrics, 11: 868, 1937.

percent and the period of hospitalization 20 percent. He voices an opinion that eventually it may be regarded as entirely unnecessary to administer any antibodies intrathecally if sufficient antitoxin is administered intravenously.[19] Clyde and Neely report that of 21 cases of cerebrospinal fever, 10 received Ferry's antitoxin with 100 percent recovery. The exact treatment of the remaining 11 cases was not known, but 8 of them died for a fatality rate of 72 percent. The patients who received antitoxin were given an average of 50,000 units of the antitoxin and averaged 137 days in the hospital. Two had permanent disability. From present knowledge, it would appear that insufficient amounts of antitoxin were given. Each of these 10 cases received antitoxin both intravenously and intraspinally. In the future the authors propose to treat all cases intravenously, and to resort to spinal puncture only for drainage to relieve pressure.[20] Morrison compares the relative efficiency of meningococcus antitoxin and antimeningococcus serum. In his analysis of two series of cases, one receiving antitoxin and the other antiserum alone, he notes a marked reduction in mortality rate and sick days and concludes that the antitoxin has a decided advantage over the antiserum.[21]

It is possible to test the specificity of antiserum by using it against a suspension of meningococci isolated from the pharynx, blood or spinal fluid. According to Herrick, in order to be effective, the serum should agglutinate in a dilution of 1 to 200. If it does not agglutinate in a dilution of 1 to 50, it will rarely be of benefit. If it is impossible to run this test, or if it is performed and the agglutionation titer is too low, then serum from another source should be tried.[8]

Case 12 was the only one in this series to receive antiserum. This case was by far the most severe. His condition was critical on admission and for a time it was feared that he would not survive. After receiving massive doses of antitoxin both intravenously and intraspinally during the first 3 days this case finally responded with a slow but satisfactory improvement, denoting control of the disease. He then suffered a combination of severe serum reaction and relapse simultaneously and his clinical condition showed a rapid downhill course. It was believed that re-institution of antitoxin therapy involved too grave a risk and since fatal outcome appeared imminent it was decided to try antiserum therapy. He was desensitized and received a total of 48 cc. of antiserum over a period of 36 hours with very little response. It was then decided to try the use of sulfanilamide. Due to his critical condition the maximum dosage as advocated was not given. No effect was noticeable from use of this compound.

[19] Hoyne, A. L.: Meningococcus meningitis; a new form of therapy, J. A. M. A. **104**: 980, March 23, 1935.

[20] Clyde, W. A., and Neely, M. G.: Meningococcic meningitis; ten cases treated with meningococcus antitoxin without a fatality, Alabama Medical Assoc. Journ., **6**: 227, Jan. 1937.

[21] Morrisson, H. J.: Antitoxin treatment of meningococcic infections, Georgia Med. Assoc. Journ., **25**: 365, Oct. 1936.

However, it is felt that this was not a fair test of the efficacy of this drug. After the acute stage of serum sickness had passed, antitoxin therapy was again instituted and the disease was finally brought under control. The patient was ultimately invalided from the service, after 126 days of hospitalization, because of a persistent foot drop.

Among the many other forms of treatment used either in lieu of or supplementary to specific antibody therapy, the outstanding is the use of the sulfanilamide compounds. Schwentker, Gelman, and Long report preliminary observations on the successful use of these compounds in the treatment of 10 cases of meningococcic meningitis and of 1 case of meningococcic septicemia. Their treatment is outlined as follows:[22]

> After diagnosis was established, sulfanilamide solution (0.8%) was injected intraspinally in amounts varying from 10 to 30 cc. As a general rule, they injected from 5 to 10 cc. less than the amount of spinal fluid withdrawn. Larger amounts were also given subcutaneously, approximately 100 cc. for every 40 pounds of body weight. This treatment was repeated every 12 hours for the first 2 days and once each day thereafter until definite improvement was evident. With some cases, the spinal fluid cell count dropped off rapidly. Culture of the spinal fluid of a number of patients was sterile after the first treatment, while for others several treatments were required, but in no case was the organism recovered later than 3 days after treatment was begun. None of the patients had any untowards effects which could be directly attributed to sulfanilamide. Among the group there was one death resulting primarily from pneumonia.

Willien reported on the use of sulfanilamide on five cases of cerebrospinal fever, and on one case of recurrence. All cases recovered. Two of the five had received massive doses of meningococcus antitoxin prior to starting on sulfanilamide therapy. This was discontinued on starting the latter treatment. His routine of treatment was as follows: [23]

> (*a*) Initial dose of sulfanilamide sufficient to saturate the tissues and to build up a high concentration in the blood. (0.8% solution in amounts up to 0.04 gm. per kilo of body weight.)
>
> (*b*) Oral administration every 4 hours day and night with a dosage graduated downward from an upper limit of 15 grains, dependent upon age and size of patients.
>
> (*c*) The drug is continued in reduced dosage for about 10 days after symptoms and laboratory findings have returned to normal.
>
> (*d*) Sodium bicarbonate is given with the sulfanilamide grain for grain in order to combat acidosis.
>
> (*e*) Avoidance of sulfate therapy in order to avoid methemoglobin formation.

Willien lists the following advantages of this form of therapy:

1. It obviates the necessity for frequent spinal punctures.

[22] Schwentker, F. F., Gelman, S., and Long, P. H.: The treatment of meningococcic meningitis with sulfanilamide. J. A. M. A., 108: 1407, April 24, 1937.

[23] Willien, Leon J.: Sulfanilamide therapy in meningococcus meningitis, J. A. M. A., 110: 630, Feb. 25, 1938.

2. It eliminated time, trouble, and expense of intravenous and intrathecal therapy.

3. It eliminates the danger of anaphylaxis and the discomfort of serum sickness.

4. It saves the high expense of antitoxin and antiserum.

The manufacturers of Prontosil and Prontylin point out that it is important to produce a high concentration of the drug in the blood stream as quickly as possible, and to maintain this as long as necessary. Also that absorption is rapid and that about 48 hours are required before the maximum therapeutic effect is obtained. They recommend large doses during the first 24 hours, or until acute symtomatology has subsided, then that treatment be continued in progressively smaller doses until a cure has been effected.[24]

A few German authors have reported success with the use of roentgenotherapy. Koehler has reported curing a case of meningococcus meningitis by applying repeated doses of x-rays to the spinal cord in fields of 6 x 8 or 10 x 12 cm. beginning at the caudal end of the cord and working upward successively. He reported the second irradiation of the lower fields as having the most noticeable effect, for the fever ceased after its application.[25] Hippe and Gruninger believe that they have secured a very favorable effect with the use of x-rays in treating two children suffering with cerebrospinal meningitis. In one case, treatments were applied to five cranial fields and to one cervical. Their object was to reduce secretion of the choroid plexus and thereby relieve the pressure on the brain and spinal cord rather than to destroy bacteria.[26] It would seem that this form of treatment is of doubtful value inasmuch as antiserum was being administered simultaneously.

Neal states that in prolonged cases of cerebrospinal fever, or in cases which have become intolerant to serum, the use of autogenous vaccine has been of certain value in some instances. She believes that the vaccine should be administered both subcutaneously and intraspinally.[15] Beckman claims that such a procedure seems illogical.[27]

Winer and Bakkal treated 50 cases of cerebrospinal fever and report on the use of autocerebrospinal fluid therapy. Seven received polyvalent antimeningococcus serum. Nineteen were treated by spinal drainage and supportive measures alone, and 24 by the subcutaneous injection of the patient's own cerebrospinal fluid alone. The authors do not report very favorably on the use of polyvalent serum. The results obtained from treatment by spinal puncture were poorest, since

[24] Winthrop Chemical Co., Balto., Md.: Leaflet on "Dosage of Prontosil & Prontylin."

[25] Koehler, G. B: Treatment of Meningitis by Roentgen Ray, Deutsche Medizinische Wochenschrift, Leipzig, 15: 304, 1936.

[26] Hippe, H., and Gruninger, U.: Klinische Wochenschrift, Berlin, 15: 304, 1936.

[27] Beckman, Harry: Treatment in General Practice, 3d ed. published by W. B. Saunders Co., Phila., 1938.

15 of 19 cases died. Of the 24 treated by autocerebrospinal fluid therapy, 19 recovered. The authors regard this simple method of treatment as favorable and promising.[28]

A combination of continuous lumbar drainage and continuous venoclysis with 0.4 percent saline is described by Herrick. The rationale of this treatment depends upon a continuous physiological flushing out of organisms from the subarachnoid spaces.[8] Fonde reports a therapeutic method based on experiments by Kolmer and others which have shown that spinal fluid contains little or no complement and that the addition of fresh serum to antiserum definitely increases its bacteriocidal power and opsonic activity. Fonde treated two cases in this manner and recommends that fresh human serum in quantity less than the amount of spinal fluid withdrawn be given within 24 hours after the initial intravenous and intraspinal injections of antiserum and that this in turn be followed within 24 hours by more antiserum. Within 48 hours after the injection, the spinal fluid of both patients became sterile. He believes that this is a valuable addition to the serotherapy of the disease.[29]

Complications encountered in this series of cases are outlined in table 6.

Eight of the 13 cases had serum sickness which appeared, ranging in time from the 4th to the 16th day with an average appearance time of 7.6 days. It manifested itself by symptoms and signs of pruritis, urticaria, arthralgia, myalgia, swelling of joints and angioneurotic edema. Transitory edema of the larynx appeared in two cases. Mild degrees of anaphylaxis appeared in cases 1 and 9. Symptoms were chills, dyspnoea, cyanosis, and rapid feeble pulse. These appeared while the patient was receiving intravenous antitoxin. Case 12 underwent a true anaphylactic shock, during which the patient became unconscious and pulseless and ceased breathing. This occurred 21 days after admission, during a relapse, and while the patient was receiving intravenous antitoxin. The man had been tested, found sensitive and had undergone the usual method of desensitization. Every patient was tested for protein sensitivity and whenever this was present, the patient was desensitized. The above reactions occurred despite such measures. Case 12 was tested each time serotherapy was administered. He had a series of complications, including a generalized hyperesthesia on the 30th day and symptoms suggestive of appendicitis, followed by petechiae and hematuria, to establish the correct diagnosis as hemorrhagic nephritis. Cases 3, 4, 5 and 9 developed a hydrathrosis of one or more joints. Recovery was complete. Case 6 developed a nonvenereal epididmyitis and

[28] Weiner, J. S., and Bakkal, S. F.: Autocerebrospinal Fluid Therapy in Epidemic Meningitis in Children, Acta Medica Scandinavica, Stockholm, **90**: 214, 1936.

[29] Fonde, Edgar C.: The Use of Fresh Human Serum (Complement) in Combination With Antiserum in the Treatment of Meningococcic Meningitis, J. A M. A., **105**: 110, 1935.

orchitis on the 15th day of the disease. Recovery was slow but complete.

Case 2 had motor weakness of all muscle groups of the left forearm and a severe prolonged neuritis of the left third, fourth and sixth cranial nerves. He failed to recover from this and was invalided from the service. In case 5, a lateral nystagmus appeared. This lasted about 10 days, then disappeared. Case 7 developed a paralysis of the right external rectus muscle. Recovery was complete. Case 12 developed complete paralysis of the right peroneal muscle group. Recovery was prolonged and incomplete.

TABLE 6.—*Anaphylaxis, serum reactions, and complications*

Case No.	Paralysis eye muscles	Peroneal paralysis (foot drop)	Neuritis	Anaphylaxis (chills dyspnoea rapid feeble pulse	Anaphylactic shock	Serum sickness urticaria	Serum sickness angioneurotic edema	Motor weakness forearm and hand	Neuritis forearm	Hydrarthrosis (serum sickness)	Epididymitis & orchitis	Nystagmus	Generalized hyperesthesia	Hemorrhagic nephritis
1				X		X								
2			X					X						
3						X			X	X				
4										X				
5						X	X			X		X		
6						X	X				X			
7	X					X								
8														
9				X		X	X			X				
10														
11						X								
12		X			X	X	X						X	X
13														

According to Herrick, the most common early complications encountered are purulent conjunctivitis, panophthalmitis, transitory amaurosis, optical neuritis, retinitis, and deafness due to involvement of the eighth cranial nerve. Other complications which may appear are pericarditis, endocarditis, meningococcus arthritis, hydrocephalus, and occasionally brain abscess.[8] Wood reports complications in 18 percent of his cases. They consisted chiefly of spinal block, blindness, deafness, and muscle paralysis.[30] Lazear encountered 33 major ocular complications among 266 cases of cerebrospinal fever. On the theory that perhaps the large number of ocular complications might be due to the intraspinal method of treatment, he discontinued intrathecal injections and administered all specific therapy intravenously. This resulted in a considerable reduction in the number of ocular complications and he is of the belief that the introduction of antitoxin therapy has also reduced the number of ocular complications very materially.[31]

[30] Wood, R. B.: Tenn. State Med. Journ. **28:** 471, 1935.

[31] Lazear, N. K.: Ocular Complications of Epidemic Meningitis, Archives of Ophthalmology, **16:** 847, 1936.

There is one hazard incident to intraspinal treatment which should be emphasized. Occasionally it is reported that patients have expired suddenly while receiving or shortly after receiving intrathecal treatment. It can usually be shown on autopsy that an acute hydrocephalus has developed.[5] Withdrawal of spinal fluid apparently causes too rapid a disturbance of pressure which results in herniation of the medulla through the foramen magnum and death from compression of the vital centers contained in this part of the brain. If while administering intraspinal therapy, or withdrawing spinal fluid for the relief of pressure, the patient suddenly develops respiratory embarrassment or begins to show evidence of shock, measures should be taken immediately to increase the intraspinal pressure by injection of the spinal fluid withdrawn, antitoxin, antiserum, normal saline, or even air.

Reference should be made to the work of Ferry and Steele who are pushing their researches on meningococcus antitoxin farther into the field of immunization. They have devised a skin test for determining susceptibility to the meningococcus by use of a mixed toxin prepared from cultures. With this, they tested a large group of children and found that approximately 50 percent were susceptible. Those who exhibited sensitivity were injected with a standardized immunizing toxin mixture. All were retested at the end of 8 weeks to determine how many had become immune. Of the 232 who received the full number of injections, 155 or 66.8 percent gave immune reactions. These investigators are of the opinion that this percentage would have been higher had larger immunizing doses of the toxin been used.[32] These studies open a new field for the control of the disease.

CONCLUSIONS

1. The so-called increase in carrier cases during an epidemic of cerebrospinal fever is not due to an increase of actual carriers. The increase is due to true infections of the disease which have become arrested in the first stage of the disease.

2. Meningococcemia is a definite disease entity. It is a low grade, subacute or chronic form of cerebrospinal fever which has become arrested in the second stage.

3. Undoubtedly many cases of meningococcemia are overlooked.

4. A patient having exacerbations of temperature, chills, skin eruptions, arthralgia, and myalgia should be suspected of having meningococcemia.

5. Diagnosis of meningococcemia can be established by positive blood culture or by the presence of a meningitic colloidal gold curve even though all other spinal fluid findings are negative.

[32] Ferry, N. S., and Steele, A H.: J. A. M. A. 104: 983, 1935.

6. A marked lowering of cerebrospinal fluid sugar content when associated with signs and symptoms of meningeal irritation, is pathognomonic for cerebrospinal fever, even though meningococci cannot be found in the spinal fluid or blood.

7. A lowering of spinal fluid sugar occurs before the meningococci appear in the spinal fluid. There is also a decrease of spinal fluid sugar preceding a relapse.

8. In the absence of other specific findings, diagnosis can be established by the blood or spinal fluid precipitin test for the presence of meningococcic precipitins.

9. Meningococcemia may simulate staphylococcus bacteremia, undulant fever, relapsing fever, malaria, subacute bacterial endocarditis, or rheumatic fever.

10. To guard against relapse, all cases of cerebrospinal fever should be kept under observation for a period of several weeks after all symptoms and findings have disappeared.

11. At the present time, meningococcus antitoxin is the most specific form of therapy known for the treatment of cerebrospinal fever.

12. Intravenous therapy alone or in combination with intraspinal therapy is superior to intraspinal therapy alone, since larger amounts of antitoxin can be administered and the antitoxin is carried by the blood stream to all parts of the body, thereby neutralizing the greatest amount of toxin.

13. Large initial doses of antitoxin should be given intravenously as soon as the diagnosis of cerebrospinal fever has been made.

14. The skin test for cerebrospinal fever susceptibility opens a new field of approach in the control of the disease.

15. Recent experiments on establishing an acquired immunity in susceptibles promises to be the final step in relegating this disease to the class of controllable contagious diseases, thereby paralleling the history of diphtheria.

IMMUNE GLOBULIN EXTRACT FOR MEASLES

By Lieutenant (jr. gr.) L. G. Llewelyn, Medical Corps, United States Navy

During the months of April, May, and part of June 1939 we were able to observe the administration of immune globulin extract (placental, human) in an epidemic of measles among the children of the post at Marine Barricks, Quantico, Virginia. As these patients were all children of service personnel, we were in a better position to observe, instruct and administer to them than to similar groups in civil life.

Injections were given only to children who had been definitely exposed to an actual case of measles. There were 105 of these definitely exposed children, ages ranging from 9 months to 18 years. Seventy

received the immune globulin for the purpose of modification only, that is, in order to allow the establishment of an active immunity as the result of a mild case. Four received the extract in an attempt at complete prevention, due to their detachment from the post and the consequent necessity of traveling. Thirty-one exposed children did not receive any extract, subsequently developed typical cases, and acted as controls. These were cases not brought to the attention of the medical department until after the prodromal symptoms had appeared.

The disease in the control cases followed its usual course, the patients being very sick for a period of 3 to 7 days, with temperatures ranging from 102 to 105 degrees and, in all cases, spasmodic coughing and vomiting. The rash appeared on about the fourth day of the illness and in all cases was very intense. Koplick spots were noted in approximately 60 percent of the cases. The white blood count varied somewhat from a leucopenia to a normal count, but the greatest percentage showed a typical blood picture, i. e.;

WBC	4, 150	RBC	4, 260, 000
Differential		Hgb	80–85 percent
Segs	45		
Lymphs	29		
Bands	14		
Monos	12		

The duration of the disease averaged 11 days from the onset of prodromal symptoms to disappearance of the rash. Complications followed in 38 percent of these 31 cases, otitis media occurring in 11 cases and a severe cervical adenitis in one. Although bronchopneumonia is considered the most frequent complication,[1] we were fortunate in that we did not have a single case, nor did we have any fatalities.

The four cases that received placental extract as a prophylactic measure were given two intramuscular injections of 2 cc. each, one day apart. In each case the first injection was given on the fourth day after the appearance of the rash in the case to which each had been exposed. Complete protection was accomplished in all four cases, and no reaction to the extract was noted. This confirms the findings of Joseph and Gleich[2] who report that none of the cases to whom they gave 4 c.c. of the extract developed any symptoms of the disease.

The 70 children who were given a modification dose of the immune globulin, with few exceptions, received the one intramuscular injection of 2 cc. on the fourth day after the contact case had developed the rash. Within 12 hours 11 children developed a mild reaction to the extract consisting of a slight fever and malaise for a period of 24 hours. No local reactions were noted. Twelve children (17%) who received this modification dose were completely protected and did not develop any

[1] Practice of Pediatrics—Holt & McIntosh: Sec. XIX; Chap. CXIX; 920.
[2] Archives of Pediatrics: 54: 307-310, May, 1937.

symptom of the disease whatever. Six children (8.6%) derived no benefit from the injection and suffered the usual more severe form of the disease.

The disease in the remaining 52 children who received a modification dose was mild, with temperatures averaging 101° and returning to normal within 2 days. The rash developed on about the fourth day but was in all cases much less intense than that observed in the control cases. Koplick spots were observed in 42 percent of these cases and a characteristic development was a macular eruption over the soft palate and uvula appearing about the first day of the illness and approximately 3 days before the appearance of the body rash. Coughing was observed in practically all cases but did not have the spasmodic character noted in the control cases. Emesis was completely absent. The blood picture in these modified cases, although tending toward a leucopenia, was not as marked as in the control cases. The following is a typical blood count for these cases:

WBC	7,400	RBC	4,400,000
Differential		Hgb	90 percent
Segs	45		
Lymphs	40		
Bands	8		
Monos	7		

The duration of the disease from the onset was materially reduced in these cases, by an average of 3 days. The only complication to follow this milder type of measles was otitis media, which occurred in five (9 percent) of the 52 cases.

SUMMARY

A modification dose of immune globulin extract (placental, human) was administered to 70 cases within 4 days after the appearance of the rash in the contact case.

It proved of value in 91.4 percent of the cases and had no beneficial result in 8.6 percent.

It afforded complete protection in 17 percent.

A prophylactic dose of 4 cc. administered to 4 cases afforded 100 percent protection.

The administration of a modification dose of 2 cc. of immune globulin is indicated in individuals exposed to a case of measles, particularly when it can be given within 4 days of the appearance of the rash in the contact case.

LOBAR PNEUMONIA, RECENT ADVANCES IN THERAPY

By Commander Kenneth E. Lowman, Medical Corps, United States Navy

According to mortality statistics, pneumonia takes about 100,000 lives a year in this country. It is the most devastating of the respiratory infections and the third commonest cause of death in the

registration area. For generations, of every 100 persons ill with lobar pneumonia, about 25 died. Of all deaths from pneumonia, lobar pneumonia accounts for slightly more than half. The pneumococcus is the causative organism in about 96 percent of all cases of lobar pneumonia. Pneumococci of types I and II produce about half of these cases.

The treatment of lobar pneumonia is in general similar to that of other acute infectious diseases. There are the same indications for bed-rest, quietness, careful nursing, regulation of elimination, and copious intake of fluids. As the disease is of relatively short duration, the caloric requirements are not so important as in prolonged infections such as typhoid fever, nor does hyperpyrexia or prolonged elevation of temperature require such attention. The outcome of a case of lobar pneumonia is complete cure unless death ensues from intoxication, suffocation, or suppuration. Therefore, our therapeutic measures should be directed toward the prevention or correction of any or all of these before they assume such a degree of severity as to threaten life. The following general management is recommended:

GENERAL MANAGEMENT

1. Absolute physical and mental rest in an airy, well ventilated room is essential. Fresh air at ordinary room temperature is all that is required.

2. Fluids, at least 3,500 cc. daily should be forced. The question of a high salt diet to replace lost chlorides is still a moot question.

3. Physical examinations should be discouraged. One a day is amply sufficient.

4. Morphine is the drug of choice, particularly in those cases who have severe pleural pain, and persistent hacking cough. We believe there is no other drug which will allay restlessness so well and permit the conservation of strength and energy.

5. The throat and nasal pharynx, should be carefully observed and treated accordingly; very often mildly astringent antiseptic nose drops and throat swabs will suffice. It is obvious that if an acute nasopharyngitis or tonsillitis goes unobserved or untreated, there will be that much more toxemia for the patient to overcome.

6. Distention and tympanites should be carefully observed and treated. Usually a rectal tube, hot abdominal packs and a few doses of pitressin will suffice. Tepid sponges in hyperpyrexia and early alkalinization should be attempted.

RESPIRATORY THERAPY

The danger of death from suffocation is a real one in lobar pneumonia although not so much so as in bronchiolitis and bronchopneumonia.

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1. Dyspnea.—Respiration, characteristically, is rapid and shallow, whether pain be present or not. But if it be present the shallowness may be still further accentuated on account of the restriction of respiration induced by the pleuritis. This type of respiration is one of the most portentous features of the disease, particularly if the rate increases above 45 per minute. Morphia, although it relieves the pain and thus may improve the respiratory excursion, is also a respiratory depressant and, therefore, must be used with caution. It has been contended by some that the initial stages of pneumonia are accompanied by a pulmonary collapse which is further accentuated by the shallow breathing. They contend that both of these features may be corrected by the inhalation of a 5 to 10 percent mixture of carbon dioxide in oxygen. This is well tolerated provided the pleuritic pain is not too severe. It greatly aids in overcoming any atelectasis, and also tends to reduce the respiratory rate and increase the depth as well as remove any anoxemia that may be present.

In addition to the tachypnea so characteristic of pneumonia, there is that feeling of respiratory oppression which is often relieved by the air in the sick room being kept fresh and free from dust and all irritating or unpleasant odors. Cold air is in itself a great aid to respiration, but care should be taken not to expose the patient to a draught which may produce too rapid cooling of the body surface.

If the outside temperature be above 40° F., the patient may with advantage, be moved in bed to the open air, or the windows of the room opened wide with the patient protected by screens:

Unnecessary movement of the patient is to be avoided, particularly from the recumbent to the sitting position. However, much comfort and relief of respiratory distress is often obtained by keeping the patient in a semi-orthopneic position either in a hospital bed or by an improvised back rest. But care must be taken that the abdominal respiration is not impaired by the patient being allowed to assume a position in which the abdomen is too concave. The lumbar curvature must be supported.

2. Cyanosis.—There are two types of cyanosis in pneumonia. First, there is that of the deep purplish hue which is usually present in the early stages and is due to a combined anoxemia and carbon dioxide retention. This is usually overcome by the inhalation of oxygen by proper methods with or without carbon dioxide. As the disease progresses the second type of cyanosis develops. This is characterized by pallor and a lavender hue. In this type of anoxemia there is also a reduction of the carbon dioxide content of the arterial blood and a loss of vasomotor control. The anoxemia *per se* is best treated by enriching the inspired air with oxygen to about 40 to 50 percent by placing the patient in an oxygen chamber; but, as few of

these are available, an oxygen tent, face mask, or even nasal catheter in children may be used. The glass funnel method is useless. The objective is to enrich the inspired air with oxygen to a concentration sufficient to raise the oxyhemoglobin in the arterial blood to 90 percent or over. This is indicated by the dissappearance of the cyanosis. The practice of prescribing its use for 5 to 15 minutes every hour is about as useless as bringing a submerged man to the surface intermittently for a like period of time.

3. Circulation.—The older conception that the myocardium was principally damaged led to the administration of the so-called cardiac stimulants, mainly digitalis. Critical analysis of the results led to many doubts as to its efficacy until now it is conceded that it has little or no value in this disease unless there is present auricular fibrillation or flutter, or congestive cardiac failure, when there are the same indications for digitalis as when pneumonia is not present. The other so-called cardiac stimulants are similarly useless. The mass of clinical and experimental evidence points to a much broader disturbance of function; namely, a loss of vasomotor control as shown by pallor, sweating, tachycardia, tympanites, and falling blood pressure. Although the disturbance is a complicated one, it would seem that the burden of disability has fallen upon the sympathetic system, probably through intoxication of the centers of the midbrain. In this conception an explanation is at hand for the many circulatory signs and symptoms which are found, indicating that not only the heart but also the whole circulation is at fault. The circulatory disturbance must be approached through removing the cause; namely, the pneumococcic toxemia and the anoxemia which is usually present, although frequently not recognized or its dangers sufficiently appreciated. Therefore, the two principal means of accomplishing this are the early use of antitoxic serum and oxygen therapy. As an emergency measure, slow (1 cc. per hour) intravenous injection of adrenalin in 5-percent glucose saline, or citrated whole blood, has rational indications.

4. Oxygen.—It is logical to assume that the vital organs, depleted of oxygen, are rendered thereby more susceptible to degenerative changes due to toxemia, and it is also logical to assume that by keeping them well supplied with oxygen they will thereby retain a definite resistance against toxemia and will be better able to function when called upon to do so. Oxygen therapy should be started as soon as the diagnosis of pneumonia is made, regardless of whether the patient is cyanotic or not. We know there is a varying degree of air deficiency at all times in this disease. To wait until the patient becomes definitely cyanotic is inexcusable, as by this time the devitalizing effects of anoxemia have already set in. The efficiency of oxygen in the treatment of pneumonia is practically in direct proportion to the

day the disease started. Perhaps the best and easiest obtainable method of administration is the tent. These are so perfected at present as to permit maintenance of an even atmosphere of 40 to 50 percent oxygen at an even temperature. Other methods are by nasal catheter and also by face masks. Most larger hospitals are equipped with oxygen rooms.

5. Diathermy.—Much has been written *pro* and *con* about the use of diathermy in pneumonia. The body's defense against pneumonia is believed to be largely due to the destructive action of enzymes produced by the leucocytes on the pneumococcus; this is mainly chemical and takes place more readily in the presence of heat. Diathermy is a convenient method of generation of heat in deep seated tissues. It is certainly a valuable addition to our treatment in that it affords immediate relief from pleural pain, produces deeper and slower respiration and promotes a general sense of relaxation to the patient. To obtain maximum benefit, it must be employed early; after red hepatization is well established, it is of no avail. A review of the literature indicates that the use of diathermy has resulted in a definite lowering of the mortality rate. Diathermy is contraindicated in tuberculous patients.

6. Pneumothorax.—If properly used there would seem to be a definite place for pneumothorax in our therapy of this disease. In favorable cases there is little doubt that it definitely renders the patient more comfortable by the reduction of pain and dyspnea. Early crisis or rapid lysis occurs in many cases in which it is employed. It should be used only in unilateral involvment when pleural adhesions are not present; never in children on account of the frequency of empyema following. This does not apply to adults. In cases with bacteremia the death rate is not reduced nor is it so efficacious after the third day. Good, even spectacular results, may be expected when used before the third day in unilateral and preferably single lobe lesions without adhesions or bacteremia. Positive pressure should not be induced on account of its tendency to cause contralateral spread. It should not replace antitoxic serum if this be available, although it may supplement it.

SULFAPYRIDINE THERAPY

Animal experiments and clinical studies suggest that this drug causes capsular degeneration and ultimate decapsulation of the pneumococcus with resultant loss of type specificity. Any bactericidal effect of the drug is apparently dependent upon the presence of leukocytes, and there is some evidence that the drug may be more effective when combined with specific sera. Its reactions are apparently no more frequent and are much less severe than those encoun-

tered with sulfanilamide. As with sulfanilamide, these reactions include nausea, diarrhea, skin eruptions, cynosis and leucopemia and may necessitate its discontinuance.

Dosage.—A suitable dose for the commencement of treatment in an adult appears to be 4 tablets (2 gm.) followed by 2 tablets 4-hourly for 20 hours and thereafter by the same dose 6-hourly. Administration of the drug should last at least 5 days, no matter how favorable the clinical course. The drug must not be discontinued on the first appearance of a favorable turn. This gives a total dosage of about 56 tablets (28 gms.) in 5 days. A reduction in the dose may be made for children; this, however, need not be proportional to body weight. There is strong clinical evidence to suggest that best results are obtained by giving the drug as soon as the disease is suspected, instead of waiting until the clinical diagnosis is established.

Comment.—A remarkable effect of the drug is an early and rapid fall in temperature and pulse rate and a corresponding improvement in the general condition of the patient. It would appear from experimental data that this group of drugs probably acts by neutralization of some bacterial metabolic function or enzymic activity.

SERUM THERAPY

Serum therapy is available for cases of pneumonia caused by pneumococcus only. The great majority of the cases of lobar pneumonia, a considerable percentage of cases of broncho-pneumonia, and an undetermined percentage of cases of postoperative pneumonia are caused by the pneumococcus. A reasonable effort should be made to determine the etiologic organism in every case of pneumonia at the earliest possible moment. By use of the Neufeld reaction, a bacteriologic diagnosis can be made within an hour or less. Determination of the type of pneumococcus is essential because serum is of no significant value except against its homologous type of organism. The sputum specimen must represent exudate expectorated from the lung and it should be examined promptly. A blood culture should also be obtained in every case of lobar pneumonia on admission to the hospital as an aid in prognosis and therapeutic approach.

Serum therapy should be started early when it is most effective. It should be employed even though the initial symptoms are mild, to prevent extension of involvement of the lungs, and because there is no reliable method of determining what cases will become severe. Pneumococci have been classified into at least 32 serologically specific types. Rabbit antisera have been developed for these types and are available commercially for therapeutic use for many of them. According to clinical reports, it may be administered in larger doses and more rapidly than horse antiserum. Also fewer doses are required.

The value of serum therapy is established. It has at least halved the mortality rate. It has shortened the period of illness. This has resulted in a saving of expense from hospitalization and concurrent treatment, such as nursing service and oxygen therapy.

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RECENT PROGRESS IN TROPICAL MEDICINE[1]

By Elliston Farrell, M. D.

Text books of tropical medicine have as their prototypes the early manuals on sea diseases written for the guidance of ship's surgeons in the spacious days of Elizabeth. This close association between tropical medicine and naval practice has persisted and there are many names like James Lind's which are remembered with equal pride by naval doctors and by civilian physicians in the tropical field. Knowledge of tropical medicine has always been particularly useful to the naval medical officer. Today familiarity with this subject is increasingly important ashore in the realm of general medicine, for modern transportation ignores ancient geographical disease barriers, and wider investigation into disease causes reveals the fundamental similarity of disease processes in all parts of the world. Tropical medicine is

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being incorporated into the main body of general medical knowledge. To this it imparts a much needed feeling for environment, a recognition of racial, climatic, geographic, economic, and social factors and a wider view of animal pathology. This survey of new additions to our knowledge of tropical medicine is necessarily incomplete but it can serve as a guide to recent literature.

PROTOZOAL INFECTIONS

MALARIA remains the major concern of tropical physicians. Explanation has finally been given for the old observation that *P. vivax*, in distinction from other human plasmodia, occurs in red blood cells which appear "swollen" in thin Wright's-stained smears (1). Actually these "swollen" cells are reticulocytes and their larger size is due not to parasitic invasion but to immaturity of the red cell. *Plasmodium malariae* and *P. falciparum* on the contrary both tend to occupy mature rather than immature red blood cells and so explanation is given for the oft-noted "shrunken" appearance of the infected red cells in these two infections (2).

What happens to the sporozoite after its inoculation into the human subject by the mosquito? Even yet no one knows, but is has been established for *P. falciparum* (3), as previously for *P. vivax*, that sporozoites certainly disappear from the peripheral circulation for about 1 week after infection. This has been determined by allowing infected mosquitoes to bite a susceptible individual, subinoculating his blood daily into other susceptible patients, and observing all for malaria. The original patient's blood remained innocuous for 6 days after he was bitten by infected mosquitoes, but blood taken the seventh, eighth, and ninth days gave malaria to others although it was not until the tenth day that clinical symptoms appeared in the patient from whom the daily blood samples were being drawn.

Though clinical symptoms of malaria occasionally develop at a time when the parasite count is as low as ten per cubic millimeter of blood, the average minimal density attained before the clinical disease appears is more likely to be 10 times as great (4).

Present knowledge of the host-parasite relationship in malaria has been summarized by two well-known workers in that field (5). The importance of immune processes in modifying the course of malaria has long been suspected but only recently has it been proved that a cellular and humoral defense mechanism actually does exist. Immune serum has been used to prolong the course, or even forestall the usual outcome, in ordinarily fatal infections of monkeys.

The terminology in use throughout the English-speaking world to describe the three commonest human plasmodia grossly violates rules of nomenclature, but has the important sanction of long usage (6).

Clinical reports accumulate of infections due to *P. ovale*, the species of human plasmodium most recently accepted as authentic. A study has been made of 108 primary induced infections with four strains of the parasite and the first of a series of detailed reports (7) describes an absence of natural resistance to this species, which may give it special therapeutic value. With few exceptions, *P. ovale* infections have been reliably reported only from equatorial and central Africa.

As regards diagnosis of malaria, detailed morphological studies with excellent color plates have been published (8) to elucidate the differences in appearance of the various stages of *P. vivax* in thick and thin smears. Diagnosis from thick film preparations is a matter of routine in the tropics. In temperate zones this simple technique should be more widely taught and utilized.

An improved biochemical test for malaria has appeared (9) but like other tests for increased serum euglobulin it is nonspecific. One of the most puzzling controversies in malariology, whether or not malaria influences the serological tests for syphilis, has apparently finally been decided (10). Wassermann and Kahn tests were studied before, during and after 25 mosquito-induced attacks of malaria and in every clinical case a positive reaction was obtained by one or the other method. In 21 of the 25 cases both tests became positive at some time, principally during the third week after the final chill.

Comparison of the therapeutic value of atebrin and plasmoquin with that of quinine has confirmed the special value of the two newer drugs but quinine is still regarded as the premier anti-malarial remedy because of its effectiveness, relatively low toxicity and the widespread knowledge of its use (11). Atebrin may have slight advantages over quinine in the treatment of individual cases under close medical observation but generally there seems little to choose between the two, certainly nothing as manifest as plasmoquin's marked gametocidal effect in *P. falciparum* infections.

It was inevitable that the sulfonamide derivatives should receive trial as anti-malarials. Their therapeutic efficacy in *P. knowlesi* infections of monkeys has been described (12). It has also been reported (13) that patients receiving proseptasine are resistant to the bites of mosquitoes infected with *P. falciparum*. For the rest, the evidence is conflicting (14) and data *pro* and *con* lead one to suspect that in fully developed clinical infections the sulfonamide drugs may cause the plasmodia to disappear from the blood so far as can be determined by clinical laboratory examinations but this effect is probably uncertain and temporary.

Mapharsen, a trivalent arsenical preparation, in intravenous doses of 0.04 to 0.06 gram given 3 or 4 times at 5- to 7-day intervals, has proved effective in *P. vivax* infections (15).

In the field of preventive malariology the most portentious happening of recent years has been the introduction of *A. gambiae* into Brazil. The profound significance of this event and its possible consequences are fully appreciated there (16). It is to be devoutly hoped that spread of this vicious malaria vector to the Amazon valley can be prevented.

The interesting experiment in control of malaria in rural Panama by drugs alone has now been continued for 9 years, and the observations on the eighth year have been published (17). This valuable work of the Gorgas Institute staff materially contributes toward a solution of the problem of malaria control in areas where, due to economic reasons or other causes, the naturalistic factors affecting mosquito propagation cannot be regulated. A summary of naturalistic measures now in use has been prepared by an expert League of Nations Commission (18), and the publication of this constitutes an important summary of modern methods, the introduction of which we owe to, among others, Sir Malcolm Watson (19), whose work in Malaya was once described by Ross as "the greatest sanitary accomplishment in the British Empire."

AMEBIASIS.—Knowledge of amebiasis has advanced rapidly on the wave of interest which followed the Chicago epidemic of 1933. Strains of *Entameba histolytica* can be assigned a pathogenic index (20) based on the extent and severity of the lesions in experimentally infected kittens. It can no longer be assumed that the only infective forms are the cysts and that the trophozoites are harmless, because a proportion of trophozoites given to dogs by mouth pass through the stomach and small intestine in a viable condition (21). The presence of ingested red cells is commonly regarded as a reliable point of differentiation between the trophozoites of *E. histolytica* and those of *E. coli*. An instructive case report (22) has revealed the fallacy of this belief by citing an instance where amebae containing ingested red cells were proved to be *E. coli*, not *E. histolytica*, though the stools were dysenteric. The bleeding ceased when a sigmoidal polyp was removed and the patient was spared futile courses of amebicidal drugs because of the unusually thorough parasitologic and clinical examination.

The efficacy of carbarsone in the treatment of amebiasis has been examined and confirmed by work at the Gorgas Institute (23). Divided doses of carbarsone given after meals over a 10-day period are recommended. The dosage varied from 0.5 gm. daily for 10 days in the case of carriers weighing 70 kg. to as much as 1.75 gm. daily for 10 days in selected active cases of similar body weight. Certainly the maximal dose suggested should be employed with much caution. Late results in amebic hepatitis and liver abscess at the Mayo Clinic have been reviewed (24). There they recommended emetine to check

the acute symptoms and arsenic to wipe out the amebas. Emetine gr. 1 is given twice daily for 3 days; then, after a week's interval, it is resumed in b. i. d. doses of gr. 2/3 with treparsol gr. 4 by mouth after meals for 4 days.

The infected food handler is apparently a less important agent in the dissemination of amebiasis than hitherto has been believed (25), for controlled study of 919 individuals in fourteen naval messes served by *E. histolytica* carriers showed no significantly greater incidence of infection in these exposed groups than in the control units served by entameba-free individuals.

LEISHMANIASIS.—American priority in the discovery by J. H. Wright in 1903 of *L. tropica*, the causative organism of oriental sore, is questioned by an authoritative writer (26) who has translated and republished the contribution on the subject made in 1898 by P. F. Borovsky, a Russian army medical officer, who died in 1932.

TRYPANOSOMIASIS.—Laboratory infections offer regrettable but rich opportunity for case study. A mischance in Professor Brumpt's laboratory in Paris which resulted in the development of Chaga's disease in one of the workers has been well reported (27) and is of interest because of the apparently effective use of a new Bayer product known as 7602 and because the diagnosis was not made by recovery of the organisms directly from the blood, in which they could never be found, but by their subsequent demonstration in clean insect vectors of *T. cruzi* which were allowed to feed on the patient the fifteenth day of the disease.

VIRAL DISEASES

YELLOW FEVER.—The Rockefeller campaign to eradicate yellow fever from the Americas has been actively pressed for 25 years. The measures of control developed in Brazil are chiefly three: viscerotomy,[1] anti-aegypti measures and vaccination (28). The pathological changes in the liver typical of yellow fever may occur under other circumstances (29), but examination of viscerotome specimens under the controlled conditions obtaining there is a reliable guide to the probable incidence of fatal yellow fever in certain parts of Brazil. Anti-aegypti measures have proved effective against urban yellow fever but of course have no part in the control of the non-aegypti-transmitted type of yellow fever of the Brazilian jungle (30). From the standpoint of the individual and the community, yellow fever vaccination seems safe and practicable (31) but little hope is entertained that in the jungle yellow fever districts, as distinguished from the urban aegypti areas, vaccination

[1] By viscerotomy is meant the postmortem removal of a specimen of liver tissue through a stab wound by use of an instrument known as a viscerotome. These specimens are taken in cases of suspected yellow fever and are sent to a central laboratory for examination. A number of silent Brazilian yellow-fever foci have been revealed. For details of organization and for pictures of the instrument see Rickard, Am. J. Trop. Med., 17: 163, 1937.

can do more than prevent human carriers from transporting the disease to uninfected areas. Furthermore, there is disquieting evidence that vaccination does not always confer protection (32), due to faulty preparation of the vaccine, its inactivation or to individual variation. Yellow fever vaccination in the past has not always been without untoward complications, particularly jaundice (33). An important article on this subject (34) offers evidence that this jaundice probably has resulted from accidental transfer of a hypothetical virus of infective hepatitis along with the attentuated strain of yellow fever virus used in immunization. Pooled human serum, it is concluded, should not be used for human inoculation until the medical history of all the donors can be followed at least 1 month, the probable incubation period of infective hepatitis.

RICKETTSIAL INFECTIONS

The discovery in the United States of a new tick-borne rickettsial virus pathogenic for man (35) may have considerable significance. The possible identity of this new infection with the "Q disease" of Australia is suspected but not yet established. Other recent work in connection with the rickettsial diseases (36) has furnished the first experimental evidence of the development in animals of a true acquired immunity to a blood-sucking arthropod. Guinea pigs acquire an effective immunity against *D. variabilis* larvae and this immunity can be produced artificially and can be passively transferred. The guinea pig escapes infection by being immunized not against the invading rickettsial organism, but against the organism's insect vector, the dog tick.

BARTONELLA INFECTION

Carrion's disease or verruca peruviana has distinctive clinical features (37) but interest is currently excited by its sudden epidemic appearance in southern Colombia (38), along the Ecuadorean border. Here it was first recognized in 1936. By the spring of 1939 the epidemic area included at least 1,000 square kilometers inhabited by 100,000 people. The mortality reached 40 percent in some areas, and during 8 months of 1938, some 1,800 deaths from the infection apparently occurred within the epidemic zone.

PROFLAGELLATE INFECTIONS

PINTA.—The number of known proflagellate infections has seemingly been increased by the addition to this disease group of pinta (39), hitherto regarded as of mycotic origin. Skin sections from a case diagnosed as such contained organisms which were regarded as treponemata and named *T. carateum*. These observations await confir-

mation particularly as to whether these treponemata are present as primary, not secondary, invaders.

LEPTOSPIROSES attract increasing attention in the United States (40) and other parts of the world (41). From Dr. Schüffner's laboratory in Amsterdam has come an important English review (42) making accessible to English-reading workers the valuable Dutch literature. The disease is uncommon in the United States but not as rare as many have believed (43).

HELMINTHIC INFECTIONS

Diagnostic methods in protozoal and helminthic infections of the gastro-intestinal tract have been critically re-assessed by workers at Tulane (44) with results of practical value. Examination of direct fecal films stained with either iodine or hematoxylin and of a loopful obtained from the surface of a centrifuged suspension of feces in a solution of zinc sulfate (sp. gr. 1.180) is recommended for clinical purposes. The original article must be consulted for important details of technique. Carbon tetrachloride in doses of 4 cc. has been found more efficient and more convenient than traditional remedies for tapeworm (45).

For that mysterious phenomenon, microfilarial periodicity, the simple explanation has been offered (46) that they settle to the bottom of the thoracic duct by gravity during the day and escape into the peripheral circulation by night when the infected person is recumbent. No theory of filarial periodicity explains all the known facts. The treatment of filarial elephantiasis has been advanced by the report of the successful use of bandaging in 105 cases treated at St. Croix (47). Lymphedema was removed, symptomatic relief was obtained and recurrent attacks of lymphangitis ceased under this management. The fresh interest with which this difficult problem was attacked and the details of technique presented make the original report worth reading.

PIN WORM surveys continue to reveal the surprisingly high prevalence of this infestation. In a large, well-run, charitable institution in New Orleans 96.3 percent of the children were infested (48), and in Washington 51 percent of a group of school children and 22 percent of adults examined had oxyuris infestations (49). One surprising finding was the demonstration of *E. vermicularis* ova in 91.7 percent of 241 dust samples examined at the National Institute of Health (50), a fact which suggests at once that infection can be air-borne.

TROPICAL HYGIENE

The success of the Italian army in Ethiopia was in no small part due to their unusually high standard of health (51). Wide authority and full support was given to the Inspector-General of Medical Services. Medical stores on many occasions received preference over

munitions in transport schedules. Medical officers passed through a period of training at the Hospital for Tropical Diseases in Rome before being sent to Ethiopia. Men in all ranks took quinine gr. 3 three times daily. By simple urine tests defaulters were detected and punished. Only twenty-three deaths from malaria occurred among 2,334 cases developing in an army of 500,000 men during the 7 months between October 3, 1935 and May 9, 1936. Dysentery was combatted by rigid supervision of drinking water and by provision of simple hygienic facilities adjacent to the latrines.

NEW LITERATURE

Within the short period covered by this review, the American literature has been enriched by the addition of two relatively young textbooks (52) of parasitology and by the ninth reissue of Stitt (53), dean of American manuals for use in the tropics, and equally useful outside them. The section on hematology has been vastly improved. From the Liverpool School has come a third edition (54) of a guide to parasitology which enjoys deserved popularity. Mühlens (55) is now in its fourth, and Rogers and Megaw (56) in its third avatar. An international authority on the dysenteries has presented the results of his experience (57). An important monograph published by the American Geographical Society deals with the effects on the white man of a tropical environment (58). Finally, the history of tropical medicine has at last been written (59).

These, in summary, seem to have been the major advances in knowledge of tropical diseases which have been made during the last 2 years. Important contributions no doubt have escaped attention, but Time, not the reviewer, assesses work at its real value. Enough has been cited for one to feel that in this stimulating field of tropical medicine frontiers of knowledge are being extended with a rapidity at least equal to that affecting the boundaries of other medical domains

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DENTAL CARIES [1]

PREVALENCE EXPERIENCE AT INITIAL EXAMINATION AND THE INCIDENCE OF NEW CARIOUS LESIONS AFTER A ONE-YEAR INTERVAL IN 1,047 OFFICERS AND ENLISTED MEN OF THE UNITED STATES NAVY

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INTRODUCTION

In addition to findings on other dental pathology, a detailed clinical dental examination provides quantitative information on the following

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two major aspects of dental caries: (*a*) The amount of present caries existing in the mouth at the time of examination, measurable in terms of the number of frank carious lesions, and (*b*) the amount of past involvement by caries which is measured by adding together the number of tooth areas with fillings, and the number of permanent teeth missing.

The total number of tooth areas observed to be affected by past and present caries at a particular examination is constituted by accumulations of all the caries episodes which occurred each year from the time of eruption of the permanent teeth until the time of the initial examination. Thus the number of actively carious areas observed at any age, for example at 20 years, is made up of those areas in which caries may have been initiated between the 19th and 20th year plus others which may have been initiated as early as the 7th, 8th, 14th or 15th year. In like manner the number of filled areas observed at this age represent previously carious areas which were initiated at certain intervals earlier than the time of examination. Similarly, the number of missing teeth observed at age 20 is constituted by teeth lost perhaps as early as the 6th or 7th year of age as a result of caries.

Counts of the number of tooth areas with active caries, with fillings, plus those absent from the mouth presumably because of caries, provide information which defines in substance the extent of involvement of the teeth of a particular mouth by past and present caries attack. Such counts of caries experience make available a quantitative measure of the intrinsic tendency of a particular person or groups of persons to experience attack of dental caries.

From these considerations it is clear that the total caries involvement at any age after eruption of the permanent teeth is the resultant of summation of lesions which develop each year as the individual progresses on the scale of chronological age.

As pointed out in a recent communication,[2] and as may be seen from the foregoing discussion, the number of new carious lesions which develop each year forms the basic ingredient of the caries problem. This has been shown by indirect analysis to approximate 1.3 permanent tooth surfaces per year per grade school child.[2] Also by means of an indirect approach, Hollander and Dunning[3] have shown that the yearly rate of attack by caries in a group of persons ages 17–65 and over approximates 1.7 permanent tooth surfaces per adult person per year.

Among the few direct (seriatim) investigations on the number of carious defects which develop over a yearly period, the studies of

[1] Klein, Henry, and Palmer, C. E.: Pub. Health Rep., 53: 751, 1938.
[2] Hollander, F. and Dunning, J. M.: J. of Dent. Research, 18: 43, 1939.

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Mary Moore [4] are of considerable interest. This worker followed 80 children for 16 years. Analysis of her findings reveals that on the average the Philadelphia children studied developed approximately 1.3 carious defects per child per year. Direct information on the amount of new caries which appears each year in an adult group is as yet unavailable.

The present study was undertaken at the suggestion of Captain H. E. Harvey (D. C.), U. S. Navy, for the purpose of obtaining information on this question. The material collected included data on the following two items as revealed by examinations on a reasonably representative sample of men in naval service: (*a*) The number of carious lesions (past and present) observable at initial examination, and (*b*) the number of new and recurrent carious lesions which developed in a one year interval following the first examination. The ships and stations where these examinations were made, the examining officers, and the personnel, examined by age groups, are reported in table 1.

Statistical analysis of the collected material indicates that the sample of men examined showed, at first examination, on the average slightly more than 24 permanent teeth areas with past or present caries involvement, per man, and an incidence of new or recurrent carious defects, after a one-year interval, of almost one and a quarter lesions per man.

MATERIAL AND METHOD

The material on which the present analysis is based was obtained from dental examinations of 1,047 men whose ages covered the range from 17 to 51 years. The men included in the study each received a thorough clinical dental examination. Routine roentgenographic examinations were not made.[5] In most cases the men were again examined 6 months later, and in all instances each received a clinical dental examination 1 year following the first examination. All past (filled or missing) and present (unfilled or recurrent) carious defects were recorded and all details of dental reparative treatment were noted.

In order to delineate the dental status of the men in respect to the amount of dental caries which they had experienced up to the time of the initial examination, the number of permanent tooth areas which contained active carious lesions, filled carious lesions, and the number with recurrent caries were added together. Each missing tooth was considered the equivalent of three carious defects, and the total defects which were assumed to have existed in the missing teeth were added to the sum mentioned above.[6] Though the procedure used and outlined

[4] Moore, M. M.: Annals of Dentistry, 3: 77, 1936.

[5] Roentgenographic examinations were made on 209 men of the U. S. S. *Houston* but the findings have not been included in this report because of the small number observed.

[6] Bodecker, C. F., and Bodecker, W. C., Dental Cosmos, 73: 707, 1931.

TABLE 1.—*Stations and ships, examining officers, and personnel in specified age groups*

Ship or station	Examining dental officer	Age groups (years)						
		17–19	20–24	25–29	30–34	35–39	40–51	All ages
U. S. S. *Houston*	Lt. (jg) C. A. Schlack, (D. C.), U. S. N.	74	262	76	51	25	10	498
U. S. S. *Portland*	Lt. (jg) S. Seidel, (D. C.), U. S. N.	56	196	43	31	13	8	347
U. S. S. *Ogiala*	Lt. (jg) F. I. Gonzales, (D. C.), U. S. N. Lt. (jg) J. L. Townsend, (D. C.) U. S. N.	1	13	8	14	9	2	47
U. S. S. *Astoria*	Lt. (jg) C. C. DeFord, (D. C.), U. S. N.	1	7	1	0	0	0	9
Guantanamo Bay	Lt. T. E. Crowley, (D. C.), U. S. N. Lt. (jg) R. F. Redden, (D. C.), U. S. N.	9	43	11	7	9	12	91
N. A. S. *Seattle*	Lt. (jg) W. W. VanZile, (D. C.), U. S. N.	2	19	10	12	7	5	55
Totals		143	540	149	115	63	37	1,047

here was followed for all missing teeth in the present study, it is necessary to recognize that a proportion of the missing teeth were probably lost through reasons other than caries. The incidence of caries, that is, the amount of new caries which appeared within an interval of 1 year following initial examination, is expressed as the number of new initial or recurrent carious defects per man.

In almost all the 1,047 cases, the untreated or recurrent carious lesions seen at the initial examination were repaired. The new and recurrent lesions subsequently recorded were those which were observed a full year after the date of the first examination. It is of interest to point out that these new defects developed in spite of the fact that all previous lesions, in the majority of instances, had been corrected.

FINDINGS

DENTAL CARIES OBSERVED AT INITIAL EXAMINATION.—Table 2 presents findings on the prevalence of dental caries experience at first examination in the sample of naval personnel studied on specified stations. The data are tabulated as to total number of areas in the permanent teeth affected by past or present caries (filled, with active caries, with recurrent caries, or missing) and are arranged by specific age groups on a per-man basis. When the rates for single age groups are considered, irrespective of ship or station, it may be seen that, on the average, men aged 17–19 years show an involvement by (past and present) caries of approximately 16 tooth areas per man. As age increases, the prevalence of past and present caries experience increases. Thus for the age group 20–24 years, almost 21 tooth areas show evidence of caries attack per man; in the group 25–29 years, the prevalence rate is more than 25 tooth areas per man; in the group

30–34, the rate increases to approximately 32, and finally in the last age group, 40–51, it is evident that, on the average, of the order of 43 tooth areas are observed to be affected by past and present caries at initial examination. The total group of 1,047 men examined showed on first examination approximately 24 tooth areas affected by caries experience per man.

TABLE 2.—*Dental caries, incidence on initial examination*

Ship or station	Age groups (years)						
	17–19	20–24	25–29	30–34	35–39	40–51	All ages [a]
U. S. S. *Houston*	17.8	21.3	27.4	34.8	40.5	44.1	24.5
U. S. S. *Portland*	12 2	17.7	20.4	26.1	39.8	36.9	19.2
U. S. S. *Oglala*	18.0	24.8	30.9	33.9	36.6	61.0	32.2
U. S. S. *Astoria*	10.0	11.0	17.0	[b]	[b]	[b]	11.6
Guantanamo Bay	27.4	31.1	22.8	47.1	57.8	47.5	35.3
N. A. S. *Seattle*	13.0	24.0	32.5	27.4	33.9	36.2	28.3
Average [a] all stations or ships	16.1	20.8	25.5	32.3	41.5	43.5	24.1

[a] Weighted average.
[b] No observations in this age group.

INCIDENCE OF NEW CARIOUS DEFECTS DURING 1 YEAR.—Table 3 presents data on the number of new carious defects observed in these same age groups and on the same ships and stations. The number of areas in the permanent teeth affected by new or recurrent caries, as observed 1 year following the first examination are expressed on a per-man basis. From an inspection of the rates for all stations, it may be noted that some variability exists in the yearly incidence of new defects among the several age groups. Calculation of a weighted average reveals that the men examined 1 year after initial examination show of the order of 1.2 new and recurrent carious defects per man.

TABLE 3.—*Incidence of new carious defects during 1 year*

Station	Age groups (Years)						
	17–19	20–24	25–29	30–34	35–39	40–51	All Ages [a]
U.S.S. *Houston*	0.76	0.95	0.94	0.57	0.24	1.10	0.85
U.S.S. *Portland*	1.61	1.36	2.12	2.95	.61	1.75	1.62
U.S.S. *Oglala*	0	1.69	2.00	2.43	1.33	0	1.79
U.S.S. *Astoria*	3.00	.60	4.00	[b]	[b]	[b]	1.22
Guantanamo Bay	1.56	1.98	.36	3.29	1.89	1.49	1.77
N.A.S. Seattle	1.00	.87	.80	1.15	0	.80	.82
Average all stations and ships [a]	1.15	1.19	1.30	1.66	0.68	1.27	1.23

[a] Weighted average.
[b] No observations in this age group.

SUMMARY

1. Clinical dental examination of 1047 naval officers and enlisted men indicates that the prevalence of past and present caries as ob-

served on initial examination is of the order of 24 permanent tooth areas per man.

2. Re-examination of the 1047 men 1 year later reveals that the incidence of new carious defects (after repair of the major proportion of lesions found on first examination) is approximately 1.2 carious areas in the permanent teeth, per man.

The earnest cooperation of C. E. Palmer, Passed Assistant Surgeon, and H. Kline, Dental Officer, Division of Public Health Methods, National Institute of Public Health, United States Public Health Service, in their review and suggestions as to manner of presentation of this paper is greatly appreciated.

SURGICAL HIGH-LIGHTS OF 1939 [1]

By Captain L. W. Johnson, Medical Corps, United States Navy

The exchange list of this Bulletin, consisting of many journals published in various countries and languages, constitutes a unique group. It gives an excellent cross section of the medical thought of the entire world, and in military medicine and surgery its coverage is most complete. Many have expressed the wish that this valuable material might be made available to all medical officers of the Navy. To meet this demand, these annual reviews represent an effort to skim the cream of surgical progress, and to serve it in palatable form. Special thought has been given to the needs of medical officers in ships and remote stations, who have little access to medical literature.

ACUTE INFECTIONS OF THE FACE.—Because of the potential danger attending infections about the skin of the face, the problem of treatment is one of extreme importance. The reasons for this are clearly shown in a recent article by Dingman,[2] which reminds us how the blood vessels and lymphatics are distributed in the musculofascial planes. This is common knowledge, but often forgotten.

The most innocent-appearing infections about the upper lip and nose may have a fatal outcome if meddlesome treatment be instituted. This area is drained by branches of the anterior facial vein which have no valves, thus permitting the blood to flow in either direction, either downward into the external jugular, or through the angular and superior ophthalmic veins into the cavernous sinus. It is suggested that patients with minor infections about the face splint the lesion by covering it with adhesive plaster, then leave it absolutely alone. Most of them will heal without complications. If the lesion shows a tendency to spread, the patient should be put to bed at once and large hot wet dressings applied. Visitors should be forbidden, and

[1] Submitted for publication November 2, 1939.

[2] Dingman, R. O.: Penn. Med. Journ., 42: 499, Feb. 1939.

the patient must not be allowed to talk because movements of the lips and facial muscles tend to favor the spread of the infection. After complete localization of the infection, it may be opened by a cautery knife, without manipulation.

For postoperative parotitis, Dingman recommends treatment by Roentgen ray or radium, with careful attention to the fluid balance. If no improvement in the general condition is noted at the end of 48 hours, conservative treatment should be abandoned. Incision and drainage should be done without hesitation. The incision should begin 1 cm. in front of the tragus of the ear and pass obliquely downward behind the posterior border of the mandible, thence around the angle of the mandible and about 2 cm. in front of it. One should extend the incision through the capsule of the parotid, but not deeper, because of the facial nerve which lies between the superficial and deep portions of the gland. The gland should then be explored by blunt dissection. Ample drainage and voluminous hot, wet dressings are advised.

The infections involving the deep tissues of the floor of the mouth, known as Ludwig's angina, should be similarly treated. There are pain, tenderness, swelling, and boardlike hardness in the floor of the mouth. Immediate surgical drainage is necessary. Some prefer a midline incision, others a transverse one, (I prefer a crucial one, combining the advantages of both with wide retraction of the 4 flaps. LWJ) but the essential thing is that it must incise the deep fascia to reach the seat of infection. It is somewhat terrifying to the doctor who is handling his first case, the depth to which he must go to find the deep fascia. When it is incised, the feeling of giving way is very definite. Free drainage and large hot, wet dressings are recommended. If a plastic repair is undertaken later, every effort should be made to bring together the cut edges of the deep fascia.

Ludwig's angina is always a difficult condition for the doctor to handle, more so if he understands the real pathology and the treatment that is necessary. An instructive summary of the condition is given by Caballol [3] with an account of a case. He emphasizes the importance of going beneath the deep fascia with the incision. Frequently no pus, simply a cloudy serum will be encountered. Free drainage is essential. A crucial incision allows wide retraction of the woody, edematous tissues. Local anesthesia is usually not desirable because of the wide diffusion of the infection, but if the case is seen early enough, a cervical-plexus block may be used. General anesthesia is preferable in most cases, but must be given with great care because of the interference with respiration. Intratracheal anesthesia is best of all.

[3] Caballol Y De Vera: F. Revista de la Sanidad Militar de Cuba, 3: 45, March 1939.

WOUND HEALING.—A statistical study of the effect of roentgen rays on wound healing leads Dobbs [4] to conclude that skin wounds irradiated with 300 roentgens immediately after operation show significant increase in tensile strength, though there is no anatomical evidence of a stimulating effect on the healing of the wounds by this dose. When 1020 and more roentgens are given, there is a significant decrease in the tensile strength.

COLEY'S FLUID.—For many years, the value of Coley's toxins in treatment of sarcoma has continued to be an interesting topic for discussion. The serious reactions frequently observed were a definite obstacle to their general use and the general opinion appeared to be that while nobody could prove that they were entirely ineffective, there were real dangers in their use. Coley's associates at the Memorial Hospital, New York, did not recommend their employment, feeling that much of the supposed favorable results had occurred in giant-cell tumors rather than in sarcomas.

A study of the efficacy of cultures of *Streptococcus erysipelatis*, *Bacillus prodigiosus*, and *Bacillus coli* has recently been made by Brunschwig,[5] an outstanding research worker and a former member of the Medical Corps of the Navy. He employed rats, inducing the growth of tumors by injection of carcinogenic substances. No evidence of inhibition of growth of the tumors was obtained. In a number of instances there was a marked hyperemic and hemorrhagic reaction, which was assumed to be a result of the injection. These reactions did not appear to restrain the growth of the neoplasm.

PRIMARY PULMONARY MALIGNANCY [6] was until recently considered an infrequent condition. Now we realize that it is not only frequent, but increasing in frequency. Irritating gases originating from motor cars, and the prevalent habit of inhaling tobacco smoke are regarded as the most probable reasons for the increase of primary lung cancers. All cancers of the lung are bronchogenic and most are of hilar origin. The right lung is involved slightly more often than the left.

Symptoms are generally not produced by the neoplasm itself but by secondary changes resulting from its presence. Cough, thoracic discomfort and hemoptysis are prominent. Atelectasis with infection or pleurisy with effusion may occur. Pulmonary abscess, without antecedent pneumonitis or foreign-body aspiration, should be considered of malignant origin until proved otherwise. Lung cancer should be suspected in every patient over 40 years of age with unexplained cough, hemoptysis or thoracic discomfort.

Diagnosis is best made by bronchoscopic visualization and biopsy. Presence of malignant cells in expectorated material can frequently be

[4] Dobbs, W. G. H.: Amer. Journ. Roent., 41: 625, April 1939.
[5] Brunschwig, A.: Ann. Surg., 109: 109, Jan. 1939.
[6] Ochsner, A., and DeBakey, M.: Surg., Gynec. & Obstet., 68: 435 Feb. 1939.

demonstrated. Thoracoscopy is of value when there are peripheral extension and pleuritis.

Surgical removal, by complete excision of the entire lung, offers the one chance of recovery. It should be preceded by a pneumothorax gradually induced in several stages. The authors prefer inhalation anesthesia with cyclopropane, though many use a combination of local and spinal analgesia. Eighty-six collected cases had mortality of 63.9 percent. An excellent discussion of the surgical technique is contained in this article.

Cancer of the lung can be treated today only by x-ray, radium and surgery. The first two have failed completely, since the most frequent forms of bronchiogenic neoplasms are highly radio-resistant.[7] Surgical excision of one lobe or an entire lung offers the patient, at present, by far the best chance of survival. The majority of survivors of pneumectomy in the series of these authors have been able to return to their previous occupations.

The general opinion today is that all these tumors arise from a single parent cell located in the basal layer of the bronchial epithelium, 76 percent of them in the stem bronchus. In 90 percent of their cases, bronchoscopy yielded tissue for biopsy.

They believe that any patient who develops a persistent cough, alone or associated with hemoptysis, wheezing, dyspnea, chest discomfort or other suggestive symptoms that cannot be definitely accounted for by the presence of some other lesion, should be given the benefit of bronchoscopic examination. This is especially true if the patient is middle aged or past and previously has been in good health.

GASTRIC CANCER.[8]—A review of the subject reminds us that little progress has been made in the treatment of this condition and that radical surgical removal is still the best procedure. Early lesions give a high percentage of cures, but only 20 to 25 percent come to surgery early enough.

SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM [9] is becoming more firmly established and more widely used from year to year. Its full scope of usefulness is yet unknown. From the anatomical viewpoint, one should realize that this system is represented by a continuous nerve trunk which lies in the paravertebral region on either side of the spinal column, and extends from the skull to the end of the spine. We are concerned chiefly with the thoracic and upper lumbar areas, because it is only in these that any connection with the central nervous system exists. We have to deal with two pathways, a motor and a sensory. Interruption of these is indicated in a number of clinical conditions. The result to be expected can be determined by preliminary blocking with novocain and by other tests.

[7] Overholt, R. H., and Rumel, W. R.: Journal-Lancet, **59**: 155, April 1939.
[8] Geary, T. C.: Hahnemannian Monthly, **74**: 167, March 1939.
[9] Smithwick, R. H.: New England Journ. Med., **220**: 475, March 16, 1939.

Surgery of motor pathways is mainly employed for relief of peripheral vascular disease due to vascular spasm. When vascular spasm is the principal cause, as in Raynaud's disease, brilliant results may be expected from a properly-executed sympathectomy. When obliteration of the main vessels is the cause, interruption of the sympathetic pathways is of much less value. Hyperhidrosis, with its occupational and psychological handicaps, has been most satisfactorily relieved by sympathectomy. Hirschsprung's disease, essential hypertension, and sudden arterial occlusion by embolism or thrombosis are other conditions in which section of the sympathetic nerves has been found beneficial.

Surgery of the sensory pathways is employed for relief of severe, persistent pain, when the cause cannot be removed. Atypical neuralgias of the head and face, causalgia, angina pectoris, pain from lesions of the abdominal tract, or the bladder, offer a wide field for this procedure.

Ménière's disease[10] is very common. It is recognized by recurring attacks of dizziness in which objects whirl, with subtotal deafness in one ear, tinnitus in the same ear, nausea, and vomiting. It is curable by dividing either the whole eighth nerve, or preferably, only the vestibular branch. Once it begins, it never ends spontaneously. It is never the result of a middle-ear infection, nor of a focal infection referable to the teeth, sinuses, or other organs. The lesion must lie in the auditory nerve or in the brain stem. The statements in text books, that this disease is due to apoplexy in the internal ear, is absurd and impossible. In about 10 percent of cases the condition becomes bilateral.

Pseudo-Ménière's disease has the same dizzy attacks, but without loss of hearing, and without tinnitus. It may be a beginning case of Ménière's, in which the tinnitus and deafness have not yet appeared. A tumor along the auditory nerve may give the same objective findings in its early stages, but one does not have the dizzy attacks.

Division of the eighth nerve on the affected side cures the disease, but produces total deafness on that side. Division of only the vestibular branch avoids the deafness. The branches are not definitely separated, but one can cut the anterior half and this will include all the vestibular fibres. If one cuts three-fourths or even four-fifths, the hearing is unaffected, so there is great redundance of auditory fibres. The end results of the operation have been practically perfect. There has been no death in 264 cases. Patients who have had the bilateral operation are totally and permanently relieved of all tendency to seasickness.

Another approach to the treatment of Ménière's syndrome is

[10] Dandy, W. E.: Journ. Indiana State Med. Assoc., 32:117, March 1939.

recommended by Biancalana.[11] The various forms are divided into two large groups, depending on the presence or absence of ear involvement. For the type with antecedent disease of the ear, he recommends extirpation of the stellate ganglion and has had good success in the treatment of five cases. Where there is no involvement of the ear, he recommends a trephine opening of the posterior fossa.

CRANIOCEREBRAL INJURIES.—"Dropped on his head when he was young" has long been used in vaudeville skits as an alibi for goofy behavior. Now it had been transferred from the field of ridicule to that of reality. An editorial [12] comments on the recent literature on the subject of subdural hematoma following injury to the head during birth and infancy. This lesion occurs much more frequently at this time than is ordinarily believed. Then there is a condition termed 'relapsing juvenile chronic subdural hematoma'. This includes cases showing subdural hematoma originating apparently in early childhood, continuing without symptoms during adolescence and suddenly becoming reactivated following another trauma. Roentgenograms reveal variations from the normal skull on the side of the lesion, also encephalographic changes which are unlike those seen in the chronic subdural hematomas occurring in adults after a single injury. These cases suggest that the condition occurs more often in the earlier years, perhaps, than in adult life, and it is important to consider this lesion in cases of juvenile injury with persistent symptoms.

Further light on this obscure condition comes from the Mayo Clinic.[13] Symptoms in their patient developed slowly after head injury, and 4 years later, the diagnosis of probable brain tumor was reached. Exploration by needle through a bur hole revealed a cystic accumulation of old, thick blood, of which 30 cc. were removed. Complete recovery followed.

A case of encephalitis with involvement of the hypophysis, coming on about 3 years after a severe trauma of the skull by shrapnel, calls attention to another possible remote effect of head injury.[14] In this case there was obesity, with disturbance of water metabolism, complete sexual impotence and marked psychic derangement. The authors have collected from the literature a number of somewhat similar cases.

APPENDICITIS still kills its thousands. In the United States it stands ninth in the list of causes of death, and the deaths from this cause average 17,114 per year. A scholarly article [15] on the subject states that appendicitis is essentially a closed loop obstruction of a short segment with no interference with continuity of the main in-

[11] Biancalana, L.: Minerva Med., Torino., 30:, 643, May 26, 1939.
[12] Am. Journ. Roent., 41: 122, January 1939.
[13] Ecker, A. D., and Love, J. G.: Proc. Staff Meet. of Mayo Clinic, 14: 107, Feb. 15, 1939.
[14] Gambigliani-Zoccoli, A., and Bocuzzi, G.: Minerva Med., Torino, 30: 155, Feb. 17, 1939.
[15] Wangensteen, O. H.: Proc. Inst. Med., Chicago, Vol. 12, Feb. 15, 1939.

testinal channel. The only early evidences of a closed-loop obstruction are intermittent crampy pain and local tenderness. Vomiting may be absent. Fever, rapid pulse, and leukocytosis are evidence that the infective characters of the disease have been set in motion. Following the obstruction, the secretions of the appendiceal mucosa collect and produce sufficient pressure to shut off the blood pressure. Fecaliths and swelling of lymphoid tissue are probably the most frequent causes of appendiceal obstruction in man. Kinks, bands, position (retrocecal), and stenoses may also cause obstruction.

Wangensteen has little patience with the observation, so frequently made by naval medical officers in Guam, the Philippines, Haiti, and Samoa, that as long as primitive man clings to his indigenous diet he rarely has appendicitis, but when he adopts the white man's diet the appendicitis rate is greatly increased. This, he states, is a myth. It reminds me of the time a distinguished statistician visited Haiti in search of evidence to support his hypothesis that cancer does not exist among primitive people. I showed him case after case in the wards, of cancer in primitive people from the remote mountain districts. His conclusion was, "Your trouble is that you are too near to the problem to see it in proper perspective. Now I, sitting in my study with the statistics of all the world before me, can prove that cancer does not exist in primitive people."

Proof of the positive increase in appendicitis during recent years is found in an elaborate statistical study of the disease.[16] They find evidence that the increase is real and not due merely to greater knowledge and skill in diagnosis. In studying the relation of diet to the disease, they find a very close parallel between diabetes and appendicitis. The mortality from both fell during the war years when food restrictions were imposed, and subsequently increased. But there is no more than suggestive evidence that excessive eating may be a cause of appendicitis. (In the Navy, the observation has frequently been made that cases of appendicitis are unusually frequent between Thanksgiving and New Years, a period usually marked by repeated and continued excesses in eating and drinking. This alleged seasonal increase would be an interesting subject for investigation by some of our budding statisticians.)

The general fatality rate in uncomplicated cases is found to be about one percent in this report, whereas in frank general peritonitis it is 28 to 30 percent, and when appendicitis is complicated by abscess, approximately 6 percent. The fatality rate is largely dependent on the interval of time that elapses between the onset of symptoms and admission to hospital, or operation. In Philadelphia, a propaganda campaign was conducted for 5 years to promote early hospitalization and the avoidance of laxatives. During that time the

[16] Young, M., and Russell, W. T.: Special Report Series No. 233, H. M. Stationery Office, 1939.

case fatality rate fell from 34.0 to 27.5 percent. They conclude that the only hopeful method of further reducing the fatality of the disease is to encourage, by the education of the public, earlier entry to hospital and the avoidance of laxatives or purgatives.

Primary closure of the peritoneum in cases of ruptured appendix with peritonitis continues to excite great discussion. Warren[17] has analyzed a series of 111 cases and finds that those patients in whom the peritoneum was closed had a lower mortality rate and a lessened incidence of fecal fistula, but a higher postoperative reaction and increased incidence of secondary abscess. One must not fail to drain down to the peritoneum because the muscle and aponeurotic layers which lie superficial to it do not have the same high degrees of resistance to intestinal bacteria that the peritoneum enjoys. Warren concludes that primary closure of the peritoneum is a safe procedure, worthy of further use in the effort to reduce the high mortality rate.

Peritoneal drainage still holds a prominent place in medical literature and a late review gave more than 1,500 recent references to the subject. Opinions differ as widely as ever, but most authors now agree that there is a strong trend away from routine drainage and toward conservative use of this measure which should be employed only when there is a definite indication for it. A leading surgeon of Mexico City[18] calls attention once more to the fact that within 12 hours, drains are effectually walled off and are no longer useful. The laws of gravity are less important in determining the flow of peritoneal fluid than the varying intra-abdominal pressure and the movements of the organs with peristalsis and respiration. Thus pockets of fluid may be found high up in various planes. The secretions contain antibodies and antitoxins which are essential in fighting the infection and should not be wasted by draining them away. If the septic focus can be entirely removed, the peritoneum should be closed without drainage.

PERFORATED PEPTIC ULCERS produce a great outflow of peritoneal fluid, which is usually sterile, if cultures be taken within the first 6 hours.[19] When organisms are present, the prognosis is poor. *Bacillus coli* and streptococci are most commonly found. Sterile cultures indicate a smooth convalescence, and have a very low mortality. Drainage is indicated only in patients operated on many hours following perforation. Early diagnosis and operation, with no drainage, are still the essential factors in treatment of this condition.

GASTROSCOPY has recently received increased notice because of the invention of a more flexible gastroscope.[20] The ease and assurance with which experts introduce this instrument into the stomach are

[17] Warren, R.: Ann. Surg., **110**: 222, Aug. 1939.
[18] de los Rios, A.: Cirurgia y Cirujanos, **7**: 1, Jan. 1939.
[19] Davison, M., Aries, L. J., and Pilot, I.: Surg., Gynec. & Obstet. **68**: 1017, June 1939.
[20] Kerns, H. M.: Journ. Iowa State Med. Soc., **29**: 161, April 1939.

amazing. The patient suffers very little discomfort and, if both the examiner and his assistant are skillful, there is no danger of puncturing the esophagus. The interior of the stomach is so clearly illuminated that the view which one obtains leaves little to be desired. For these reasons the method is being used ever more widely by gastro-enterologists and, as a result, we are beginning to realize that our former conceptions of the gross appearance of the gastric mucosa and of many gross lesions of the stomach need considerable revision. Its field of future usefulness is believed to be at least as great as that of the cystoscope.

The results depend entirely on the examiner's ability to recognize what he sees, which means that extensive experience is necessary. At present the instrument does not provide sufficient light for color photography, but rapid development along this line is expected. The gastroscopic method supplements, but does not replace, the roentgenologic and other diagnostic methods. In the study of various forms of gastritis it has proved particularly useful. In many cases it has made possible the diagnosis of carcinoma of the stomach before it could be detected by other methods, and it has proved very useful in diagnosis of gastric ulcer and hemorrhage.

PERITONEOSCOPY developed slowly after its first use in 1901, but in recent years the invention of improved instruments has renewed interest in the subject.[21] The technique, dangers and contraindications are now well understood. It does not replace any of the diagnostic methods now in use, but it does make possible accurate diagnoses by inspection and biopsy without subjecting the patient to the dangers and discomforts of laparotomy. It is a minor procedure, performed under local anesthesia, and requires hospitalization for only 1 day. Its greatest use is found in cirrhosis of the liver, splenomegaly, malignancy of the liver, suspected malignancies, identification of unknown masses, ascites of unknown nature, tuberculous peritonitis, and chronic pelvic conditions in the female. The instrument is inserted through a small incision with local anesthesia, usually just below the umbilicus. Pneumoperitoneum is then instituted. A tilting table is desirable, so that the aid of gravity can be gained by changes of level. The only death in Ruddock's series of 500 cases followed bleeding from biopsy of the liver. Adhesions may be cut by direct vision, as in thoracoscopy. The method is not advised in patients with serious cardiac or pulmonary disease; when numerous abdominal adhesions are present; in acute inflammatory conditions.

Another writer[22] believes that the subject has been neglected. Besides visualization, it permits aspiration, biopsy, cauterization, coagulation, and radon-seed implantation. The indication for its use is

[21] Flocks, R. H.: Journ. Iowa State Med. Soc., 29: 162, April 1939.
[22] McHardy, G.: New Orleans Med. & Surg. Journ., 91: 528, April 1939.

any intraperitoneal complaint that cannot be diagnosed by more conservative measures, especially disorders of a chronic nature. It bids fair to displace exploratory laparotomy as a diagnostic aid. More can be seen with the peritoneoscope than with exploratory laparotomy, and it avoids the expense, danger, and discomforts of that formidable operation. One can diagnose adhesions, hepatic cirrhosis or malignancy, tuberculous peritonitis, ovarian cysts, cholecystitis, splenomegaly, bladder and sigmoid diverticula, uterine tumors, and extra-uterine pregnancies.

HERNIA.—The fact that hernias recur after operation in an astonishingly large number of indirect as well as direct hernias, is one to which the surgeon is likely to close his eyes. This is especially true if he is unable to follow up his cases, which is the rule in the Navy. The recurrences happen in the hands of the most competent surgeons and under circumstances which are considered favorable to cure. So the technique cannot by any means be considered as perfected. Bisgard [23] submits another method of employing the external oblique aponeurosis in heniorrhaphy. A strip approximately 1.5 cm. wide is cut from the superior mesial flap of the aponeurosis, severing it at the musculo-aponeurotic juncture above, but leaving it attached below. Usually it is possible to obtain a strip of 18 cm. or longer. The free end is fastened to a special fascial needle, which does not require doubling of the fascia. This is passed through the conjoined tendon, then through Poupart's ligament at its juncture with the triangular ligament, and continued as a suture approximating Poupart's ligament to the transversalis fascia, the internal oblique muscle and its tendon. The fascial loops are fixed with interrupted silk sutures.

For this method are claimed the advantages of the security obtained from the use of living fascial sutures, without increasing the duration or trauma of the operation. It utilizes the triangular ligament to solidify the repair by closing the vulnerable area immediately above the pubic spine.

There is a common delusion that the operative cure of inguinal hernia is nearly an ideal surgical procedure, since the results are good and the mortality so low as to be negligible. Longacre [24] helps to debunk this Pollyanna point of view by showing that the mortality is sufficiently high to warrant serious consideration of all factors in the general condition of the patient. Recurrences are sufficiently frequent to justify a careful weighing of all the benefits to be derived before recommending operative repair. Even the simplest surgical procedures carry a definite risk of death or an unsatisfactory result.

[23] Bisgard, J. D.: Surg., Gyn. & Obstet., 68: 113, January 1939.
[24] Longacre, A. B.: Surg., Gyn. & Obstet., 68: 238, Feb. 1939.

His series includes 752 patients operated on by 60 surgeons in the Presbyterian Hospital, New York. Follow-up by actual examination is more accurate than by letter, for only 3.4 percent of recurrences were reported in writing, while 8.3 were found on examination. There was very little variation in the recurrences with different anesthetic agents. Wound infections occurred in 53 cases, all but two of which were trivial. Of those repaired with silk, 2.55 percent showed infection, contrasted with 15.04 percent of those in which gut was used. A marked and impressive lower incidence of recurrence is found in the cases repaired with silk as compared to those repaired with chromic gut. In sliding hernias 6.6 percent of the silk repairs recurred, while 25 percent of the chromic repairs recurred.

Effective use of the transversalis fascia is recognized as important, but it did not materially lower the incidence of recurrences when careful treatment of this tissue was mentioned in the record. Transplantation of the cord beneath the external oblique appeared to be the most effective method for there were only 2.7 percent of recurrences in this group. Living fascial sutures were used in 139 hernias. Recurrences were noted in 6.7 percent of the cases in which a strip of the external oblique aponeurosis was used, 16 percent of cases sewed with fascia lata, and 13.3 percent of the cases repaired with rectus muscle or fascia. Fascia lata was used more frequently in recurrent hernias, which may affect the higher rate of recurrences. There were 10 deaths in the series, a mortality rate of 1.3 percent.

Many Navy surgeons regard themselves as specialists in surgery of hernias, and I believe there is a common impression that our rate of recurrences is lower than that of the average surgeon. I have even heard Navy surgeons say that they never had any recurrences. Properly interpreted, such a statement means simply that there are no worth-while figures on recurrences after operation in the Navy, and no effective follow-up. There is an excellent field for some studious young medical officer, to work up the statistics on a large number of cases which have been operated on by surgeons in the Navy.

Postoperative complications in 2,000 cases of simple oblique and direct inguinal hernias have been carefully studied.[25] Wound infection and respiratory complications occurred about twice as often in cases of bilateral hernia as in single hernia. Age was an important factor. Up to 30 years of age, complications were relatively infrequent. From then on, there was a steady rise in their frequency in each succeeding decade. Respiratory complications were distributed more or less uniformly through all the decades of life. The more serious ones, however, were found more often in older individuals. The period of time used in performing the operation played an im-

[25] Beekman, F., and Sullivan, J.: Surg., Gynec. & Obstet., 68: 1052, June 1939.

portant part in the rate of frequency of wound infections and severe respiratory complications. The curve for the more severe infections rose rapidly from less than 0.5 percent for a 30-minute period, to 25 percent when the operation took longer than 1½ hours. Respiratory complications also increased rapidly with increase in the time of operation.

The morbidity from wound infection was slightly lower when local anesthesia was used. Morbidity after general anesthesia was a quarter higher, and that for spinal was twice as high as that for local anesthesia. Atelectasis appeared almost twice as frequently when local or spinal anesthesia was used as it did when a general anesthetic was used. On the other hand, pneumonia occurred more than twice as often following the use of general anesthesia.

The frequency of wound infection with the use of silk was about one-half that of gut, one-third that of kangaroo tendon or ox fascia, and one-fifth that of autogenous fascia. The more serious pulmonary complications occurred about twice as often among those cases in which kangaroo tendon, ox fascia or autogenous fascia was used, as among those in which silk or gut was used.

Cure of hydrocele by injection of sclerosing fluids appears to continue in favor. A report from Italy describes the use of picric acid for injection.[26] The author empties the hydrocele of its contents and injects 50 to 70 cc. of a saturated aqueous solution of picric acid at body temperature. He then massages the scrotum gently, after which he withdraws the fluid, and then once more injects the same amount. After 5 to 10 minutes he withdraws as much of the fluid as possible. He keeps his patients in bed for 24 hours following the injection. He endorses the method and states that he has had no complications and no recurrences in a large series of cases.

(One must not forget, however, that acute hydrocele is sometimes the first tangible evidence of a malignant growth arising near or in the tunica. This was indelibly impressed on the present writer when he was involved in such a case. A urologist of international reputation missed the diagnosis, but operation revealed the minute growth on the tunica, and also explained the mystery of a hard, tender swelling which had appeared above the left clavicle.)

Sprains.—Treatment of sprains by injection of procaine is recommended as a pain- and time-saving method.[27] The anatomically-slight injury starts a reflex which is followed by a vasomotor response, with local edema and rise of temperature, pain, and limitation of movement. Injection of a local anesthetic puts an end to the whole vicious cycle, and it does not often recur after the effects of the injection have worn off.

[26] Di Pace, I.: Minerva Medica, Torino, **30**: 62, Jan. 20, 1939.
[27] Frankel, E. L.: Lancet, **237**: 597, Sept. 9, 1939.

A 2 percent solution of procaine is injected into the most tender spot, and the whole area infiltrated. The area is then massaged lightly. A dramatic relief is usually experienced. Some times it may be necessary to repeat the injection on the second or third day. The injection should be made as soon as possible after the injury, but older sprains may also be treated in this way. After the injection, the joint may be strapped with adhesive, and the patient told to use it carefully.

COMPOUND FRACTURES still constitute a very live problem, in spite of advances achieved during the World War. Orr [28] reviews the gradual development of modern methods, and makes recommendations based, on his great experience.

Weight and pulley traction, the Hodgen splint, and even the Thomas splint should be abandoned now, except as emergency expedients, in favor of fixed traction in plaster casts. In large hospitals there is division of responsibility, loss of continuity of treatment, and lack of personal interest in the patient. Under these conditions, earlier methods which were successful under the personal supervision of a devoted physician, do not suffice. The lesson of the World War was that, with inadequate reduction of compound fractures, and with traumatization of wounds by frequent dressings, local and general septic complications were encouraged and the healing of wounds and fractures prevented or delayed. Orr has discontinued the use of Kirschner wires or other less efficient skeletal transfixion devices. He believes that mechanical devices which can be adjusted by the patient or attendants, are less efficient than transfixion pins embedded in plaster. Complete immobilization of the patient and his limb in plaster is the secret of success in the use of skeletal fixation. If pins are properly inserted and locked in the cast, they do not become loose. There is no irritation, necrosis, or other complication.

The large soiled wound, the hemorrhage, shock, pain, swelling and deformity are the surgeon's first interest when he sees a compound fracture, so that the first fundamental, which is reduction of the fracture, may be overlooked. With an injured limb immobilized in proper position, the only indications for disturbing it are those familiar evidences of complications with which every surgeon is familiar.

Closed treatment of fractures was used in a great number of cases during the Spanish Civil war.[29] Despite the use of methods learned during the World War, infected wounds with osteomyelitis and gas gangrene were numerous. Then there was a trend toward setting wounded limbs in plaster and cutting windows, leaving the wound alone so long as it progressed favorably. This was quite successful

[28] Orr, H. W.: Ill. Med. Journ., 76: 71, July 1939.
[29] Trueta, J.: Lancet, 236: 1452, June 24, 1939.

and was adopted. Trueta developed a combination of the methods recommended by L. P. Friedrich in Germany, and Winnett Orr, of the United States. This was so satisfactory that it eventually supplanted other methods.

After injection of tetanus-gas antitoxin, the patient was anesthetized with ether and the wound thoroughly cleaned with soap and water. The skin was shaved and swabbed with iodine, which was not allowed to enter the wound. Debridement was done, saving as much skin as possible, but with radical removal of all injured muscle and aponeurosis. Bony fragments were preserved wherever possible, even those deprived of periosteum. Gauze drainage was introduced between the muscle layers, and a rubber tube to the lowest part of the cavity. The fracture was reduced as much as possible, the limb placed in an appropriate position and fixed by a plaster case which included the joints above and below. In most cases the first cast remained throughout the healing, but in others it was necessary to replace it when it became bad-smelling or wet and soft.

This treatment provided absence of pain, rapid disappearance of shock, elimination of sleepless nights and quick return of appetite. In 1,073 cases treated by this routine, 976 had good results, 91 had bad results and 6 died.

KNEE INJURIES are frequent enough in the Navy to make us interested in their outcome. A series of 193 cases has recently been anaylzed.[30] Eighty-nine percent of the cases of simple synovitis and 81 percent of those with a lateral-ligament sprain showed good final results, so that they were able to continue active athletics. Semilunar cartilage injuries gave much better results when treated by immobilization (46 percent good) than when treated merely by bandaging, rest, crutches, and physical therapy (36 percent good). The authors recommend that the limb be immobilized in plaster as soon as a diagnosis can be made. The results of conservative treatment are so good that they feel it should be generally tried before resorting to operation. Of the cases which have disabling symptoms after this treatment, 66 percent can be sufficiently improved by operation so that they can engage in active athletics.

REMOVAL OF THE PATELLA after open fracture is recommended as a routine measure.[31] Several cases are mentioned in which this procedure has been followed by restoration of function and no disability. Leclerc believes that it should be a part of the debridement of compound fractures of the bone. Splinters, blood clots, and bone marrow that offer breeding places for infection can thus be eliminated. The torn edges of the patellar ligament may be sewn to the quadriceps tendon and the adjacent muscles, to close the knee joint.

[30] Hopkins, F. S., and Huston, L. L.: New Eng. Journ. Med., 221: 95, July 20, 1939.
[31] Leclerc, G., Memoires de L'Academie de Chirurgie, 65: 475, March 1939.

From England comes another report indorsing this procedure.[32] The author has treated a number of cases in this manner, and finds that one result is increased power in the knee after recovery. Use of the joint can be commenced very early, and new bone formation gradually replaces the patella.

VOLKMANN'S CONTRACTURE is a spectre which haunts the surgeon who has the care of patients with fractures about the elbow. Until recently, most medical men believed that the condition was due to pressure of splints or tight dressings on the nerves. Courts were generous in awarding damages based on this belief, and many surgeons suffered financially from suits arising from such cases. A more complete understanding of the condition now changes the picture, and a summary of our knowledge, coming from a most reliable source, should be of value.

Meyerding,[33] writes that it may occur in cases which have not been treated by a physician, or by any form of fixation. The physician who carefully observes, and has the cooperation of his patient, who is cognizant of the early symptoms, and who carries out measures to prevent interference with the circulation of the forearm, may avoid the occurrence of this contracture. A record of the findings at the time of the first visit of the patient should be made, and should include the interval between the injury and the examination. The character of the pulse and presence of paralysis may determine the type of treatment. When the physician finds serious impairment of the circulation, he should request consultation to protect himself and to give his patient the benefit of added skill in meeting the impending complication.

The common fracture associated with this contracture is of the supracondylar type, in which the force that breaks off the lower fragment of the humerus carries the condyles backward and strips the periosteum away from the posterior surface of the humerus. The space thus formed promptly becomes filled with blood. The upper fragment is carried forward, pierces the periosteum, and may impinge on or cut blood vessels or nerves. The blood which collects about the site of the fracture infiltrates the antecubital spaces, compresses the soft tissues, and if there is any large amount of blood it may pass downward into the forearm. Should arterial hemorrhage continue, the sub-fascial hematoma produces distention, extreme pain, and tenderness at the antecubital space, with cyanosis, loss of pulse and numbness. The forearm, hand and fingers may become cold, wet, pulseless, and cyanotic, and blebs may appear. Pain is very severe, and unless intrinsic pressure is soon relieved, contracture occurs in a matter of hours, not days or weeks.

[32] Mehriz, M. M.: Lancet, **236**: 91, Jan. 14, 1939.
[33] Meyerding, H. W.: Minnesota Med., **22**: 100, Feb. 1939.

When the swelling is sufficient to make reduction difficult, or immobilization in flexion with splints or casts results in pain, it is advisable to remove constricting dressings, place the patient in the recumbent position and elevate the arm. Should pain and swelling increase sufficiently to cause cyanosis or interference with the circulation, it is advisable to abandon the treatment of the fracture and care for the soft tissues. Suspension of the arm above the recumbent body for a few days usually will permit reduction of the fracture after swelling subsides, without risk of incurring excessive pain or ischemic contracture.

When patients are seen after the antecubital space is distended and tender and the pulse is faint, with bluish-red color of the skin and unbearable pain, all dressings should be removed and the arm elevated on pillows with the elbow extended beyond a right angle. Warm, but not hot, moist dressings should be applied. If, within the next hour, the swelling increases and the pulse is absent, the relatives must be warned of the serious outlook and preparations made for operation.

Other patients may be encountered after several manipulations have been performed, and considerable time has intervened since the injury. The arm may be distended to such an extent that immediate surgical relief is demanded.

Free incision over the hematoma and down through the deep fascia liberates muscle tissue and blood which have been under tension. An incision mesial to the biceps permits the bicipital fascia to be divided close to the tendon, and the artery, vein and nerve can readily be inspected. The deep fascia over the forearm may require division. the fracture may then be reduced and internal fixation by bone screws may be introduced, or a Kirschner wire may be passed up through the condyles into the proximal fragment. The entire arm is then inclosed in a large, moist alcohol and boric-acid dressing, and suspended in abduction.

ANESTHESIA.—The annual reports from the section on anesthesia of the Mayo Clinic are always worth watching for, if one is interested in this field.[34] There has been an increase in the use of local anesthesia combined with inhalation, also an increase in the use of the intratracheal method. Cyclopropane is stated to be of such value that one regrets placing a ban on it, because of the danger of ignition of the gas within the machine by static spark. Ethylene tends to return to the place it occupied before cyclopropane became available. Helium may be used to advantage in the presence of a marked respiratory obstruction.

The use of intravenous anesthesia has increased. Pentothal sodium is given in a 2.5 percent concentration. One gram is dissolved in 40 cc. of distilled water. With this reduced strength there is less

[34] Lundy, J. S., Tuohy, E. B., Adams, R. C., and Mousel, L. H.: Proc. Staff Meet. Mayo Clinic, 14: 273, May 3, 1939.

delayed phlebitis than before. If cyanosis appears, oxygen is given with the gas machine. The addition of oxygen is also desirable when intravenous anesthesia is used for intra-abdominal operations, to improve muscular relaxation. Regional block of the abdominal wall helps to reduce the amount of the pentothal sodium required. This combination has proved excellent for patients who are poor surgical risks. It is not felt to be safe for children under 10 years of age, for most operations on the respiratory tract, for those with advanced cardiac disease, or for those who have taken sulfanilamide within 24 hours before operation.

Morphine sulfate intravenously is becoming more useful as an adjuvant to intravenous, spinal, local, or block anesthesia. It helps in tranquilizing the patient before esophagoscopy or bronchoscopy. An ampule of 1/6 grain should be diluted to make 2 or 3 cc., and administered slowly watching the effect of the drug.

Rectal anesthesia is used chiefly for children under 10 years of age, for such procedures as encephalography and bronchoscopy. Avertin is used in doses sufficient to produce basal anesthesia, and supplemented by local or inhalation agents.

Procaine hydrochlorid and metycaine were the local anesthetic agents of choice. The latter, in spinal anesthesia, is believed to produce longer anesthesia than the former. Spinal anesthesia has been found valuable for certain types of thoracic surgery, especially lobectomy. The space between the second and third lumbar vertebrae is used as the site, and the metycaine is diluted with 10 to 14 cc. of spinal fluid. No more than 200 mg. was used in any case. For operations on the urinary bladder and lower part of the genitouninary tract, they introduce the agent between the 4th and 5th lumbar vertebrae. This appears to reduce the fall of blood pressure.

Shock.[35]—An article on recent advances in surgical therapeusis contains some pertinent remarks on the subject of shock, and the present conception of this syndrome. There is no subject in surgery of which more has been written, and of which we have a poorer conception than surgical shock. In 1893, Malcom came to the conclusion that shock was associated with arteriolar constriction rather than relaxation. This was overshadowed by Crile's theory that it was the result of exhaustion of vital nerve centers by painful stimuli. Later workers found that failure in the cerebral circulation, for only a very short period, produced nerve-cell changes identical with those that Crile had observed. These changes were the result of shock, not the cause of it. There is now universal agreement upon the fact, that the low blood pressure of surgical shock is the result of a diminished blood volume. This diminished flow of blood to the peripheral tissues is the dominant feature of surgical shock. Shock begins when the

[35] Ravdin, I. S.: Ann. Surg. 109: 321, March 1939.

tissues of the body receive too little blood for the maintenance of tissue metabolism.

There is a loss not only of fluid, but also of protein. It is necessary that some substances which do not easily escape from the blood vessels be administered with the fluid. Normal or hypertonic glucose or saline solution do not remain in the circulation long enough to be of any permanent value. The most effectual substances are blood, plasma and acacia. Warmth improves the peripheral circulation. Trauma causes vasoconstriction. The rougher the surgeon, the greater will be the trauma and the higher the incidence of shock.

A somewhat different approach to the problem of shock is the use of faradic stimulation to the muscles. Henderson has called attention to the venous collapse which occurs in shock. The authors [36] of this article attempted to raise the pressure in the veins by stimulating contractions in the voluntary muscles. Large electrodes were applied to the legs and lower abdomen, with proper precautions against burning, and faradic stimulation was applied. Use of this method in several cases of shock has given very encouraging results.

VITAMIN K.—The danger of hemorrhage in jaundiced patients who require surgery has long been recognized. During the operation no abnormal bleeding may be noticed and hemostasis is generally secured without difficulty. The bleeding takes place later, and the period of greatest danger is between the third and the sixth days, usually as a slow ooze into the depths of the wound or from the mucous membrane. The principal factor appears to be the impaired coagulability of the blood, due to a deficiency of prothrombin. Illingworth [37] describes a test for prothrombin estimation, which appears to be a reliable criterion of the hemorrhagic tendency. He has found that the danger of hemorrhage may be considerably reduced by the administration of vitamin K. This should be done for some time before the operation, and it should be continued until the dangerous postoperative period is past. The lack of prothrombin may be caused by defective absorption from an intestine in which there is no bile or by inability of a damaged liver to synthesize prothrombin from the vitamin.

Last year's article [38] mentioned vitamin K as an interesting novelty which might evolve into something of value for prevention and treatment of hemorrhage associated with jaundice. Developments during the past year have been rapid. The substance has been identified and prepared synthetically in concentrated form by extraction from alfalfa with petroleum ether. It can be assayed biologically by its effect on chicks that have been previously fed on a diet deficient in

[36] Ornstein, G. G., Light, S., and Herman, M.; Quart. Bull. Sea View Hospital, 4: 333, April 1939.
[37] Illingworth, C. F. W.: Lancet, 236: 1031, May 6, 1939.
[38] Johnson, L. W.: Naval Med. Bull., 37: 11, Jan. 1939.

vitamin K. Its dosage has been determined for various types of patients under differing conditions, and it has been firmly established as a valuable therapeutic agent.

BLOOD TRANSFUSION.—The Spanish war served as a laboratory for experimentation in many lines of endeavor, not the least of which was the organization of blood-transfusion services under war conditions. A detailed report of the working of the Barcelona service is now available, and is important enough to justify a lengthy abstract.[39]

The blood-transfusion service in Barcelona began to function in August 1936 and carried on till January 1939. The technique invented by it was instituted throughout the whole of the Republican army. The organization was devoted to the performance of blood-transfusions on a large scale—i. e., to the problem of providing as large an amount of blood as possible to meet the requirements of an army. The solution of this problem is the establishment of an organization dedicated to the preparation of stored blood in a large town. Any organization that tries to solve the problem by the use of fresh citrated blood or by direct transfusion from donors is likely to fail, because in a rush of wounded it is impossible to give all the required transfusions, both on account of their number and the amount of blood required. The only solution is a stock of well-prepared and preserved blood. Not only must a service of blood-transfusion of citrated and stored blood be organized, but also it must be established in a large town where the altruistic spirit of the citizens can be aroused and organized so that they volunteer as donors. A main object of a blood-transfusion service is the proper organization of donors.

The organization of donors was threefold: by groups in (1) workplaces and clubs, (2) small towns and villages round the city, and (3) districts of the city. With this triple organization the service could mobilize many donors at any moment and yet preserve the secrecy of military movements. The Barcelona service included 28,000 donors, whose blood was classified in September 1938 as follows:

Blood group	Number	Percentage
IV-O	9,190	41.54
II-A	10,080	45.57
III-B	2,071	9.36
I-AB	779	3.55

Biologically, the blood was prepared in the following manner:

The donor's blood was examined as regards its agglutinins and agglutinogens. A blood count was done and the hemoglobin estimated. The Barcelona service instituted its own technique for this

[39] Jorda, F. D.: Lancet, 236: 773, April 1, 1939.

study. The blood was studied serologically, two reactions at least being tried for syphilis, one based on deviation of complement and the other on flocculation. Citrate should be added in the proportion of 4 in 1,000 and glucose 1 in 1,000. The solutions should be made with fresh doubly-distilled water. The extraction of blood must be carried out with the greatest of care. In Barcelona 150 donors were bled in 2 hours, obtaining 25–30 litres of blood an hour. This technique must comply with both surgical and bacteriological rules. Blood was aspirated by suction so regulated as to avoid collapse of the donor's vein and consequent cessation of flow. All the material was sterilized in an autoclave, except the needle, which was kept immersed in ether. The donor's arm was perfectly sterilized and the selected area protected with a sterile towel. The donor was bled fasting, so as to avoid post-prandial colibacteremia, excess of fat, glucose and protein, allergens of alimentary origin that sometimes occur in blood-plasma during digestion, and incompletely broken-down amino-acids due to hepatic insufficiency or to too large an intake of certain foods.

Yudin's method of using cadaver blood is useless when large quantities of blood are needed (in September 1938, there were 832 litres of blood prepared), for its efficacy is limited by the difficulties attending the collection of blood and by the need for all samples to be of the same blood-group. The problem of obtaining sufficient blood was solved by mixing all bloods of the same group, and the experience of thousands of transfusions amply confirmed the value of this technique. The mixture of bloods of the same group gives a very homogenous blood, biologically speaking, with a quantity of cells, hemoglobin, glucose, urea, and other constituents, which tend to approximate a normal blood.

A method of transfusion, to be perfect, must provide for the possibility of transfusion in any place whatever, at any moment whatever, and by any member of the medical personnel. To fulfill these three conditions, the authors developed an apparatus consisting of a sealed glass tube containing blood under a pressure of two atmospheres exerted by filtered air. This pressure is the force that drives the blood into the veins. It also acts as a most important automatic bacteriological control. Physiologists have shown that a pressure of 16 mm. Hg. is enough to convert 99 percent of the hemoglobin to oxyhemoglobin if the blood is in contact with oxygen. This action takes place in the tube owing to the amount of oxygen in the air. All the blood pigment is changed to oxyhemoglobin and the blood becomes a ruby red. If the blood is accidentally contaminated, the organisms will not grow unless they are aerobic, because the growth of anaerobic organisms is inhibited by the oxygen in the tube and by the intense oxygenation of the blood. The growth of aerobic organisms will

take place at the expense of the oxygen in the tube, and, since the tube is sealed, the oxyhemoglobin will be reduced and the blood changed from ruby red to dark red, thus showing that the blood is not sterile. If the bacterial action proceeds further, the blood pigment alters to hematoporphyrin and the blood turns black. This fundamental fact of this technique provides colorimetric control of sterility.

The blood is stored in refrigerators at 2° to 4° C. The Barcelona institute had its own means of transport to enable the blood to be taken to the required place with the greatest safety. There were two lorries and a railway van, each fitted with a refrigerator. Advanced military posts were provided with refrigerators which functioned well either by electricity, petrol, or paraffin. For transport to places which could not be reached by vehicles the blood was carried in isothermic cases supplied with a refrigerating mixture.

Prior to use, the following points were noted: (1) The sterility of the blood was observed as shown by the color, ruby red for sterile blood, dark red or black for infected blood. (2) The vitality of the blood was estimated by observation of the degree of hemolysis evident in the overlying plasma. The plasma should be amber and not red. A note should also be made as to whether the line of division is evident.

The tube is prepared for use complete with the necessary apparatus required for administration of blood. The blood must be mixed by inverting the tube. It is then carefully heated to 45° C. in a water-bath. The temperature must be exact. If the water-bath is too hot, hemolysis of the peripheral parts of the blood may take place. If the correct temperature is not reached, there may be severe reactions in the transfused patient. A venepuncture having been made with a needle attached to the two-way cock, it is only necessary to break the capillary neck of the tube for blood to be driven into the vein by the pressure of air present.

The institute at Barcelona had a complete organization for following up the results of each transfusion and the exact fate of each tube. A card was made out for each tube and another for each transfused patient in addition to his clinical history. At the end of January 1939, the institute had obtained 9,000 litres of blood as a result of 20,000 withdrawals and prepared more than 27,000 tubes. A list of 28,900 donors, well studied and classified, was available. The institute further took over the preparation of all the sera necessary for grouping; the plasma of group 1 (AB) and group 11 (A) for the hemostatic treatment of hemoptysis, hematemesis, and hemorrhagic states; and antipoliomyelitic and antityphoid sera from cured and convalescent patients. One whole section was devoted to the treatment of measles, scarlet fever, smallpox, and other fever. After a time the institute also took over the investigation of paternity and the study of agglutinogens and agglutinins in bloodstains. Finally, a

section was established for experimental biology. Such was the institute that grew up out of the blood-transfusion service in Barcelona, and a similar institute, wherever established, will be able to render great help not only in civil practice, but also in time of war.

Transmission of syphilis by blood transfusion has become a very live subject with the recent developments in blood banks and kindred activities. In the service we have had some very awkward incidents, and medical journals carry a number of reports of such infections. In most of the cases the donors were in the primary or secondary stage, with negative blood reactions, in one case an intraurethral chancre. McCluskie [40] recommends that donors be informed that syphilis can be transmitted from donors who are clinically and serologically negative, so that by careful questioning one may be able to rule out those who have been exposed to infection during the preceding 2 months. He states that the risk of syphilis transmission could be minimized by the use of cadaver blood, or by the discovery of some means of rapidly destroying spirochetes in the blood withdrawn from the donor. Certainly, the knowledge of this danger imposes on us the duty of taking every precaution to avoid infection of the recipient.

Syphilis inoculation by blood transfusion has been closely studied and 41 cases were found in the medical literature.[41] It is believed that if such a number have been reported, several times as many have actually occurred, which makes the problem a serious one. In 16 of the cases studied, an examination of the blood would not have revealed the presence of the disease. Eichenlaub and Stolar recommend the use of mapharsen, .01 gm. being added to the sodium citrate solution. Other writers have recommended the use of arsphenamine for this purpose. It is too early to make a definite recommendation, but the seriousness of the problem and the amount of research being devoted to it make it probable that a solution will soon be found.

At the Mayo Clinic, 3,295 blood transfusions were given during 1938.[42] Only occasionally are the bloods of the donor and recipient cross matched, usually in cases with previous transfusion. They feel that fewer reactions occur when refrigerated blood is used. Blood for nonemergency transfusions is not used after it has been in the refrigerator more than 10 to 12 days. The direct method of transfusion was not used during 1938. One must be guided in the amount of blood given by the response of the patient. Thus the use of refrigerated blood becomes of increasing importance.

In treatment of shock, plasma is more effective than whole blood. This is true because in shock without hemorrhage there is no loss of red cells, but a great concentration of red cells in the capillaries. A

[40] McCluskie, J., Brit. Med. Journ., 4075: 264, Feb. 11, 1939.

[41] Eichenlaub, F. J., and Stoler, R.: Penna. Med. Journ., 42: 1437, Sept. 1939.

[42] Lundy, J. S., Tuohy, E. B., Adams, R. C., and Mousel, L. H.: Proc. Staff Meet., Mayo Clinic, 14: 283, May 3, 1939.

paper soon to be published discusses the use of plasma in treatment of the emergencies of war.[43]

Colloids suitable for raising blood pressure in conditions of shock are acacia, serum and plasma. In time of emergency, blood plasma has the following advantages over whole blood: (*a*) It can be stored safely for long periods without refrigeration. (*b*) It can be transported safely over long distances. (*c*) It can be given without typing or cross matching.

The authors describe a method of utilizing blood which has been kept for the safe period in the blood bank. It may be made useful by extracting the plasma and preserving it in vacuum bottles until needed. They conclude that plasma will prove an ideal substitute for whole blood in emergency treatment of hemorrhage and shock from war wounds. It can be prepared at the source of blood, and preserved for long periods of time if necessary before being shipped to the point where it is needed. Laboratory facilities are not needed.

Now the people at home can do their bit by donating blood which may be used either whole or as plasma for the wounded soldiers at the front. The next logical step will be for the laboratory to develop an artificial plasma, with all the good qualities of the human product.

[43] Tatum, W. L., Elliott, J., and Nesset, N.: personal communication.

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.

TREATMENT IN GENERAL MEDICINE, edited by *Hobart A. Reimann, M. D., Magee Professor of Practice of Medicine and Clinical Medicine, Jefferson Medical College, Philadelphia*, with 34 contributors. Set of 3 volumes and desk index. (First volume 895 pages, second 896, third 834.) F. A. Davis Co., Publishers, Philadelphia, 1939. Price $30.00.

This is the first edition of a work destined to become internationally known. The physical appearance of the set is excellent. Designed for durability and easy reading the cover, binding, paper and type combine to make it the finest on the shelf.

Such an encyclopedic work cannot be reviewed, item by item. It covers the field of medicine. Doctor Reimann is supported by 33 contributors, each an authority in his field, each active in the practice and teaching of the specialty upon which he writes. This wise division of labor emphasizes the fact that medicine has become so complex, so many sided, that whether we like it or not we are forced to specialize. No man can know all of medicine. It is difficult to know all of one specialty.

The trite argument that, once a diagnosis is made, anyone can treat the disease has caused the slighting of therapy in text books and systems of medicine. Treatment is itself becoming so technical, and the advances in serotherapy and chemotherapy as well as many other specialized lines are so important and yet often so confusing that the need for specific advice in each procedure is amply demonstrated. Our ignorance or confusion in therapy makes us easy prey for the manufacturers and marketers of proprietaries who always have a readymade therapeutic agent, with their trademark upon it.

Recognizing the fact that intelligent treatment is based upon a knowledge of the disease, discussion of the treatment of each disease is preceded in this work by a short presentation of highlights of the history, geographical distribution, etiology, epidemiology, pathology, symptomatology and diagnosis. Especial attention is given to the newer methods of approach in etiological diagnosis. In this the

importance of the laboratory in diagnosis and treatment is emphasized. Brief discussion of the pathological physiology of each disease and the relation of symptoms to such alterations in body processes would have added still further to the enormous value of this work. It would, of course, also have added greatly to the scope and size of the volumes. After all a limit to the scope of a book must be set. The writers have all followed the recent developments in therapy. For example, in pneumonia chemotherapy with sulfapyridine is mentioned. The many uses of sulfanilamide are given consideration although its use in the treatment of chancroid is omitted. The value of active immunization with tetanus toxoid, in the prophylaxis of tetanus, is mentioned.

In addition to medication other fields too often neglected in our handling of cases are fortunately given adequate attention. These are psychotherapy, physical therapy, heliotherapy, and mechanotherapy. For though, in therapy, none of these forms of treatment are specific, they are unquestionably of great value in the relief of symptoms and the improvement of morale. The neglect of these fields by legitimate medicine has too often left them open to the quack and the charlatan.

Extremely well prepared sections on minor surgery, gynecological and obstetrical treatment and one on geriatrics are included and increase the value of the set to the profession as a whole.

Seldom has a more valuable addition been made to medical literature. Appreciation of this work will grow in the mind of the doctor as day by day the set is called upon for aid in the treatment of his patients. This simple statement is the greatest praise one can give.

Each contributor to "Treatment in General Medicine" has agreed to revise his section annually. The revision material thus submitted will be edited by Doctor Reimann and appear in the form of a supplement, which will be for sale to subscribers only. Through these revisions the owner will always have at hand the last authoritative word on treatment.

HYPERTENSION AND NEPHRITIS, by *Arthur M. Fishberg, M. D., Associate in Medicine, Mount Sinai Hospital, New York City.* Fourth edition, enlarged and revised. 779 pages. Illustrated with 40 engravings and a colored plate. Lea & Febiger, Philadelphia, 1939. Price $7.50.

Due to the rapid progress and numerous investigations in this field, the author has found it necessary to completely revise this book. A new chapter on azotemia has been added; other chapters have been brought up to date by the addition of items such as: Goldblatt's experimental work on renal ischemia and hypertension, clearance tests, Addis count, mercurial diuretics, pathogenesis and treatment of hypertensive encephalopathy, Cushing syndrome, pathogenesis of malignant phases of hypertension, paroxysmal hypertension in chromaffine tumors and surgical treatment of hypertension.

The reviewer feels that some mention should have been made of the treatment of amyloidosis with liver extract. While this treatment is still in the experimental stage, it appears to be quite promising.

In the majority of instances the author gives the experiences and opinions of other authorities and then adds his own experiences and frankly states his opinion of various treatments and procedures.

The majority of cases of hypertension and nephritis are treated by the general practitioner, whose laboratory facilities are limited, and, for this reason the author has emphasized simple reliable tests which can, as a rule, be carried out in the office.

This book is outstanding and undoubtedly the most valuable, comprehensive and authoritative contribution on the subject that we have today. Medical students, practicing physicians, clinical instructors and investigators will find this work an invaluable aid.

INTERNAL MEDICINE, Its Theory and Practice in Contributions by American Authors, edited by *John H. Musser, B. S., M. D., F. A. C. P., Professor of Medicine in The Tulane University of Louisiana School of Medicine; Senior Visiting Physician to the Charity Hospital, New Orleans, Louisiana.* Third edition, thoroughly revised, large octavo, 1428 pages, illustrated. Philadelphia: Lea & Febiger, 1938. Price $10.00.

The third edition has undergone a careful and extensive revision. In the fields of internal medicine that have shown rapid progress in the past few years, the contributors have found it necessary to practically rewrite such sections. Several new sections have been added, such as, erythemia arthriticum epidemicum (Haverhill fever), glycogenosis, the syndrome of hypertension, hyperglycemia and obesity.

There are exceptionally few typographical errors in this book, it is accurately indexed and has an ample bibliography.

The few minor faults the reviewer noted are hardly worth mentioning, for example no mention is made of the roentgen ray treatment of herpes zoster, atebrine treatment in giardiasis and the impression is given that sulfanilamide is of no value in undulant fever.

With the rapid advances in some of the phases of internal medicine any book on the subject necessarily shows some signs of beginning obsolescence almost as soon as it is released by the publishers.

There are 26 eminent contributors, all of whom are teachers in prominent medical schools; most of them are also practicing specialists and they are recognized authorities in the various branches of internal medicine.

This book can be unhesitatingly recommended for medical students, general practitioners, and internists.

THE ENDOCRINE GLANDS, by *Max A. Goldzieher, M. D., Endocrinologist, Gouverneur Hospital and Brooklyn Women's Hospital, New York; Former Professor of Pathology, Royal Hungarian University, Budapest.* First Edition, 916 pages, illustrated. D. Appleton-Century Company, New York and London, 1939. Price $10.00.

During the past several years the literature dealing with the various phases of endocrinology has been so voluminous that the average

physician has found it extremely difficult, if not impossible, to keep abreast of the scientific development in this field. If he has made the effort, all too often he has been unable to see the forest because of the trees. The author admirably reviews this literature and evaluates it against a background of 30 years actively spent in the clinical and experimental study of glandular disorders. His judgement is sound and the text is built on the concepts which we hold today.

Following a discussion of the general principles of endocrinology, the various glands are taken up separately and considered with equal thoroughness from both the theoretical and practical aspects. The study of each gland opens with a brief historical, embryological and anatomical résumé which is followed by thorough physiological and pathological considerations before taking up the various clinical conditions associated with dysfunction. The clinical disorders associated with each gland are orderly and thoroughly considered from the standpoint of symptomatology, morbid anatomy, pathogenesis, diagnosis, differential diagnosis, treatment, and prognosis. The sections on diagnosis and treatment are outlined in considerable detail, with emphasis on the author's personal experience, and illustrated by case histories from his own practise.

The book is well bound, well printed, and illustrated with 271 figures in black and white. The bibliography is extensive and is so arranged as to offer the source for more detailed information in support of statements made.

It is recommended to all interested in this field of medicine and especially to the general practitioner who first sees the great majority of patients with endocrine disorders.

DISEASES OF THE NOSE AND THROAT, by *Charles J. Imperatori, M. D., F. A. C. S. Professor of Otolaryngology, New York Polyclinic Medical School and Hospital, formerly, Professor of Clinical Otolaryngology, New York Post-graduate Medical School, Columbia University, New York; consulting Laryngologist to Nyack General Hospital and Harlem Hospital, New York; consulting Bronchoscopist, Manhattan Eye, Ear and Throat Hospital, Fifth Avenue and Flower Hospital and Riker's Hospital, New York*; and *Herman J. Burman, M. D., F. A. C. S., adjunct professor of Otolaryngology, New York Polyclinic Medical School and Hospital; formerly assistant professor of Clinical Otolaryngology, New York Post-graduate Medical School, Columbia University, New York; director of the Department of Otolaryngology, Harlem Hospital, New York; consulting Bronchoscopist to Broad street Hospital and Pan American Clinics, New York.* Second edition revised. 726 pages, 480 illustrations. J. B. Lippincott Company, Philadelphia, London and Montreal, 1939. Price $7.00.

Doctors Imperatori and Burman have in the second edition of their book on the diseases of the nose and throat given the general practitioner as well as the student a revision and numerous additions to their original well written volume. The entire book is concisely and simply written and yet has sufficient incorporated detail of necessary material whether anatomical, pathological or surgical as to be of great help to the student of nose and throat whether in general practice or one confining his work to the specialty alone. Information is readily

available and so simply written that the hurried physician can quickly gain the needed knowledge and not have to wade through a series of discussions on the thoughts and methods of numerous authorities as well as those of the authors. The additional chapters of X-ray Examinations by Dr. Frederick M. Law, Radium and X-ray Therapy by Dr. I. I. Kaplan and Laboratory Methods by Dr. Andrew A. Eggston tend to make the book a well rounded treatise for ready reference as well as thoughtful consideration.

ROENTGEN TECHNIQUE, by *Clyde McNeill, M. D., Louisville, Kentucky,* Chas. C. Thomas, Publisher, Springfield, Ill. and Baltimore, Md., 1939. Price $5.00.

This book is a valuable contribution to the technical literature of roentgenology. It is not however, designed to cover the entire subject of technique in all its manifestations; it largely omits x-ray physics, dark-room technique, soft-tissue technique and consideration of apparatus. Its great value is that it constitutes a complete and exceedingly well illustrated treatise on the actual technique of radiography with special emphasis on positioning. All the many positions used in radiography of sinuses and mastoids are shown with precision and clarity. The same holds true for other parts of the anatomy. Photographs and anatomical diagrams are well used to accomplish this end.

Brief but fairly adequate consideration is given to the kymograph, spinograms, uterosalpingograms, urograms, pelvimetry, etc. The book closes with a good though highly condensed chapter on exposure technique.

DISCOVERY OF THE ELEMENTS by *Mary Elvira Weeks, Associate Professor of Chemistry at the University of Kansas.* With illustrations collected by *F. B. Dains, Professor of Chemistry at the University of Kansas.* Fourth Edition enlarged and revised. Pages 470. Photographs, illustrations, and drawings 334. Copyright 1939 by The Journal of Chemical Education, Easton, Pennsylvania, and printed by the Mack Printing Company, Easton, Pennsylvania. Price $3.00.

This book is a history of the discovery of the chemical elements, and a biography of the scientists who made possible these discoveries. For the first time the story of the discovery of the chemical elements has been told as a connected narrative, which for the research student has obviated the necessity of delving into old chemical journals and obsolete text books for source material. The necessary data are available in chronological sequence and at the end of each chapter is a complete bibliography. Even to the layman this book provides entertaining reading—in fact, it reads like a novel.

SCLEROSING THERAPY. The Injection Treatment of Hernia, Hydrocele, Varicose Veins and Hemorrhoids. Edited by *Frank C. Yeomans, M. D., F. A. C. S., M. R. S. M. (Lond., Hon.), Professor of Proctology and Attending Surgeon, New York Polyclinic Medical School and Hospital, etc.* Williams and Wilkins Co., Baltimore. 1939. Pp. 317. Ill. 117. $6.00.

In 1931, at the University of Minnesota, the injection treatment of hernia was begun in the clinic for ambulant treatment of hernia. Dr.

A. A. Bratrud, the director of the clinic, now gives his report on the cases treated by this method, and contrasts them with the results in cases surgically treated. He shows how to decide which cases should be operated on, which injected, and which should wear trusses. The results, when this intelligent sorting of cases is used, are very satisfactory. He warns of the dangers of the injection treatment, and tells how to avoid them. The anatomy, histopathology, and technique are fully described. He concludes that the injection treatment is a most valuable adjuvant to the surgical treatment, and will not supplant it.

Injection treatment of hydrocele is described by Dr. George F. Hoch, attending urologist at St. Luke's Hospital, New York. He discusses the anatomy, pathology and diagnosis of hydrocele, and gives in full detail the technique of the injection treatment.

Dr. Harold J. Shelley, assistant surgeon at St. Luke's Hospital, devotes 120 pages to the injection treatment of varicose veins, and handles the subject in a most competent manner. Treatment of hemorrhoids by injection is fully discussed by Dr. Yeomans.

Whether or not one desires to adopt the sclerosing methods, a surgeon cannot afford to remain ignorant of them, or to resist too long the strong modern trend toward them. This book will prove an excellent guide for their study and use.

FUNCTIONAL DISORDERS OF THE FOOT. By *Frank D. Dickson, M. D., Orthopedic Surgeon, St. Luke's, Kansas City General, and Wheatley Hospitals, Kansas City, Missouri. Providence Hospital, Kansas City, Kansas* and *Rex L. Diveley, M. D. Orthopedic Surgeon, St. Luke's, Kansas City General; and Wheatley Hospitals, Kansas City, Missouri; Providence Hospital, Kansas City, Kansas.* J. B. Lippincott Co., Philadelphia, Pa. 1939. Pp. 292. Ill. 202. $5.00.

This is an excellent book. It is easy to read and covers the subject matter well. The anatomy and physiology of the foot are presented. The disorders of the foot occurring in childhood, adolescence, and in the adult are taken up separately and covered in detail.

The treatment of disorders of the foot is not standardized. The authors include many of the accepted methods and have added methods of treatment which have proven successful in their vast experience in treating foot disorders.

This book should not only be of interest to the orthopedic surgeon but also to the general practitioner who is frequently called upon to treat disorders of the foot. A better understanding of the foot disorders by the medical profession as a whole would greatly relieve the chiropodist and shoe salesmen of the responsibility they have assumed in the treatment of disabled feet.

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PRACTICAL DERMATOLOGY AND SYPHILIS, by *Harry M. Robinson, M. D., Professor of Dermatology, and Director of the Syphilis Clinic, University of Maryland, School of Medicine, Instructor in Medicine, Syphilis Division, Johns Hopkins Medical School,* First edition, 397 pages, illustrated. P. Blakiston's Son and Company, Inc. Philadelphia, 1939. Price $4.50.

In most books on skin diseases the aim seems to be to make the book as large as possible. Most dermatologist authors have had great success in this aim and have been able to produce books of at least 3-inch thickness and of unusually large format. The result for the reader is a laborious process in the seeking of information and much confusion in extracting the meat from a sea of more or less extraneous matter.

In this present book, however, the author has departed from the aim of size and has succeeded very notably in achieving in a work of only 1-inch thickness a condensation of the knowledge of actual practical value in dermatology.

This is not to say that it resembles a quiz compend either in arrangement or in text or in purpose. On the contrary, the arrangement is such that conditions may be looked up according to lesion appearances rather than according to disease entities, thus enhancing the search of the puzzled practitioner who has the patient and his lesions on one side and the book with the answers on the other.

The text and the very excellent collection of illustrations give the answers quicker, more easily than any skin text this reviewer has seen, and with unquestionable authenticity.

The section on syphilis, even though condensed, is a 1939 presentation quite adequate for the general practitioner.

The book in all its phases is something new, something original and should be something that will prove to be highly welcomed by anyone who sees a skin lesion, knows not what it is or what to do about it.

The best feature of the book is the viewpoint of the author who sees dermatology as cutaneous medicine, a broad field of casative factors with their resulting manifestations in the skin.

DISEASES OF THE SKIN by *Richard L. Sutton, M. D., Sc. D., L. L. D., F. R. S. (Edin.), Professor of Dermatology, University of Kansas, School of Medicine,* and *Richard L. Sutton, Jr., A. M., M. D., L. R. C. P. (Edin.), Associate in Dermatology, University of Kansas, School of Medicine.* With 1,452 text illustrations and 21 color plates. Tenth Edition. 1,549 pages, C. V. Mosby Company, St. Louis, 1939. Price $15.00.

This is a veritable encyclopedia of dermatology. It is enclopedic in weight and size as well as in the exhaustive manner with which the authors have attempted to cover every branch and phase of skin diseases. This present volume, being the tenth edition, gives the appearance that its authors have added much new material with each succeeding edition but without deleting much obsolete material that could easily be discarded. In short, it appears that their strenuous efforts to make the succeeding editions bigger and better have unfortunately resulted only in making them bigger.

The book is, of course, a most excellent and authoritative reference work on the subject and very probably would serve as well as, if not indeed better than other similar works of recent publication. The enormous listing of reference sources at the end of each chapter suggests that the authors must have hired a considerable office force to digest and compile this literature, perhaps with some assistance by themselves.

COMPLETE DENTURE PROSTHESIS by *Rudolph O. Schlosser, D. D. S., F. A. C. D., Professor of Prosthetic Dentistry, Northwestern University Dental School,* with 583 illustrations on 285 figures. W. B. Saunders Company, Philadelphia and London, 1939. $5.00 net.

The purpose of this book is to furnish to the student, undergraduate, graduate, or postgraduate a "comprehensive presentation of the clinical and laboratory procedures necessary to render a service in accord with the highest professional standards."

This textbook employing nomenclature used in the report of the Curriculum Survey Committee of the American Association of Dental Schools, is presented in two sections. The author believing that the hand cannot execute that which the mind cannot conceive, has devoted the first section to oral anatomy and the fundamentals of mandibular movements, while the second section is devoted to diagnostic and technical procedures.

While the text primarily describes the technic taught by the author, various contemporary methods are included, which, though differing somewhat in detail of technical procedure, approach in principle the same fundamental considerations. These inclusions widen the scope and usefulness of the book.

The principles and the conclusions as set down in this book represent the acme in effort of a man known throughout the United States for his contributions to the advancement of dentistry. It truly is "a guide to a better understanding of the fundamental principles involved in complete denture prosthesis and a means to a solution of the problems."

PARTIAL DENTURES, a System of Functional Restoration, by *Ferdinand G. Neurohr, D. D. S., F. I. C. D., Author of chapters in the American Textbook of Prosthetic Dentistry; Fellow of the New York Academy of Dentistry; Attending Prosthodontist at the New York Polyclinic Medical School and Hospital; Special Lecturer in Partial Denture Prosthesis at Columbia University School of Dentistry;* 244 pages. Illustrated with 206 engravings. Lea & Febiger, Philadelphia, Pa., 1939. Price, $6.50.

This volume is a concise, compact textbook describing in detail the technique used in constructing the insertion pin and spring wire lock partial denture restoration. It may be heartily recommended to those practitioners working in the partial denture and crown and bridge fields of dental prosthodontia.

THE DIVISION OF PREVENTIVE MEDICINE

Commander C. S. STEPHENSON, Medical Corps,
United States Navy, in Charge

TOXIC EFFECTS OF ARSENICAL COMPOUNDS

AS EMPLOYED IN THE TREATMENT OF DISEASES IN THE UNITED STATES NAVY, 1938

By Commander C. S. Stephenson, Medical Corps, United States Navy and Chief Pharmacist's Mate, W. M. Chambers, 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 report of each case in which ill effects are noted. During the 14 years, 1925–38, in which this information has been compiled, 1,432,970 doses of arsenicals have been administered and 1,013 reactions have been reported.

Previous articles dealing with the information obtained from these reports have been published in the following issues of this bulletin.

September 1925.	October 1933.	January 1937.
January 1927.	October 1934.	October 1937.
January 1929.	January 1935.	January 1938.
July 1930.	October 1935.	October 1938.
October 1931.	January 1936.	January 1939.
October 1932.	October 1936.	October 1939.

Cases of arsenical dermatitis occurring during the year 1938 were published in the October 1939 issue.

The present article includes all cases, except arsenical dermatitis, which were reported during the year 1938. Comparative figures from the experience of previous years are also presented.

TABLE 1.—*Arsenical reactions, 1938*

Classification	Neoarsphenamine, mapharsen, and acetarsone reactions *			
	Mild	Severe	Fatal	Total
Arsenical dermatitis	13	10	2	25
Vasomotor phenomena	7	0	0	7
Blood dyscrasias	2	0	1	3
Liver damage	0	3	0	3
Gastrointestinal	1	1	0	2
Total	23	14	3	40

* Case histories were published in the October 1939 number of the bulletin. Included in the above table is 1 mild reaction caused by acetarsone and 7 reactions (5 mild and 2 severe) caused by mapharsen.

TABLE 2.—*Arsenicals administered during the year 1938 for all diseases*

Drug	Dose (grams)				
	0.9 to 3	0.9	0.6 to 0.9	Less than 0.6	Total
Acetarsone:					
Navy	0	0	0	26	26
All others	0	0	0	0	0
Bismarsen:					
Navy	0	0	0	368	368
All others	0	0	0	189	189
Mapharsen:					
Navy	0	0	0	37,517	37,517
All others	0	0	0	5,238	5,238
Neoarsphenamine:					
Navy	0	163	25,623	27,371	53,157
All others	0	41	4,292	14,204	18,537
Sulfarsphenamine:					
Navy	0	0	15	121	136
All others	0	0	25	1,790	1,815
Tryparsamide:					
Navy	4,467	0	0	0	4,467
All others	1,726	0	0	0	1,726
Total	6,193	204	29,955	86,824	123,176

TABLE 3.—*Arsenicals administered during the 7-year period, 1932–38, for all diseases*

Drug	Doses (grams)				
	0.9 to 3	0.9	0.6 to 0.9	Less than 0.6	Total
Acetarsone:					
Navy	0	0	0	166	166
All others	0	0	76	729	805
Arsphenamine:					
Navy	0	0	149	10,297	10,446
All others	0	0	7	706	713
Bismarsen:					
Navy	0	0	0	1,544	1,544
All others	0	0	1	844	845
Mapharsen:					
Navy	0	0	0	48,611	48,611
All others	0	0	0	7,678	7,678
Neoarsphenamine:					
Navy	0	5,091	254,942	352,105	612,138
All others	0	437	32,491	103,915	136,843
Silver arsphenamine:					
Navy	0	0	0	350	350
All others	0	0	0	204	204
Sulfarsphenamine:					
Navy	0	18	296	7,416	7,730
All others	0	7	234	11,403	11,644
Tryparsamide:					
Navy	26,125	0	0	10	26,135
All others	12,183	0	0	5	12,188
Total	38,308	5,553	289,196	545,983	878,040

TABLE 4.—*Deaths and severe reactions, following administration of 1,244,537 doses of neoarsphenamine, 1925–38; ratio of deaths and severe reactions to doses*

Classification	Deaths		Severe reactions		Deaths and severe reactions	
	Number	Ratio to doses 1 to—	Number	Ratio to doses 1 to—	Number	Ratio to doses 1 to—
Hemorrhagic encephalitis	16	77, 784	1	1, 244, 537	17	73, 208
Arsenical dermatitis	12	103, 711	187	6, 655	199	6, 254
Vasomotor phenomena	6	207, 423	56	22, 224	62	20, 073
Blood dyscrasias	6	207, 423	18	69, 141	24	51, 856
Acute renal damage	2	622, 269	5	248, 907	7	177, 791
Acute yellow atrophy of the liver	2	622, 269	0	----------	2	622, 269
Vascular damage (probable renal hemorrhage)	1	1, 244, 537	0	----------	1	1, 244, 537
Liver damage	0	----------	18	69, 141	18	69, 141
Jarisch-Herxheimer	0	----------	2	622, 269	2	622, 269
Gastro-intestinal	0	----------	3	414, 846	3	414, 846
Polyneuritis	0	----------	1	1, 244, 537	1	1, 244, 537
Border-line hemorrhagic encephalitis	0	----------	1	1, 244, 537	1	1, 244, 537
Arsenical neuritis	0	----------	1	1, 244, 537	1	1, 244, 537
Optic neuritis	0	----------	1	1, 244, 537	1	1, 244, 537
Total	45	27, 656	294	4, 233	339	3, 671

TABLE 5.—*Deaths following administration of arsenical compounds, 1919–38*

Year	Arsphenamine	Neoarsphenamine	Total	Year	Arsphenamine	Neoarsphenamine	Total
1919	3	0	3	1930	0	3	3
1920	1	1	2	1931	0	0	0
1921	3	1	4	1932	0	4	4
1922	0	4	4	1933	0	7	7
1923	0	1	1	1934	0	3	3
1924	1	2	3	1935	0	2	2
1925	0	2	2	1936	0	3	3
1926	0	4	4	1937	0	1	1
1927	1	4	5	1938	0	3	3
1928	0	6	6				
1929	0	3	3	Total	9	54	63

NUMBER OF PERSONS TREATED FOR SYPHILIS AND OTHER DISEASES

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

In the table which follows, treatment data have been separated into that given to active service personnel and that given to all others. The term ALL OTHERS includes Veterans' Administration patients, dependents of naval personnel, retired naval personnel, and native populations of insular possessions.

TABLE 6.—*Syphilis and arsenicals, U. S. Navy, 1938*

Item	Persons		
	Navy and Marine Corps	All others	Total
Strength, Dec. 31, 1938	139, 128		139, 128
Syphilis census, Dec. 31, 1938	14, 059		14, 059
Number of persons treated for syphilis with—			
Arsenicals:			
Acetarsone	0	3	3
Bismarsen	19	27	46
Mapharsen	2, 488	488	2, 976
Neoarsphenamine	3, 422	678	4, 100
Sulpharsphenamine	16	60	76
Tryparsamide	208	102	310
Total persons treated with arsenicals	6, 153	1, 358	7, 511
Heavy metal compounds:			
Bismuth compounds	5, 073	1, 345	6, 418
Mercury compounds	145	27	172
Mixed treatment (specific mixture, etc.)	45	54	99
Potassium iodide	109	98	207
Total persons treated with heavy metal compounds	5, 372	1, 524	6, 896
Number of persons treated for disease other than syphilis with—			
Arsenicals:			
Mapharsen	30	0	30
Neoarsphenamine	271	1, 569	1, 840
Sulpharsphenamine	0	116	116
Tryparsamide	1	0	1
Total persons treated with arsenicals	302	1, 685	1, 987
Heavy metal compounds: Bismuth compounds	1	103	104

In table 6 it will be noted that 302 service personnel and 1,685 non-service personnel were treated for diseases other than syphilis with arsenical compounds during the year 1938.

Of the 302 naval personnel, 272 were treated for Vincent's infection, 16 were treated for acne, 1 treated for yaws, and 13 treated for other diseases and conditions.

Of the 1,685 persons in the group ALL OTHERS, 1,682 were treated for yaws and 3 treated for other diseases and conditions.

VASOMOTOR PHENOMENA

Neoarsphenamine—(26–1938).—A patient who was exposed to infection on April 21, 1938, developed a small lesion on superior medial surface of posterior prepuce. Repeated darkfield examinations and Kahn blood tests were negative. On June 13, 1938, the patient complained of a sore throat and a rash on his body. Examination revealed a maculopapular rash resembling that of secondary syphilis. Kahn blood tests were strongly positive on June 14 and 16.

Arsenical treatment began on June 16 with a 0.3 gram injection of neoarsphenamine, followed by 0.45 gram injections on June 23 and 30. Bismuth subsalicylate was given as concurrent treatment.

Four hours after the last injection of neaorsphenamine the patient complained of severe headache, chills, and palpitation of the heart. Examination showed marked flushing of the face and body and suffusion of the conjunctivae. Temperature, 103.2°; pulse, 112; respirations, 32. He vomited profusely, mostly water, colored with bile, which was followed by a slight nose bleed. One gram of sodium thiosulphate was given intravenously. Recovery in 4 days.

(*27–1938*).—A patient, exposed to infection on December 18, 1937, developed a penile lesion which was positive for *Treponema pallidum*.

Arsenical treatment was instituted on January 27, 1938, with a 0.3 gram injection of neoarsphenamine. Six hours after the injection the patient suffered a typical Herxheimer reaction with localized edema at site of chancre, stiffness of neck and knees, general malaise, and fever of 101°. No specific treatment was administered and patient completely recovered in 12 hours after onset of symptoms.

(*28–1938*).—This patient was exposed to infection on September 2, 1937, and developed a small ulcer on the penis, generalized adenopathy, and generalized maculopapular rash. Repeated Kahn blood tests were 4-plus.

From November 2 to December 21, 1937, he received 8 injections of neoarsphenamine, a total of 3.75 grams, and from February 22 to March 31, 1938, 6 injections, a total of 3.05 grams.

Three and one-half hours after the last injection of neoarsphenamine the patient complained of headache and chilly sensations. The face was flushed and the temperature was 100°. He was put to bed, covered with blankets, with ice cap to head and hot water bottle to feet. In ½ hour the patient broke out in a profuse perspiration and all symptoms quickly subsided. Recovery in 6 hours.

(*29–1938*).—After exposure to infection this patient developed a primary lesion on the penis on April 14, 1933. Kahn blood tests and darkfield examinations were negative.

A diagnosis of syphilis was established on June 4, 1934, when the patient developed femoral adenopathy and numerous patches in the mouth. No history of initial lesion since the above on April 14, 1933.

Two courses of eight injections each of an arsenical compound (type not stated) were given during the period from June 5, 1934, to July 7, 1935, a total of 2.7 grams. Mercury succinimide was given as concurrent treatment.

The third course of arsenical treatment began with 0.3 gram injection of neoarsphenamine on March 16, followed by 0.3 gram injections on April 6, 13, and 21, 1938. Because the patient complained of feeling badly following his first injection of this series, 0.3 gram injections were given. Signs of illness appeared immediately following completion of the fourth injection. He gagged, appeared pale, and his gait faltered. There was an increase of cyanosis, dyspnea, weakening of the pulse, and vomiting.

One gram of sodium thiosulphate was given intravenously, and 7 minims of epinephrin, 1–1,000, intramuscularly. Vomiting continued and the patient appeared in semi-shock for about an hour. A blood streaked vomitus and bowel movements of bloodly mucus occurred intermittently. Excoriated papular rash appeared on volar surface of forearms. Caffeine sodium benzoate and 500 c. c. of 50 percent glucose in normal saline were administered immediately. The condition improved considerably and the symptoms subsided the following day. Recovery in 1 day.

(*30–1938*).—This patient was exposed to infection on February 6, 1938, and was given a diagnosis of syphilis because of positive darkfield examinations.

From March 15 to May 11, 1938, he received 5.55 grams of neoarsphenamine, and from May 11 to July 20 he received 1.3 grams of bismuth salicylate.

The second course of arsenical treatment began on July 27, 1938, with a 0.3 gram injection of neoarsphenamine, followed by a 0.45 gram injection on August 3, and 0.6 gram injections on August 10, 17, 24, 31, September 21, and October 5, a total of 4.35 grams.

Ten minutes after the last injection the first symptons of a reaction appeared with cutaneous flushing, injection of the conjunctiva, puffiness of the eyelids,

choking and asthmatic wheezing, cough, headache, spasm of the abdominal muscles, and retching, but not actual vomiting. There was moderate prostration. Temperature, 99.6°; pulse, 110; respirations, 28.

One gram of sodium thiosulphate was given intravenously 15 minutes after onset of symptoms. Recovery in 4 hours.

Mapharsen—(*31–1938*).—After exposure to infection in August 1937 this patient devcloped a chancre on the penis which was positive for *Treponema pallidum.* A Kahn blood test was 3-plus.

He received 12 injections of mapharsen from August 13 to October 26, 1937, and 19 injections of bismuth from October 13, 1937, to February 16, 1938.

The arsenical reaction in this case occurred during the second course of mapharsen, 4 hours after the fourth injection on March 30, 1938. The patient was admitted complaining of headache, fever, nausea, and vomiting; temperature 100.4°. No specific treatment given.

The following day all symptoms had subsided and the patient appeared completely recovered. On April 12 he was given a 0.03 gram injection of mapharsen as a therapeutic test. Three hours later he became nauseated and vomited and the temperature was elevated to 100°. Injections of neoarsphenamine were given on June 7, 14, 21, and 28, with no evidence of a reaction. Recovery in 2 days from onset of first symptoms.

(*32-1938*).—A patient, exposed to infection on May 18, 1938, developed a primary lesion on the penis, generalized rash, and general adenopathy. The Kahn blood test was 4-plus.

From June 29 to August 23, 1938, he received 11 injections of mapharsen, a total of 0.63 gram. Two minutes after the last injection the patient developed a cardiovascular type of reaction accompanied by severe shock as evidenced by pallor weakness, nausea, and sweating, One gram of sodium thiosulphate was administered intravenously. Recovery in 4 hours.

Gastro-intestinal

Neoarsphenamine—(*33–1938*).—A patient who was exposed to infection on December 15, 1938, developed a primary lesion on shaft of penis. A darkfield examination was positive for *Treponema pallidum.*

Arsenical treatment began with a 0.3 gram injection of neoarsphenamine on December 20, 1938, followed by 0.6 gram injections on December 23 and 26.

Two days after the last injection of neoarsphenamine the patient developed a generalized rubellaform rash, accompanied by nausea, vomiting, and diarrhea. A urinalysis on December 29 showed a trace of albumin. WBC, 6,400; hgb,100; bands, 5; segs, 40; lymphs, 39; eosins, 2; basos, 2; monos, 12. No specific treatment was administered. Symptoms gradually subsided. Recovery in 12 days.

(*34–1938*).—After exposure to infection, this patient developed an initial lesion on frenum of penis. Darkfield examinations were repeatedly positive for *Treponema pallidum.*

Arsenical treatment was instituted with 0.3 gram injections of neoarsphenamine on October 18 and 24, 1938, followed by a 0.45 gram injection on October 27.

Five hours after the last injection the patient was suddenly seized by a profuse diarrhea and was removed to the sick bay. Temperature, 103°; pulse, 110; respirations, 25. Examination revealed an extreme degree of hyperemia and injection. WBC, 19,200. Scaling of skin over back, scrotum, face, and legs occurred 4 days later.

One gram injections of sodium thiosulphate were administered on October 27 and 28.

The skin condition terminated by desquamation, and the other symptoms gradually subsided. Recovery in 96 days.

Liver Damage

(*35–1938*).—The source of infection in this case is unknown. The patient was given a diagnosis of syphilis because of repeated positive Kahn blood tests, and a rash on the trunk and extremities. It was thought that the initial lesion might have been intraurethral.

Arsenical treatment began with 0.3 gram injections of neoarsphenamine on January 20 and 27, 1938.

Two days after the last injection of neoarsphenamine the patient complained of headache, earache, fever, and general malaise. Examination revealed a generalized fine, red macular eruption over the entire body except head, hands, and feet. WBC, 12,200; myelocytes, 3; juveniles, 1; bands, 32; segs, 33; lymphs, 24; basos, 1; and monos, 2. The urine was positive for albumin with occasional hyaline casts, few pus casts, and some mucus. Some of the stools were clay colored and there was a definite icteric tint to the sclera and skin.

February 14: The patient was markedly jaundiced.

March 11: Duodenal drainage daily. Sodium phosphate each morning.

March 22: Marked improvement. Antiluetic treatment continued with bismuth.

The patient's condition gradually improved under treatment and he was returned to duty 75 days after the last injection of neoarsphenamine.

(*36–1938*).—One week after exposure to infection on April 4, 1938, this patient developed a penile lesion which was positive for *Treponema pallidum*. He received a 0.3 gram injection of neoarsphenamine on April 13 and a 0.5 gram injection on April 19.

Three hours after the last injection of neoarsphenamine the patient developed a headache and a temperature of 103°. He was immediately given one gram of sodium thiosulphate intravenously. The following day he developed a generalized confluent rash, which was confined to large irregular purplish areas apparently underlying the stratum corneum. The patient was transferred to a hospital on April 21. Upon admission, examination revealed a brilliant erythema of the face, arms, and trunk, with edema of the skin over the cheek bones and of the eyelids. He was given 10 cc. of 1 percent sodium thiosulphate.

Urinalysis showed 2-plus albumin, few course granular casts, and a small amount of mucus. WBC, 8,250; segs, 48; bands, 28; eosins, 3; lymphs, 20; and monos, 1.

April 22: Skin shows an increase in erythema and a tendency toward purpura. The sputum is positive for pus.

April 23: The urine shows the presence of bile. Icterus index 30. Glucose therapy instituted.

April 26: The skin shows a slight tendency toward desquamation, erythema still marked. The patient was placed on the serious list due to a severe liver damage.

April 27: Icterus index 40. Urinalysis shows a trace of albumin and is positive for bile. The erythema has disappeared, but the icterus has increased. Large amounts of glucose by venoclysis, covered by insulin.

May 2: There has been a cutaneous relapse at site of chancre. Patient placed on soluble bismuth salt, one injection weekly.

May 10: Icterus index 105. Urinalysis essentially negative except for the presence of bile.

June 2: Jaundice steadily improving. Penile lesion completely healed under bismuth therapy.

August 29: Patient's general condition improved.

November 26: Patient returned to duty this date with recommendation that he receive mercury and bismuth to limit of tolerance, but that arsenicals be avoided. Recovery in 221 days.

(*37–1938*).—Two months after exposure to infection this patient developed a pronounced inguinal adenopathy. A Kahn blood test was 3 plus on July 18 and 4 plus on July 25, 1938. Patient denied initial lesion, but there were numerous scars on the penis.

Arsenical treatment began with 0.3 gram injections of neoarsphenamine on July 18 and 25, followed by a 0.45 gram injection on August 2.

Ten hours after the last injection the patient became nauseated and feverish. Two days later he became jaundiced, but denied any skin rash. He had, in addition, a painful swelling in left groin of 6 or 7 weeks' duration. A urinalysis showed 1-plus albumin with 10–15 leukocytes per HPF. One gram of sodium thiosulphate was given intravenously.

August 9: WBC, 3,700; segs, 7; lymphs, 76; monos, 4.

August 10: Icterus index 53.

August 17: Frei test positive.

August 19: Icterus index 25.

August 25: Diagnosis changed to lymphogranuloma inguinale (concurrent).

September 3: Patient no longer jaundiced.

October 4: He was given a 0.01 gram injection of mapharsen with no untoward after effects except that a white blood count revealed slight suppression of neutrophiles.

A 0.02 gram injection of mapharsen was given on October 17 and a 0.04 gram injection on October 24 with no apparent ill effects.

Antiluetic treatment continued with mapharsen, bismuth being given as concurrent treatment.

Blood picture

Date	White blood count	Segmented	Band forms	Eosinophiles	Lymphocytes	Monocytes
Aug. 9, 1938	3,700	7			76	4
Aug. 16, 1938	8,050	54	8	6	32	
Oct. 4, 1938	5,850	66	2	4	26	2
Oct. 7, 1938	7,100	56	2	6	36	
Oct. 12, 1938	6,300	68	4		28	
Oct. 13, 1938	7,300	62	2		36	
Oct. 20, 1938	5,000	50	4	2	42	2
Oct. 27, 1938	6,500	51	7	1	39	2
Oct. 31, 1938	6,250	59	3	1	32	5
Nov. 3, 1938	9,050	58			31	4
Nov. 10, 1938	6,700	65			31	4

The patient was returned to duty 110 days after onset of first symptoms.

Blood Dyscrasias

(*38–1938*).—This patient was first infected in January 1929, and was given a diagnosis of syphilis because of positive darkfield examinations. Arsenical treatment consisted of 21 injections of neoarsphenamine and 23 injections of a heavy metal (type not stated) as concurrent treatment.

The patient was infected the second time on February 1, 1930, and one month later developed a primary lesion on scrotum, a macular rash over chest and upper extremities, sore throat, headache, and general glandular enlargement. A Kahn blood test was 4 plus.

He received 25 injections of neoarsphenamine from March 31, 1930, to June 30, 1931, and 61 injections of bismuth from March 31, 1930, to December 7, 1937.

The fourth course of arsenical treatment began on January 22, 1938, with a 0.3 gram injection of neoarsphenamine, followed by 0.45 gram injections on February 2 and 10. No concurrent treatment was given during this course.

One hour after the last injection the patient complained of nausea, vomiting, abdominal cramps, and bleeding from the gums. Examination was essentially negative except for a persistent ooze of blood from around the lower anterior teeth.

Blood picture

Date	WBC	Hgb	Bands	Segs	Lymphs	Eosins	Basos	Monos
Feb. 10, 1938	8,150	90	2	81	9	1	2	5
Feb. 11, 1938	13,450	90	------	83	11	5	1	------
Feb. 13, 1938	8,050	90	2	67	19	7	------	5
Feb. 14, 1938	7,650	90	4	67	22	5	------	2

Patient was put to bed and symptomatic treatment immediately instituted. Recovery in 7 days.

(*39–1938*).—Following exposure to infection on August 14, 1938, this patient developed an ulcer on the left flank, and a ham-colored maculopapular rash on trunk, arms, and thighs. A darkfield examination of the ulcer was positive for *Treponema pallidum*. Repeated Kahn blood tests were 4 plus. During the period from September 10 to September 27, 1938, he was given seven 0.1 gram injections of bismosol.

Arsenical treatment began on September 22, 1938, with a 0.3 gram injection of neoarsphenamine, followed by a 0.4 gram injection on September 29. Eight hours after the last injection of neoarsphenamine the patient complained of slight headache and insomnia. Examination revealed flushed and swollen face, red and swollen gums, a slight swelling of the right jaw, and tenderness of teeth. Temperature, 103.2°; WBC, 4,500; RBC, 4,010,000.

The following day there was marked edema of skin overlying the cheeks and eyelids. The gums were swollen and inflamed but no bleeding was noted. There was an ulceration covered with a grayish membrane over the left lateral incisor. No rash was evident.

Blood picture

Date	WBC	Hgb	Segs	Bands	Eosins	Lymphs	Monos	Juvs	Basos
Oct. 3, 1938	3, 950	----	8	4	3	82	3	----	----
Oct. 3, 1938	7, 600	90	13	9	9	46	21	2	----
Oct. 4, 1938	4, 200	----	34	10	12	34	10	----	----
Oct. 5, 1938	6, 500	----	26	12	6	50	8	----	----
Oct. 6, 1938	9, 850	----	56	8	6	24	6	----	----
Oct. 7, 1938	10, 800	----	34	12	6	36	12	----	----
Oct. 10, 1938	12, 350	----	42	------	2	52	4	----	----
Oct. 11, 1938	8, 100	----	44	6	2	38	8	----	----
Oct. 12, 1938	8, 200	----	55	4	6	30	4	----	1

Two 0.5 gram injections of sodium thiosulphate were given intravenously Mouth lesions healed and white blood count slowly returned to normal. Recovery in 26 days.

CASE HISTORY OF FATAL REACTION

(*40–1938*).—The source of infection in this case is unknown. Patient denied venereal infection and there was no evidence of a former penile lesion. A diagnosis of syphilis was made because of general glandular adenopathy and repeated 4-plus Kahn blood tests. Patient was on the sick list with a diagnosis of hydrocele, scrotum, when syphilis was discovered.

Antiluetic treatment began on January 11, 1938, with a 0.3 gram injection of neoarsphenamine, followed by a 0.45 gram injection on January 18, and 0.6 gram injections on February 1, 8, 15, and 23. He received 14 injections of bismuth subsalicylate as concurrent treatment.

January 10: Urinalysis normal. Feces positive for ascaris lumbricoides and trichuris trichiura.

January 12: Patient received routine treatment for intestinal worms.

January 25: Hydrocelectomy this date under spinal anesthesia. Post-operative condition good.

February 10: Incision healed. Swelling of scrotum and testicle decreasing.

March 1: Patient complained of headache, sore throat, and sore gums. Examination revealed acute tonsillitis, pharyngitis, and gingivitis. Diagnosis changed this date to poisoning, neoarsphenamine, acute. Placed on critical list. This reaction was agranulocytosis, apparently caused by injections of neoarsphenamine. Fifteen grains of scdium thiosulphate were given intravenously and 250 c.c. of whole blood were given by direct transfusion. Patient placed on pentnucleotide therapy.

March 2: Temperature, 102 to 103°; pulse, 120 to 130; respirations, 25. General condition poor. Large ulcerated and sloughing areas in hard and soft palate. WBC, 350, of which 100 percent are lymphs. Urine shows 2-plus albumin with many red blood cells.

March 4: Two hundred and fifty cc. of whole blood given by direct method Pulse, 140 to 150; temperature, 103° to 105°; and WBC, 300. Urine contained 3-plus albumin with a few red and white blood cells.

March 5: Patient moribund. Daily intake of fluids maintained by hypodermoclysis of normal saline and glucose.

Blood picture

Date	WBC	Segs	Bands	Juvs	Lymps	Monos	Eosins	Basos
Jan. 10, 1938	8,750	70	------	------	28	------	2	------
Mar. 1, 1938	350	------	------	------	100	------	------	------
Mar. 1, 1938	300	------	------	------	100	------	------	------
Mar. 2, 1938	350	------	------	------	100	------	------	------
Mar. 3, 1938	250	------	10	2	88	------	------	------
Mar. 5, 1938	300	6	20	------	72	1	0	1

Patient's condition became steadily worse, respiration became labored, and the patient died at 12:23 p.m., March 5, 1938, 30 days after the last injection of neoarsphenamine and 25 days after the onset of first symptoms.

Autopsy findings: Body was that of a Filipino, male, 34 years of age, showing beginning dependent lividity and no rigor mortis. Both antecubital spaces are ecchymotic and show evidence of venipuncture. Skin shows a yellowish discoloration. Sclerae are not icteric. The muscles and subcutis tissues were very wet. When the thorax was opened a moderate increase in pleural fluid was found. The right lung was extensively involved in a massive hemorrhagic consolidation which also involved the dependent portions of the left lung. On section the consolidated areas in the left lung were solid, but those in the right exuded frothy fluid. The pericardium showed many petechial hemorrhages and an increase in the amount of pericardial fluid. The myocardium showed multiple petechial hemorrhages and thickening of the coronary vessels. The endocardium and valves appeared grossly normal.

The kindneys were hemorrhagic. The capsules stripped easily and left a smooth surface. The cortical-medullary ratio was normal. The adrenals appeared pale. The spleen was small and dry on section.

The gastro-intestinal tract was normal except for acute congestion of the appendix with hemorrhagic discoloration of the peritoneum making up its bed. A minute rupture was present at the tip, but no pus or exudate was present.

There was a healed incision on the left scrotum (hydrocele).

Bone marrow from the long bones (tibia) was pale and yellowish. That from the sternum appeared dry and less red than normal.

When the calvarium was removed the dura showed small petechial hemorrhages. The cortex of both lobes showed fairly large multiple hemorrhages up to 3 cm. in diameter. The brain was not sectioned.

Gross Pathology: (*a*) Poisoning, acute, neoarsphenamine; (*b*) agranulocytosis; (*c*) pneumonia, lobar, bilateral; (*d*) petechial hemorrhages, pericardium, myocardium, and dura; (*e*) hemorrhages, cerebal cortex; (*f*) ulceration, mouth; and (*g*) appendicitis, acute.

THE HOOKWORM PROBLEM

SOME GENERAL AND MILITARY ASPECTS
A REPORT OF THE EXAMINATION OF 1169 NAVAL RECRUITS[1]

By Lieutenant James J. Sapero, Medical Corps, United States Navy

It is a prevalent opinion that hookworm infections in individuals in apparently perfect health are not of serious consequence. This view, together with reports that hookworm in southern naval recruits

[1] From the Gorgas Memorial Laboratory, Panama, R. de P. Read before the Medical Association of the Isthmian Canal zone at its three hundred and eighty-first meeting on August 16, 1938.

is no longer of common occurrence, has led to the discontinuance of routine examinations for necatoriasis in men enlisted from endemic hookworm centers. In this report, however, data are presented which indicate that hookworm infections are both injurious to the health of these navy men and highly incident in this group. These findings have led to a reconsideration of certain military and general phases of the hookworm problem.

INTRODUCTION

For a 14-year period from 1918 to 1932, it had been the policy to examine incoming recruits at the naval training station in Norfolk, Virginia for hookworms. In 1932, however, routine fecal examinations of recruits from the endemic hookworm centers of the southern United States were discontinued. In this year it appeared from the examinations that necatoriasis had become so rare that there was no longer need to consider this parasitic infection a problem. Previously, Wildman and Betts [2] had reported an incidence of 23.3 percent hookworm in 15,929 southern naval recruits entering the service between 1918 and 1925. The results of the examinations at the naval training station, Norfolk, Virginia in subsequent years are shown in table I.

TABLE 1.—*Results of routine examinations of recruits from Southern States* [3]

Year	Number of examinations	Positive for hookworm	Incidence
1925	2,949	586	19.87
1926	4,478	828	18.49
1927	3,162	398	12.59
1928	3,801	481	12.65
1929	887	95	10.71
1930	827	39	4.72
1931	987	20	2.03
1932	967	7	0.72

It is to be noted that the reported incidence from this station gradually fell in the ensuing years. This fall in incidence paralleled, in general, the splendid hookworm control measures initiated by the Rockefeller Foundation workers. However, in 1932 out of 967 stool examinations, only 7 cases of necatoriasis were found, a remarkably low incidence of 0.72 percent; yet Stiles [4] in this same year noted that hookworm was still widespread in the South. This apparent discrepancy emphasized the need for a resurvey.

[2] Wildman, O., and Betts, N. S.: U. S. Nav. Med. Bul., **23**: 241, Sept. 1925.
[3] The data used to compile this table were taken from the Annual Reports of the Surgeon General, U. S. Navy, for years 1925 to 1932 inclusive.
[4] Stiles, C. W.: Jour. Parasit., **18**: 169, 1932.

INCIDENCE

Simple smear examinations were done on 257 Norfolk recruits since this was the method used in previous naval hookworm studies. Likewise, only men from the same endemic Southern States were included, and thus the incidence obtained was comparable to the previously reported rates. Surprisingly, hookworm infections were found in 11.3 percent of the men, an incidence, which contrary to that found in 1932, indicates a current prevalence of some importance.

It was recognized, however, that the simple smear technique would not detect all of the infections. Therefore, a survey on 696 recruits at the naval training station, Norfolk, Virginia, was conducted using the more efficient Willis brine floatation method. The results are shown in table 2. By this technique an incidence of 21.0 percent hookworm was found.

TABLE 2.—*Incidence of hookworm found in recruits from 14 Southern States*

State	Number men examined	Number cases hookworm	Percent incidence
Alabama	58	17	29.3
Arkansas	6	1	16.7
Florida	19	8	42.1
Georgia	51	14	27.5
Kentucky	98	21	21.4
Louisiana	21	7	33.3
Mississippi	52	11	21.2
N. Carolina	128	36	28.1
Oklahoma	2	1	----------
S. Carolina	42	11	26.2
Tennessee	61	6	9.8
Texas	2	2	----------
Virginia	98	6	6.1
West Virginia	58	5	8.6
Total	696	146	21.0

The incidence by states gives an interesting cross section of the varying prevalence of hookworm in the different endemic areas, though in some cases the numbers included are too small to be of significance. Of the "deep South" States, only one, Tennessee, showed an incidence of less than 20 percent while the others varied from 20 to 42 percent.

In all, 1,169 men were examined. In addition to those already reported, *Necator americanus* was not encountered in any of 94 men from nonendemic hookworm areas. It is also of interest to report that in a group of 212 colored recruits, no necator infections were found, despite the fact that these men had resided in the same regions as the white recruits in which the incidence was 21 percent.

There were no significant numbers of infection with helminths other than *N. americanus*. *T. trichiura*, *H. nana*, and *A. lumbricoides* were encountered only infrequently.

HEMOGLOBIN EXAMINATIONS

In order to obtain a measure of the injurious effect of hookworms in naval recruits, hemoglobin estimations were made on 159 parasitized men. Comparisons were made with a control group in which the examinations had not revealed *N. americanus*. The Dare instrument was used in all determinations. Thus, any inherent error in this method would be expected to occur uniformly in both groups. The results of hemoglobin examinations in hookworm-positive and hookworm-negative men are shown in table 3.

TABLE 3.—*Comparative hemoglobin values in hookworm-positive and hookworm-negative cases*

Hemoglobin percent by group	Hookworm cases		Control (hookworm-negative)	
	Number in group	Percent of total number examined	Number in group	Percent of total number examined
90-100	11	6.9	8	22.2
80-89	40	25.2	14	38.8
70-79	81	50.9	13	36.1
50-69	27	17.0	1	2.9
Total	159	100.0	36	100.0

Average hemoglobin of hookworm-positive cases: 76.04%.
Standard error of the mean: 0.736.
Average hemoglobin of hookworm-negative cases: 82.79%.
Standard error of the mean: 1.352.
Odds against chance occurrence more than 15,000 to 1.

There is a significant difference between the average hemoglobins of the hookworm-positive group and the control group, but this difference does not in itself indicate that any great amount of injury has been caused to the individuals of the infected group taken as a whole. On the other hand, by subdividing the two groups according to the hemoglobin readings, it will be noted that few parasitized individuals have as high hemoglobins as do the uninfected controls, and further, that a rather surprising proportion of men in the former group show a well-defined anemia. This measure of the amount of injury caused by the parasite indicates that few supposedly healthy individuals escape some degree of harm, and with the proportion that have the more severe grades of anemia, it is evident that therapeutic measures should be considered.

COMMENT

MILITARY ASPECTS.—The opinion that hookworm infections in naval recruits are not of serious consequence, is one which has been well supported. The basis for this conclusion, therefore, should be examined in the light of the findings in the present investigation.

Smillie and Augustine [5] and others, have stated that persons harboring only a few worms are healthy carriers and not appreciably harmed by the presence of their parasites. This view especially pertains to naval recruits, for it would seem that the rigorous physical requirements for entry into the service would eliminate all but lightly infected, healthy carriers who would not require treatment. Moreover, there is no danger of dissemination in naval environment, and it is maintained that eventually the infected individuals will spontaneously lose their worm burdens without the necessity of treatment. Again, the earlier reports had indicated a progressive decrease in the prevalence of hookworm infections in recruits, findings which were in agreement with a report of the International Health Board of the Rockefeller Foundation [6] which as early as 1927, had stated that hookworm disease had almost disappeared from the United States. All of these considerations substantiated the view that necatoriasis among recruits might quite properly be ignored.

This conclusion, however, does not seem tenable with the demonstration of a 21 percent incidence of hookworm in naval recruits, and together with the further evidence of an injurious effect of the worm burdens, as shown by the anemia in parasitized individuals, these are facts which in themselves indicate the advisability of control measures.

Furthermore, it does not appear that the spontaneous loss of worms proceeds rapidly enough to justify witholding treatment on this basis. In another survey of intestinal parasites in Navy men,[7] numerous cases of hookworm were found in sailors who had been in the Navy for periods varying from 1 to 16 years. It does not appear that these infections had been acquired in naval environment, but rather, had persisted since their residence in endemic centers of the United States, as necatoriasis was encountered solely in men whose homes had been in the Southern States. This observation on the longevity of hookworm infections is supported by such experimental work as that of Caldwell and Caldwell [8] who report a case in which there was no apparent loss of worm burden during a 4-year period of observation, and again, that of Kendrick [9] who infected volunteers with *N. americanus* and detected ova during 5 years of observation.

Resistance.—Another phase of the problem which is too often ignored is that of host resistance; yet, from the standpoint of the parasitized individual's health, its consideration appears to be of great practical importance. Little is known of the extent of the operation of this factor, but experimental work indicates that the

[5] Smillie, W. G. and Augustine, D. L., J. A. M. A. 85: 1958, Dec. 1925.
[6] International Health Board of Rockefeller Foundation, Thirteenth Annual Report, pp. 5–6, 1927.
[7] Sapero, J. J., and Johnson, C. M.: Nav. Med. Bul., 37: 279, April 1939.
[8] Caldwell, F. C., and Caldwell, E. L.: Jour. Parasit., 17: 209, June 1931.
[9] Kendrick, J. F.: Amer. Jour. Trop. Med., 14: 363, Sept. 1934.

effect of host resistance is to modify the seriousness of hookworm in either direction, a heavy infection remaining latent in the host where resistance is high, and a light infection further debilitating the host where resistance is low. Foster,[10] working with dogs infected with *Ancylostoma caninum*, found that as a result of any factor causing the host to become anemic or poor in general health, the injurious effects of the hookworms were augmented. This concept applied to humans is exceedingly important, indicating that the injurious effects manifested in the host at any one time are but one phase of a changing disease process which may become aggravated or improved as the resistance varies. Such seems to be the explanation of illnesses due to hookworms in patients who have carried their worm burdens for some years. An evaluation of the importance of an infection at a time when an individual is otherwise in excellent health, thus gives a false impression of the harmlessness of necatoriasis.

On the other hand, hookworms appear to predispose to other sicknesses. In a recent report by Reed [11] it was noted that 12 out of 14 recognized authorities in tropical medicine believed that in the presence of hookworm disease, susceptibility to other disease is increased. Striking evidence to confirm these opinions may be found in a study by Kofoid and Tucker.[12] In studying the relationship of hookworm infection to morbidity in some 20,000 men in an army camp, they found in the hookworm group as compared to non-parasitized soldiers, 27.9 percent more men sick, 88.6 percent more men at sick call, and 76.6 percent more hospital admissions. This statistical relationship is a clear-cut, impressive demonstration of the importance of resistance in necator infections.

INDICATIONS AND METHODS FOR CONTROL.—In summary of all factors, the high prevalence of the parasite, the presence of an anemia in those harboring *N. americanus*, the indications that the rate of spontaneous loss of worms is prolonged, and the importance of the resistance factor to the health of the parasitized men, there seems little justification for the withholding of routine preventive measures which can hardly fail to increase the mental and physical efficiency of these southern recruits.

The method of control should be simple, not adding disproportionately to the duties of the medical department. When a new platoon of recruits is formed, men who have lived for any considerable time in the known endemic areas should be segregated for instructions concerning the collection of fecal specimens. Each man may be provided with a tongue blade and ointment tin. The stool may be passed as usual directly into the toilet, and a small portion of the

[10] Foster, A. O.: Amer. Jour. Hyg., 22: 65, July 1935.

[11] Reed, A. C., "Ultimate prognosis of hookworm disease, malaria and amebiasis." Medical Section, American Life Convention, 27th Annual Meeting, 1937, pp. 176–205.

[12] Kofoid, C. A. and Tucker, J. P.: Amer. Jour. Hyg., 1: 79, Jan. 1921.

specimen placed in the tin which has been labeled with the man's name, platoon, and State. The tins are sent to the laboratory where a single examination by the Willis brine flotation method will reveal to an efficient degree the cases of necatoriasis. The use of tetrachlorethylene, a single treatment in routine cases, would appear to be the treatment of choice and may be given ambulatory since the men in detention are already under close medical supervision. The employment of this newer drug obviates the objection to treatment by the older, more toxic remedies. Lambert [13] reports 46,000 treatments with tetrachlorethylene with no deaths and without untoward symptoms. Checking the stools after treatment, while desirable, would not be essential in the routine case.

STATUS OF HOOKWORM IN THE SOUTH.—Some interpretations of the naval data are also of interest from a general viewpoint. Thus, there has been a tendency on the part of many to regard necatoriasis as no longer a public health problem due to the effectiveness of control measures instituted in the past years. The high incidence of hookworm recorded for naval recruits is, however, an indication of the present widespread occurrence of hookworm in the South. Considering also the presence of minor hookworm disease in the naval group, these facts postulate a serious situation as regards hookworm incidence and disease in the general civilian groups of the southern United States. One may well question the statement of those who maintain that hookworm disease is no longer a problem in the United States.

RACIAL DIFFERENCES.—It is well known that hookworm is less prevalent in the colored race, Keller *et al.*[14] reporting the infection to be 12 times as infrequent. There was, however, an entire absence of *N. americanus* in 122 colored recruits examined in this study, in contrast to the 21 percent incidence in white men of the same age group, like physical status, and presumably representing a similar economic level. It is therefore difficult to explain this discrepancy as due to differences in exposure or habit. Rather, there appears to be in negroes a high degree of true racial immunity which, however, seems to be maintained absolutely only in the absence of disease or lowered vitality. This view agrees well with the previously quoted experimental work of Foster,[10] who in this regard, notes that the general condition of health of the animals appeared to be the regulating factor in their resistance both to the entry, and the establishment of hookworms within the host.

[13] Lambert, S. M.: J. A. M. A., 100: 247, Jan. 1933.
[14] Keller, A. E., Leathers, W. S. and Ricks, H. C.: Amer. Jour. Hyg., 19: 629, May 1934.

ABSTRACT SUMMARY

A current incidence of 21.0 percent hookworm infections in southern naval recruits is reported. Hemoglobin determinations showed a significant anemia in a considerable proportion of the positive cases. It was concluded that the reinstitution of preventive measures is clearly indicated. Adding support to this decision, are other considerations which indicate that even in such apparently healthy individuals as these naval recruits, hookworm infections tend to decrease resistance to other illnesses; conversely, that the injurious effects of the parasitism are augmented when the host becomes debilitated. Finally, it was observed that the spontaneous loss of worm burden in naval environment appears to be too prolonged to substitute this natural process for treatment.

Of more general interest is the observation that although there is a tendency to regard the hookworm problem as no longer of major public health importance in the southeastern United States, the naval data indicate that the contrary is so. Lastly, a suggested explanation for racial immunity, as observed in colored recruits, is given.

ACKNOWLEDGMENTS

This work was initiated and made possible through the interest and coöperation of Captain John B. Kaufman, Medical Corps, United States Navy, senior medical officer, Naval Operating Base, Norfolk, Virginia. Appreciation is expressed for the interest and many helpful ideas of A. O. Foster, helminthologist to the Gorgas Memorial Laboratory, Panama, R. P.

FOOD POISONING

U. S. S. RICHMOND

A sudden outbreak of food poisoning occurred aboard the U. S. S. *Richmond* on April 27, 1939, while off Cristobal, C. Z., just prior to transit of the Canal. Two hundred and thirty-one men of the 423 in the general mess were affected to a greater or less extent. No mess other than the general mess was affected. The onset of symptoms was about 2½ hours after the noon meal. Symptoms were marked in about half of those made ill and consisted of a sudden attack of abdominal cramps, nausea, vomiting, diarrhea, and prostration. Temperatures were normal or slightly elevated. There was slight leukocytosis and neutrophilia. The acute phase lasted from 1 to 6 hours and was followed by considerable prostration; however, all but four were ready for duty the next day. These were retained on the sick list until the following morning. Treatment consisted of saline cathartics, opiates for some, and all were given sodium chloride with their drinking water the next day.

The noon meal consisted of vegetable soup, boiled ham, boiled peeled potatoes, stewed corn, horseradish, apple and celery salad, nut spice cake, bread, butter, and lemonade. The boiled ham was served cold and was immediately suspected as the cause of the outbreak due to a history of faulty handling in its preparation. No bacteriological study could be made of the suspected food, however, because all left overs from the meal had been destroyed.

The history of the preparation of the ham was as follows: Government inspected, cured, frozen hams were taken from the ice box about 11 a. m. April 26, 1939, the day before serving, and allowed to thaw until 5:30 p. m. when they were boiled until midnight. They were then boned and sliced. This work was completed about 2 a. m. From 2 a. m. until it was served at the noon meal the ham remained in covered pans at room temperature in very hot weather.

All of the ill men had eaten of the ham. Men who did not eat the ham were not ill. There would seem to be no doubt that the ham was contaminated by the cooks during the process of boning and slicing and the organism was then allowed to incubate for 10 hours at very warm room temperatures. The galley was free of flies, roaches, and rodents. No peculiarity in appearance, taste, or odor of the food was noticed and since symptoms appeared within 2 to 3 hours after ingestion, all of which are characteristics of staphylococcic food poisoning, it is believed the staphylococcus was the causative organism.

In questioning the cooks, even those who had graduated from service cooking schools, it was found that they were completely ignorant of the fundamental causes of food poisoning. Outbreaks of food poisoning are of frequent occurrence in the service and routine inspections by the inspecting officers can not always detect or prevent it since the appearance, taste, and odor of the poisonous food may be normal. Its prevention is usually dependent upon the galley personnel understanding why and under what conditions food becomes unsafe. To understand this they will have to be taught the rudiments of very elementary bacteriology by the medical officer. In various parts of ships safety instructions are posted for the handling of guns, magazines, motor boats, etc., and the medical officer of the U. S. S. *Richmond* suggests that some such safety rules for prevention of food poisoning as the following should be promulgated for posting in food issuing rooms, galleys, and pantries.

SAFETY RULES FOR PREVENTION OF FOOD POISONING

Severe illness will occur unless the utmost care is exercised in the handling and preparation of food.

The following precautions shall be rigidly adhered to:

1. It must be presumed that all food is contaminated with bacteria while being handled during its preparation. The growth of these bacteria to a dangerous

degree must be prevented by keeping the food **too hot** or **too cold** for their multiplication prior to serving either by continued cooking or by placing in a refrigerator immediately following the cooking.

2. Some spoiled food can be detected by the smell and appearance and must be discarded. Other contaminated foods are not changed in appearance, taste, or odor. Therefore the proper preparation and handling of food is of the utmost importance.

3. Whenever possible frozen meats and poultry shall be thawed in the chill box. If meat is thawed at room temperature the time between thawing, cutting, and cooking must be reduced to a minimum.

4. Warm ground or cut up meats when stored in the chill box must be in shallow pans and be not of greater depth than 6 inches to insure quick cooling. Otherwise warmth in the center of the mass will provide for bacterial growth.

5. If boiled ham is to be served, it must be first boned, then cooked, sliced, and served while hot. If cold ham is served, it must be placed in the chill box immediately after boiling or baking and allowed to remain until just time to slice before serving.

6. Hash shall not be served when it would be necessary to prepare the ingredients the night before, or even several hours before the hash is to be cooked and served.

7. The following foods are especially apt to produce bacterial growth if improperly handled: Meat, meat products, gravies, milk, custards, fish, shell fish and any other food having a high protein content.

8. A dangerous form of spoilage is caused by the *Bacillus botulinus* and is known as botulism. Botulism particularly affects uncooked or precooked canned foods, such as olives, sausage, fruits, vegetables (particularly spinach), and fish. This develops in foods insufficiently processed at the time of packing. Cooking destroys the poison produced by this germ. All canned vegetables that are to be served in the form of salads shall be removed from the cans and cooked thoroughly prior to serving. Food contaminated by this germ possesses a characteristic and offensive odor, similar to the odor of rancid butter, but the odor may be barely discernible. No canned food should be tasted before cooking unless the odor and appearance are normal.

9. Any can, containing food, which shows nail holes or is bulged shall be discarded. Contents of cans dented or damaged in handling frequently spoil due to slight perforations of the cans which cannot be detected by the eye.

10. The galleys and pantries shall be kept free from rats, mice, flies, roaches, and dirt, and food shall be properly covered to prevent possible contamination from same. All scraps and waste matter shall be disposed of as rapidly as possible in order to prevent the attraction of flies.

11. Food handlers must keep themselves, their clothing, and utensils scrupulously clean. The stowing of or washing of clothes in the galleys, bakeshop, and pantries is forbidden.

12. Food handlers showing evidence of skin or intestinal diseases shall report to the medical officer.

13. Avoid handling of food with hands whenever possible.

14. The stowing of sandwiches and other food in lockers by individuals for later consumption is forbidden.

15. Food from ashore for the officer and chief petty officer messes must be inspected by the medical officer in the same manner as for the general mess.

16. The chief commissary steward, the senior cook, senior baker, officers' cooks, and stewards shall be responsible that personnel under them comply with these safety rules.

STATISTICS

HEALTH OF THE NAVY

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of April, May, and June 1939.

ENTIRE NAVY

	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1934	385	67	452	35	116	64
1935	371	67	438	28	85	62
1936	337	49	386	30	140	42
1937	276	36	313	18	98	59
1938	331	50	382	9	79	78
1939	339	48	387	7	85	90
FORCES ASHORE						
1934	621	88	709	76	230	58
1935	491	80	571	54	110	45
1936	518	50	568	59	226	26
1937	312	36	347	34	131	27
1938	364	51	415	15	105	45
1939	332	48	380	12	104	41
FORCES AFLOAT						
1934	271	57	328	15	62	67
1935	312	60	372	16	72	71
1936	229	49	278	13	89	51
1937	256	37	293	8	78	78
1938	313	50	363	5	64	96
1939	343	47	391	4	75	117

Common infectious diseases of the respiratory type.—A total of 3,155 admissions for these diseases were reported for the second quarter of the year 1939, or a 46.9 percent decrease from the number of cases notified for the preceding quarter. Catarrhal fever was responsible for 2,290 of the total admissions for these diseases.

There were 1,829 admissions for these diseases reported by forces afloat, 1,183 from shore stations in the United States, and 143 from

outlying naval stations and activities. The largest number of cases were reported from the following ships and stations:

Ship or station	April	May	June	Total
Naval training station, Norfolk, Va	127	63	32	222
U. S. S. *Yorktown*	23	89	3	115
Naval training station, San Diego, Calif	41	38	27	106
Naval training station, Newport, R. I	49	39	17	105
Fleet air detachment, San Diego, Calif	45	40	12	97
Marine Corps base, San Diego, Calif	54	25	12	91
Naval Academy (midshipmen)	49	33	2	84
Marine Barracks, Quantico, Va	37	31	13	81
Naval training station, Great Lakes, Ill	34	26	14	74
U. S. S. *Arizona*	52	8	7	67
U. S. S. *Pennsylvania*	15	38	10	63

Two cases of scarlet fever were reported in May—one from the Naval training station, Great Lakes, Ill., and one from the U. S. S. *Trenton.*

Headquarters, 11th Naval District, San Diego, Calif., reported one case of diphtheria in May.

The Marine Barracks, Quantico, Va., reported 17 cases of mumps in April, 3 in May, and 4 in June.

Chickenpox.—Seventeen cases of chickenpox were reported for the quarter as follows:

Ship or station	April	May	June	Total
U. S. S. *Henderson*	0	1	2	3
Navy yard, Cavite, P. I	2	0	0	2
U. S. S. *Beaver*	1	0	0	1
U. S. S. *Case*	0	1	0	1
U. S. S. *Dobbin*	0	0	1	1
U. S. S. *Melville*	0	0	1	1
U. S. S. *Nashville*	0	0	1	1
U. S. S. *Saratoga*	0	1	0	1
Naval Academy (midshipmen)	1	0	0	1
Naval Academy (others)	1	0	0	1
Naval air station, Norfolk, Va	0	1	0	1
Receiving ship, San Diego, Calif	1	0	0	1
Navy yard, Pearl Harbor, T. H	0	1	0	1
Navy yard, Washington, D. C	0	1	0	1

The senior medical officer of the naval station, Tutuila, Samoa, reported as follows in the sanitary report for the month of June:

Station personnel have suffered from the extensive epidemic of influenza which began on the island June 17th. As was to be expected, the incidence of cases was much higher among the native personnel than the whites, there being 30 cases among the Fita Fitas and only 6 cases among the whites. There were no complications or deaths.

MORBIDITY

Summary for the quarter ending June 30, 1939

Average strength	Forces afloat, 92,078		Forces ashore, 49,516		Entire Navy, 141,594	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	8, 996	390. 80	4, 701	379. 76	13, 697	386. 94
Diseases only	7, 904	343. 36	4, 107	331. 77	12, 011	339. 31
Injuries and poisonings	1, 092	47. 44	594	47. 98	1, 686	47. 63
Communicable diseases (Class VIII):						
A	100	4. 34	143	11. 55	243	6. 86
B	1, 729	75. 11	1, 287	103. 97	3, 016	85. 20
Venereal diseases	2, 690	116. 86	503	40. 63	3, 193	90. 20

DEATHS

During the second quarter ending June 30, 1939

Cause		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		10, 246	2, 006	109, 680	1, 349	17, 874	439	141, 594
Disease								
Appendicitis, acute	Poisoning, anesthesia, ether (inhaled).			1				1
Edema, lung	None			1				1
Embolism, cerebral	do			1				1
Gastro-enteritis, acute	Dilataion, cardiac, acute			1				1
Hemorrhage, cerebral	None			1		1		2
Hodgkin's disease	do	1						1
Mastoiditis, acute	do			1				1
Nephritis, chronic	Thrombosis, coronary artery.			1				1
Nephritis, chronic	Hemorrhage, subarachnoid, brain.	1						1
Pneumonia, lobar	None			1				1
Do	Pleurisy, suppurative			1				1
Do	Ulcer, duodenum			1				1
Pleurisy, suppurative	Abscess, brain			2				2
Septic sore throat	Septicemia					1		1
Status lymphaticus	None			1				1
Thrombosis, coronary artery.	do			1				1
Do	Arteriosclerosis, general	1						1
Teratoma, testis	None			1				1
Tuberculosis, pulmonary, chronic.	do					1		1
Do	Hemorrhage, pulmonary			1				1
Do	Tuberculosis, spine			1				1
Tumor, malignant, mixed cell, parotid gland.	None			1				1
Tumor, malignant, mixed, teratoma, testicle.	do			1				1
Ulcer, stomach (perforated).	Peritonitis, general, acute			1				1
Do	Pneumonia, broncho-			1				1
Do	Raynaud's disease					1		1
Do	None			1				1
Valvular heart disease, aortic and mitral.	Myocarditis, chronic			1				1
Total for diseases		3		23		4		30

DEATHS—Continued

Cause		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or Contributory	Officers	Midshipmen	Men	Officers	Men		
Injuries and poisonings								
Burns, multiple	Fracture, compound, skull.	1						1
Crush, abdomen and pubis.	None			1				1
Drowning	....do	1		31		1		33
Do	Psychosis, unclassified			1				1
Electric shock	None			1				1
Fracture, compound, skull.	....do			5		1		6
Do	Meningitis, cerebral			1				1
Fracture, simple, frontal	Intracranial injury	1						1
Fracture, simple, skull	None			1				1
Do	Intracranial injury			2				2
Fracture, simple, temporal.	Hemorrhage, traumatic, intracranial.			1				1
Fracture, vertebra, cervical.	Pneumonia, broncho-			1				1
Do	Intraspinal injury			1				1
Injuries, multiple, extreme.	None	3		6	1			10
Do	Alcoholism, acute			1				1
Intracranial injury	None			2				2
Wound, gunshot, abdomen and chest.	Psychosis, unclsasified					1		1
Wound, gunshot, head	None					2		2
Do	Psychosis, unclassified					1		1
Poisoning, acute, potassium cyanide.	None			1				1
Poisoning, acute, strychnine.	....do			1				1
Total for injuries and poisonings.		6		57	1	6		70
Grand total		9		80	1	10		100
Annual death rate per 1,000:								
All causes		3.51		2.92	2.97	2.24		2.82
Disease only		1.17		.84		.90		.85
Drowning		.39		1.17		.22		.96
Poisonings				.07				.06
Other injuries		1.95		.84	2.97	1.12		.96

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for second quarter ending June 30, 1939

The following statistics were taken from sanitary reports submitted by naval training stations:

April, May, and June, 1939	Naval training station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period	1,265	676	700	1,151
Recruits appearing before Board of Medical Survey	13	0	3	(a)
Recruits recommended for discharge from the Service	13	0	3	(a)
Recruits discharged by reason of Medical Survey	12	0	4	(a)
Recruits held over pending further observation	4	0	(a)	(a)
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	0	8	(a)	18

[a] Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess, periapical	2	Flat foot	8
Absence, acquired, teeth	3	Focal infection, teeth	1
Amblyopia	1	Foreign body, left leg	1
Arthritis, chronic, left sacro-iliac joint	1	Gonococcus infection, urethra	3
Asthma	1	Hernia, inguinal, indirect	3
Cardiac arrhythmia, premature contractions.	1	Hypertension, arterial	1
		Myopia	1
Cardiospasm	1	Myositis, chronic	1
Caries, teeth	3	Nephritis, chronic	4
Chorioretinitis	1	Osteochondritis dissecans	1
Color blindness	3	Osteomyelitis	1
Constitutional psychopathic state, inadequate personality.	1	Otitis, mdia, chronic	5
		Psoriasis	1
Constitutional psychopathic state, sexual psychopathy	1	Psychoneurosis, hysteria	1
		Psychoneurosis, neurasthenia	1
Curvature, spine	2	Psychoneurosis, unclassified	1
Deafness, unilateral	3	Stuttering	1
Deformity, acquired	3	Syphilis	2
Deformity, congenital	4	Union of fracture, faulty	1
Dislocation, articular cartilage	3	Valvular heart disease (mitral insufficiency).	1
Effort syndrome	1		
Enuresis	2	Vertigo	1
Epilepsy	8		

VOLUME XXXVIII APRIL 1940 NUMBER 2

United States Naval Medical Bulletin

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

Issued Quarterly by the Bureau of Medicine and Surgery

Navy Department

Washington, D. C.

VOL. XXXVIII APRIL 1940 NO. 2

UNITED STATES NAVAL MEDICAL BULLETIN

FOR THE INFORMATION OF
THE MEDICAL DEPARTMENT OF THE NAVY

DIVISION OF PUBLICATIONS
THE BUREAU OF MEDICINE AND SURGERY

THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY

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

Compiled and published under the authority of Naval Appropriation
Act for fiscal year 1940, approved May 25, 1939

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON: 1940

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

NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary.

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

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Volume XXIV, 1926, Nos. 1 and 4.
Volume XXV, 1927, No. 1.
Volume XXVII, 1929, Nos. 3 and 4.
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Subscription should be sent to Superintendent of Documents, Government Printing Office, Washington, D. C.

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means 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 Medical Department personnel, and reports from various sources, notes, and comments on topics of professional 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 professional interest.

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

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

ROSS T. MCINTIRE,
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.

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EBEN E. SMITH, *Editor*,
Commander, Medical Corps, United States Navy.

U. S. NAVAL MEDICAL BULLETIN

VOL. XXXVIII APRIL 1940 No. 2

SPECIAL ARTICLES

THEY TOLD ME ABOUT THEIR HOSPITALS[1]

By Captain Lucius W. Johnson, Medical Corps, United States Navy

Our Navy is expanding rapidly, with new ships, new stations, and many additional thousands of men. This entails an increase of accommodations at many of our naval hospitals, and the construction of a number of new ones. For example, there is the new Naval Medical Center, in Washington, D. C., a $4,850,000 project which includes a 500-bed hospital, medical school, and dental school. Then there are dispensaries for new air bases in the islands of the Pacific, in Alaska, and in the Caribbean. These numerous structures, with varying climatic conditions, and of differing sizes, involved many problems to which nobody appeared to have satisfactory answers. So the Surgeon General of the Navy arranged for me to visit about 30 of the newest, largest, and best-managed hospitals in the United States and Canada, to study their construction, equipment, and administration.

Everywhere I met with the greatest courtesy. Hospital superintendents, doctors, nurses, dietitians, mechanics, orderlies, and employees of all sorts talked freely to me about their methods, their gadgets, their institutions, and their organizations.

From these hundreds of interviews, certain definite ideas have crystallized. They are set down here with the hope that they will stir up discussion and stimulate others to express their opinions. Not to arouse criticism, they say, is a reflection on a writer, so conflicting points of view will be welcomed.

FLOOR MATERIALS

The many hospital administrators with whom I talked were pretty generally agreed on the relative merits of various floor materials. I will set down what appears to be the consensus.

Concrete is the cheapest flooring for the ordinary building of modern construction. When steel troweled to a hard finish it is likely to

[1] This article was originally prepared for publication in *Modern Hospital* where it is being published serially, the first installment appearing in the March issue. The editor of *Modern Hospital* kindly consented to concurrent submission to this BULLETIN for publication of the entire article in this issue.

powder unless the finishing is done in just the right way at just the right time. Proper curing of the floor is also an important item. There is a large business organization which specializes in this work, and guarantees against cracking and powdering. Concrete is likely to be slippery when wet or greasy. It cracks, but the cracks can be repaired so that the surface is restored, though not the appearance. It stains readily and is difficult to keep looking well. For basements, storerooms, engine rooms, and areas which receive hard usage, it is quite satisfactory. Some hospitals are using for sick rooms various types of rubber-, mastic-, or cork-composition materials, cemented directly to concrete floors.

Painted concrete floors are seldom satisfactory. The trouble is that no paint has yet been prepared which will resist traffic and also adhere to a concrete surface. The paint soon wears away, where the traffic is heaviest, and the appearance quickly becomes very shabby.

The once-popular hardwood floors had many very definite virtues, but one sees very few of them being installed for ward use in modern hospital construction.

Quarry tile is highly satisfactory for floors of x-ray dark rooms, kitchens, cafeterias, and dining rooms. It is easily cleaned and has low absorption, but it is likely to be slippery when wet or greasy. The tile should not be brought too close to ranges or pressure cookers because the heat and grease have a bad effect on the cement in which they are set. Under steam kettles, also, the cement quickly erodes, loosening the tiles, unless care is taken to prevent dripping from the spouts.

Ceramic tile continues in wide use for bathrooms, operating rooms, sterilizer rooms, and other spaces where floors must be kept clean though frequently wet. Its principal competitor for such use is terrazzo, which averages somewhat lower in price. I saw several private rooms done in colored patterns of tiles as part of the general decorative scheme. They were attractive enough to give the impression that hospital architects have been missing a trick by not using more colored tiles. Tiles with a high abrasive content are used to prevent slipping in shower baths, dressing rooms, and other areas where people may walk with wet or soapy bare feet. This same material is becoming widely used in stair nosings to prevent slipping.

Terrazzo continues to increase in favor. Many believe it to be the best flooring for lobbies, operating suites, wards, and private rooms. It is important to specify the grade of marble chips to be used in the mix, for the cheaper grades powder with use, and the floor soon acquires a pitted appearance. For operating rooms, some consider that it is likely to give off more dust than other materials, such as marble or ceramic tile.

It has certain very definite defects. It stains with oil, tobacco juice, iodine, and other agents. It is prone to crack unless skillfully laid on a solid foundation, with dividing strips, and then carefully cured. Acids and strong alkalies affect the surface, and nothing but neutral soap solution should be used as a cleanser.

In spite of its shortcomings, it is very highly regarded. Hospital wards having terrazzo floors where the beds rest, and a center strip of some resilient material appear to be today's best bet.

Travertine is seen occasionally in floors. It is a rather soft stone, with high decorative value in walls. When used for flooring, the irregularities which give it beauty, wear unevenly. Holes large enough to catch the spike heels of ladies' shoes soon develop, with danger of falls and lawsuits. Efforts to repair the holes with plaster or cement give a shabby appearance.

Linoleum is very popular, and reasonable in price. It should be laid by an expert, on a smooth surface, with suitable lining and adhesive. I saw several floors of this material, in use 10 to 15 years, and still in excellent condition. This was true not only of battleship linoleum, but also of the cheaper grades.

Careful housekeeping is necessary for such a result. In some hospitals they wax the linoleum every 6 months. Some do it oftener, cleaning between times with a dry hair brush. At least two preparations are now on the market which, applied every 6 months, do away with all necessity for waxing. Certainly, it would be a great boon to staff and patients alike if all floor polishers could be banished from hospital wards and sick rooms.

Linoleum in the form of tiles received more general condemnation than any other material, except cork tiles. Presumably made of the same material as sheet linoleum, it does not appear to give the same satisfaction to the user. They say that it scratches, cracks and pits badly; that it comes unstuck, the corners curl and get broken. It has the advantage that a defective, broken, or stained tile can be removed and replaced without making an unsightly patch.

Cork-tile floors were seen in several libraries, but nobody spoke well of them, except on the one point, that of quiet. The squares appear to curl, break, chip, pit deeply, and are difficult to keep looking well. One institution was about to replace the cork-tile floor in its library after only 1 year of use.

Asphalt or mastic tile is widely used and is very satisfactory if its limitations are recognized. I saw a number of floors of this material which had been in use for 10 years or more that looked very well. Others, in less than half of that time were very badly pitted and scarred. Asphalt tiles will pit where heavy furniture rests with small bearing surfaces. It is generally accepted that the

dark colors stand up better, because the asphalt content is greater, and these are also less expensive. The light colors show foot marks less, but are more easily stained, and do not wear so well. Asphalt tile floors appear to wear better in cold climates than in the warmer areas. A grease-proof asphalt tile is marketed, but this is said to be softer and to pit more easily. The cost of asphalt tile is moderate, not far from that of the best grades of linoleum.

Rubber-tile floors appear to be the best of the various composition floorings. If one is in a position to balance the high cost of installation against the low cost of maintenance, they will be most satisfactory. I saw several that had been in use for 12 to 15 years and appeared to be in perfect condition. They were criticized for use in laboratories, because ether, chloroform, acetone, and other reagents dissolve the cement which holds them in place. In operating suites they are said to be very slippery when wet, or when soap is splashed on them in the scrubroom. They should not be used in plaster rooms because it is very difficult to remove plaster from them. Aside from these minor points, I heard no criticism, and there was much praise because of the ease of maintenance. They do not pit so badly as the asphalt tiles.

All types of composition tile are subject to the common defect that moisture and certain reagents tend to loosen them from the adhesive cement. So their use in laboratories, sterilizer rooms, and washrooms is a matter of careful consideration. But they have the advantage that tiles are easily replaced when that becomes necessary.

A great deal can be done to prevent marring of floors. All furniture should be fitted with glides not less than 1 inch in diameter, or with wide casters which will give an ample bearing surface. Desks and heavy tables should have glides of a size proportionate to the weight they bear.

Floor drains in operating rooms, dressing rooms, kitchens, and ice boxes are going out of fashion. In many communities they are forbidden by building codes, unless provided with a constant flow of water which will maintain the water level in the traps. Washing down the operating room with a hose is no longer a sacred part of the ritual, as it used to be.

AIR CONDITIONING

When I discussed this feature I found on all sides a strong impression that one should go slowly in making commitments because great changes in methods and equipment are impending which will revolutionize the whole industry. A considerable number of hospitals now

have air-conditioning equipment and do not use it. Their reasons for abandoning it were, in order of frequency:

1. High cost of operation.
2. Technical difficulties—too much trouble to keep in repair.
3. Capacity of machines too small.

I did not find anybody, even in the South, who thought that a completely air-conditioned hospital was desirable. All agreed that operating rooms, delivery rooms, nurseries, and small wards for special medical cases should have this equipment. In one hospital which had the executive offices air conditioned, the director said he would prefer not to have it, as it caused too many respiratory-tract infections.

A large institution should be divided into several independent units, for in some places recirculation of air is permissible, in others not. Operating rooms should not have recirculation if explosive gases are used, for they may build up to dangerous concentration. When bad odors or poisonous fumes are produced, there should be no recirculation. At one institution where there is a single system with recirculation, the visitor on entering is immediately assailed with a nauseating odor of animal excreta, which pervades the entire place. Individual portable machines are very satisfactory for offices or sickrooms where they are desired, and they are being rapidly improved. Their cost, when used in the sickroom, can be added to the patient's bill.

In one hospital an installation costing $50,000 was not being used, as their mechanical force could not keep it running. In another, they were spending several thousand dollars to have the faults of their equipment corrected. One operating room had the inlet and outlet louvers close together on the same wall, so that there was no circulation of air. Another room had openings so small that there was a strong cold draft between them. A midwest hospital had air conditioning of the operating room, but not of the corridor of the operating-room suite, which was hard on the personnel and overworked the machinery. In another hospital I saw a small cooling unit sparking away in an operating room where explosive gases were being used. No explosion had occurred—yet.

Several complaints were heard of chronic colds, sinusitis, and neuritis being greatly increased after air conditioning was installed. A differential of 10° to 12° in temperature appears to be the most that is comfortably endured by persons who must pass in and out of an air-conditioned area.

Control of moisture will usually be found to be the most important detail affecting comfort. It is also the most costly item in air conditioning. Where an abundant supply of water from wells at a low temperature is available, its use will greatly reduce the cost of air

conditioning. In several municipalities restrictions are being placed on the use of water for air-conditioning plants.

The whole field of air conditioning has developed so rapidly, and is changing so frequently, that the faults and errors which are so evident today will soon be corrected. Then, it will be regarded as one of the most valuable of modern improvements in hospitals.

BATHING FACILITIES

Bathtubs are being supplanted by the shower bath. In some departments, notably the obstetric and neuropsychiatric, tubs are regarded as actually dangerous.

What facilities should be provided for nurses and female patients? One chief nurse covered the subject pretty well when she said, "The youngsters who play tennis and such games want showers, but we old gals who get most of our exercise at the bridge tables want to soak in a tub." When tubs are installed, their usefulness will be greatly increased if a shower is placed over each tub. If designed for the use of women, the shower head should be about 5 feet above the floor, so that they can avoid wetting the hair. The best ratio of tubs to showers appears to be about 1 to 4.

Tubs provided for the use of ward patients appear to be seldom used, so seldom that in some cases the rooms had been converted to storerooms. Showers are more popular and will be used more.

VACUUM AND PRESSURE OUTLETS

Air pressure and suction are an important adjunct to many surgical operations. The danger of explosion of anesthetic gases makes it desirable to have the machinery in another room, with outlets near the operating table for attachment of tubes. There appears to be widespread difference of opinion concerning the best location for the outlets.

Some have been placed in the floor beneath the operating table. The weight of the modern table with the patient, may approximate a thousand pounds. This weight may be enough to break or bend the covering of the outlet so that it is no longer waterproof, and it may provide another obstruction to stumble over. Others have suspended the outlets over the table, but there is a growing prejudice against unsterile gadgets hanging above the sterile operative field.

Most hospitals are now installing the outlets in neat metal cabinets on the side wall, a foot or more above the floor. This involves a line of tubing across the floor, but appears to be the best arrangement until somebody thinks of a better one.

ACOUSTIC MATERIALS

Sound control in hospitals has come to stay, but it has developed so rapidly, and there are so many unknown factors concerning its application that kinks are constantly developing. While it is true that efficiency is often in proportion to price, there are many other factors that should be considered, such as resistance to fire, light reflection, appearance, method of mounting, distribution of the material, reverberation time, also the effect of cleaning and painting.

Sound absorption should not be too complete. Ordinary noises of daily life may better be allowed to continue to some extent. Otherwise patients listen for them, with much tension, like waiting for that other shoe to drop, and so they do not relax.

The use of acoustic materials is desirable in corridors, wards, sick rooms, consulting and examining rooms, baths and toilets, utility rooms, operating suites, and wherever dishes and utensils are handled.

Opinions of the value of acoustical plaster varied greatly, some stating that it was entirely useless, while others found it satisfactory. This is quite understandable, because so much depends on the method of its application, and it is difficult to get the average plasterer to apply it correctly. The directions of the manufacturer should be strictly followed, for the surface finish and the time and method of applying the several coats greatly affects its efficiency. Some of these plasters can be cleaned and painted, others cannot.

There is a very satisfactory preparation consisting of asbestos fibres which are sprayed on a prepared surface together with an adhesive material. Its efficiency increases with the thickness of the layer. It can be spray painted, and is attractive in appearance as well as efficient. Until recently it has been higher in price than others but is said to be considerably reduced in cost. The method of application is said to be such an important factor that one should always have it applied by the company owning the American rights, rather than by a local contractor.

Acoustic material sold in the form of tiles, sheets or large blankets may be divided into cellulose (from sugar cane or other vegetable matter), wood fibre, asbestos, and other minerals. Methods of suspension are by cementing, nailing, and various mechanical devices. Application by an adhesive material, such as glue or cement, was formerly quite common, especially in hospitals already built, but it is being used less and less. I saw some dreadful examples of this method. Tiles are liable to come loose, especially if there is much heat or steam in the place. There have been some serious injuries and many narrow escapes from falling acoustical tiles. In a number of hospitals I saw tiles that had fallen and had been nailed back in place. The nails had rusted badly and produced a very shabby appearance.

Mechanical suspension is much safer, and nearly every important manufacturer will supply special devices for suspending his materials. Nearly all of these require a void above the acoustic tiles. This space may increase the sound absorption, but it also may make a snug harbor for rats, mice and other vermin. I saw a number of places where the excreta of such animals had soaked through the tiles, producing conspicous stains which could not be removed. In spite of this minor fault, mechanical suspension is better.

There are a number of acoustical tiles on the market which are made of bagasse, or sugarcane waste. They are attractive in appearance, efficient, and reasonable in cost. One type comes with small holes regularly spaced over the surface. In kitchens and dining rooms it has happened more than once that roaches moved into the small holes, finding them just the right size for breeding purposes, and the slight residue of sugar helped to make a happy home. The cellulose materials should not be used in rooms where grease and steam may soak into them. They are very satisfactory in wards, corridors, toilets, bedrooms, and auditoria.

Acoustic tiles of asbestos are usually more costly than those made of cellulose, since it is largely an imported material. They are more resistant to fire, and some of them can be painted and cleaned. A material that is now becoming very popular is made of powdered stone with a binder. The price has recently been reduced so that it competes with other moderately priced materials.

Another type has a perforated metal case containing a muslin bag which encloses the sound-absorbing material. Some of these are as high as 80 percent in efficiency, and costly in proportion. They can be both cleaned and painted, and are quite attractive in appearance. Their one defect appears to be that rats and insects find the absorbent material perfect for building nests, and their excreta may produce unsightly stains. I saw a number of places where such tiles were badly rusted and discolored.

Most of the large manufacturers have acoustic materials in all price grades. Bulletins issued by the United States Bureau of Standards, and the Acoustic Materials Association, give the coefficient of sound absorption, the degree of fire resistance, and the effect of painting on the efficiency. Some materials can be painted and cleaned, others cannot. This is an important detail to consider. For example, the greasy finger marks of mechanics around access panels are very conspicuous. Rapid progress is being made in developing the decorative value of acoustic materials. Some very pleasing colors and designs are now available, which in no way suggest, as the earlier materials did, a mechanical feature which it was unfortunately necessary to have exposed.

Another use of sound-absorbent material is found in its application to the under sides of metal desks, tables, trucks and other noise-producing furniture.

LIGHTING

Doctors are frequently mentioned as being outstanding individualists, with no liking for standardization. Their attitude toward lighting would support this idea, for their opinions were marked by a wide diversity.

All agreed that there should be no bright points of direct or reflected light to attract and dazzle the eyes of the patient. Overhead lights need an opaque reflector, designed to protect a patient lying in bed from glare.

Indirect lighting of the room or ward, with a reading light for each patient, is regarded as best. Luminous tubes are coming into more general use. They provide a large area of illumination, of low intensity, use little current and produce very little heat. There is no doubt that satisfactory fixtures will soon be developed to adapt them for use in hospital wards and rooms.

Recessed lighting is increasing in use, and several attractive types of fixtures are on the market, designed to be set flush in walls or ceilings.

Night lights, set below the level of the bed, are generally approved. Several persons suggested that they be placed beneath the lavatory. Some night lights which were otherwise very satisfactory, were activated by switches noisy enough to wake the soundest sleeper. Listening to them convinced me that switches for night lights should be of the silent type. A wakeful patient will then be saved the annoyance of listening to the loud click of the switch.

The huge domelike illuminators over operating tables are not generally liked. Nurses complain that they cannot be properly cleaned Doctors fear that dust from inaccessible crevices will sift down on the operative field as the light is adjusted. Others find it simpler to move the table, patient, and anesthetist into focus, than to adjust the light itself.

I saw some very attractive installations of lights set flush in the ceiling, with focusing lenses. They leave a space above the table entirely clear of dust catchers, are easily cleaned, and give superb illumination on the operative field. The disadvantages are that they cost considerably more to install, and use more electric current.

The idea of an emergency unit to take over the lighting when the usual source fails has been thoroughly sold to the hospital world. Originally this service was largely limited to the operating suite, but it is now recognized that other areas are equally important when the

lights go out. The admitting office, accident room, stair wells and panic exits are now being included in the emergency circuit.

INTRAVENOUS FLUIDS

Many of the larger hospitals prepare their own solutions, and claim savings running to many thousands of dollars per year. On the other hand, one large clinic, which has made its own solutions for many years, is now changing to the use of commercial fluids. This is due to the fact that a State law has recently been passed which regulates very stringently the preparation and sale of drugs. It was considered simpler to buy the fluids ready made than to make and dispense them under the new law. Small hospitals usually find it better to purchase their intravenous solutions as needed.

There is a system on the market by which the distilled water is mixed with the sodium chloride, dextrose, or other agents and bottled ready for the autoclave, without being exposed to the air or to handling. Hospitals of 300 beds or more find it most satisfactory but for smaller institutions its cost might be prohibitive.

Preparation of these fluids is usually placed in the hands of the pharmacist or a graduate nurse. At one hospital they claimed to have reduced the cost per liter from 70 cents, which they formerly paid for the commercial preparation, to 8 cents. Others estimated that their home-made solutions cost as much as 23 cents per liter.

The time required to prepare the solutions, using the closed system, averaged about 4 hours for 200 flasks. Reactions were said to be very rare, and one enthusiast claimed over 30,000 infusions without a reaction, a record seemingly beyond the bounds of human achievement.

Stills used in preparation of the solutions were of all types and makes. Most hospitals, however, used a single-effect still. The belief is pretty general that, if cleaned daily, it is just as good as the double-effect or triple-effect type. Daily cleaning is so important that a still should be chosen that can be quickly and easily cleaned, and it should be conveniently located for this purpose. Also, it is essential that all distillate that comes over during the first 15 minutes after starting the operation of the still be thrown out, as well as all that comes over after the heat is turned off. Distillates come over at these periods of lower heat that may not be sterile, and may contain pyogenic material. Most of those with whom I talked believed that the use of chemically-pure chemicals in mixing the solutions is of very great importance in avoiding dangerous effects.

In some hospitals the flasks are dated and, if not used within 2 weeks, put through the autoclave again. Others regard them as

good for 2 months, while many pay no attention to age, believing them good until used.

Washing of flasks and rubber tubes is regarded as of very great importance. At one institution the flasks are washed by hand, 7 times in tap water and 7 times in distilled water, and drained after each washing. At another hospital the pharmacist has made a very simple and ingenious device, by which 12 flasks are washed at one time.

The flasks are autoclaved after being filled. Some screw the caps down before they are placed in the sterilizer. This requires very careful management to avoid breakage from differences of pressure within and without the flasks. Also, if the temperature is not completely reduced before the autoclave is opened, the flasks may explode and endanger the workers. Others sterilize the flasks with the tops open, and then, with sterile forceps, cap or cork them and apply a sterile paper cover.

It is customary to issue the flasks of solution with the sterile needles, tubes, and fittings from the central issue room. Some affix the tube to the flask before issue, so that one has only to remove the sterile wrapper, and it is ready for use. Others issue the fittings separately, to be attached just before use. Most hospitals make it a routine to sharpen the intravenous needles every time they are used, which is truly an act of mercy toward the patient.

A careful accounting and follow-up system is needed to insure the prompt return of flasks and fittings to the issue room. Otherwise they drift away to wards and rooms, where they are useful for other purposes. Their cost is sufficient to make this an important item.

OPERATING-ROOM EXPLOSIONS

Operating-room explosions are tremendously impressive affairs which hang in the memory of the community for years. Enough of them have happened in recent years so that the lay public is demanding assurance of safety from this danger. The point of view expressed at different clinics, hospitals, and medical schools with regard to explosions shows wide variance of opinion. At one large clinic the thought was expressed that cyclopropane represented the perfect inhalation anesthetic, except for the one characteristic—explosiveness, and, because of this one feature, they were abandoning its use. At another large hospital the anesthetist said that he saved so many lives by the use of cyclopropane that its dangers were far outweighed by its advantages. At most institutions they were taking all known precautions and hoping that old man percentage would keep explosions away from their operating rooms.

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The American Hospital Association is now sponsoring an extended investigation to determine the exact extent and nature of all hazards of operating-room explosions, which should help to clarify the situation. In the meantime, they recommended making haste slowly.

The most dangerous gases are ether, ethylene, and cyclopropane. Their explosiveness is probably in the order named, but there is some difference of opinion on this point. Nitrous oxide with oxygen, vinethene, and ethyl chloride when mixed with oxygen are also very dangerous. Nitrous oxide alone is not flammable, but it supports combustion. The mixture of nitrous oxide with oxygen becomes highly flammable when mixed with minute quantities of oil. Vaporized alcohol is also flammable. Most of the explosive gases are heavier than air, an important consideration in the prevention of accidents.

A discharge of static electricity across an air gap has been the commonest cause of explosions. Second comes the use of cautery or sparking devices. One explosion was caused by a dentist using a hot-air syringe in the mouth of a patient under ether anesthesia. So far as is known, operating-room lights have never caused an explosion.

Static electricity is the greatest danger. Woodbridge and Horton [2] state that an anesthetist wearing cotton garments acquired a potential of several hundred volts merely by sliding forward on the cushioned stool and then rising. When woolen street clothes were worn the potential was greater. A draft of air accompanying the opening of a door raised the potential of the operating table 50 volts. Placing the drapes gave the patient a potential of 40 volts, while insertion of gauze pads between the sterile sheet and the neck raised the patient's potential 150 volts. A recent explosion was believed to be due to static electricity generated by drawing a woolen blanket over a rubber pad on the operating table. Flow of gas into a bag or tank containing another gas may cause an explosion. Friction of clothing, sheets, blankets, rubber pads, or the patient's hair may generate a dangerous charge of static electricity.

It must be remembered that the patient's breath may remain a hazard for some time after he leaves the operating room. Several fires have occurred from smoking in patients' rooms after operations. The danger zone in the operating room probably does not extend more than 2 feet from the source of the gas with most agents, but with ether it may extend as much as 15 feet.

Precautions may conveniently be grouped under construction, equipment, and administration. Under the first heading would come brass grilles in the floor, properly grounded. All electric outlets, telephones, signal and time apparatus, or other devices which may produce a

[2] Woodbridge, P. D., and Horton, J. W., J. A. M. A., 113: 740, August 26, 1939.

spark, should be located in the corridor, not in the operating room. All switches should be of a sparkproof type and preferably located in the corridor. Air conditioning should provide for a relative humidity of 65 percent, which rapidly dissipates most static charges but is not an absolute protection. The air should not be recirculated, for gases may build up to an explosive concentration. Operating-room lights have received a great deal of attention, though they have not been a cause of explosions so far as is known. A type has been developed which is called explosionproof. This means that the fixture is able to withstand an internal pressure of about 500 pounds per square inch. It makes the lights heavy and less adjustable, and may materially decrease the amount of illumination. Their desirability is doubtful. Operating lights recessed into the ceiling and vented into an adjoining compartment are giving satisfaction in some hospitals, and would appear to give perfect freedom from danger from this source.

A special room should be provided for storage of gas machines when not in use, and reserved solely for this purpose. It should have temperature and humidity control, ventilation to the outside, a fireproof door, and "no smoking" signs.

Under the head of equipment, a ban is placed on all woolen blankets or clothing, rubber tires on operating table or on wheel stretchers, electric pads, or other objects which may generate static electricity or prevent its discharge. Floor lights and all motor-driven machines must have enclosed, sparkproof motors, switches, and connections. Each operating room requires a hygrometer, so that the relative humidity can be constantly regulated. A device known as an intercoupler should be provided. This connects the patient, the gas machine, and the anesthetist, so that they have always the same electrical potential, which avoids sparks on contact.

Administrative precautions will consist of education of the personnel and constant vigilance to see that the protective measures are carried out. Humidity should be maintained above 60 percent, with good circulation of air and no recirculation while explosive gases are being employed. There should be no smoking, no open flame, no use of cautery or any sparking device until gas machines have been removed to a safe distance and the patient protected from danger. Gases should be administered only by skilled anesthetists. No rubber pads, woolen blankets, woolen clothing or rubber-soled shoes should be allowed in the operating room. Silk or woolen underclothing does not appear to be dangerous. No rubber-tired stretchers should be allowed near the operating table until all safety precautions have been taken. Intercoupling of patient, anesthetist, and gas machine should always be required.

TREATMENT OF LUETICS

IN THE DESTROYERS OF THE BATTLE FORCE

By Captain William E. Eaton, Medical Corps, United States Navy

It is well known that men suffering from lues in the smaller vessels, especially the destroyers, where medical officers are not usually on board have not received the treatment considered necessary for their infection and that such treatment as men have received has been of the catch-as-catch-can variety due largely to the character of this type of service. The presence of so many long-standing cases with positive serology evidences that in the past our early luetics were not properly treated or followed up. A review of medical records reveals the great irregularity of the doses and courses of medication and the extended and varying duration of the periods governing their administration. There was no continuous uninterrupted treatment.

These circumstances came particularly to notice not only in surveying the health records of men attached to ships in which the writer has been medical officer but also while he was on duty in the physical qualifications and medical records division of the Bureau of Medicine and Surgery, Navy Department. Upon reporting for duty on the staff of commander destroyers, battle force, the matter was investigated and much the same conditions were found to prevail. The exceedingly active operations of destroyers require that all men of the crews must be in the best of physical health. The chronic and untreated luetic has no place in such an organization nor can those liable to central nervous system involvement be tolerated in an organization where all men are expert technicians and hold positions and duties of great responsibility.

In view of these factors and the very definite and comprehensive program of civil agencies concerning the venereal-disease problem, the writer felt that he could no longer be remiss in our own handling of the treatment of lues, subject as it is here to administrative control and that all that was needed to provide our men with treatment in keeping with the most modern views was to establish this control with and by the authority of the force commander.

The success of our program, therefore, has been due largely to the unusual interest, consideration, support, and direction of the present commander destroyers, battle force, Rear Admiral Walton R. Sexton, United States Navy. Furthermore, the methods and results show what it is possible to accomplish under authoritative control and how the problem could be removed from our concern were it possible to have such control of the remedy and cooperation on the part of the patients in civil status.

The propaganda in the public press as well as our own advice to our men had aroused attention to such a point that the attitude of en-

deavoring to escape or dodge treatment on the part of the infected individual, who has in some measure in the past been at fault, was replaced by a demand for treatment not heretofore experienced. Infected men found that we were working in their interests and that their problem of receiving treatment was more theirs than that of the medical officer. It was therefore apparent that if a system of administering the remedy could be once started on a definite regular basis it would operate itself. The system, however, had to be consistent with the operation schedules and availability of the ships. Well defined and uninterrupted treatment was the goal.

The interest and reaction of several commanding line officers to the proposed system was investigated and it was found that there was no objection but rather encouragement provided the peculiar personnel and operations complications could be adjusted satisfactorily, which became quite possible once the system was put into effect.

Consequently on September 16, 1937, an order was issued by the Admiral whereby continuous alternating treatment became established with the responsibility of getting the patients to the place of administration vested in the commanding officers of ships as advised by the division medical officer. Division medical officers were required to review all case records and govern the treatment to be given using such consideration as was necessary and desirable, in connection with the personnel problem and movements of the individual ships, when transfers of men were obligatory in order for them to receive the courses of arsenicals.

The administration of arsenicals on board destroyers was prohibited, this being confined exclusively to tenders or shore stations when tenders were not available. Bismuth preparations subsequent to arsenicals were given on destroyers.

Our mission was to place the primarily infected men, in the seronegative stage if possible, under immediate control and treatment until they were rendered noninfectious and to regulate the subsequent treatment and supervision of these men and also the chronically diseased, inadequately treated cases, while they continue their employment and duties, in order that they may not become again infectious and may reach a status approaching cure. The unusually close association of men on destroyers makes this essential to the interests of the Navy as well as to all men, infected or otherwise.

The following system was therefore evolved:

All primary cases of luetic or other venereal infection, upon appearance or discovery, were transferred at once to tenders; at first on the sick list, later to pass to temporary duty status. Positive diagnosis was confirmed by microscopical, serological, and clinical evidence. Those cases with venereal disease other than lues were placed under appropriate treatment and observation for the development of possible luetic infection.

Luetic cases began to receive administration of 10 consecutive weekly doses of neoarsphenamine given intravenously, the first two doses of 0.3 gram, the remainder 0.6 gram. Untoward effects and reactions were watched for and urinary examinations carried out. If the man seemed to be doing well he was discharged from the sick list and allowed to perform duties compatible with his condition in the tender, thus saving him loss of pay in misconduct status, and thereby gaining his utmost cooperation.

Upon the completion of this period of 10 weeks' arsenical administration the man returns to the ship from which he came, where he promptly continues a course of weekly injections of a bismuth preparation, the first 2 or 3 doses of which have overlapped the last arsenical injections, until 10 doses have been received.

The patient is now ready for his second course of arsenical and joins the group of luetic patients whose cases are to be given treatment in the tender under the following weekly procedure. This group consists of those men whose health records have been reviewed and the need for treatment established and coordinated. These men are those with broken courses, insufficient treatment and chronic latency; those in whom we try to rectify the previous irregularities and neglect of treatment and try to regularize, and if possible terminate the dosing with arsenic which has been going on for years yet with positive serology persisting in spite of it all.

By order of the force commander it is required that these men are to be sent on board the tenders by 9:00 a. m., every Saturday morning. If there is a large number of men from a division of destroyers, the division medical officer is to accompany the group to assist with the injection procedures. Subsequent to the injection the men rest and are observed for reactions and are then returned to their own ships where they have the week-end to recuperate. Upon the completion of this period these patients again are given on board their own ships the weekly injections of bismuth preparation, the number of doses being determined by the duration of the treatment.

This system of procedure continues until under the decision of the division and tender medical officers sufficient treatment has been given. This is determined by a conjoint review of the man's records, computing the amount of remedies already given, the duration of treatment or period of time elapsed since treatment first began (often several years), the serological reactions and general condition of the patient. Any man released from treatment is to receive serological examination every 4 to 6 months and no case is released without spinal tests. In case of doubt or disagreement the case is referred to the force medical officer for decision.

Kahn tests are done on all men at frequent intervals whenever opportunity affords. A total of 756 Kahn tests were done between January 1 and July 31, 1938, on the U. S. S. *Dobbin* alone and the three other tenders will show a similar record.

Division medical officers are enjoined to keep a close check on all men under treatment and their courses of arsenicals and bismuth to the end that no broken courses are permitted and that courses of arsenicals are not begun unless completion is possible. Upon ships going

on a cruise wherein it is impossible or impracticable to send men to tenders for treatment the procedure is modified as follows:

The bismuth course is extended until such time as it is possible to place the man under circumstances permitting him to receive a complete course of arsenical. If the man is due for an arsenical course in the near future he must be transferred to temporary duty on a tender or the flagship before his ship leaves in order that the remedy may be given at the proper time—this particularly if the arsenical course is the second or third in the scheme—and return to his ship at the next meeting place.

At first no definite order as to the scheme of treatment to be carried out was issued. It was found however among the several medical officers concerned, that various conceptions of regularized treatment existed. Therefore in order that there might be a more or less definite understanding of our procedure all medical officers were informed by the force medical officer that the general scheme of treatment as outlined by the Cooperative Clinical Group and the United States Public Health Service as it appears on page 87 of Volume X, No. 2, dated February 20, 1929, of "Venereal Disease Information" issued by the United States Public Health Service would be the guide with the exception that the initial course of neoarsphenamine would be of 10 doses and that the alternating courses of bismuth will be of 10 doses also.

It was proposed to follow the American continuous alternating system of treatment which serves our purposes much better than any other, in that it not only provided under our military arrangement an uninterrupted weekly administration of the required remedies but also gave us the advantage of confining our treatment periods to definite terms, a very important consideration in a fleet of ships as numerous and as exceedingly active as is our naval force. The loss of man-hours of service is reduced and what is more important there is greater cooperation and less annoyance. Likewise we stand a better chance of effecting a more probable arrest of the disease by this very definite organized regime.

This procedure has now been in effect for nearly 18 months. Those men who commenced treatment at the beginning of this period for the most part were seronegative and have so remained or become so. They have received continuous, uninterrupted therapy and may well be considered on the way to eradication of their infection, if such is possible. Those chronic inadequately and improperly treated cases with serological findings of questionable value are showing improvement and renewed hope for cure.

We are getting cooperation from the patients and from line and staff officers alike because they find in this system a well-defined procedure and results. We are affording the luetic men better than the minimum standard continuous treatment without interruption or rest

periods. In all cases and especially the old ones we depend on and are guided by the amount of the remedies given and not by the serological findings, and there is "no rest without a spinal test."

The author has long been interested in establishing a definite scheme of treatment for luetics in the naval service, one which could be governed by necessary authority for its application, with the belief that not only are all persons infected with syphilis entitled to proper diagnosis and treatment but also all are obliged to submit to complete treatment in consideration for their fellow associates. He has felt that more success would attend our efforts at treatment under a definite outline of procedure and it is believed that the foregoing account of treatment has satisfied in a measure the principle advanced.

The following instructions are now in effect:

Primary cases, and those within the first 4 years after infection.—Courses of neoarsphenamine will consist of 10 doses, the first (and if indicated the second) of 0.3 gram and remainder of 0.6 gram. Courses to be given on tenders. Courses of bismuth preparation will consist of 10 doses, the first three and the last three to be given to overlap the last 3 and the first 3 doses of neoarsphenamine courses.

Three 10-dose courses of neoarsphenamine and three 10-dose courses of bismuth preparation will be carried out continuously and alternately. No interruption whatever will be allowed, 45 weeks required. Neoarsphenamine and bismuth subsalicylate are the preparations to be used.

Latent cases of more than 4 years' duration.—We see a great many of these cases with broken courses and often a large amount of arsenic, bismuth, and other remedies spread out over several years with intervals or rest periods of great variation. It is difficult to determine just where the case stands or what remedies should be given.

The health record will be carefully reviewed and a digest of it made and decision drawn. A complete thorough physical examination will be made with special reference to cardiovascular and central nervous systems, and a spinal test obtained before starting further treatment.

Put the patient under treatment in accordance with scheme outlined above until the patient has had at least three consecutive alternating courses each of arsenic and bismuth as shown by the health record. At the conclusion of the third bismuth course allow a rest period of 3 months then repeat this course of bismuth twice again with another intermediate rest period of 3 months. Treatment may now be concluded if the spinal fluid is normal regardless of blood serology, but observation should continue.

Medical officers must exercise careful judgment in applying these fixed schedules to the end that the procedures may be carried out only through adequate tolerance on the part of the patient and that this tolerance and the interests of the patient may be conserved in every way. Kahn tests will be made if practicable at the beginning and end of each arsenical course and spinal test will be taken at the conclusion of the third arsenical course.

SOME TRENDS IN SYPHILIS

By Commander R. P. Parsons, Medical Corps, United States Navy

If one studies the data tables compiled over the 14-year period 1925–38 for various phases of the general problem of syphilis in the

Navy one becomes struck with certain important and very definite trends all of which appear to this writer as most encouraging. Whether these same trends find parallels among the civilian population of the United States is not known with certainty because of the impossibility of collecting this type of data with any high degree of accuracy among a civilian population. But it does seem very reasonable to suppose that the figures for these conditions in the Navy are in large measure a reflection of the same conditions in the civilian world.

The trends in question can be stated in outline as follows:

1. Better diagnosis.
2. More intelligent—thus more effective and less hazardous—treatment.
3. A drop in the incidence of syphilis and a drop in the syphilitic census.

One of the first principles in the diagnosis of primary lesions is that about 90 percent of all genital lesions are syphilitic. The amount that we vary from this ratio in our annual figures is a fair index of how good, or rather how poor, our diagnosis of primary lesions has been during the year. During each of the first 7 years of the 1925–38 period we were actually reporting more "chancroidal" than syphilitic infections, as follows:

TABLE 1.—*Incidence of "chancroid" and syphilis, 1925–31, rate per 1,000*

	1925	1926	1927	1928	1929	1930	1931
"Chancroid"	27	25	36	29	31	35	33
Syphilis	20	22	25	23	22	25	25

It is evident that about 1932 we began a practice of more thorough examination of primary lesions and a more thorough follow-up of cases of genital lesions by periodic serological and general physical examinations, because the year 1932 is the first year in which we reported more syphilitic than "chancroidal" infections. And in each of the last 7 years of the period we continued to report more syphilis than "chancroid." The reason for the quotation marks on the word chancroid is our certain knowledge that all but a very few of such reported infections are really not chancroid. For the last 7 years of the period the figures are as follows:

TABLE 2.—*Incidence of "chancroid" and syphilis, 1932–38, rate per 1,000*

	1932	1933	1934	1935	1936	1937	1938
"Chancroid"	24	12	13	9	9	7	10
Syphilis	28	24	21	14	11	11	12

As to improvements in the field of treatment, we can trace these back to the beginning of the period, in 1925. In that year we gave a total of 48,887 injections of arsenicals and increased the annual total with each succeeding year until we reached the peak of 141,534 in 1933. This shows an increase of about 300 percent in annual treatment volume and in spite of no appreciable change in the incidence of syphilis in the Navy from 1925 to 1933. Since 1932 there has been a sharp decline in the Navy syphilis rate—28 in 1932 to 12 in 1938 but we have kept our total arsenical figure close to the peak point of 1933, giving a total of 123,176 injections in the last reported year, 1938. Thus, it is commendable that despite a syphilis incidence rate decrease of 57 percent since 1932 there has been a treatment volume decrease of only 10 percent.

That these figures do reflect conditions in the civilian world is indicated by figures showing the large increase in the sale of antisyphilitic arsenical drugs in the United States. This has increased from the sale of 5,787, 278 doses in 1935 to 10,656,253 in 1938. Table 3 shows the use of the various types of arsenical drugs in the Navy with their totals, since 1925.

TABLE 3.—*Arsenical therapy in the Navy, 1925–38*

Year	Arsphenamine	Fatal reaction ratio	Neoarsphenamine	Fatal reaction ratio	Maphar-sen	Sulph-arsphen-amine	Tryp-arsamide	Total
1925	5, 282	.....	41, 791	1–20, 896	.....	644	1, 160	48, 877
1926	6, 501	.....	55, 651	1–13, 913	.....	1, 011	1, 232	64, 395
1927	5, 749	1–5, 749	68, 340	1–17, 085	.....	389	2, 054	76, 532
1928	5, 093	.....	71, 754	1–11, 959	.....	3, 153	2, 551	82, 551
1929	5, 018	.....	76, 688	1–25, 563	.....	1, 345	2, 383	85, 492
1930	1, 393	.....	85, 646	1–28, 549	.....	1, 421	4, 418	92, 878
1931	1, 353	.....	95, 442	.....	.....	1, 157	5, 907	103, 859
1932	174	.....	128, 540	1–32, 135	.....	2, 614	6, 783	138, 269
1933	89	.....	138, 490	1–19, 000	.....	2, 921	4, 679	141, 534
1934	2, 865	.....	117, 799	1–39, 266	.....	2, 541	2, 980	126, 388
1935	3, 574	.....	113, 686	1–56, 843	573	5, 311	5, 397	129, 453
1936	2, 611	.....	92, 907	1–30, 969	2, 302	2, 525	5, 072	106, 041
1937	1, 846	.....	85, 760	1–85, 760	10, 660	1, 507	7, 219	107, 553
1938	0	.....	71, 694	1–23, 898	42, 755	1, 951	6, 193	123, 176
Total	41, 558	1–41, 558	1, 244, 537	1–27, 656	56, 290	28, 495	58, 056	1, 428, 936

In the above table the arsphenamine column is not a cause for pride. Despite the well-recognized superiority of this drug over neoarsphenamine we gave only 41,558 injections of it during the period as against 1,244,537 injections of neoarsphenamine. While the more difficult technique of preparation and injection of arsphenamine makes its use rather awkward under ship conditions this does not begin to explain its surprisingly small use in the Navy since the hospitals and other shore activities could well have used 10 times the annual amounts shown in the table. Arsphenamine seems to have faded out of general use in the Navy about 1933. The table indicates that it was revived during the 1934–37 period but this is

only an apparent reviving, since it is known to this writer that all the arsphenamine figures for that period were produced by one hospital. The fatal reaction ratio of 1 death to 41,558 injections of arsphenamine for the 14-year period as compared to the neoarsphenamine ratio of 1 to 27,656 adds some statistical support to the other long-known arguments in favor of arsphenamine.

Neoarsphenamine was our real mainstay during the period, although we did kill 45 people with it. However, we seem to have become somewhat more expert in avoiding fatal reactions. The first 7 years gave us a ratio of 1 death to 22,500 injections of neoarsphenamine while the ratio for the last 7 years was 1 to 32,559. No deaths have occurred from the other three arsenicals—mapharsen, sulpharsphenamine, and tryparsamide.

As for nonfatal reactions for the 14-year period, these can be listed by ratio of reactions to injections, as follows:

TABLE 4.—*Incidence of nonfatal arsenical reactions, 1925–38*

Arsenical	*Ratio*
Arsphenamine	1 to 989.
Sulpharsphenamine	1 to 1,140.
Neoarsphenamine	1 to 1,335.
Mapharsen	1 to 7,036.
Tryparsamide	1 to 14,514.

Sulpharsphenamine seems never to have gained much favor in the Navy, having reached its peak of 5,311 doses in 1935, and having been on the decline since then. It stands second on our reaction list and is known to produce dermatitis about seven times as commonly as neoarsphenamine. Other objections have also probably held its use to a low point, these being principally the relatively small doses in which it must be used and the excessive pain incident to its intramuscular injection.

It is encouraging to note the rise of tryparsamide in the Navy. Although this drug is indicated only in the neuro types of syphilitic involvement and although there are probably relatively and actually fewer neurosyphilitics in the Navy now than there were 14 years ago, the use of tryparsamide has increased from 1,160 doses in 1925 to 6,193 doses in 1938. This increase undoubtedly has resulted in large part from our increasing practice of spinal fluid examinations. And this is another trend upon which the Medical Corps is to be commended and congratulated.

The most striking drug trend has been in the case of mapharsen. This drug came into use in the Navy in a small way in 1935, when 573 doses were given. Since then its rise has been so rapid—42,755 doses in 1938—that we have cause to speculate whether it is on the road of neoarsphenamine displacement. The suspicion is strength-

ened by observing that the use of neoarsphenamine has been falling with an equally rapid rate over the same period—113,686 doses in 1935 to 71,694 doses in 1938. Reference to table 4 may furnish the principal reason for this trend. Besides the table 4 figures it can also be noted in table 3 that 56,290 doses of mapharsen have been given to date without fatal reaction.

Further evidence of the decline in syphilis is seen in the annual syphilitic census figures which have been compiled every year, beginning with 1933.

TABLE 5.—*Incidence of syphilis in the Navy, 1933–38*

Year	Syphilitic census	Navy strength includes Marine Corps	Percent of population	Year	Syphilitic census	Navy strength includes Marine Corps	Percent of population
1933	15,111	105,691	14.4	1936	14,427	126,583	11.3
1934	15,039	111,840	13.4	1937	13,887	133,501	10.4
1935	14,897	117,149	12.7	1938	14,059	139,307	10.0

Taking it all in all, these tables indicate quite strongly that during the past 14 years the Navy has made steady progress both in the diagnosis and the treatment of syphilis, with the result that there has been during the past 7 years a steady decline in the incidence and prevalence of syphilis in the Navy. It is believed (and certainly hoped) that parallels of these trends exist in the civilian world.

A FOOTNOTE ON YAWS AND SYPHILIS[1]

By Ellis Herndon Hudson, M. D., D. T. M. & H. (Eng.)

Let us speak first in abstract terms. What is the accepted procedure for unravelling the differential diagnosis between two diseases which resemble each other in some respects and differ in others? Etiology deals with the specific causative agent and epidemiology with the sum total of environmental influences. The characters of a given disease stem either from etiology or epidemiology. Epidemiological influences vary from time to time and from place to place, and are therefore worthless for differential diagnosis. The causative agent, however, is a fixed point, and from this point differential diagnosis can take true bearings.

The correct trend in differential diagnosis is to reduce the number of diseases to the point where it equals the number of etiological agents. The same etiological agent cannot cause two different diseases, and, on the other hand, two disease conditions caused by the

[1] Read before the annual meeting of the American Society of Tropical Medicine, Memphis, Tenn., November 22, 1939.

same agent have no right to separate entities merely because they look different. This is the law of "parsimony" in diagnosis and we apply it in medicine every day. We first eliminate all intercurrent and environmental causes for difference, and then we compare what we have left. If the two residual pictures are identical, then the etiology is identical; if not, then we truly have two different diseases. In diagnosis as in everything else we cannot compare two things until we have them on a common basis. A given disease will have a clouded title to separate entity so long as there are environmental factors which might account for the differences between it and its fellows.

Let us now speak specifically of yaws and syphilis. It has been the misfortune of the syphilis-yaws discussion that the abstract propositions stated above have often been disregarded. Several years ago Blacklock[2] pointed out that most comparisons of the two diseases fail to reduce them first to a common basis. Discussions have ranged over ground that was obviously environmental, and epidemiological factors have been used unjustifiably to prove etiological difference.

I am not here today to discuss the differential diagnosis of syphilis and yaws. I know yaws only from the published descriptions of that disease. I acknowledge the eminence of Butler, Craig, and many others in the field of yaws, and of such men as Fox and Wile in the field of syphilis. It has been my fortune, however, to see syphilis propagated under environmental conditions comparable to those of yaws, and this is my justification for raising some questions concerning the commonly accepted differential criteria for the two treponematoses. Bejel, the syphilis of the Euphrates Arabs, seems to me to provide the requisite base line for true comparison.

In a series of papers,[3] I have taken up the commonly accepted differential points in turn and have shown that when syphilis is propagated under the environmental conditions of yaws, these points lose their differential character. For example, "mode of infection" is obviously epidemiological; and whether a treponematosis is acquired by adults, as in syphilis, or by children, as in bejel or yaws, determines in great measure the whole subsequent trajectory of the disease. The immunological course, the pathological result, and the influence on the next generation are all different in the two cases. Relative humidity, the presence of insects, the influence of trauma, are all environmental factors influencing the picture, and there are many others which present themselves to the student of the epidemiological aspect of treponematosis. That bejel-syphilis resembles

[2] Blacklock, D. B.: Yaws and syphilis, Ann. Trop. Med. Parasit., 26: 423, 1932.

[3] Hudson, E. H.: Bejel: Syphilis as a contagious disease of children, Am. J. Trop. Med., 18: 675, 1938. Hudson, E. H.: Can syphilis exist apart from sex? N. Y. State J. Med. 39: 1840, 1939. See second article for all references of this series.

yaws so closely is presumptive evidence that though yaws and syphilis look so unlike, they may stem from the same root.

Where such a problem of differentiation has arisen in certain other diseases, the solution has been found in animal inoculation or laboratory culture. Unfortunately, differentiation of the spirochetes by culture or biological test has yet to be accomplished. The experiments of Pearce [4] and of Bessemans [5] show how versatile and nimble the spirochete is, how readily adaptable to new environments. *T. pallidum* steps easily into new roles. The experimental biologist can produce new strains almost at will, but the organism remains the same species. Nature may also have played upon *T. pallidum*, and on a far grander scale, without change in its specific nature. In short, conditions for crucial experiments in this field have not yet been obtained, and conclusions based on present information must be exceedingly cautious.

The present situation of the treponematoses might be pictured in the following terms. Etiology is the bedrock of diagnosis, and the problem is to determine whether yaws and syphilis are founded on an identical ledge of bedrock, or whether they represent truly different rock strata. Bedrock, however, is not exposed to the eye, but concealed by layers of gravel and earth, and crowned by a thorny and dense undergrowth—all these the products of environment. One set of excavators has made a clearing and sunk a shaft to bedrock, establishing the disease called syphilis. At some distance another group has excavated another area where there is different topsoil; they have also come down to bedrock, establishing another disease called yaws. All admit that the bedrock in the two cases is remarkably similar. Debate hinges upon the question, Is the ledge identical?

The debate will go on until the two shafts are connected up by the removal of overlying and intervening debris. A good deal of time and energy has already been consumed in debating about bushes and topsoil! The so-called "syphiloids"—bejel among them—represent trial shafts sunk at points between the main shafts, and these offer some positive evidence that complete clearance of ephemeral factors will establish beyond doubt the biological identity of *T. pallidum* and *T. pertenue*.

It is curious that the idea of an identical organismal cause for yaws and syphilis has met such vigorous opposition. Proponents of the synthesis of other mutually related diseases have been hailed as

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[4] Pearce, Louise: Experimental syphilis: transmission to animals and the clinical reaction to infection, Syphilis, p. 58, Science Press, 1938.

[5] Bessemans, A.: Morphologic variations of syphilitic germ, Am. J. Syph., Gonor., Ven. Dis., 22: 294, 1938. Bessemans, A.: Functional variations of *T. pallidum*, ibid., 22: 301, 1938.

prophets, and the elimination of redundancies is nosology have been regarded as scientific triumphs. Note the bracketing of such clinically diverse entities as bubonic and pneumonic plague, and dermal leishmaniasis with kala-azar; note the present enthusiasm for the correlation of the numerous "Proteus-X reacting" organisms, and the acceptance of one spirillum as the cause of relapsing fever, whether tick or louse borne. Conversely, however, contumely has often been the lot of the proponent of the causal identity of yaws and syphilis, and proof of this identity—if ever established—would apparently be greeted with disappointment in some quarters.

It might be profitable to ask ourselves why this should be. One reason may be the emotional aura which inevitably surrounds venereal disease. Of all diseases it is the most personal, and approach to it is to be made with circumspection. Every new fact or fresh viewpoint must be scrutinized for its possible repercussion upon a lay opinion which is itself now being educated to new attitudes toward spyhilis. Related to this is the desire—long and widely held—to think of syphilis as confined within certain geographical and historical limits. To admit the common nature of syphilis and yaws would be to admit that treponematosis is of globar distribution and a disease of ancient lineage; this in turn would require a reorientation of viewpoint respecting the history and geography of treponematous diseases, and a surrender of the present orthodox position.

In the second place, orthodox opinion (*sic*) has limited yaws by definition to the tropics, furnishing apparently the only instance where geography has been used as a crucial differential criterion. This glaring example of *petitio principii* precludes comparison of syphilis and yaws in temperate and tropical zones. In regions where there may be some overlap, a further principle is invoked, *viz.*, that if a given case is acquired in childhood it is yaws, whereas it is syphilis if there is venereal history and a chancre—diagnosis again seduced by epidemiology. Consequently the acquaintance of syphilologists with yaws is limited either to medical literature or to impressions gained in visits to the tropics, and the investigations of yaws *in situ* have been handicapped at the start by confused or wrong premises.

A third reason may be the conventional linkage of syphilis and yaws with dermatology. This specialty is at present in the process of hoisting itself from a morass of morphological concepts to a platform of sound etiological classification founded upon biochemistry of the body and parasitology of the skin. Syphilis, upon the discovery of the spirochete, broke away from dermatological tradition and won a place for itself among the constitutional diseases, but yaws has not been so fortunate. Old habits of thought cling, and yaws

in many minds still trails the outworn habiliments of a skin disease. For example, Hasselmann,[6] as late as 1937 made the following surprising statement:

> Yaws is an infectious and contagious skin disease caused by a spirochete, *Treponema pertenue* * * * Syphilis, on the other hand, is an infectious and contagious disease of the skin and inner organs caused by another spirochete, *Treponema pallidum* * * * The principal difference between these two treponematous diseases consists, therefore, in that yaws affects the skin only, and occasionally the adjacent mucous membranes of the face orifices, whereas syphilis attacks not only the skin, but sooner the inner organs too * * *.

It has been said that the reception of each new medical truth comprises three stages. First it is dubbed not true, next true but not important, and lastly not new. Those who have argued for the identical etiology of yaws and syphilis must feel that they are approaching the end of the first stage. There are some who still insist that this is not true, but others have already begun to say it is not important. Before long there will be many who will say the idea is no longer new, and the proponents of identity will suddenly realize from the faces of their audience, as Langdon-Brown said in another connection, that they are trying to force an open door. As I have met individuals and groups interested in this subject both in this country and abroad during the past 3 years I have been impressed with their evident desire for new light from whatever angle, and their sincere respect for those who have not allowed the weight of authority or orthodox opinion to stifle their search for the truth about yaws and syphilis.

SUMMARY

I have advocated the disentangling of epidemiology from etiology in the discussion of yaws and syphilis, and the elimination from the differential table of differences found to be produced by environmental agencies. I have raised the question whether many, if not all, of the differential points may upon investigation eventually be proven to be of epidemiological origin.

Opinions largely dominate this field at present. If *T. pallidum* and *T. pertenue* represent different biological species, let us have factual proof for the statement. If, on the other hand, yaws is an artefact, let us remove the word from the textbooks.

Finally, I have suggested some explanations for the tendency to complacent acquiescence in the present obscure position.

[6] Hasselmann, C. M.: What is yaws? and what is syphilis? St. Luke's Hosp. Alumnae Ass'n News Letter, 14: 3, 1937.

SYPHILIS OF THE SKULL[1]

AMONG ALEUTS, AND THE ASIAN AND NORTH AMERICAN ESKIMO ABOUT BERING AND ARCTIC SEAS

By Captain Richmond C. Holcomb, Medical Corps, United States Navy, Retired

Today, a study of syphilis in bone in the human skull presents some obstacles. The literature on the subject is scattered; there is a great diversity of opinion as to the primary cause of the lesion, the full extent of which seems often to go unrecognized; and not the least of these obstacles is to secure the patient's skull for study.

One must turn then to the museums of pathology, and the museums of the natural sciences for specimens, and for those who are so inclined there is, though scattered, plenty of material in America. Most of the textbooks published here and abroad, in order to illustrate their texts, exhibit specimens from such museums. In recent years, among such museums whose collections total more than 29,450 skulls, largely of American aborigines, I have had the opportunity to examine some 86 complete, or parts of syphilitic skulls, some of which I have been privileged to photograph, make roentgenograms, transilluminate, or otherwise study.

This particular study grew from a long interest in the condition anciently known in Rome, and even in parts of Europe at the time of the discovery of America as *mentagra*. In the vernacular of Spain it was known as *gafedad* and *gangoso*. In parts of France its victims were called *cagots*, *ladres*, and several other names, and recently the condition has been called by Leys, of the United States Navy, *rhinopharyngitis mutilans*. It is a late or tertiary lesion of the treponematosis called *yaws* and *syphilis*. Circumstantial descriptions of it, under various names have been given in the literature of many cultures, Chinese, Hindu, Greek, Arabic, Latin, and the romance languages. It may be seen today in many parts of the world, including several of the United States. One of the remarkable descriptions of syphilis of the center of the face, and an important contribution to the evolutionary concept of modern syphilis, is the chapter on venereal leprosy, by Bernard de Gordon, the model upon which Guy de Chauliac and later writers laid the descriptive foundations for the metamorphosis of leprosy into syphilis. This ultimately came about with little more change than the substitution of one name for the other. As the name syphilis was not invented by Fracastoro until 1530, it follows that no one wrote of this disease under this name before this time.

[1] Presented at a meeting of the section on Medical History of the College of Physicians of Philadelphia, March 11, 1940.

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In the course of this study of skulls in the museums, I was struck with the number of syphilitic skulls found about the basin of Bering Sea, showing the involvement of the bones of the cranium, a condition often accompanying the involvement of the bones of the center of the face, and this naturally turned my attention to the early official medical reports from Alaskan regions, and brought to light the evidence of widespread syphilis about the shores of this basin. These reports, long buried in the archives, confirm the evidence of the bones, which testifies to the extensive prevalence of these late lesions, which with lesions of the center of the face, still persist in certain parts of Australia, New Guinea, Solomon Islands, Fiji, Borneo, Java, Philippine Islands, Guam, Marshall Islands, the Pelew Islands of the Japanese mandate, and other regions of the Pacific basin, even into the frozen areas of the Arctic Ocean. Called *yaws* in some locations, it differs in no essential from syphilis.

This paper deals with 17 skulls from 11 localities. The map (fig. 1), shows the locations from which these skulls were secured. Point Barrow and Point Hope are each north of the Arctic circle, too far to consider yaws or "tropical syphilis," although it possesses the same characters of stages, lesions identical to each stage of clinical progress, etc. Yaws, although so extremely contagious that it infects 80 per cent or more of the native populations where it prevails, some will have us believe, for incomprehensible reasons, shies away from the white race. This enigma, with the added absurdity that it is a disease entity peculiar to the tropics where the inhabitants are immune to syphilis, should be easily correctable. Personally I reject this doctrine, choosing rather the evidence of careful scientific investigations which have been verified. To me the doctrine of duality for these two conditions, rests largely on rumor, traditionalism, abandoned theory, a single type of incidental skin lesion, and failure to give due consideration to racial insanitary habits. Those who still cling to the sophistry that syphilis cannot, or does not exist in the presence of yaws will find but little consolation in Choisser's study of 700 consecutive autopsies, or in Weller's study of the material selected by Chambers from more than 1,000 autopsies at Haiti.

The term *nodular osteitis* as used in the following descriptions, applies to the lesion which, when existing without a superinfection, is sometimes called *caries sicca*, and as such is described by Bertrandi, Cullerier, Ricord, and Virchow, and is one of the most characteristic lesions of syphilis of the cranium. It presents thickened bumpy areas, often spoken of as gummatous, and accompanied with irregular tortuous linear areas of atrophy, the depressed walls of which give a puckered appearance. The depth of these scars varies, and in isolated areas they are spoken of as stellate cicatrices. These areas of infil-

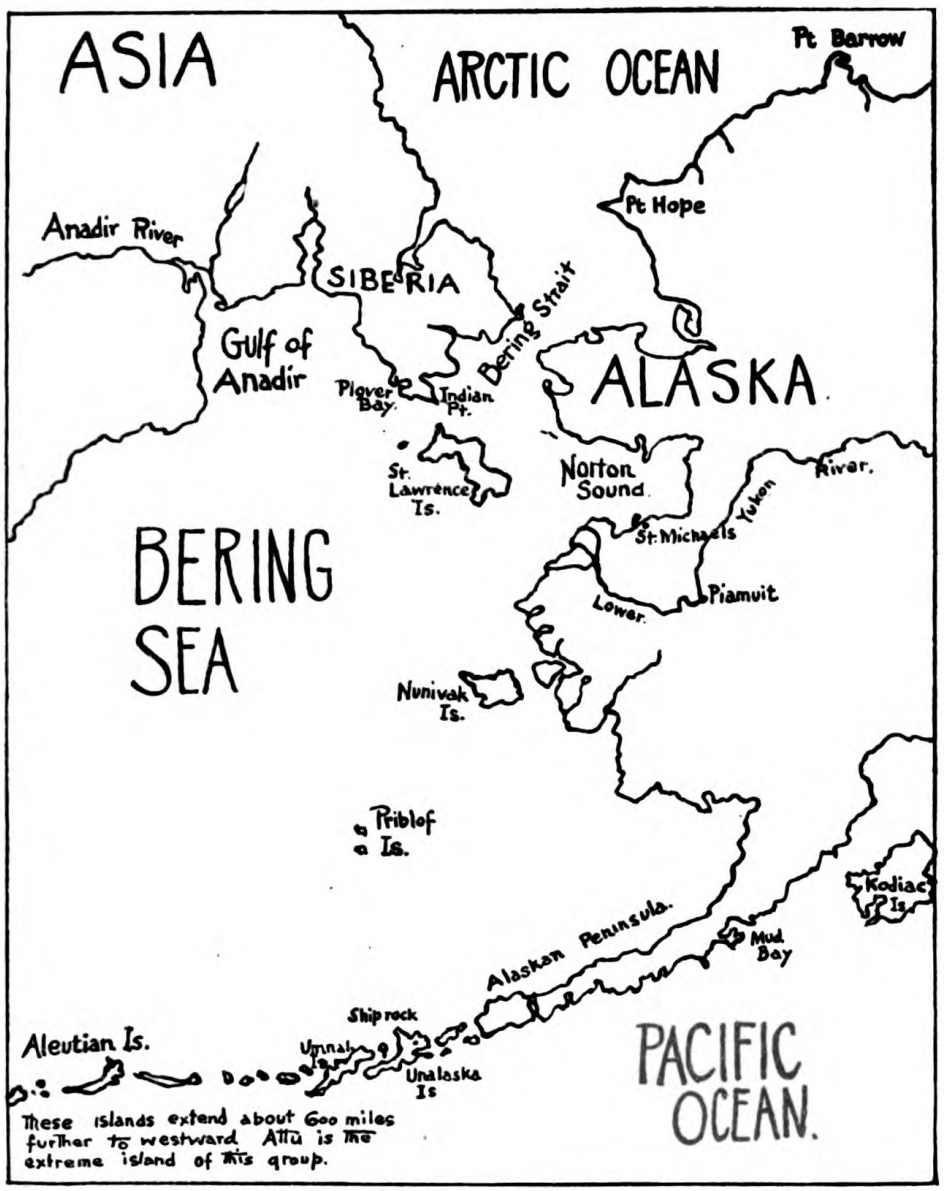

FIGURE 1.—Map showing locations of sites in Siberia, Alaska, and Aleutian Islands where syphilitic skulls have been found.

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FIGURE 2.—Vault of Eskimo skull from Plover Bay, Siberia, U. S. Army Medical Museum, accession 12,815. Photograph through courtesy of Lt. Col. J. E. Ash, U. S. Army.

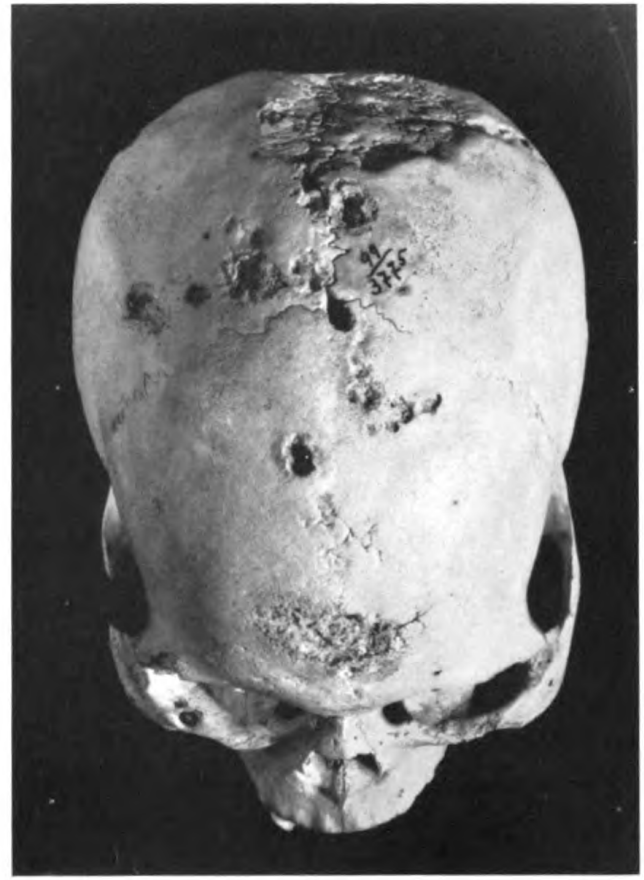

FIGURE 3.—Eskimo skull from Indian Point, Siberia. American Museum of Natural History, New York, N. Y., accession 99/3775. Photograph by the Museum.

tration and scarring are mentioned in particular, and although other less characteristic lesions consequent upon these changes might also be noted, we would prefer to discuss them in another paper, dealing with clinical material as well, and where the process has been known to exist for a long period, say from 2 to 37 years.

Lesions of the nose are difficult to determine in these dried and weathered specimens, particularly as often the bones of the nasal chamber are missing, and there remain only the palatal processes and the ethmoid and sphenoid, which in turn are badly weathered or possibly damaged by the root-erosion of a reddish arctic moss.

The following is a description of these skulls and the locations where found.

Skulls From the Siberian Side of Bering Strait

Accession 12,815 (fig. 2).—Through the kindness of Lieutenant Colonel J. E. Ash, M. C., United States Army, I was offered every facility and aid in the examination of this and other skulls at the United States Army Medical Museum. This skull was not in the exhibit case, but among a miscellaneous collection in the basement of the museum.

Title.—Syphilis? of vault of a skull. Found at Plover Bay, Siberia, southwest of Bering Strait by Dr. W. H. Dall of the Smithsonian Institution about 1873 (see map).

This skull is that of an Eskimo about 35 years of age. Bones of the face are missing. The frontal bone shows several areas of caries with exfoliation of the external table exposing the diploe. Both frontal sinuses are open. Both parietals are involved, particularly the left in moderate nodular areas.

Pathologic opinion.—Syphilis of the skull. This specimen was examined by Prof. H. U. Williams, who cites it in his paper as probably syphilitic, though he erroneously attributes it to Alaska.

Anthropologic opinion.—The data at the Army Medical Museum were meagre on this score, simply furnishing information that the specimen had been transferred from the Smithsonian Institution. Dr. Aleš Hrdlička, who is familiar with this skull and who has made extensive explorations in Alaska, was consulted. In his opinion the skull was post-Russian in period. That is to say, the victim existed since the exploitation of these regions by the Russians. This subject will be touched upon more at length below.

There are two syphilitic skulls and possibly a third from Indian Point, Siberia (see map). These are all in the American Museum of Natural History, New York, N. Y. Through the kindness of Dr. Shapiro, I was permitted to examine specimens in the skull room of his department. The collection comprises something over 9,000 skulls. I found no syphilitic skull in the large South American collection. Among 33 skulls from Indian Point, Siberia, I found two and possibly a third syphilitic skull. Accessions 99/3775 and 99/3780 are considered syphilitic by me, but no pathologic classification is made at the museum.

Accession 99/3775 (fig. 3), has several deep depressions of caries on the surface of the frontal bone and in the supraorbital region. Both parietal bones are involved in nodular osteitis at the vault with destruction of the outer table and reveal a few areas of healing. There are several deep punctate areas of caries in right malar bone. The nasal chamber shows turbinates gone and openings into the antri.

PATHOLOGIC OPINION.—Syphilis of the bones of the skull.

ANTHROPOLOGIC OPINION.—Female Eskimo. This specimen was obtained at Indian Point, Siberia, by Waldemar Vogoras, during an expedition for the museum in 1901. No additional information is available. Probably a surface find and post-Russian.

Accession 99/3780 (fig. 4), from Indian Point shows both parietals over the vault involved with an area of nodular osteitis. The greater part of the structures within the nasal chamber are gone, as is also the posterior border of the hard palate from irregular erosion.

PATHOLOGIC OPINION.—Syphilis of bones of the skull

ANTHROPOLOGIC OPINION.—Eskimo skull found on the same expedition as the preceding in 1901.

In addition to these two skulls there is a third skull from the same locality, same expedition, with a few points on the external surface of the frontal bone, resembling the early stage of syphilitic invasion from the periosteum, but not enough advanced to justify an opinion.

SKULLS FROM ST. LAWRENCE ISLAND

St. Lawrence Island lies at the southern entrance to Bering Strait, nearer the Siberian Coast, and is a sort of stepping stone between Asia and North America (see map). There are two skulls from the north shore of this island, and these are located at the United States National Museum, Washington, D. C. Dr. Hrdlička placed 21 syphilitic skulls from his collection at my disposal with facilities for examining them. The collection of skulls at this museum is in excess of 16,000 and fine in Alaskan and Aleutian specimens.

Accession 280,093.—Shows marked areas of nodular osteitis of both parietals near the occipital and along the sagittal and lambdoid sutures. Slight caries of the left side of the frontal bone. Most of the structures of the nasal chamber are missing.

PATHOLOGIC OPINION.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—This specimen was secured from the north coast of St. Lawrence Island, by Riley D. Moore on exhibition under the joint auspices of Smithsonian Institution and the Panama-California Exposition at San Diego, Calif. Accessioned Sept. 11, 1913. The specimen is that of a male Eskimo, and in the opinion of Dr. Hrdlička is post-Russian.

Accession 280,095 (fig. 5).—Shows extensive nodular osteitis of frontal bone and both parietals along the sagittal suture.

PATHOLOGIC OPINION.—Syphilitic nodular osteitis involving the whole vault and left temporal region.

ANTHROPOLOGIC OPINION.—Eskimo skull from north coast of St. Lawrence Island, collected by Riley D. Moore in 1901 on the same expedition as the foregoing. The skull is post-Russian in period.

FIGURE 4.—Eskimo skull from Indian Point, Siberia. American Museum of Natural History. Photograph by the Museum, accession 99/3780.

FIGURE 5.— Eskimo skull from St. Lawrence Island near the Siberian coast. U. S. National Museum, accession 280,095. Photograph by U. S. Naval Medical School, Washington, D. C.

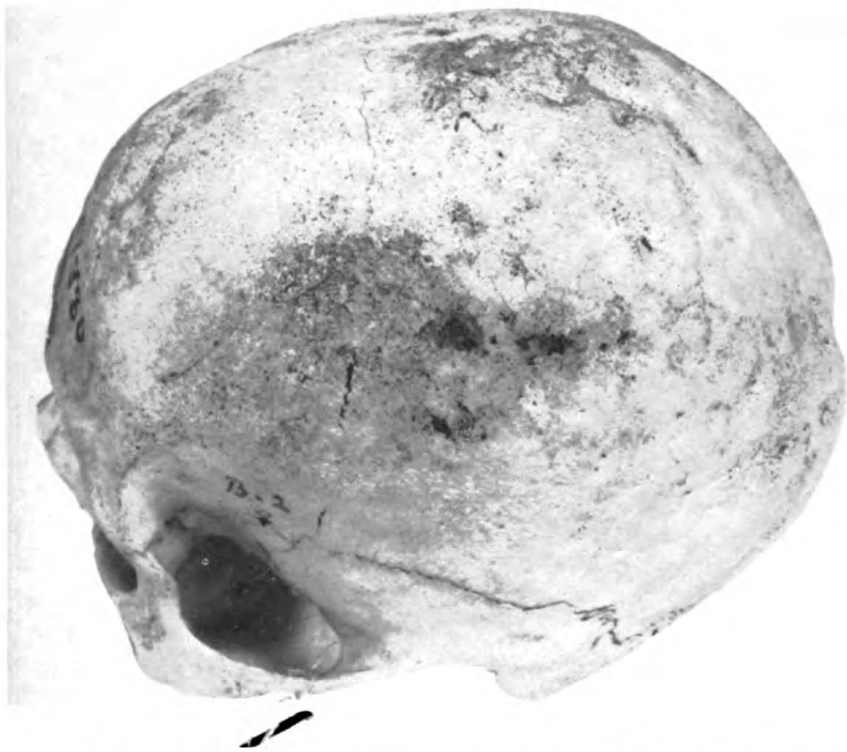

FIGURE 6.—Eskimo skull from Point Barrow, Alaska. Wistar Institute of Anatomy and Biology, Philadelphia, Pa., accession 17,380. Photograph by U. S. Naval Hospital, Philadelphia, Pa.

FIGURE 7.—Eskimo skull from Point Barrow, Alaska. Wistar Institute of Anatomy and Biology, Philadelphia, Pa., accession 17,399. Photograph by U. S. Naval Hospital, Philadelphia, Pa.

Skulls From Continental Alaska

The northernmost specimens are from Point Barrow, which is also the northernmost settlement in Alaska, and is well within the Arctic circle (see map). This point was reached by the British explorer Captain Beechey in the *Blossom* in 1826. We have examined many skulls from this location in different museums, collected by different expeditions. The two skulls to be here noted are at the Wistar Institute of Anatomy and Biology, Philadelphia, Pa. Through the kindness of Drs. Farris and Aptekman, I was permitted to examine the collection of skulls here, and would cite two from Point Barrow as syphilitic.

Accession 17,380 (fig. 6).—The frontal bone, including the area over the right orbit shows the linear and stellated scar of atrophy. Both parietals show nodular osteitis, particularly the right where there is atrophic scarring in the region of the lambdoid suture. In this region are three small circular perforations through both tables. Most of the structures of the nasal chamber are missing. There is a slight erosion of the palate not considered pathologic.

Pathologic opinion.—Syphilis of the skull.

Anthropologic opinion.—Female Eskimo, secured at Point Barrow by W. B. Van Valin on John Wanamaker expedition of University Museum (Pennsylvania) 1917–19, released to Wistar Institute April 1928. Apparently a surface find and probably post-Russian.

Accession 17,399 (fig. 7).—There is a deep erosion of the outer table in mid-frontal region. The bone about the edges is thickened and much weathered. Both the right and left parietals have areas of erosion. Examination by x-ray and by transillumination of the bone shows a deep area of opaque infiltration of the diploe about the edges of this erosion. The palate is intact, much of the nasal structure is missing, and the postnasal surface of the body of the sphenoid shows disintegration.

Pathologic opinion.—A positive diagnosis here is difficult. Root-erosion of the reddish arctic moss has been considered and rejected. Weathering and exposure have destroyed the surface evidences. The lesion is probably syphilitic.

Anthropologic opinion.—Male Eskimo secured at Point Barrow by W. B. Van Valin on John Wanamaker expedition of University Museum (Pennsylvania), 1917–19. Apparently a surface find and post-Russian.

The next locality is Point Hope, Alaska, which like Point Barrow is within the Arctic circle, on the shores of the Arctic Ocean, and north of Bering Strait (see map). There are two accessions in the United States National Museum from this region, one male, the other female.

Accession 333,390 (fig. 8).—This skull shows an extensive ulcerating nodular osteitis, with areas of perforation in frontal bone, both parietals, and in the occipital bone along the lambdoid suture. Some of the disintegration is possibly post-mortem. Much of the structure of the nasal chamber is missing, and there is disintegration of bones of the face possibly from weathering.

Pathological opinion.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—Male Eskimo, specimen collected by Dr. Hrdlička at Point Hope, Alaska. Accessioned October 1926. Specimen of post-Russian period.

Accession 333,441.—This skull shows several areas of syphilitic caries of the outer table of the frontal bone, with stellate scars and nodular osteitis. Both parietals, particularly the right, are involved. The latter has a perforation at the lambdoid suture. The left malar is intact, the right is gone. There is extensive involvement of the occipital bone along the lambdoid suture. Right side of the face is gone, palate missing, also the structures of the nasal chamber.

PATHOLOGIC OPINION.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—Female Eskimo. Specimen collected by Dr. Hrdlička at Point Hope, Alaska, accessioned October 1926. Specimen of post-Russian period.

The next locality is the southern shore of Norton's Sound at St. Michaels (see map). There is one accession in the United States National Museum from this region.

Accession 228,285 (fig. 9).—The frontal bone shows evidence of syphilitic nodular osteitis, also both parietals with caries and perforation. Deep erosion in the occipital bone, also the left temporal area. Just posterior to the bregma is a "worm-eaten" demarkation characteristic of sequestrum formation.

PATHOLOGIC OPINION.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—Male Eskimo skull collected by E. W. Nelson (possibly about 1895, see references), while on expedition for Bureau of American Ethnology from where this skull was transferred. This skull is post-Russian, and antedates the gold rush into this region.

The next skull comes from a locality described as "Lower Yukon, below Paimute and above Alex Viska's camp" (see map). This accession is in the United States National Museum.

Accession 245,333.—The frontal bone shows evidence of nodular osteitis. Both parietals, particularly the left, show erosions, as does also the occipital.

PATHOLOGIC OPINION.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—Female Eskimo of post-Russian period. Collected by Dr. Hrdlička.

The balance of the skulls are from Aleuts, the first of these from Mud Bay, Alaskan peninsula (see map). With one exception (9667), these accessions are in the United States National Museum.

ALEUT SKULLS

Accession 372,956 (fig. 10).—There is extensive ulcerative rarefaction of the nodular osteitis of the frontal bone, and both frontal sinuses are open. Both parietals show an extensive caries particularly in the atrophic scars surrounding the nodular infiltration. At the rear part of the left parietal is an area of syphilitic nodular osteitis not yet involved in caries. Both malar and mastoid bones are involved.

PATHOLOGIC OPINION.—Tertiary syphilis of the skull.

ANTHROPOLOGIC OPINION.—Female Aleut, collected by Dr. Aleš Hrdlička at Mud Bay, Alaskan peninsula, accessioned January 1935. Post-Russian in period.

FIGURE 8.—Eskimo skull from Point Hope, Alaska. U. S. National Museum, Washington, D. C., accession 333,390. View of face and vault. Photograph by U. S. Naval Medical School, Washington, D. C.

FIGURE 9.—Eskimo skull from St. Michaels, Alaska. U. S. National Museum, Washington, D. C., accession 228,285. View of face and vault. Photograph by U. S. Naval Medical School, Washington, D. C.

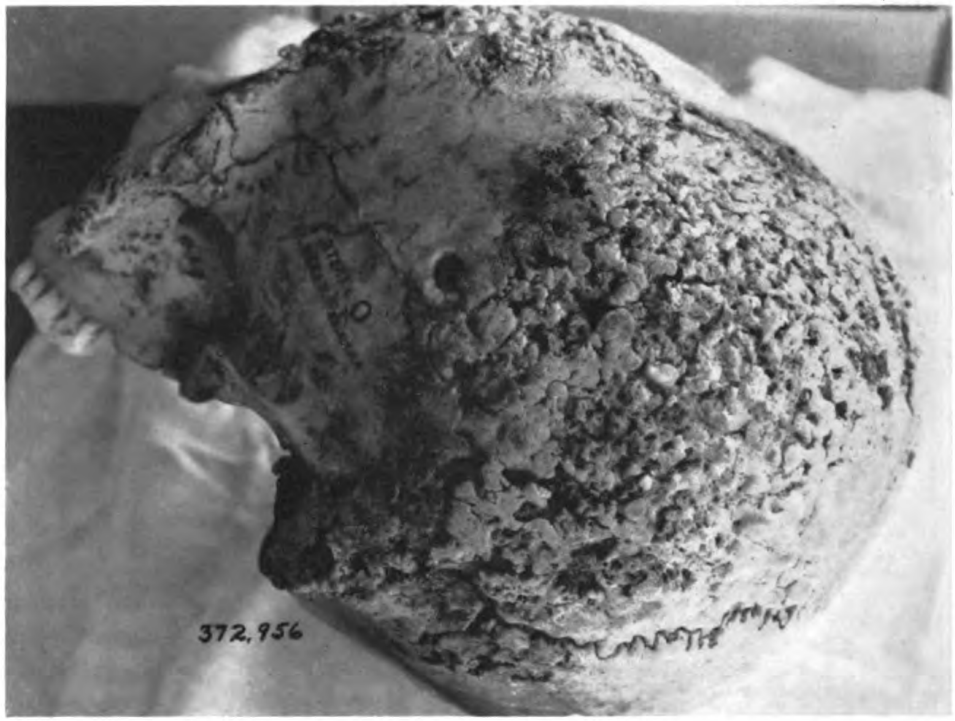

FIGURE 10.—Aleut skull from Mud Bay, Alaskan Peninsula. U. S. National Museum, Washington, D. C., accession 372,956. View from the front and the left parietal region. Photograph by U. S. Naval Medical School, Washington D. C.

FIGURE 11.—Aleut skull from Chenoffsky, Unalaska Island. U. S. Army Medical Museum, Washington, D. C., accession 9,667. Photograph by courtesy of Lt. Col. J. E. Ash, M. C., U. S. Army.

Accession 9,667 (United States Army Medical Museum) (fig. 11).—This skull is deeply browned, and is the earliest syphilitic skull brought from the Alaskan regions, having been acquired less than a decade after the purchase. It is labeled: "Skull of prehistoric Aleut of Chenoffsky, Alaska, collected by Dr. W. H. Dall, Smithsonian Institution." The frontal bone shows nodular osteitis, both supraorbital ridges are eroded, and there is an area of atrophic rarefaction with rounded margins, perforating the inner table, located near the coronal suture. Both parietals also show nodular areas, with several circular craters of deep atrophic scarring. The turbinates and palate are intact.

PATHOLOGIC OPINION.—Syphilis of the skull. Prof. H. U. Williams writes of this skull: "One can be as sure as ever possible in the case of a dried specimen, that this skull is syphilitic."

ANTHROPOLOGIC OPINION.—Aleut skull from Chenoffsky (Chernovski), Unalaska Island (see map), collected by Dr. Dall, who made the first American investigations of this area in 1873. Dr. Hrdlička who has made extensive investigation at this site (see next skull) as well as other districts of Alaska, was consulted with regard to the antiquity of this skull, and gives as his opinion that the skull is post-Russian. The area from which this and the following skull was collected, is the site of a post-Russian settlement. He states there is located in this vicinity the mound of an earlier settlement, but it is as yet untouched.

Accession 378,304.—This skull is also from Unalaska Island. The frontal bone is slightly involved, both frontal sinuses eroded and open. The left parietal contains a small area of nodular osteitis, the right is extensively involved.

PATHOLOGIC OPINION.—Tertiary syphilis of the skull.

ANTHROPOLOGIC OPINION.—Male Aleut from Chernovski (Chernoffsky), Unalaska Island. This skull was collected by Dr. Hrdlička and was accessioned October 1937.

Accession 378,485.—This skull is from Shiprock, a high island in the passage between Unalaska and Umnak island to the westward of the former (see map). There is a small diseased area in the frontal bone, and both parietals near the lambdoid suture show nodular osteitis. Part of the skull is missing.

PATHOLOGIC OPINION.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—Adult male Aleut, collected by Dr. A. Hrdlička, and accessioned October 1937. Post-Russian in period.

The two final skulls to be mentioned come from Umnak island (see map). On this latter island extensive investigations were made by Dr. Hrdlička in 1938. He announced in his annual lecture, November 8, 1938, the advisability of directing attention now to the unexplored mounds of Asia.

Accession 378,454.—The frontal bone shows several small erosions on the right side. Erosion above the right orbital ridge. Both parietals, but principally the right, show syphilitic nodular osteitis with erosion. Occipital similarly affected near the center of the lambdoid suture.

PATHOLOGIC OPINION.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—Adult female Aleut from Umnak island, one of the Aleutian group, lying to the westward of Unalaska island. Specimen collected by Dr. Hrdlička, and accessioned October 1937. Post-Russian in period.

Accession 378,606.—The frontal bone shows extensive syphilitic caries. Both parietals are involved, particularly in their posterior part near the lambdoid suture. There is a perforation of the left parietal with some evidence of healing.

PATHOLOGIC OPINION.—Syphilis of the skull.

ANTHROPOLOGIC OPINION.—Female Aleut, collected by Dr. Hrdlička at Umnak island, Aleutian group, and accessioned 1938. Post-Russian in period.

The Russian Period

From the time the United States purchased Alaska from Russia in 1867 it has been reported many times in official documents that syphilis was one of the principal diseases affecting the Eskimo and Aleut of these regions. Before this time a veil of silence shrouds its prevalence here.

Although Bering is generally said to be the earliest explorer to visit this region, it would appear from a publication entitled "The Pacific Russian Scientific Investigation," published by the Academy of Sciences of U. S. S. R., Leningrad, 1926, that the Russians were making explorations in these regions for about 100 years before the second cruise of Bering, when he made contact with the North American continent. Between 1639–46, Ivan Moskovitin and Vasli Poyarkof had explored the Sea of Okhotsk. In 1646 Michael Stadkin had made the overland journey from Kolmia River to the Anadyr River, emptying into a gulf of this name, an arm of the Bering Sea. Plover Bay, whence came our early evidence in the bones, is at the extreme eastern edge of this gulf. In 1648 Simon Deshnef, a Cossack, went through the strait as far as Diomede Islands, a voyage that Bering repeated in 1728, before he made the famous cruise with the two ships *St. Peter* and *St. Paul* in 1741, which reached the American continent. Upon this latter event the Russians based their interest in continental Alaska, although for some time nothing was done to exploit these regions. Expeditions were made by other countries, as the Spanish expeditions of 1774 and 1775 and the voyage of Captain James Cook of the British Navy in 1778.

For at least 100 years before the purchase by the United States, the Russians were spreading their influence in these regions, establishing trading posts and church missions. The earliest of the Russian fur traders carried their wares to Canton, China, one of the most ancient ports of Asia, whose junks for centuries had carried on trade with ancient India and Persia, and at which port they found a ready market. The trade was early vested as a monopoly in the Sholikof Co., and later, in 1799, it was vested in the Russian-American Co. At this time, within the infant United States, a knowledge of the vast regions beyond the Mississippi, extending to the Pacific, was hazy and indefinite.

Of course, as recent investigations of their remote cultures have shown, the aborigines of these regions were passing across the strait or the islands long before the Russian Government took an interest in their exploitation.

The term post-Russian as used in this paper applies in the main to the period since 1741 when Bering made his cruise of exploration to the American continent in the interests of the Russian Govern-

ment. This region was then inhabited by the Eskimo and Aleut. The former as encountered in Siberia and northern Alaska, are near relatives. Gambell says that the language of the Eskimo of St. Lawrence Island is similar in most respects to the language at Indian Point, Siberia.

I have not encountered reports of the prevalence of clinical syphilis during the Russian period. But as evidence that syphilis was present and widespread when the United States took possession we have the trail of diseased bones from remote regions, and the first-hand testimony from documents by medical officers of the United States Navy, Revenue Cutter Service, and the United States Marine Hospital Service, who made the earliest observations.

With the advent of the purchase of Alaska by the United States, as will appear below, reliable evidence of a widespread prevalence there broke the silence. Incidents like this have repeatedly occurred, and are not peculiar to the time of Charles VIII, when some believe a wildfire epidemic of syphilis in all its forms spread all over the known world in the space of a year or less. At this latter time there was a complete ignorance of our present conception of those diseases which are a cause of epidemics. Properly speaking syphilis is not an epidemic disease, and was probably endemically present over wide areas before the invention of printing brought it so prominently into notice. In Alaska the disease had probably been spreading over a long period. Soon after the purchase the screen over this communicable and hereditary disease was lifted by the first American medical observers who invaded these regions to survey their resources or to preserve order. Due to the conspiracy of silence that has long hovered over syphilis as an extremely wicked and unmentionable disease of venereal origin, their observations have long been smothered in the archives and have not had the attention that they deserve.

The earliest evidence of the syphilitic skull comes from Dr. W. H. Dall, on expedition for the Smithsonian Institution, who brought back the skulls from Plover Bay, Siberia, and from Unalaska Island. These were discovered between 1873 and 1877, during the first decade of the United States ownership, and before the gold-rush of 1897.

The earliest syphilitic skull from the Alaskan continent in this series was found at St. Michaels, by Edward William Nelson on an expedition for the Bureau of American Ethnology in 1895–96. In 1901 the two skulls from Indian Point, Siberia, were acquired by the American Museum of Natural History, N. Y. These were found by Waldemar Vorogas on expedition for the museum.

It is possible that there are other native syphilitic skulls from these regions unknown to me in other American museums, for the total of Alaskan skulls in these museums comprise a vast collection. Arrang-

ing those skulls examined to date in the chronological order of accession or find, it will be seen that though the first of these skulls came to light about 6 years after the purchase of Alaska, most of them were discovered within recent years.

Chronological order of the accession

Accession			Discovery	
Location	Number	Date	Locality	Collector
Army Medical Museum	12,815	1873 ?	Plover Bay, Siberia	W. H. Dall.
Do	9,667	1873 ?	Chenoffsky, Unalaska	Do.
U. S. National Museum	228,285	1896	St. Michaels, Alaska	W. E. Nelson.
American Mus. Nat. Hist	99/3775	1901	Indian Point, Siberia	W. Vogoras.
Do	99/3780	1901	do	Do.
U. S. National Museum	280,093	1913	St. Lawrence Island	R. D. Moore.
Do	280,095	1913	do	Do.
Wistar Institute Anatomy & Biology.	17,380	1919	Point Barrow, Alaska	W. B. Van Valin.
Do	17,399	1919	do	Do.
U. S. National Museum	333,441	1926	Point Hope, Alaska	A. Hrdlička.
Do	333,390	1926	do	Do.
Do	345,333	1929	Lower Yukon, Alaska	Do.
Do	372,956	1935	Mud Bay, Alaskan Peninsula	Do.
Do	378,304	1937	Chernovski, Unalaska	Do.
Do	378,454	1937	Umnak, Aleutian Islands	Do.
Do	378,485	1937	Shiprock, Aleutian Islands	Do.
Do	378,606	1938	Umnak, Aleutian Islands	Do.

Anthropologists have variously estimated the coming of man to North America at from 5,000 to 20,000 years ago. Some would make it even longer. However, it is fairly agreed that the route of his migration was across the Bering Sea area. At the close of 1935 the work done in Alaska by Dr. Hrdlička during the preceding decade was summarized in a monograph: *The coming of man from Asia in the light of recent discoveries.* According to Dr. Hrdlička:

> The main indications are that man came over very gradually and disconnectedly over a long period of time; that he brought with him differences in types, language, and culture; that at least some of the culture he carried was already far advanced; that according to all indications he did not proceed to people America across the mainland, but by skirting the western and southern coasts of Alaska; and that the Eskimo, the latest comer, in his two types, is a blood relation of the Indian.

Early Clinical Evidence of Syphilis in Alaskan Regions

Turning now to a consideration of the clinical evidence, there is little before the U. S. S. *Jamestown* under Captain Beardslee, United States Navy, arrived at Alaska in June 1879. We learn now from a sanitary report of the medical officer of that vessel that venereal affections were predominant.

The *Jamestown* was relieved later by the U. S. S. *Pinta*, and from this ship came reports of several successive medical officers through a period of 12 years, testifying to a widespread character of syphilis among the natives.

Passed Assistant Surgeon W. G. G. Wilson, United States Navy, reporting conditions at Sitka in 1884, writes:

The population consists chiefly of Indians, Russians, and half-breeds; besides these there are a few Americans, Germans, and persons of other nationalities. Pulmonary diseases and syphilis are the most prevalent (diseases) among the natives.

Writing of the Russian element he continues:

Syphilis is also very general among this class, the most of whom cohabit with the native women.

Passed Assistant Surgeon C. W. Rush, United States Navy, reporting in 1887 from the *Pinta*, writes:

As a rule the Indians from the northward and the interior are far superior physically to those of Sitka. They are taller, more robust and muscular, and present fewer evidences of chronic disease, but syphilis is everywhere present.

Passed Assistant Surgeon H. B. Fitts, reporting from the *Pinta* in 1889, writes:

The natives of southeastern Alaska were formerly of fine physique and good constitution, but at present the whole race is completely saturated with syphilis and scrofula, both inherited and acquired.

In 1892 the U. S. S. *Mohican* cruised from Sitka to Attu (Attoo), the westernmost of the Aleutian group, on her way to Japan less than 700 miles beyond, and her medical officer, Surgeon Simons reports:

The Aleuts and Sitka Indians are very scrofulous, and it is almost impossible to find one who does not present the scars of the disease, generally on the neck. It was so when the Russians first arrived. Syphilis is also common.

Again from the *Pinta* in 1895, her medical officer, Passed Assistant Surgeon S. G. Evans, United States Navy, also reports:

The extreme prevalence of syphilis and other venereal diseases.

The following year gold was discovered in the Klondike, and the spotlight of publicity was suddenly thrown upon Alaska. Until then this region, separated from the sovereign States, as a vast little-known territory, had claimed little interest. With the advent of the gold rush there was a marked change in the life of this region. Shortly after this the population nearly doubled.

There are two other reports of interest bearing on this subject. They are both among the documents of Congress, and both are from medical officers experienced with the disease over a wide area of coastal Alaska, and before the discovery of gold there.

Passed Assistant Surgeon Robert White of the United States Revenue Steamer *Rush* in Alaskan waters reports conditions in 1879 in the vicinity of Victoria, British Columbia. The natives:

Revealed the prevalence of various chronic venereal affections and their sequelae as well by the development of a general syphilitic diathesis, which renders the

people especially prone to the engrafting of strumous affection, and to succumb to attacks of acute disease. * * *. At Kazan Bay * * * there is an Indian village of some 300 people with no white residents. I found the chief of the village completely blind from long continued ophthalmia, and several other members of the tribe suffering from various chronic syphilitic affections, especially rupia and syphilitic rheumatism.

At Kodiak he reports venereal affections, scrofula, rheumatism, chronic catarrh, and tubercular diseases:

The marked prevalence of venereal disease in all forms among the natives of Urga is attributed to the proportionately large number of whites engaged in the fisheries. Several cases of condylomata, mucous tubercle, ulcerations, and necrosis were seen that could hardly have been equaled in the venereal clinic of a Paris or Vienna hospital. Mothers and children were found in whom the disease had progressed unchecked and without treatment through its different stages until they had reached a condition horribly repungant to civilized senses.

At Attu, the most extreme western island of the Aleutian group, and more than 600 miles west of Unalaska passage, he writes:

Two children were found with marked development of congenital syphilis and many of the people (about 100 in all), suffered from chronic skin diseases.

These were conditions as they existed about 1879, or about 10 years after the purchase, along a coastal extent of about 2,000 miles.

In another Congressional document dealing with the cruise of the United States Revenue Cutter *Bear* to Point Barrow, Alaska, for the relief of 8 whaling vessels fast in the ice, there is a report of Surgeon S. J. Call, R. C. S., dated 1898, and who had been on service in Alaskan waters some 13 years. During this time although he had seen widespread syphilis among the natives, they were invariably late lesions. He had never seen a primary sore, or chancre, among them. He had come to believe that much of the syphilis might be hereditary. On this subject he writes:

Among the Eskimo this disease (syphilis) in the primary stage, is not met as often as the profession and laity are led to believe. My experience on the coast of Alaska from Point Barrow to Attu dates from 1885 and I must confess that I have never yet seen in a native of either sex the initial lesion of the disease. While at Unalaska for nearly 5 years, I visited once and sometimes twice a year professionally every village from Attu to St. Michael. The only cases of this nature were two (neither of them natives), one of the soft and one of the hard variety, the latter being on the lower lip of a Portuguese on a passing whaler. Those large deep, foul-smelling ulcers so frequently seen in villages on the coast of the Alaskan islands may be the result of hereditary syphilis; yet the life, habits, quarters, and food of the people in these sections of Alaska are productive of the very worst forms of scrofulous lesions.

Such observation as that of Surgeon Call, that is to say, the absence of a venereal chancre, among a people numerously infected with syphilis, spread over several hundred miles of coast, and during a period of 12 years observation, is the kind of testimony we hear often in relation

with yaws. It suggests the possibility of a large number of those cases called *heredo-syphilis précoce* and *heredo-syphilis tardive* by the French, and even the binary syphilis of Tarnowsky. The term *scrofula*, which appears so frequently in these reports and was so frequently used in the texts of their time, has now about disappeared from the literature. Thom, writing in 1922 says:

> I am convinced that many cases of so-called "scrofula," as it was termed by the older writers, is not tuberculosis at all, but late congenital syphilis.

For my part I studied medicine from text books, and under teachers who taught medicine with a generous use of this term. The lesions are still seen but the terms used are more discriminating.

As late as 1914, Surgeon W. S. Pugh, United States Navy, writing on Alaskan ports, and referring especially to the Pribilof Islands comments:

> The evidences of syphilis are noted everywhere, mostly tertiary lesions and of old broken-down gummata, nerve lesions, ulcers, interstitial keratitis and the like. I personally saw every individual, and I think that 60 percent would give a positive Wassermann.

The several foregoing extracts from official reports, together with the evidence of the skulls, testify in a general agreement, to a widespread distribution of syphilis through a long period, sometimes described as a hereditary syphilis of the early and late types, existing along the Siberian coast of Asia, the islands of the Bering Sea, the Alaskan coast and peninsula, and the Aleutian Islands even to Attu, the westernmost island of this group. And, there is much to show that the disease was there before the advent of the gold-rush or the purchase of Alaska from Russia.

The Origin of Syphilis in Alaska

Whence came this syphilis to Siberian and Alaskan shores? Is this a recent infection planted there by the coming of the Russians? This latter question it seems to me may be answered affirmatively, though there is substantial evidence that syphilis existed in Asia since remote time, and it should be evident that Bering Strait and Sea offer no substantial barrier to the spread of syphilis in either direction from one continent to the other provided there is communication by some means from one group of people to the other. The Russians, and later the whaling ships were of course a factor for providing this means. It is approximately 54 miles across the strait. The island of Attu, the westernmost of the Aleutian Islands, is about 650 miles from the Alaskan peninsula and 660 miles from Japan's eastmost naval base, Horomushiro, with islands of the Russian Commander group as possible way-stations.

Did man coming from Asia bringing with him certain cultures since greatly modified, also bring with him any of his chronic diseases? It certainly seems plausible that in some ancient time, syphilis might have spread from one continent to the other as we see the evidence in a circumstance of our own time. I do not propose to discuss the anthropologic phases of this question, which is complex, and exacting as a science well may be. However, there have been finds in the Americas of Indian skulls which beyond all question are syphilitic. Such skulls as the Paracas skull of Tello, from South America, or the North American skull of Haltom and Shands should be recognized as syphilitic by any experienced pathologist. So to speak, the diagnosis is written all over these. But when it comes to a consideration of their antiquity, no skull has yet been produced upon which all anthropologists can agree. Dr. Hrdlička believes that no syphilitic skull of pre-Columbian time among the American Indian has yet come to light.

It might be well here to point out that endemic syphilis in these cold regions is previously mentioned. Edward Ehlers, of Copenhagen, long ago pointed out that the earliest of the post-Columbian syphiloids, *sárasótt*, prevailed in Iceland before 1528, as evidenced by their ancient chronicles. This was before Fracastoro invented the word syphilis.

The chance that we may find a syphilitic skull of great antiquity in these Alaskan regions would seem remote. The extreme violence of the elements with the disintergrating influence of alternate freezing and thawing, and where the traditions for preservation of the dead have long been at a primitive level, all conspire to defeat this object.

Different methods of disposing of the dead once existed in different parts of Alaska. Before the advent of the Russian church with its ritual of Christian burial, some tribes are said to have practiced cremation. Some wrapped the bodies in skins and hid them in caves and crevasses of the isles. Among some far northern tribes where the ground is almost perpetually frozen burial was out of the question. Here the body might be wrapped in skins and elevated above the ground on a scaffold made of such things as the straight lower jawbones of the Right or Bowhead whale. Some were wrapped, and dragged on a sled beyond the settlement "to sleep on the ground," where they soon become the prey of animals. Incidentally this practice is not unlike a custom still used in Asia, about Urga, in Outer Mongolia. Furthermore, there are three syphilitic skulls at the Army Medical Museum, and one at the United States National Museum, Washington, D. C., brought from Urga by Dr. Hrdlička, from the scene of such a practice.

Such crude and primitive methods of disposing of the dead hold forth little promise in our search. Today, even in the farthest north one now finds cemeteries along the coast, walled in with such whale jawbones as mentioned above.

The meager evidence from the bones collected thus far is not sufficient to prove that the present invasion of syphilis existed in these regions before their exploitation by the Russians for a fur trade and fisheries.

Wong's account of syphilis in ancient China should be read by those who wish authentic information on the subject from the ancient Chinese medical classics.

In conclusion I wish to acknowledge the generous assistance given to me by the curators of the several museums mentioned above and by their assistants. I am also especially indebted to Captain William Chambers and Henry L. Dollard, Medical Corps, United States Navy, of the United States Naval Medical School, Washington, D. C., and the United States Naval Hospital, Philadelphia, Pa., respectively.

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PATHOLOGY OF SCHIZOPHRENIA

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Man has been afflicted with insanity since time immemorial. There are frequent references to it in the Bible where it is mysteriously alluded to as possessed with devils. Deep in the background of many of our minds even today there appears something mysterious about the mind which to some extent at least tends to place it on the dark shelf with the metaphysical phenomena. Scientists, however, keep pulling it down from that shelf from time to time in attempting to subject it to scientific analysis. Such analysis, however, is extremely difficult and their success has been only partial. While they have succeeded in bringing to light many facts more or less related to the mind and its nature, there remains much unexplored territory, which requires further illumination before these facts can be adequately understood and correlated.

Probably the most complete description of the insanities was made by Kraepelin about 1896 when he classified some of them on a behavioristic basis. His classification of dementia praecox into the well-known simple, katatonic, hebephrenia, and paranoid forms is considered classic.

He also described and directed attention to certain abnormalities of the brain structure, particularly the ganglion cells, and evidences of disordered metabolism in dementia praecox cases coming to autopsy which have been to a great extent corroborated by most subsequent investigators in this field. His pathological studies were made on post mortem tissues exclusively, which leaves us in the dark in the great field of the vital forces of the living cells and their changes incident to health and illness, particularly as related to schizophrenia. Chemists are working with living cells attempting to understand what goes on in them during life. Here several more or less isolated facts have been learned but so far living cells have defied complete analysis. It is perhaps in the living cells that the source of trouble is to be found.

It appears that the mind is a function of the nervous system which guides and directs the activities of the individual. Upon such a basis it is easy to correlate in a general way a healthy nervous system with a clear and sound mind, and a diseased nervous system with a defective mind. In some instances this correlation is manifest, for example, advanced hydrocephalus and idiocy, or syphilis and general paresis. In many other cases, however, the pathological condition of the tissues has not been demonstrated and there is only the irrational behavior of the patient to guide us. Of course here we can assume much and many assumptions have been made but too much of their scientific proof is lacking.

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The literature, particularly that of recent date, abounds in descriptions of postmortem findings with efforts at correlation, and more recently vital reactions have received considerable attention. It is interesting in this connection to note the findings of Nolan D. C. Lewis [1] who in a very elaborate article in 1922, reported on 600 autopsies on schizophrenics. In this work he demonstrated a consistent aplastic circulatory apparatus for the katatonic and hebephrenic types. The heart was approximately one-third smaller than the normal. The aorta and other blood vessels were decidedly smaller than normal. There were also frequent abnormalities of the endocrine glands, particularly the thyroid and adrenal glands.

Hoskins [2] in 1933 reported an extensive and interesting study of the organic functions of schizophrenia. A large series of cases was studied and averages were compared with average normals for control. This study showed enormous variability in all the known organic functions. The pupils varied more in size, shape, and reaction than the normals. They were often pin point in size and would rapidly dilate to the full excursion of the iris and again contract, regardless of the influence of light or other conditions which ordinarily affect the pupils. They were often oval in shape, a condition seldom seen in normal individuals. Cyanosis of the extremities was common, the pulse irregular, and on the average the rate was slow. Blood morphology was suggestive of secondary anemia.

In 1932 Winkelman [3] made a very extensive study of injury to the brain structure and its relation to mental affect. In this report he points out that circulatory stoppage for a period of 8 to 10 minutes is sufficient to produce permanent ganglion cell death and necessarily permanent impairment of the function of the cells involved; but in spite of this ganglion cell death the brain has the remarkable ability to compensate the loss in some cases through the activity of other cells. He cites a case of motor aphasia from hemiplegia, which is not uncommon, in which loss of speech was complete. The patient lived to recover the speech function to such an extent that there was only slight hesitation in speech at times. When the patient eventually died the autopsy showed complete absence of the entire Broca area. Winkelman's observations in another case show how anoxemia may, as previously claimed cause ganglion cell death with consequent mental derangement. This case was a woman who, during an anesthetic for a surgical operation, stopped breathing for a period of

[1] Lewis, N. D. C.: Congenital Megacolon, J. Nerv. & Ment. Dis., 56: 193, Sept. 1922.

[2] Hoskins, R. G.: Organic Functions in Schizophrenia, Arch. of Neur. & Psyc., 30: 123, July 1933.

[3] Winkelman, N. W.: Importance of Small Blood Vessels in Psychiatric Problems. Am. Journ. Psyc., 12: 775, Jan. 1933.

6 minutes. Respiration was then restored and she lived for considerable time, but she did not recover mentally. Eventually she died and at autopsy he found intense ischemic death of all ganglion cells.

These observations suggest several avenues for speculation as to the pathology of schizophrenia. They add scientific evidence to the usual assumption that the mind is a function of brain cells, probably ganglion cells and that interference with the supply of necessary substances to these cells will cause injury to them or even their death if such interference is too extensive or continued over a sufficient period of time.

The defective circulation in schizophrenia as demonstrated by Lewis, Hoskins, and others suggests a plausible explanation of the manner by which the interference to the supply of the necessary substances to the ganglion cells may obtain in this disease.

A low blood pressure, defective blood cells, toxic substances in the blood, etc., offer ample reasons for such interference with the required supply of proper substances to the ganglion cells.

The fact that recovery takes place sometimes is compatible with this idea. As shown by Winkelman and many others, nerve function may be restored by compensation through the activities of other nerves even when those nerves which were developed specifically for that function are completely destroyed. Some cells are capable of taking over the functions of others. In schizophrenia it is likely that not all nerves concerned with mental functions are completely destroyed. Perhaps some are destroyed completely while others are spared. Those which are spared could very well take over the function of those which are destroyed. Probably a more likely assumption would be that all the ganglion cells are injured sufficiently to cause the symptoms and that later when the damage is repaired they resume their normal functions which is in keeping with such facts as are known. All this fits in perfectly with the familiar fact that some people are more intelligent than others. More intelligent men have better nerve cells to begin with, and having a more efficient circulatory apparatus keep their nerve cells better supplied with the substances which they require to properly guide and direct them in their daily adjustment to the conditions under which they live.

We are familiar to some extent with the effect of toxic substances on the sensibilities. It is the general assumption that those ganglion cells concerned with thinking, feeling, etc., are affected when toxic substances obtain in the circulation. In some cases it appears that the vital functions of the cells are accelerated while in other cases they are retarded.

Physiologists have shown experimentally that cerebral anemia will cause convulsions in animals, and it has been observed in man with

cerebral exposure at operations. Stanley Cobb[4] has shown that when the carotid and cerebral arteries of a cat are tied off even for short periods of time convulsions regularly occur. In cats 10 minutes complete anemia is a convulsive dose and in man 18 seconds is a convulsive dose. Cobb found that anemia sufficient to cause convulsions produced demonstrable damage to the nerve structure.

Schizophrenia, in the great majority of cases, occurs during puberty and the climateric, a time when the endocrine glands, in fact the entire human system is undergoing a revolution in which it is adjusting itself to the requirements of adult desires and ambitions incident to mature life. Some of these glands are growing rapidly at this time, for example the gonads; while others, the thymus for example, are undergoing regressive changes. The secretions of these glands have a profound effect on the nervous system in a way which further illumination is needed to clarify. They profoundly affect the vital chemical reactions of the cells concerned in mental reactions as well as all cells of the body. Chorobski,[5] Penfield,[6] Finesinger,[7] and others have shown, contrary to former teaching of physiologists and anatomists that the blood vessels of the brain have a nervous mechanism similar in all respects to those of the rest of the body. This fact suggests the possibility that the abnormal organic functions such as cyanosis of the extremities, altered circulatory action, abnormal pupillary reactions etc., are indications of what is going on in the cerebral tissues, perhaps producing relative anemia of the brain and damage to the ganglion cells concerned with normal mental reactions, all of which result in schizophrenia.

As opposed to the view that schizophrenia is caused by organic changes in the brain structure, either chemical or physical, is the view of Freud and his school, who contend that schizophrenia is a functional condition and independent of any physical or chemical abnormalities that may be found. Freud and his associates appear to have shown that there is a very close resemblance between dream states of the normal on the one hand, and the psychotic states of schizophrenia on the other, and that both dreams and psychoses are wish-fulfilling phenomena, which lead him to conclude that psychoses are unconscious attempts to possess in phantasy that which is desired but denied in reality, and a regression from a higher to a lower biologic level of adjustment which is very unsatisfactory in the conscious states, but in the unconscious states, dreams, phantasies, and psychosis are entirely

[4] Cobb, S.: Cerebral Circulation; Cerebral Anemia. Am. Journ. of Psyc., 13: 947, March 1934.

[5] Chorobski, J.: Cerebral Vasodilator Nerves. Arch. of Neur. & Psyc., 28: 1257, Dec. 1932.

[6] Penfield, W.: Intracerebral Vascular Nerves. Arch. of Neur. & Psyc., 27: 30, January 1932.

[7] Jacob and Finesinger: Cerebral Vessels. Arch. of Neur. & Psyc., 29: 1057.

satisfying even though uncompatible with society. Dreams and phantasies seem to be directly related to the personality and there seems to be conflict between desire and possession which results in complexes which some individuals are unable to face in reality and who are forced to regress to the low biological level of adjustment, to phantasy, dreams, and psychoses for the necessary satisfaction. Upon this functional assumption which seems quite plausible in some respects the Freudian School has devised a system of treatment known as psychoanalysis. By this method an attempt is made to learn by a study of the personality, what the disturbing complexes are and to teach the patient how to face them in reality. Some psychiatrists report glorious success with this method of treatment while others fail.

It seems, however, that only a few are able to master the technic, and consequently the method has lost much of its former popularity. Granting that the Freudian view of this problem is correct from a psychobiological angle, still it fails to satisfactorily explain why some individuals are unable to face their problems in reality while others do, and yet this is probably the crux of the solution to this problem. The fact that psychoanalysis fails to cure in a high proportion of cases suggests the possibility that the pith of the proposition has escaped attention in the functional view, and we are forced to search further for it.

The effect of treatment of schizophrenia offers great fields for speculation as to its pathology. It is almost universally recognized that simple hygienic care almost invariably results in improvement in the physical condition and behavior of these cases. The use of more abundant and varied diet, regular physical exercise, recreation in sunlight together with proper rest periods is almost always followed by noticeable improvement.

When proper food is supplied to the nerve cells they have the correct chemical elements, whatever they are, for complete integration. Exercise aids the cells in procurement of those elements from the circulation.

Sunlight is essential to proper oxidation by all animal as well as vegetable cells. By these facts the attention is directed to the tissue cells, even to their vital processes, whether chemical, physical, other phenomena, or all of them, as the possible seat of the pathology. Assuming for sake of argument that the vital reactions of the ganglion cells are being gradually but constantly retarded by lack of some or all of the essential substances or forces which they require, one can explain to a very large extent the bizarre behavior. Ingham,[8] in a paper read before the American Psychiatric Association at San Fran-

[8] Ingham, S. D., Nielsen, J. M., and Von Hagen, K. O.: Correlation of Insulin Shock to Symptoms. Am. Journ. Psyc., 95: 819, Jan. 1939.

cisco, suggests that the site of greatest effect in insulin shock treatment of schizophrenia is in the region of the basal ganglion, the gray matter and the brain stem, and intimates as his feeling, that the pathology responsible for schizophrenic symptoms is located in these areas. But here again much of the proof is lacking. Pharmaceutical shock treatment is now used widely in institutions for the care and treatment of schizophrenia, and further light on the pathology may evolve from observations of the effects of this treatment. It would be interesting to see insulin in much smaller and less dangerous dosage used in the treatment of these cases before they get to the stages requiring institutional care. This, of course, is exceedingly difficult. Public opinion prevents these individuals from receiving scientific attention until they have committed some damning crime for which they are forced into an institution to escape criminal prosecution. It has been suggested that insulin is necessary to the proper oxidation of sugar within the tissue cells, ganglion cells included, of course. The effect of insulin in the treatment of diabetes mellitus is well known. Just how it operates is probably unknown but it suggests possibilities for further research. The suggestion that it operates in the treatment of schizophenia by interrupting the troublesome complexes through unconsciousness and convulsions, thereby permitting the psyche to flow in its normal channels, is interesting but appears vague and inadequate. It will be interesting to watch what further light this work may shed on the subject.

CONCLUSION

A review of some of the more recent literature relating to the pathology of schizophrenia has been made and an attempt has been made to abstract parts of some articles, and to show how some of the facts disclosed in this great raft of literature may relate to the subject.

Some of these articles appear to make certain definite advancement to our knowledge of the nervous mechanism of the cerebral vascular apparatus, and to add weight to the opinion that the pathology of schizophrenia is organic and that the psychological nature of the psychosis as propounded by the Freudian school is incidental to the operation of the diseased tissues.

I am not sure that I have interpreted Freud's teachings correctly as the literature is so voluminous and so ambiguous in some respects that I could not be certain at times what it meant.

In the preparation of this article the author gratefully acknowledges his appreciation to Dr. W. C. Davidson, dean of Duke University Medical School, to Dr. E. A. North, professor of neurology and psychiatry of the University of Cincinnati, and to Dr. E. A. Baber, superintendent of Longview Hospital, for valuable assistance.

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METRAZOL THERAPY IN SCHIZOPHRENIA

By Lieutenant Roger G. Osterheld, Medical Corps, United States Naval Reserve[1]

Since 1935, when von Meduna[2] published his paper, *The Convulsive Therapy of Schizophrenia*, there has been an ever-increasing interest in the treatment. It calls for the use of camphor in the form of metrazol (pentamethylentetrazol) as a therapeutic agent in mental disease, especially schizophrenia. Many relatives of patients in mental hospitals have inquired about this treatment and in many cases have practically demanded its use. This is because the layman has been led to believe that the so-called shock treatment is a "cure-all" for any type of mental illness.

Metrazol is a white, crystalline, odorless, faintly bitter powder which is freely soluble in water. It belongs to the class of substances known as analeptics which stimulate the vasomotor and respiratory systems when they are in a depressed state. In large doses, injected rapidly into the vein, metrazol acts as a powerful convulsant. It causes a change in the physical metabolism; the patient puts on weight, the appetite is improved. A number of cases that had been tube fed began to eat after 2 to 4 convulsions. The theory is advanced—that the different cells of the organs of the body (brain included) are sluggish because there is a lack of proper nutrition. This sluggishness occurs when the walls of the arterioles encroach upon the lumen of the vessel causing only a small blood supply to pass through for the nourishment of the cells. During the convulsion with its numerous clonic contractions, there occurs marked congestion of the sclera, of the lungs, and no doubt of the other organs of the body. This forceful pushing of the blood through the arterioles during the clonic contractions gives nourishment to the cells and recanalizes the arterioles, reestablishing a better blood circulation. With this increased supply of nourishment the sluggish cells take on new life and waste products are eliminated.

Observations have shown that schizophrenia and epilepsy rarely, if ever, are found together. Whether there is a biologic antagonism between the two disease processes is a conjecture. However, in producing epileptic attacks in schizophrenic patients, there is a change in the biochemistry and metabolism which changes the somatic economy. Friedman,[3] believes there has been a functional barrier to the

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[2] von Meduna, L., Ztschr. f. d. ges. Neurol. u. Psychiat., 152: 235, 1935.
[3] Friedman, E., The Irritative Therapy of Schizophrenia. N. Y. State J. Med., 37: 1813, Nov. 1937.

assimilation of nutritive elements in the brain and that with the use of metrazol, which is a medullary stimulant, the vital centers are affected and the sluggish vegetative nervous system is again stimulated into action.

During the past 11 months I have given metrazol to 105 patients. Knowing of the danger of this therapy to the patient, it has been my practice to explain the situation thoroughly to the relatives and to get a signed request for treatment from a responsible person. Then, a very searching physical and neurological examination is done. Cardiovascular conditions, acute infection, history of head injury, chest disease, arteriosclerosis, or other organic disturbances are contraindications for this form of treatment. During the treatments, urinalysis and physical examinations are continued. Any indication of poor response to the drug is a signal to stop further administration. Because of this very careful preparation and watchful procedure, there has been no undesirable outcome.

Some writers have advised chemical alkalization of the patient before the treatments begin because this lowers the convulsant threshold. We have not found it necessary to use alkalies to produce the desired reaction with relatively small doses of the drug. Convulsive treatments are given twice a week for a varying number of convulsions, depending upon the physical and mental reactions of the patient. They are continued if there are no contraindications until no further improvement is expected in the person under treatment. The largest number of convulsions I have given is 42 with little improvement while treatments have been discontinued after 6 to 8 convulsions because of apparent remission of mental symptoms.

From 6 to 10 patients are treated daily in separate rooms, in a portion of a ward that can be closed off from the other patients. The bed is protected with a rubber sheet as occasionally there is loss of control of the sphincters. The patient is not given anything by mouth for several hours before treatment; otherwise there would be danger of inhalation of the vomitus which might be ejected from a full stomach during convulsion. To make this food problem as simple for the patient as possible, the treatments are given at 7:30 a. m. Thus the patient is ready for a lunch at 10:30 and dinner at noon. Care is exercised to see that the patient does not have false teeth, gum, or other objects in his mouth before treatment is given.

I have found it very satisfactory to have a tray containing all of the necessary equipment which can be carried from one room to another. This tray contains:

1. Rubber diaphragm bottle of 10 percent sterile metrazol solution, 100 cc. size.
2. Rubber tourniquet.

3. Sterile glass syringes of 5 cc. and 10 cc. done up in individual sterile towel with 2 hollow needles, gauge 19.

4. Mouth gags made from tightly rolled cotton covered by gauze, length about 6 inches, diameter ¾ inch.

5. Alcohol solution 70 percent and cotton balls for sponging arm.

6. Stop watch for timing phases of convulsion.

7. Ampules of stimulants and sterile 1 cc. syringe (adrenalin, caffeine-sodio-benzoate, etc.).

8. Notebook for careful recording of date, dose of medication, time of phases of convulsion, reaction following the convulsion, pupillary changes and other neurological or physical findings.

After the preliminary physical and neurological examination, we are now ready to give our first treatment. The first dose of the 10 percent metrazol solution is about 3½ cc., but this amount varies with the weight of the patient. The tourniquet is applied, the arm cleansed, and the solution is injected very rapidly into the vein. After the injection the arms are held closely to the body, the lower limbs are extended and the jaw is supported. At least three nurses or attendants are necessary at the time of injection, one nurse remaining with the patient after the convulsion has ceased so the patient cannot roll out of bed.

Almost immediately the patient gives a little cough and complains of a queer feeling. This sensation often causes him to ask for help because he believes he is dying. In from 6 to 13 seconds he begins to have short clonic contractions, with blinking of the eyes, lasting about 5 seconds. This is followed by a tonic phase lasting from 5 to 20 seconds. The patient yawns, occasionally dislocates the jaw; all extremities are rigid and extended; the hands are in the position of tetany; the corneal reflexes are absent; and the eyes are rolled upward or outward. Opisthotonos is the usual position. During this stage cyanosis is present, often to a marked degree.

The clonic state again enters, and from a beginning of fine rapid tremors the patient goes into massive convulsive movements lasting from 20 to 40 seconds. These movements slow down, the patient takes a deep breath and stertorous breathing with marked salivation begins. The cyanosis disappears and there is often a flushing of the skin. No definite reaction of the eyes may be anticipated at this point. That varies, not only with different patients, but in the different convulsions of a single patient. There may be either a divergence or convergence of the pupils which are somewhat dilated, or a turning of the eyes in any direction, but usually upward. At this stage, the patient falls asleep. The sleep is sometimes preceded by a few minutes of restlessness, confusion, and rolling around. The period of sleep varies from a few minutes to half an hour. He may remain somewhat confused for another half an hour or an hour, but

is able to care for himself. Others regain their senses within 10 minutes after the convulsion and are allowed to get up and walk. They are closely supervised by a nurse because they may be unsteady. Within an hour all signs of the convulsions have disappeared and each patient is able to attend the occupational therapy class or carry on any other of his activities as if no convulsion had occurred. No serious accident has occurred in this series, partial dislocation of the jaw being the only unusual finding. This dislocation is easily reduced before the convulsion is ended and in no case has the patient complained of pain after returning to consciousness.

In my effort to keep the dose of metrazol at a low level, about 20 percent of the injections did not produce convulsions. The practice of not increasing the dose until the patient no longer reacts has kept most of the patients at 6½ cc. throughout their courses of treatment.

A summary of our results shows:

Result	Male	Female	Total
Unchanged	9	7	16
Improved+	4	7	11
Improved++	5	10	15
Improved+++	12	6	18
Dismissed on visit	12	8	20

The patients in the above group have been at the hospital for periods varying from 13 years to new admissions who have been ill only a few months. In general, those who have been mentally ill for several years made little improvement or were unchanged. The recently admitted cases showed the marked changes and were the ones who went home, although not all of the recent cases did improve after treatment.

Case Reports

Case 1.—G. M. 33571. Male, white, age 39, married, laborer. Family history is negative—moderately alcoholic. Early life uneventful, grammar school education. Physical condition good.

Diagnosis.—Dementia praecox, paranoid.

Admitted October 11, 1935. He was actively hearing threatening voices, believed his name was called out over the radio, and accused his wife of infidelity. He was suspicious of those about him, irritable, and stayed by himself.

Treatment.—Metrazol therapy was started. After four convulsions there was a marked change in his attitude. His hallucinations ceased, he became cooperative and developed insight into his condition. After eleven convulsions he was allowed to return to his home. At the present time he has obtained work and is supporting his family.

Comment.—This case illustrates apparent recovery in a moderately old illness.

Case 2.—H. N. 33091. Female, white, single, age 30, registered nurse. Family history is negative. Early life, school, and hospital training school life were normal. She was following her profession until admission on December 3, 1936, to another mental hospital where she was actively hallucinated in the auditory

sphere, very impulsive and, although apparently cooperating, would without warning attack those near her and where she received 41 insulin treatments with little or no improvement.

She was transferred to this hospital and continued to have periods when she seemed slightly confused; at other times she reacted violently with no apparent cause except the auditory hallucinations.

TREATMENT.—Twenty-six convulsions were induced by metrazol with very little improvement. During the last 6 months there has been no change. The patient remains in a disturbed ward due to her impulsive tendencies.

COMMENT.—This case illustrates no improvement following both insulin and metrazol.

Case 3.—C. McA. 33667. Female, white, aged 24, single, housework at home. Family history is negative for mental disease. Early life is not abnormal. High school graduate.

DIAGNOSIS.—Dementia praecox, catatonic type.

This girl has had a shut-in personality all of her life. Although she had some girl friends, she showed no interest in the opposite sex. Her work as housekeeper was confining even though there were only adults in the family circle. She was always interested in church and for several months before her commitment, spent some time each day in church. Hallucinations of hearing began to appear. She believed Jesus and the Virgin Mary were talking to her. She assumed poses for long periods of time; finally became mute, blocked, showed stiffness and resistance to motion of the extremities. Food was refused and, at the time of admission, the physical condition showed emaciation.

TREATMENT.—Metrazol treatments were started, with considerable improvement after the second convulsion. Hallucinations ceased, resistiveness and refusal of food changed to cooperation and a good appetite. There was interest in what was going on about her and after six convulsions, the treatments were discontinued. Physical and mental improvement continued, and the patient left the hospital for a visit 4 months ago. She is continuing to make a very normal appearance and has complete insight.

COMMENT.—This case illustrates apparent recovery following a short illness.

Case 4.—M. M. 27701. Male, white, married, millworker, admitted July 1927. The family history is negative as far as mental illness is concerned. The patient was born in the Azores, coming to America as a young man. Until a short time before admission, no evidence of mental illness was apparent. He was always of a quiet, reserved nature and had few friends. He was a steady worker, not inclined to mix with his fellow workers, but was well thought of by them. A few weeks before admission he was very irritable and assaultive to his wife and child. His wife found it necessary to go to live with a friend because of his actions. This led him to believe that she was interested in some other man. He began to watch the house where she was living. Hallucinations of hearing developed, the patient thought God was talking to him.

On admission he continued to be impulsive, was mute, sat in one position for long periods of time, paid little or no attention to his surroundings. Except for an occasional impulsive outbreak, he sat in a stilted manner month after month until he reached a state where he remained in a fixed position. All efforts to interest him in activities about him failed. He was mute to questions.

TREATMENT.—Metrazol was given at this late date to see what effect it would have on the patient. After the first five convulsions, considerable improvement was seen. The patient wandered around the ward, leaving this chair where he had remained for several years. He answered questions in monosyllables and

gained in weight. Fourteen injections in all were given and the patient was sent out to work with a group. He attempted to run away so often that he could not be held in a group and was returned to the occupational therapy class. The last metrazol treatment was given 4 months ago. Since that time there has been a gradual decline in the patient's activities. He is spending more time in a rigid position, speaks less frequently and is slowly returning to his former condition.

COMMENT.—This case illustrates no improvement in a chronic condition.

SUMMARY

In many cases of schizophrenia, metrazol therapy appears to be of value.

The dose varies with the response of the patient. The smallest dose which produces a convulsion should be used.

Acute cases show a greater percentage of mental improvement. Return to former condition occurs frequently.

In our series of 80 cases, sufficient improvement was seen to allow 20 patients to return to their homes.

SOME POLLENS OF HAWAII[1]

BOTANICAL CHARACTERISTICS IN RELATION TO ALLERGY

By Commander Louis H. Roddis, Medical Corps, United States Navy

Three most important characteristics of the individual pollen grain in relation to allergy are: Size, adhesiveness, and power to carry the allergic-provoking substance.

SIZE is of great importance. In general, the larger the pollen grain, the less likely it is to cause allergic manifestations. The ragweeds, and the grasses and trees that cause the most hay fever and asthma are all small. Low ragweed (*Ambrosia elatior*) has a pollen grain only 16 microns in diameter; Bermuda grass (*Capriola dactylon*), 26 microns; and white ash (*Fraxinus americana*), 24 microns. As a rule plants with pollen grains larger than 35 microns in diameter are not likely to cause serious trouble. Field corn (*Zea mays*), has a pollen of approximately 90 microns in diameter and though a member of the allergic-producing grasses causes little hay fever even in the great corn-producing States like Iowa, Illinois, and Nebraska where thousands of square miles of corn, when in the tassel and silk, produce large amounts of pollen.

The principal reason that the large pollens cause little allergic disease is primarily a matter of gravity. The larger the pollen grain the more quickly it falls to the ground. Similarly, when a grain of

[1] Presented before the Forty-ninth Annual Meeting of the Hawaii Territorial Medical Association (a branch of the American Medical Association), May 5, 1939, at Honolulu, T. H., and published with the permission of the Territorial Association.

dust and a rock of considerable size are tossed into the air, the small grain of dust will be carried some distance, the rock falls to the ground.

Another cause is the number of pollen grains produced. A basket of the same size will hold many more peas than baseballs and pollen cases of the same size can of course hold many more small pollen grains than large ones.

The pollen grains of the tropical trees and flowers are all generally large and in consequence they probably are of less importance as causative agents in hay fever and asthma than the small and light pollens of the grasses, weeds, and flowering trees listed in our textbooks. Because of the almost total absence of measurements of tropical pollens in the literature of allergy, the measurements and characteristics of some of the most common ones are given in table 1. In the past year I have made measurements and studies of the pollens of about 45 tropical species mostly from the Pearl Harbor area.

TABLE 1.—*Size, shape, and structure of certain pollen grains of Hawaii*

Common name	Scientific name	Size in microns	Shape	Adhesiveness	Quantity
Monkeypod	*Samanea saman*	94.25	Spherical	No	Moderate.
Hibiscus	*Hibiscus sinesis*	159.5	do	Yes	Do.
Night-blooming Cereus	*Hylocereus undatus*	72.5	do	Yes	Do.
Cycad	*Cycas revoluta*	75.25	do	Yes; slight	Profuse.
Ginger	*Hedychium coronarium*	65.25	do	Yes	Scanty.
Royal Poinciana	*Poinciana regia*	45.5×58	Ovoid	Yes	Do.
Fountain Tree	*Spathodea companulata*	56	Spherical	Yes	Moderate.
Pink Shower	*Cassia grandis*	21.5×43	Oblong	Yes; slight	Small.
Plumiera	*Plumiera acutifolia*	21.75	Spherical	Yes	Do.
Angel Trumpet	*Datura arborea*	49.5	Cuboidal	Yes	Do.
Allamanda	*Allamanda hendersoni*	58	Spherical	No	Do.
Kukui	*Aleurites Moluccana*	60	do	Yes	Do.
Koa	*Acacia koa*	52.5×65	Oblong	Yes	Moderate.

It must be kept in mind that a pollen that is of large size cannot on that account be entirely absolved of guilt in a given case for if people sit under a flowering tree, or flowers are carried into the house, or worn on the clothing, there may be intimate contact with a sufficient quantity of the pollen to produce symptoms.

ADHESIVENESS or stickiness, is found in all the insect pollinated plants of which there are an unusually large proportion in the Tropics. Of course the purpose of this characteristic is obvious, to make the pollen adhere more readily to the insect carrying it. Such pollen, however, is not readily removed from the plant by the wind and in consequence, insect-carried pollens are usually of little importance as causative agents in allergy. Nevertheless they are incriminated in certain cases. The writer had three cases, all women, due to hibiscus pollen. The hibiscus, which is the floral emblem of the Territory of Hawaii, so designated by legislative act, is much used as a decorative

flower about the home and often worn by women in their hair or on their gowns. All three of these cases were due to this latter use of the hibiscus and the symptoms were much alleviated by giving up this practice. Yet as the hibiscus has both a large and very adhesive pollen it would on both these counts not be likely to cause allergic difficulties.

ALLERGIC-PRODUCING SUBSTANCE.—The white pine is a heavy producer of a small, easily wind-blown pollen. It is not an uncommon sight to see the ground under a white, or a Norway pine, golden with the pollen dust. Yet a patient susceptible to hay fever may wallow in the pollen with impunity for it does not contain any allergic-provoking material. This is a quality shared by all the *Coniferae* with one or two exceptions. Northern Minnesota and Wisconsin both have extensive areas covered with evergreen forest and these are havens for many hay fever sufferers. In the Tropics the cycads are heavy pollen producers but their pollen like the conifers seems to be innocuous. In this respect it is interesting to note that the cycad is a primitive gymnosperm related to the forerunners of the conifers. This ability to carry the allergic-producing antigen I have called *atopophorism.*

This possession of an allergy producing agent is probably not a common characteristic of many pollens. There are in the world and already described by botanists about 135,000 species of plants. Only a few hundred species cause practically all the hay fever and asthma, and 95 percent is caused by only three or four species.

In connection with the carrying of the allergy producing substance the number of species which are carriers of it tend to decrease in the Tropics. This is due to the difference in proportion of plant types in the temperate and the tropical latitudes. In the cooler zones of the earth's surface there is a greater proportion of grasses and such genera as the ambrosia to which the ragweeds belong. In the warmer zones the proportion of these plants is less and tropical trees, vines, and shrubs take their place. To illustrate this point, imagine all the vegetation in a northern latitude such as the Province of Alberta in Canada, collected in one large heap. About one-sixth of it would be grasses. But if all the vegetation in a province of Brazil in the Amazon valley were collected only about one-twentieth of it would be grasses. Thus as the Tropics are approached the hay fever plants cease to be relatively as numerous. The generally expressed opinion that hay fever is relatively rare in the Tropics has thus certain botanical evidence in its favor. Medical men in various parts of the Tropics have stated that hay fever was rare in their localities or even nonexistent.

In Hawaii these factors which lessen the incidence of hay fever producing plants is further enhanced by the oceanic climate. The Hawaiian group are small mountainous islands far out in the earth's largest ocean, over 2,000 miles from the nearest continental land mass, the air therefore when it reaches here is pollen free. Furthermore the high precipitation of an oceanic climate (often 40 to 60 inches of rainfall a year in the valleys), the barriers of mountains and deep mountain valleys, conspire to prevent the spread of pollen produced in the island itself. As is to be expected the air counts are very low. In table 2, Honolulu is compared with certain United States cities, and it appears that Miami alone among mainland cities is lower.

TABLE 2. *Pollen counts of some mainland cities compared with Honolulu*

Chicago	500	Miami	10
New York	150	Honolulu	28
Houston	750		

In table 3, some counts are shown taken in various parts of the island and in various situations. Of particular interest is the fact that on the windward side with the normal trade wind and at 3,000 feet no pollen was found. In the lee of a field of sugarcane 45 pollen grains were found of which 36 were from sugarcane. The traps were 100 feet from the nearest cane. Because of the extensive cane fields in Oahu there is little doubt but that a certain amount of allergic trouble is caused by the pollen during the blossoming time which is November, December, and January. The pollen grain is, however, of fair size (30 microns), is not readily windborne, and it is likely to be incriminated only when there is close association with it such as occurs among workers in the fields or those living close to the cane fields.

TABLE 3. *Various pollen counts in Oahu*

Windward side	0	Beneath mango trees	32
3,000 feet altitude	0	Beneath date palms	48
Lee side of cane field (December)	45	Beneath shower tree	6
Lee side of red top field	185		

The date palm and the algaroba are also likely sources of trouble. Though the pollen of each is primarily insect borne and wind is only of secondary importance, there is again little doubt that many cases occur when these trees are in close proximity to the houses as is so often the case. We had one case with a positive skin test to royal palm. In general, however, the most ubiquitous pollens are those of the grasses, notably Bermuda grass, and red-top. These with the date palm, algaroba and sugarcane, which it must be remembered is one

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of the grasses, are, I believe, the principal sources of trouble in the Pearl Harbor region of Oahu where most of these field studies were made. Table 4 lists these plants.

TABLE 4.—*Plants in Pearl Harbor area most likely to cause hay fever and asthma*

Common name	Scientific name	Size in microns	Shape	Adhesiveness	Quantity
Red top	*Agrostis alba*	35	Spherical	No	Profuse.
Bermuda grass	*Capriola dactylon*	26	do	No	Do.
Date palm	*Phoenix dactylifera*	25	Oval	Yes, slight	Do.
Royal palm	*Oreodoxa regia*	28	Flat-oval	Yes	Do.
Algaroba	*Prosopis juliflora*	39.5	Spherical	No	Do.
Cadena de amor	*Antigonon leptopus*	32	do	Yes	Small.
Mango	*Mangifera indica*	21.75	do	Yes	Profuse.
Sugar cane	*Saccharum officinarum*	29	do	No	Do.

One of the most pressing needs in the study of allergy at present is for some extensive statistics on its incidence. We have information on the contagious and infectious diseases, but the number of cases and age groups of asthma or hay fever is lacking. Such information would be of great value in correlation with the botanical evidence.

CONCLUSIONS

1. Tropical plants are not common causes of hay fever and asthma due to the large size and adhesiveness of their pollens and to the low proportion of allergy-producing species.
2. Pollen counts of air in Hawaiian Islands are low due to insular position, oceanic climate, and topographical features of the islands.
3. Probably less hay fever and asthma occur here than in the continental United States.
4. Relatively more asthma than hay fever occurs since the former is frequently due to other inhalants (animal dandruffs, dust, etc.) and also because food allergy is frequently a factor in asthma and not in hay fever.
5. Most frequent sources of pollens causing allergy are the common grasses, algaroba, sugarcane, date palm, and the commonly used flowers especially hibiscus and the so-called chain of love (cadena de amor) or Mexican creeper.

ENDEMIC TYPHUS OF THE SOUTHEASTERN STATES

By Lieutenant Earl F. Evans, Medical Corps, United States Navy

In recent years an increasing interest has been manifested in the rickettsial diseases. In the Southeastern States, especially in those States bordering on the Gulf of Mexico, this interest has been centered to a large degree on endemic typhus fever. That this disease warrants this recent interest is evidenced by a study of the

public health reports from this area, which show a surprising and steady increase in the number of cases reported annually (table 1). The occurrence of 2 cases of sporadic or endemic typhus in the naval hospital at Pensacola, Fla., during the fall of 1937 stimulated the author's interest in this subject. Since 1934 there have been 5 patients with endemic typhus fever admitted to the Pensacola Naval Hospital, and since 1932 there were 25 cases diagnosed in the Charity Hospital of Louisiana at New Orleans. Of the 5 patients admitted to the Pensacola Naval Hospital, 2 were service personnel and 3 were veteran patients.

TABLE 1.—*Endemic typhus*[1]

Year	Louisiana		Texas		Mississippi		Alabama		Georgia		Florida	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
1929	1	0	8	1	0	0	72	11	57	2	48	1
1930	0	0	13	0	0	0	67	4	134	9	39	2
1931	1	0	43	0	0	0	80	4	127	6	31	3
1932	17	0	227	2	0	0	237	11	308	7	42	5
1933	11	1	398	7	1	0	823	41	625	30	54	2
1934	17	3	465	16	0	0	271	18	414	26	35	7
1935	20	1	265	14	5	0	219	18	489	28	27	4
1936	12	0	327	23	6	0	369	16	817	48	55	9
1937	23	1	453	28	15	1	478	21	1,092	54	107	11
1938	27	2	497	32	24	1	341	23	1,017	47	75	10
Total	129	8	2,696	123	51	2	2,957	167	5,080	257	513	54

[1] As reported to the U. S. Public Health Service.

The presence of endemic typhus in the Southeastern States was first brought to general notice by the work of Maxcy, who made an epidemiological survey of this region in 1922. There is reason to believe that the disease has existed in this section since the earliest inhabitation. However, for the time prior to the accurate clinical differentiation between typhus and typhoid we can only speculate as to its extent and nature.

At the present time the United States Public Health Service recognizes two distinct types of typhus fever: (*a*) The classical European typhus, which occurs in epidemic form, is associated with bodily infestation with infected lice, and has a high mortality rate; (*b*) endemic typhus, which follows contact with infected fleas, does not appear in epidemic form, and has a relatively low mortality rate. Further differentiation of endemic typhus from Brill's disease on clinical and epidemiological features is stressed by certain investigators (Mooser, Anderson, and Kemp.) Anderson's contention, that the reported cases of Brill's disease were originally infected in Europe and were merely an American exhibition of European typhus, Mooser's presentation of epidemiological differences and an analysis of the cases reported from the South, all strongly support opposition to the use of the term, Brill's disease, as a synonym for endemic typhus.

EPIDEMIOLOGY OF ENDEMIC TYPHUS FEVER

The epidemiology of endemic typhus fever of the South has certain features which distinguish it from the types of typhus seen elsewhere throughout the world. Endemic typhus is primarily a disease of young and middle-aged males. Female cases, while not uncommon, are outnumbered by more than 3 to 1. In reported cases, according to Kemp, whites outnumber Negroes more than 20 to 1. However, this difference is probably due, to a large degree, to the difficulty of recognizing the exanthem in the Negro patient. The seasonal incidence of the disease is largely confined to the months from August to November. Because the disease does not become epidemic, because it manifests a relatively low mortality, and because many physicians are notably lax in reporting a disease of this nature, the annual number of cases, especially in the rural areas, must be greater than the number of reported cases.

By recent investigators, especially Dyer, Badger, Ceder, and Workman, it has been proven that endemic typhus is transmitted to man by an infected rat flea (*Xenospsylla cheopis*). Both infected fleas and infected rats are recognized as reservoirs for the disease. In late years there has been evident a spread of the disease from its early focus in the seaport towns into the rural areas. Rumreich has maintained that the increasing incidence in the rural sections is due to the spread of the virus into rodents other than the rat. This contention has been substantiated by Brigham and Dyer, who have shown that eight species of animals, chiefly rodents native to the typhus-infected section of the United States, are susceptible to infection with the typhus virus. They also recovered the virus from a rodent trapped in a rural section of southeast Alabama. In the two cases herein reported it is of interest that one patient was on duty in the commissary storeroom and admitted handling several dead rats about 10 days prior to the onset of his illness, and the second patient had, along with his neighbor, killed and handled rats about 14 days prior to onset. In this latter case investigation revealed that the patient's neighbor was concurrently ill with a similar illness and his civilian physician reported a positive agglutination for *Proteus X19* had been obtained from the Florida State Board of Health Laboratory.

CLINICAL DESCRIPTION OF ENDEMIC TYPHUS

The clinical course of the disease is distinguished from European typhus chiefly by the comparative mildness of the mental manifestations, the more rapid recovery, and the low mortality rate. The onset

in the large majority of cases is abrupt, with fever, recurring chills, apathy, severe headache, generalized muscular aches, and usually some degree of photophobia. Occasionally the onset is more gradual and follows 4 or 5 days of prodromal malaise, making differentiation from typhoid difficult in the early stage. The temperature rises rapidly to 101° to 105° and remains elevated with only slight morning remissions for 14 to 16 days, when it falls by crisis, or, as in the majority of cases seen, by rapid lysis. In a small number of cases the temperature begins to subside about the ninth day, gradually returning by regular remissions to normal. The pulse is characteristically slow in relation to the temperature elevation, but the dicrotic pulse of typhoid is absent.

The exanthem of endemic typhus may be the outstanding diagnostic feature of the disease, appearing about the third to the sixth day of the disease as a more or less characteristic discrete, pink, maculopapular rash. The lesions appear first in the axillae, spreading over the lower chest and upper abdomen and, later, on the flexor surfaces of the arm and forearms, but the palms and face are spared. In mild cases the skin manifestations may be so transient that unless looked for daily they may escape detection. In the more severe cases the color becomes deeper, possibly purpuric, and at times the lesions may present a dark central point, giving the appearance of a flea bite (Maxcy). The duration of the rash varies greatly. In mild cases a dirty pink macular eruption may last only a day, while in severe infections a subcuticular mottling may persist for about 3 weeks. In general, the exanthem is an index to the severity of the infection and reflects the degree of involvement of the central nervous system. Maxcy has pointed out that the chief characteristic of the rash is its irregularity in distribution, depth of color, elevation and outline.

The presence of conjunctival congestion, lacrymation, and photophobia is suggestive of measles, but the temperature curve, the marked prostration, and mental apathy serve to prevent this confusion before the eruptions appear. In children differentiation from measles in the preeruptive stage might prove very difficult. To add to this difficulty is the fact that typhus is often accompanied by a catarrhal bronchitis, as was the case in the second patient herein reported.

Gastro-intestinal features are usually confined to marked anorexia and obstinate constipation. At times in children nausea and vomiting may accompany the onset, and diffuse abdominal pains may be a later feature. In the average infection the cardiovascular system shows little toxic effect except for the relative bradycardia. How-

ever, in severe infections and in elderly patients with impaired cardiac reserve, the chief cause of death may be acute cardiac damage. Phlebitis and vascular thrombosis have been reported (Maxcy and Blatteis), but these complications occur in less than one percent of cases. Auricular fibrillation was observed for several days in a fatal case reported by Kemp. The nervous system manifestations of mental apathy, clouding of consciousness and mild delirium are usually prominent. There seems to be a complete absence of sequelae referable to changes in the central nervous system.

The laboratory findings, with the exception of the Weil-Felix reaction, are not distinctive. The total white blood cell count usually shows a leukopenia of 3,000 to 6,000 cells per cu. mm. However, not infrequently counts as high as 12,000 to 15,000 are encountered. There is usually a moderate shift to the left in the Schilling count. The frequency of such wide variations in the total white blood cell count may only serve to confuse the diagnostic problem. Spinal fluid findings, with the exception of an occasional moderate increase in pressure, are always normal. The Weil-Felix reaction is the best criterion in the diagnosis, but even this procedure is probably not infallible, as cases have been reported (Kemp) that present all the features of typhus with the exception of a positive Weil-Felix agglutination reaction. Agglutination of *Bacillus proteus X19* by the patient's serum begins in increasing dilutions after the tenth day. Whereas a complete agglutination in a dilution of 1:160 is usually considered necessary to establish the diagnosis, if serum taken at 2-day intervals is originally negative, then becomes positive in increasing dilutions, such as 1:20, 1:40, and 1:80, this could be considered positive before the agglutination is complete in the 1:160 dilution.

Endemic typhus may be differentiated from typhoid fever by the more sudden onset, the absence of positive blood and stool cultures, and the Widal reaction. Pneumonic plague, with its similar mental features, is easily ruled out by the relative mildness of the condition and the characteristic rash. In the preeruptive stage of typhus, influenza may be a confusing possibility. Encephalitis shows no rash, a more gradual onset, and no relative bradycardia. Differentiation from Rocky Mountain spotted fever, eastern type, may only be accomplished with absolute certainty by guinea pig inoculation. Endemic typhus virus, when injected, produces only a febrile reaction after 7 to 10 days, with none of the characteristic scrotal lesions of Rocky Mountain spotted fever. However, R. E. Dyer has stressed several clinical differences between these two conditions which are of material help in their differentiation (table 2).[2]

[2] Dyer, R. E., Personal communication.

TABLE 2.—*Differentiation between endemic typhus and Rocky Mountain spotted fever*

	Endemic typhus	Rocky Mountain spotted fever
1. Season	Late summer and fall	Spring and early summer.
2. Exposure	Urban usually	Rural.
3. History of bite	Not often obtained (flea) *	Positive tick in about 50 percent.
4. Incubation period	7 to 14 days	2 to 12 days.
5. Clinical course	Usually mild (mortality about 3 percent).	Usually severe (mortality up to 80 percent—but may be milder in East). Average for United States about 22 percent.
6. Fever and pulse	Pulse lags behind; fever gone by sixteenth day.	Both up; fever gone by twenty-second day.
7. White blood count	Usually normal or slight leukopenia	Usually between 12,000 and 18,000.
8. Rash: Onset	Fifth to seventh day	Second to fifth day.
Distribution	First on trunk, thence to extremities. *Rarely on face or head.*	First on wrists and ankles, thence to trunk, face and head.
9. Convalescence	Short	Long.

* Rash in Rocky Mountain spotted fever is centripetal in distribution and spread (as is smallpox) and also shows predilection for buttocks; in endemic typhus, it is centrifugal (as is chickenpox). In both diseases the rash is usually most marked at site where it first appears.

Endemic typhus in this country is generally considered to be a mild disease with a low mortality rate of from 1 to 3 percent. It seems, however, that this rate does not hold true when the reported case mortality is figured from the public health reports from the Southeastern States for the 10-year period 1929 through 1938, where the rate is found to vary from a high of 8.06 percent in 1929 to a low of 3.0 percent in 1932 (table 3), as will be seen from table 3

TABLE 3.—*Incidence of endemic typhus, southeastern States, 1929–38*

Year	Cases	Deaths	Mortality rate (percent)	Year	Cases	Deaths	Mortality rate (percent)
1929	186	15	8.06	1935	1,025	65	6.3
1930	253	15	5.9	1936	1,586	96	6.0
1931	283	13	4.5	1937	2,168	116	5.3
1932	831	25	3.0	1938	1,981	115	5.8
1933	1,921	81	4.2				
1934	1,202	70	5.8	Total	11,436	611	5.3

which presents the cases, deaths, and mortality rates as reported from the States of Louisiana, Texas, Mississippi, Alabama, Georgia, and Florida for this 10-year period. In 1937 there were 2,393 cases of endemic typhus reported in the United States with 142 deaths, a case mortality rate of 5.9 percent. In 1938 there were 2,233 cases reported, with 137 deaths, a case mortality rate of 6.1 percent. In the Southeastern States over the entire 10-year period there were 11,427 cases reported, with 611 deaths, a case mortality rate of 5.3 percent. It is possible that these figures do not represent the true mortality rate, there being reason to believe that there is need for more specific criteria for diagnosis and more universal reporting of

the milder cases. Fatal cases, for the most part, are confined to older patients with accompanying myocardial or renal impairment. Acute myocardial damage and bronchial pneumonia have been given as the cause of death in some cases. With the increasing number of cases occurring annually, the possibility of the virus building up its virulence year by year has been suggested. However, the case mortality rates over the 10-year period studied show no evidence of a regularly increasing rate.

The treatment of endemic typhus is purely symptomatic at the present time. Medication has little or no effect on the temperature elevation or duration. Hydrotherapy for the pyrexia and appropriate analgesia are the only measures having any beneficial effect. The disease appears to be self-limited, the temperature subsiding by crisis or rapid lysis on the fourteenth or fifteenth day. A moderate persisting bradycardia and a moderate hypochromic microcytic anemia have been the only sequelae seen.

Report of Cases

Case 1.—L. C. T. (VBP), white male, aged 44 years, was admitted September 19, 1937, with a history of sudden onset of chill 3 days prior to admission followed by fever of 103° and pulse of 80, severe headache and marked prostration, generalized muscular aches, somnolence and mental confusion, frequency and urgency of urination. Significant in the past history was the general good health prior to the sudden onset, the handling of rats about 15 days prior to onset, and the fact that he had received a course of typhoid-paratyphoid inoculations in 1918.

Physical findings.—The patient was somnolent, with clouding of consciousness and disorientation as to time and place. The conjunctival vessels were congested and several small stellate hemorrhages were seen in the periphery of both fundi. There was marked oral sordes and the mucous membranes were dry and congested. The tongue was heavily coated and protruded in midline with coarse tremor. Heart rate was 86 with irregular rhythm due to frequent ectopic beats. B. P. 132/86; T. 103.2°. There was coarse tremor of extended fingers. Deep and superficial reflexes were present and equal. Speech was slow and at times incoherent, but not impeded by slurring. Patient read printed words and recognized pictured objects with difficulty. With his eyes closed, patient was unable to recognize common objects, such as a key, pencil, and coin placed in his hand.

Laboratory findings on admission.—*Urine:* Albumin present in dilution of 1:20, few hyalin casts and occasional leukocytes.

Blood: Hgb., 80 percent (Hellig); RBC, 4,050,000; WBC, 13,500; bands 1, segs 61, lymphs 29, monos 9; negative for malaria plasmodia (Wright and Giemsa).

Spinal Fluid: 14 cells (lymphocytes). No increase in globulin. Kahn negative. L. C. G. 0000000000. Smear and culture negative for organisms.

Clinical course.—Several pink maculopapular pinhead size lesions appeared on the lower chest and upper abdomen. These lesions faded under digital pressure. WBC, 9,200; no change in differential count. Agglutination for *E. typhi* positive 1:160, for *S. schottmuleri* positive 1:80, for *S. paratyphi* positive 1:160, for undulant fever and *Proteus X19* negative. Urea nitrogen was 16 mgms. and blood sugar 93 mgms. per 100 cc. whole blood. An x-ray of the

lung fields was negative for pathology. Two days later the rash had disappeared. During the following 7 days the patient's condition remained the same, with the temperature ranging between 102° and 103.6°. Repeated cultures of blood and stools were all negative. Repeated thick smears for malaria were all negative. Obstinate constipation, requiring enemata, persisted. Blood Kahn was negative. On October 12th, 15 days after onset, temperature fell by rapid lysis and mental condition improved rapidly. Naval Medical School reported agglutinations for *Proteus X19* complete in dilution 1:1280 and partial in 1:5120 in serum obtained on the twelfth day of the disease. Temperature remained within normal limits and patient improved rapidly except for a RBC of 3,760,000. Patient was discharged well 32 days after admission.

Subsequent investigation revealed that patient's companion in the rat handling suffered a concurrent similar illness and his serum, tested at the Florida State board of health laboratory showed a positive agglutination for *Proteus X19* in dilution of 1:1280.

Case 2.—M. H., Sc. 1c, United States Navy, white male, aged 26 years, was admitted on October 29, 1937, complaining of intense headache and fever of 3 days' duration, 3 or 4 periods of chilliness since onset, but no shaking chill, severe aching pains in back, arms and leg muscles, and occasional nonproductive hacking cough. No gastro-intestinal or urinary disturbances. For 24 hours prior to admission temperature, recorded at the air station dispensary, ranged from 101° to 104°. Past medical history was irrelevant. Patient had been on duty in the commissary storeroom and about 10 days prior to onset had removed some dead rats from traps. A second course of typhoid-paratyphoid inoculations was completed on March 19, 1939.

PHYSICAL EXAMINATION.—On admission patient was somnolent but oriented, and answered questions logically when aroused. Skin was hot and sweating was profuse. There was an eruption present consisting of pink maculopapules, varying from 1 to 3 mm. in diameter, distributed sparsely over the chest, upper abdomen, and anterior surface of the forearm. T. 104.2°, pulse 78. The conjunctival vessels were congested and moderate lacrymation and photophobia were present. No Koplik spots were seen. There was moderate oral sordes with a heavily coated tongue. Lungs and cardiovascular system revealed no abnormal findings. The spleen was palpably enlarged and slightly tender. Superficial and deep reflexes were present and equal bilaterally.

LABORATORY FINDINGS.—Blood: WBC, 5,200; juv 1, bands 7, segs 52, lymphs 33, monos 7. Thick and thin smears were negative for malaria.

CLINICAL COURSE.—On day following admission rash became more pronounced and petechial in character. Patient had three light chills and clouding of consciousness developed. A blood count at this time showed WBC, 4,600, juvs 3, bands 11, segs 76, lymphs 7, monos 3. Urine was chemically and microscopically normal. Repeated smears for malaria were negative. Serum on this date (fifth day of disease) showed no agglutination for typhoid, paratyphoid, undulant fever, typhus fever, or Rocky Mountain spotted fever. Blood Kahn was negative. Blood and stool cultures produced no growth. On the sixth day after admission the rash had disappeared, but the temperature was still ranging between 101° and 104° with a pulse range of 78 to 84. During this period patient developed a catarrhal bronchitis with frequent cough, mucoid sputum and coarse rales in the intrascapular region. On the eleventh day of the illness the temperature did not go above 101.2°, and there was some symptomatic improvement. Blood count showed WBC 8,950; Hgb 70 percent; RBC, 3,880,000; Widal negative. Serum taken on this date showed *Proteus X19* agglutination complete in dilutions of 1:640 and partial in 1:1280. On the fifteenth day of the illness the temperature dropped by rapid lysis and patient was symptom free except for marked malaise. Patient was discharged to duty, well, 30 days after admission.

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THE TREATMENT OF CHEMICAL WARFARE CASUALTIES

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It may be of interest to first discuss briefly chemical warfare in the light of recent developments. This type of warfare, as developed during the World War, proved to be an effective means of covering those areas not readily reached by shell fire, such as caves or dugouts on the far side of a hill or cliff. It was found to be effective for blanketing low-lying areas and woods. It often proved to be more effective than high explosives in denying certain areas to the enemy.

With so effective an agent at hand one must wonder why chemical warfare has not been used in the present conflict. The reasons are many and of unequal importance. First, it is necessary for us to recall that the present belligerent nations signed the Geneva Protocol in 1925, abhoring the use of chemical warfare in the future. A second reason may be the effective preparedness of the leading nations in both offensive use of chemical agents and in defense against this type of warfare. Finally, it is also possible that such a valuable weapon will be reserved until troops are ready to move, in other words, its use will depend on the season, weather, and strategy.

The Navy is rather fortunate in its position, especially when its units are active at sea. Such units are very poor targets for a chemical attack. However, when these ships return to their bases they will become fair targets for this type of warfare.

Since the World War many reports or rumors have appeared concerning the development of new and deadly war gases. Only a few of the approximately 44 agents used during that conflict proved to be outstandingly effective, and in the event of the employment of chemical warfare during the present war, it would not be surprising to find that main reliance would still be placed on these same agents, and not on newly discovered compounds.

In the classification of chemical warfare agents, there are two methods which are in general use, one of which utilizes the tactical purpose as the basis for classification, while the other is based on the physiological action of these agents. This latter classification is the one we shall use in this discussion and is called the physiological classification. The various groups are not absolute in their differentiation, as some agents may have more than one action on the body, and therefor will overlap somewhat in their physiological effects.

PHYSIOLOGICAL CLASSIFICATION OF CHEMICAL WARFARE AGENTS

I. Lacrimators (tear gases):
   Examples:
      Chloracetophenone (CN).
      Brombenzylcyanide (CA).
II. Irritant Smokes (sternutators, sneeze gases)
   Examples:
      Diphenylchlorarsine (DA).
      Diphenylaminechlorarsine (DM).
III. Incendiaries
   Examples:
      Thermit.
      White phosphorus (WP).
IV. Lung Irritants
   Examples:
      Chlorine (CL).
      Phosgene (CG).
      Chlorpicrin (PS).
      Nitrous fumes (incidental gas).
V. Vesicants
   Examples:
      Mustard gas (HS).
      Lewisite (M-1).
      Ethyldichlorarsine (ED).
VI. Paralysants
   Example:
      Hydrocyanic acid (HCN).
VII. Asphyxiants (incidental to explosions)
   Example:
      Carbon monoxide (CO).

LACRIMATORY OR TEAR GAS

Lacrimatory gases are those agents which, in low concentration, produce eye irritation and weeping. Other agents such as chlorpicrin, mustard gas, and irritant smokes may also produce lacrimation, but to a lesser degree.

SYMPTOMS.—These agents, in effective concentrations, cause an immediate localized irritation of the sensory endings of the conjunctival surfaces of the eye, manifested by a stinging sensation, spasm of the eyelids and a copious flow of tears resulting in temporary blindness, and it is often necessary to lead the affected individual out of the contaminated atmosphere.

Higher concentrations, such as might be met within a closed compartment, will produce irritation of the upper respiratory tract, accompanied by a sensation of burning and constriction of the throat, discomfort in the chest, and possibly nausea and vomiting. Pulmonary irritation, if severe, may lead to lung changes and subsequent pulmonary edema.

These agents also act as local irritants to the skin, producing a burning and stinging sensation. Some persons are so sensitive that repeated exposure may cause an obstinate dermatitis.

TREATMENT.—Lacrimated individuals should leave the contaminated atmosphere, face the wind, and allow it to blow into their eyes. The eyes should not be rubbed. Clothing and equipment should be loosened and shaken so as to get rid of the entrapped gas. If the gas has been blown into the eyes, as by shell or bomb explosion, normal saline irrigations should be used. It is seldom necessary to send these casualties to the dressing station.

Men suffering from severe skin irritation should bathe with soap and water, or apply 70-percent alcohol solution to the irritated areas to dissolve and remove the agent.

Cases of acute bronchitis should receive the same care as is recommended for lung-irritant casualties.

IRRITANT SMOKES

Irritant smokes or sternutators are agents which can be dispersed by heat in the form of minute solid particles.

SYMPTOMS.—When unprotected men are exposed to low concentrations of irritant smokes, symptoms do not appear for several minutes, but then increase rapidly in severity, even though the subjects have left the contaminated atmosphere. Men who have adjusted the mask immediately after exposure, often experience an apparent increase in irritant symptoms from delayed action of the agent, and mistakenly believe that this condition is due to a leak in the mask, lose confidence

in the latter and remove it. Men should be warned concerning this latent period and the characteristic increase in severity of symptoms.

Affected men experience irritation of the nose and throat, lacrimation, and an irritation in the chest with a strangling type of cough. Excessive nasal secretion and salivation are present. Prolonged exposure produces aching pains in the stomach, numbness of the limbs, and sometimes sharp pains in the extremities. A prominent symptom in severe cases is a characteristic irritation of the accessory sinuses causing sinus headache, stinging of the face and lips, and a feeling of intense misery and discomfort. Individuals so afflicted are unable to carry on their duties.

Some casualties manifest temporary loss of mental control, often requiring restraint to prevent injury to themselves. Other cases show such weakness of the legs that walking is difficult.

Men should be warned not to use contaminated water for drinking or washing purposes. During the World War failure to observe this rule resulted in cases of arsenic poisoning.

The acute moderately severe cases gradually clear up in a few hours. The severe type of casualty will usually be able to return to duty in a day or two.

TREATMENT.—Remove casualties to fresh air and change their clothing. Very small amounts of irritant smokes in clothes or around compartments will cause persistence of symptoms. Flush the nose and throat with 5 percent sodium bicarbonate solution or normal saline solution. The nasal irritation may be relieved by breathing a low concentration of chlorine gas such as is evolved from a bottle of high-test bleach powder. Sedatives may be used to relieve headache and the more severe symptoms.

INCENDIARIES

The incendiaries are materials which can be used under field conditions to set fire to grass, buildings, and other articles of a combustible nature. Such materials cause heat burns if they come in contact with the skin. Some of the common incendiary agents are thermit, solid oils, and white phosphorus. The burns resulting from thermit and the oils are treated as ordinary heat burns. White phosphorus will be considered in detail.

WHITE PHOSPHORUS.—White phosphorus is a casualty agent, an incendiary, and a smoke producer. It can be used in bombs or shells, and the flying particles may become imbedded in the skin, where they continue to burn as long as air can reach them. Treatment consists of immersing the affected part in water or using a wet sponge to stop the phosphorus from burning. The solid particles are picked out of the skin. As phosphorus melts at 44° C. (112° F.) it is ad-

vantageous to apply hot water at this temperature or above and remove the melted phosphorus from the wounds with a gauze sponge. Care should be taken that all particles are removed.

An excellent first-aid procedure, if available, is the prompt flooding of the burning phosphorus with a 1- to 2-percent solution of copper sulphate in fresh or sea water. This solution forms a thin coating of copper compounds on the phosphorus particle which stops the burning immediately, and the coated particle can then be picked out of the flesh.

It is not advisable to use oils or fats in dressings unless it is certain that all particles have been removed. Tannic acid treatment, as used for heat burns, is recommended. There is a tendency for these burns to be slow in healing. The danger of phosphorus poisoning by absorption through the burned areas is minimal.

LUNG IRRITANTS

This group of agents was responsible for a large number of casualties during the World War. The chief agents are chlorine, phosgene, and chlorpicrin. Of these, chlorine and phosgene were used extensively in that war. Phosgene may be used in the future, and will probably be dispersed from large shells or large cylinders. The pathological findings characteristic of these gases will be considered separately.

CHLORINE GAS, when inhaled in high concentrations, has a destructive action on the mucosa of the upper respiratory passages, resulting in an acute inflammation and edema of the supporting tissues followed by a sloughing of the mucosa. The lungs are voluminous, firm in consistency, and do not collapse. The pleural cavities contain an excess of fluid. Edematous fluid fills the alveoli and bronchial tree. Microscopic examination reveals disruptive changes in the alveolar septa, the alveoli in some zones being filled with coagulated edema fluid, while the capillaries are engorged with blood and obstructed with fibrin strands. The right heart is acutely dilated due to the increased resistance in the pulmonary capillary bed, incident to the formation of fibrinous thrombi.

CHLORPICRIN poisoning is essentially similar to that of chlorine poisoning. The edema and congestion of the lungs are usually less marked, depending upon the concentration breathed and the length of exposure.

PHOSGENE, in low concentrations, does not affect the upper respiratory tract, but the lungs are found to be voluminous and heavy. Their surfaces show petechial hemorrhages and alternating patches of emphysema with consolidation. On section, irregular alternating patches of acute edema and emphysema are found. The right heart is considerably dilated. Microscopic examination shows the alveoli

filled with an albuminous fluid. Much fibrin is present in the alveoli, alveolar walls, and in the pulmonary capillaries.

Symptoms.—Acute cases of chlorine poisoning, such as those which resulted from the cloud attacks during the World War, showed the following symptoms:

Intense irritaton of the upper respiratory tract with catching of the breath, strangling cough, sense of constriction about the chest, gasping respiration, severe pain over front of chest, and rapid, shallow respiration. Deep breathing is painful. Vomiting is often present and patients soon show fatigue and prostration. When edema of the lungs appears the respiration becomes rapid and shallow and the face and extremities are cyanotic.

Acute cases resulting from moderate or low concentrations of phosgene show practically no irritation of the upper respiratory passages. Coughing is slight. After such an exposure a man may be able to carry on his duties for an hour or two with the occurrence of only slight symptoms, but he then suddenly becomes worse, cyanosis appears, and he may develop a circulatory collapse. Some of these cases during the World War walked back to the first-aid station, but a few hours later suddenly developed pulmonary edema and died from circulatory collapse.

Acute pulmonary edema is an important sequela to changes in the lung parenchyma. Study of lung changes from the beginning shows that damage to the capillaries may be present as early as half an hour after gassing. These capillaries become dilated, much engorged, and there is evidently a condition of stasis. Often minute thrombi form and block the capillaries for some distance. There is a marked increase in the viscosity of the blood due to loss of plasma into the lung tissues and a consequent retardation of the flow through the capillaries. The increased permeability of the lung capillaries and the capillary stasis in the injured lung tissues lead to pulmonary edema, dilatation of the right heart, and a tendency to circulatory failure.

During the World War the cyanosed cases were referred to as the *blue* cases. The blood which leaves the left ventricle contains an abnormal amount of reduced hemoglobin due to imperfect aeration in the lungs. Asphyxia is evident, the face having a full blue color while there is a visible distension of the superficial veins of the face and neck. The respiratory rate is increased; the pulse rate is above 100 per minute, and is full and strong in character.

The cases of circulatory collapse are referred to as the *gray* cases. The pulse is weak, thready, irregular, and the rate is 100 or above. The skin is ashen in color; extremities are cold. The heart is contracted. Bronchopneumonia often develops accompanied by a rapid rise in temperature.

Prognosis.—The slightly affected cases have a mild cough with a feeling of debility which passes off in a few days, but never become cyanosed as is the case with more severe casualties.

Severely gassed casualties develop immediate symptoms of irritation of the upper respiratory tract accompanied by cough, pain in the chest, and in a short time by more severe symptoms as dyspnoea, cyanosis, rapid pulse rate of 100 or more, and collapse.

The prognosis depends mainly upon the amount of lung tissue damaged by the irritant gas and upon the compensatory powers of the heart. Blue cases tend to recover under proper management and

treatment. However, any physical effort, such as severe coughing spells, or getting out of bed, may lead to circulatory collapse. The gray cases are always serious. In the group developing bronchopneumonia the outlook is grave.

Treatment.—Casualties must be removed from the contaminated atmosphere at once. Sometimes it is difficult to determine if a man has been gassed. A casualty may sometimes be diagnosed by having the individual smoke a cigarette. If he has been gassed the smoke will have a disagreeable taste and will cause increased irritation with coughing.

The most important therapeutic measures are as follows:

1. REST.—Patients must be made to lie down and remain quiet. Heat will increase comfort. Oxygen must be conserved as all activity rapidly leads to a decrease in available oxygen in the blood.

2. VENESECTION.—This procedure should not be used except as an emergency measure since it robs the patient of the needed oxygen-carrying power, and repeated bleeding so lowers the body resistance that bacterial invasion of the inflamed tissues is invited. Venesection should not be used for the gray cases. Oxygen therapy is preferable to relieve the cyanosis, but if not available, venesection should be used to tide the patient over the period necessary for transportation to a casualty center, where oxygen is available.

3. OXYGEN.—Oxygen inhalation is the most important measure in the treatment of these cases, and should be available in sufficient quantities for administration at a rate which will keep the patient comfortable and relieve the cyanosis. Either the mask, tent, or nasal catheter method may be used.

4. SALINE.—The judicious use of glucose or saline solutions is indicated. These are best administered by hypodermoclysis into the subcutaneous tissues of the thighs. It is best not to use the chest regions for these injections because of the danger of further embarrassment of the respiration. Fluid should not be given intravenously during the acute stage unless at a very slow rate, in order to avoid an increase in venous pressure and overloading of the right heart. Periodic hemoglobin determinations should be made for the purpose of estimating the amount of solution required to lower the viscosity of the blood.

5. POSITIONAL DRAINAGE.—Moderate elevation of the foot of the bed (about 18 inches) will often promote drainage of fluid from the upper respiratory passages. Care should be used not to elevate the foot of the bed to a degree that will allow the abdominal contents to press against the diaphragm and embarrass respiration. The consumption of water or food in large quantities may also embarrass respiration, therefore, these should be allowed only in small amounts and at frequent intervals.

6. SEDATION.—The judicious use of sedatives to lessen severe coughing and promote rest, decreases the demand of the tissues for oxygen and thereby spares the heart. In cases of extreme restlessness small doses of morphine may be used, bearing in mind, however, the depressant action on the respiratory center.

7. CONTROL OF INFECTION.—Measures should be instituted in wards to limit the spread of infection. Bronchopneumonia cases should be segregated.

NITROUS GAS (incidental).—This is an incidental gas and not a chemical-warfare agent. It is included because it may be a casualty producer during military action.

Nitrous gases are produced in toxic concentrations when high explosives, such as guncotton, cordite, or dynamite are burned, or when nitric acid is spilled on organic material such as wood or fabrics. When high explosives are detonated, however, these gases are not produced in toxic amounts.

Symptoms.—These gases produce nitric and nitrous acid in the presence of moisture and oxygen. The acids thus formed react with the alkali in the tissues and secretions of the respiratory tract to form the sodium salts (nitrates and nitrites). The nitrite is the substance which is active after absorption.

Nitrous fumes, by the formation of nitric acid, produce an irritant effect resembling that of phosgene on the lung tissues. A long latent period of 2 to 24 hours intervenes before severe pulmonary symptoms develop. The symptoms are those that characterize phosgene poisoning. The initial irritation of the nose and throat is slight. The patient experiences gradually increasing breathlessness as edema of the lungs develops. Coughing and severe pains in the chest ensue. When the edema is well developed, the extremities become cyanotic and embarrassment of the heart is evident.

Two systemic actions are exhibited by the nitrite. Patients experience the usual effect of excessive vasodilatation, that is, a fall in blood pressure, vertigo, and headache. The other action is the formation of methemoglobin by the nitrite, preventing the combination of oxygen with hemoglobin thus giving rise to an asphyxiating effect. Both of the above systemic effects of nitrous gases play a minor role in the production of severe casualties.

Treatment.—These casualties are to be treated in the same manner as phosgene cases. Remove the patient from contaminated atmosphere, secure early and complete rest, and give oxygen inhalations if cyanosis appears. Convalescence may be protracted due to cardiac weakness.

VESICANTS

This type of chemical-warfare agent proved to be the most effective casualty producer during the World War. The most important gases of the vesicant type are mustard gas (HS), lewisite (M–1), and ethyldichlorarsine (ED). These agents, in either liquid or vapor phase, have not only the ability to produce blistering and necrosis of the skin, but also have a decided irritant action on the eyes, respiratory system, and gastro-intestinal tract, and are general systemic poisons following absorption into the circulation.

MUSTARD GAS—*Symptoms.*—This gas affects the skin, eyes, and the respiratory and gastro-intestinal systems. Its action on these parts of the body will be discussed separately.

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SKIN.—Vapor or liquid mustard gas produces no immediate irritation of the skin. After a latent period of 2 hours or longer, a dark erythematous blush appears over the contaminated area and a gradually developing edema of the skin occurs in areas of deep penetration. Warm, moist areas, such as axilla and crotch, show the greatest degree of irritation. A few hours later these burned areas are covered with small blisters which coalesce to form large thin walled sacs, distended with straw colored fluid. Blister fluid is not vesicant. Beneath the blister is found a raw, red, weeping surface. Such areas become ulcerated and are later covered by a thick crust. Itching is a persistent symptom during healing. The healing period is prolonged and lesions are prone to become infected. Pigmentation and white scars mark the area which was burned.

EYES.—Definite symptoms of eye inflammation, such as pain, and signs of acute conjunctivitis appear 2 to 3 hours after prolonged exposure to effective concentrations of the vapor. In from 4 to 16 hours there is a free discharge from the eyes, severe pain, and great swelling of the lids. These severe symptoms continue for 24 to 48 hours and then gradually abate.

RESPIRATORY TRACT.—Mustard gas vapor has a corrosive effect on the mucosa of the respiratory tract, followed by an acute catarrhal inflammation. More severe effects are necrosis and desquamation of the mucosa with formation of a necrotic membrane. This destruction of the mucous membrane leaves a raw red surface which readily becomes infected. Alveolar changes in the lungs are not intense, consisting of congestion and some epithelial desquamation. Bronchopneumonia may result from secondary infection.

GASTRO-INTESTINAL SYSTEM.—The ingestion of mustard gas in food, water, or saliva will cause an acute gastro-intestinal catarrh with nausea, vomiting and epigastric distress. If poisoning has not been severe, symptoms clear up within 48 hours.

SYSTEMIC ACTION.—The action of mustard gas on the system has been studied by direct intravenous injection. The action is mainly on the heart, nervous, and gastro-intestinal systems. The pulse becomes slow and irregular, and death results from heart failure. Nervous symptoms consist of increased nervous irritability, excitement, and sometimes convulsions. The intestinal symptoms are probably due to the excretion of a toxic substance by the intestinal mucosa. A necrotic enteritis with diarrheal symptoms results. If large areas of skin are burned with mustard gas toxic absorption is evident, with resulting shock as in the case with severe heat burns.

Treatment.—The prophylactic methods of decontamination and the treatment of mustard burns will be discussed separately. Prophylactic treatment aims to remove the mustard gas from the skin before it has been absorbed, and also to remove that mustard which has penetrated the superficial layers of the skin. The surgical treatment consists of those measures and procedures which have been found satisfactory for treating mustard burns.

PROPHYLACTIC TREATMENT.—Visible droplets of mustard gas should always be dabbed off the skin at the earliest possible moment with a piece of cloth or blotting paper. Prophylactic measures to be of the greatest value must be applied immediately after contamination. Strive to demustardize patient at the earliest possible moment consistent with the circumstances. Mustard gas penetrates the skin readily. Personnel contaminated with the vapors of mustard gas should receive the same prophylactic treatment.

If the patient is still wearing contaminated clothing or carrying contaminated equipment, he should be undressed and all equipment discarded; the usual precautions being observed in handling this material.

There are three types of prophylactic materials which may be used to remove mustard gas from the skin: (1) solvents which dissolve the mustard; (2) emulsifying materials; and (3) neutralizing chemicals.

Solvents used for the removal of mustard from the skin are kerosene, alcohol, oils, and carbon tetrachloride. Rubber gloves should be worn by the first-aid personnel to protect the hands. Contaminated skin areas are swabbed thoroughly with a cloth sponge saturated with one of these solvents. Care must be used in applying the solvent so as not to allow it to run over uncontaminated areas and thus spread the dissolved mustard over a larger area than originally contaminated. Whenever possible the use of solvents should be followed by washing with soap and water.

Emulsifying agent.—Laundry soap and water are effective for the removal of mustard from the skin and are applied by thorough scrubbing with a flesh brush employing enough force to cause reddening of the skin, thus removing some of the surface cells along with grease, dirt, and absorbed mustard. If soap or material for the other methods described are not available, the contaminated area should be scrubbed with large quantities of water, shaking the water out of the brush frequently.

Chemical neutralization of mustard on the skin can be effected if undertaken early, before the mustard has become fixed in the skin. Fresh bleaching powder, either of high test or common type, may be wetted with water, and made into a creamy paste, and applied to the contaminated areas where it is allowed to remain for 5 minutes. It may be necessary to remove the bleach paste from the tender areas of the skin after a shorter period, if the treatment causes discomfort and irritation.

Care must be used to keep the bleach away from the eyes. Do not apply bleach paste to areas already showing signs of inflammation as it is too late to neutralize the mustard gas and the chlorine will further injure the inflamed skin. If liquid mustard gas hits the eyes it is necessary to immediately swab the conjunctival recesses with a cotton swab. This procedure will remove small droplets of mustard which would otherwise tend to be absorbed. The eyes should next be thoroughly flushed with a 0.5 percent solution of chloramine or a (1-5,000) potassium permanganate solution.

TREATMENT OF MUSTARD BURNS.—The burned areas showing erythema or vesication are cleansed thoroughly with soap and water. Areas of erythema and edema are treated like severe sunburn with cooling and soothing applications. As blisters appear they should be opened aseptically and the serum removed. It may be necessary to excise the blister cover and remove the coagulated serum. Raw areas should be tanned by the tannic acid, silver nitrate method.

Infection must be controlled by the use of Dakin's solution or azochloramid saline solution treatment. Open air treatment with a cradle and electric lights is of value in severe burns.

During the later stages of healing, when the crusts become thick and heavy, it is often advantageous to remove them by clean dissection along the base of the crusts. Thick crusts act as splints to burns and preclude a rapid reduction in size by preventing contraction of the newly formed fibrous tissue.

Respiratory irritation is treated by the usual sedative and inhalation medication. Bronchopneumonia is a frequent complication. Such cases should be segregated.

Discharges from burned eyes should be regularly removed by saline irrigation. Application of a sterile 1 percent atropine ointment every 12 hours will

give relief. Cocaine is contraindicated because of its tendency to loosen the corneal epithelium and facilitate ulceration. The eyes should not be bandaged tightly, a loose compress being all the dressing that is necessary.

LEWISITE.—Although lewisite was developed by professor Lewis in 1917, it was never used in the World War. It is a vesicant gas of a high degree of potency. It is presumed that this gas, if used in a future war, would be employed in the same manner as mustard gas, i. e., airplane spray, bombs, or shells.

Symptoms.—While lewisite exhibits many of the characteristic actions of mustard gas, it differs from the latter both in the rapidity with which it produces lesions and in the greater inflammation it causes. In comparison with mustard gas, lewisite burns exhibit an earlier necrosis of the epidermis, edema is more extensive and the exudate definitely fibrinous, the inflammatory reaction appears earlier and is more intense and extends deeper, and vascular thrombosis is a more conspicuous feature.

Liquid lewisite or its vapor will produce an almost immediate severe irritation of the eyes accompanied by pain, spasm of the eyelids, and a copious flow of tears. Inflammation develops rapidly in 10 to 15 minutes, the eyelids become edematous and ulceration of the cornea and a purulent discharge ensue.

The vapor of lewisite is very irritating to the nasopharynx and the entire respiratory tract. Coryza, salivation, and irritative cough occur in a few minutes, followed by bronchitis in 24 hours. Severe poisoning may lead to lung edema and bronchopneumonia.

Liquid lewisite, unlike mustard gas, causes an immediate stinging sensation in the skin which may persist for some time. Erythema and edema develop in 15 to 30 minutes. Small blisters appear within 12 hours or less on an inflamed surface, and tend to coalesce and form large vesicles. The blister fluid is cloudy and contains traces of arsenic. The healing time is prolonged and burns are often secondarily infected. The subcutaneous fibrosis is slow in being absorbed.

Lewisite or its toxic products are absorbed through the skin into the subcutaneous tissues, causing a general systemic poisoning. Arsenic is found in the burned skin, subcutaneous edematous fluid and vital tissues, and the chemical is excreted in the urine. Symptoms of severe intoxication with renal and hepatic congestion, gastrointestinal irritation and a rapidly developing condition of collapse ensue within 24 hours.

Splashes of lewisite on the skin, such as those resulting from bomb or shell explosions, would probably prove lethal due to arsenic absorption unless immediate and adequate treatment was instituted.

Treatment—PROPHYLACTIC TREATMENT.—All contaminated clothing and equipment must be discarded. The penetration of lewisite is so rapid that pro-

phylactic treatment must be started immediately. The methods used for the removal of mustard gas are effective for the removal of lewisite, if used immediately. A 5-percent solution of sodium hydroxide is useful for neutralizing lewisite on the skin. This solution should be removed after a few minutes by washing with water to prevent injury to the skin by the alkali.

TREATMENT OF LEWISITE BURNS.—It is necessary to excise and remove the blister cover and irrigate the raw surface so as to prevent absorption of the arsenic-containing blister fluid. Severe burns with considerable edema should be treated by excision of the infected skin as described by Vedder in *The Medical Aspects of Chemical Warfare*. This surgical treatment will lessen the degree of toxic absorption of arsenic present in the skin and edematous subcutaneous tissue.

ETHYLDICHLORARSINE.—This type of vesicant represents the quick acting, moderately persistent casualty and harassing agent.

Symptoms.—Ethyldichlorarsine has three physiological actions: a severe sternutative effect on the nose and throat similar to that produced by the irritant smokes; a blistering effect on the skin like that from mustard gas, but only approximately one-sixth as irritative; and a general toxic action on the body by the absorbed arsenic, producing symptoms such as weakness, faintness, and a severe paralysis and anaesthesia of the extremities.

Treatment.—Prophylactic treatment is the same as that recommended for lewisite. The treatment of burns should aim to reduce arsenic absorption through the skin. Severe burns should receive the same treatment as that used for lewisite burns.

PARALYSANT

HYDROCYANIC ACID GAS was used during the World War by the French (Vincennite), but without success, because of the difficulty of establishing an effective concentration under field conditions.

Symptoms.—Hydrocyanic acid is a true protoplasmic poison in the sense that it inhibits oxidation in the living matter thus producing a type of internal asphyxia. The venous blood is bright red in color in cyanide poisoning because the oxygen is not abstracted from the blood in the tissues. In higher concentrations a man will collapse after taking a few breaths; convulsions are rapidly followed by death.

In less severe poisoning there is constriction of the throat, giddiness, headache, and some nausea; recovery follows after several hours, unless damage to vital tissues has occured due to asphyxia.

Treatment.—Remove the patient from the contaminated atmosphere and give artificial respiration. Inhalation of an oxygen-carbon dioxide mixture is useful. Special antidotal methods of treatment may be tried, such as the inhalation of amyl nitrite fumes at frequent intervals.

ASPHYXIANT

CARBON MONOXIDE will probably not be used as a chemical warfare agent, but may be met with since it is formed when high explosive powder is burned or detonated. This gas will be found in dangerous concentrations in compartments in which bombs or shells have exploded, or in shelters and trenches into which it has diffused from exploded mines. The service gas mask canister does not protect against this gas.

Symptoms.—Carbon monoxide combines with the hemoglobin of the blood to the exclusion of oxygen since its affinity for hemoglobin is 300 times greater than that of oxygen; the carbon monoxide hemoglobin formed precludes the carrying of oxygen as oxyhemoglobin, and results in asphyxia.

Carbon monoxide has no color or odor, hence gives little or no warning of its presence even when breathed in high concentrations. Symptoms such as giddiness, ringing in the ears, blurred vision, dyspnoea, and general weakness develop rapidly. An affected individual is often unable to leave the contaminated atmosphere even though he realizes that he is being overcome by the gas. Some casualties become stimulated and exhibit symptoms of intoxication and exhilaration, but others collapse without warning.

Treatment.—The patient should be removed from the contaminated atmosphere, and if breathing has ceased, artificial respiration should be instituted immediately. A mixture of carbon dioxide and oxygen should be given by the inhalator method if apparatus is available. The patient should be wrapped in blankets, kept warm, and at rest. After partial recovery he should be watched carefully for relapse into unconsciousness, and evacuated as soon as all danger of relapse has passed.

MEDICAL SUPPLIES FOR FIRST-AID TREATMENT OF CHEMICAL WARFARE CASUALTIES

There seems to be a great individual difference in opinion as to just what supplies should be made available at battle stations for the treatment of chemical-warfare casualties. No doubt the medical officer must be allowed much latitude in the selection of supplies but, nevertheless, there are certain items which are absolutely essential.

The following list of supplies includes basic material to which the medical officer can make additions if the necessity arises.

The quantity of material which is needed must be estimated according to the complement of the ship.

FIRST-AID BOXES

1. Boric acid, saturated solution, 500 cc.
2. High-test bleach (amount calculated on basis of 20 gm. per man).

3. Sodium hydroxide solution, 5 percent, 500 cc.
4. Copper sulphate solution, 2 percent, 500 cc.
5. Sponges, gauze (2 x 2 in.)

DECONTAMINATION CENTERS

1. Ethyl alcohol, tin containers (pint).
2. Navy salt water soap.
3. High-test bleach (large containers).
4. Sodium hydroxide solution, 5 percent.
5. Sponges, gauze (2 x 2 in).

DRESSING STATIONS

1. Tannic acid solution, 7 percent.
2. Silver nitrate solution, 10 percent.
3. Ferric hydrate paste, 500 gm. jars (2 per station).
4. Oxygen cylinders, large, with masks.
5. Inhalator.
6. Amyl nitrate pearls, 2 dozen.
7. Sodium bicarbonate solution, 5 percent.
8. Saline solution, physiological, sterile (intravenous use).
9. High-test bleach.

A NEW METHOD FOR DETERMINING NIGHT BLINDNESS[1]

A PRELIMINARY REPORT

By Lieutenant (Jr. Gr.) Earle E. Metcalfe, Medical Corps, United States Navy

INTRODUCTION

It is believed that the method to be described may be properly termed a test for night blindness, inasmuch as it measures those factors chiefly deficient in this condition, light adaptation and dark adaptation.

Methods in use at the present time to determine these functions are those devised by Birch-Hirschfeld[2]; Nagel[3]; Lo Casio[4]; Derby, Chandler and Sloan[5]; Delaney[6]; Ferree and Rand[7]; Feldman[8]; and Hecht and Schlaer.[9]

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[1] Submitted October 26, 1939, for publication.

[2] Birch-Hirschfeld, A.: Zur Frage der Witwirkung des Lichtes bei der Entstehung des Altersstares; Ber. ü. d. Versamml d. deutch Oph. Gesellsch. 46: 226, 1927.

[3] Nagel, N.: Traffic Signal Apparatus; Klin. Monatsbe f. Angenk, 93: 433, October 1934.

[4] Lo Casio, G.: Appareccio per l'esame del semo luminoso nelle parti periferche della retina nell' ochio umano; Soc. ital. di oftal, 10: 27, 1925.

[5] Derby, G. S., Chandler, P. A., and Sloan, L. L.: A Portable Adaptometer; Tr. Am. Oph. Soc., 27: 110, 1929.

[6] Delaney, J. H.: Light Sense as Tested by Photometric Glasses of Tscherning; Am. J. Oph., 13: 1058, Dec. 1930.

[7] Ferree, C. E., and Rand, G.: A New Type of Instrument for Testing the Light and Color Sense; Am. J. Oph., 14: 325, Apr. 1931.

[8] Feldman, J. B.: Instrument for Determining Course of Dark Adaptation and for Measuring Minimum Light Threshold; Arch. Oph., 12: 81, July 1934.

[9] Hecht, S., and Schlaer, S.: An Adaptometer for Measuring Human Dark Adaptation; J. Optic Soc. America, 28: 269, July 1938.

The literature on light sense and dark adaptation has been amply reviewed by Adams [10] in 1929 covering all methods used prior to that time. Sloan [11] has reviewed the literature of the past 10 years, and concludes:

> It is apparent from this review of the literature that a number of problems require further investigation before tests of light sense will be of much value in clinical ophthalmology.

BASIC PRINCIPLES INVOLVED

From the standpoint of a complete academic discussion, it would be essential to review the histology, neurology, anatomy, and physiology of the eye with optics involved, but inasmuch as one interested in this work is undoubtedly well grounded in these subjects it will be necessary to recall only a few factors in order to understand how conclusions have been derived.

The stratum pigmenti is the layer of the retina we are most concerned with, the epithelium consisting of a single layer of hexagonal, nucleated cells containing pigment granules. The fovea contains only cones and therefore takes no part in distinguishing light. The rods increase in number from the fovea to the periphery. They are the end organs of the optic nerve and originate the impulses which, in the brain, are perceived as light.

All vision near light minimum is peripheral vision and concerned only with rods, and is dependent upon the metabolism of visual purple contained in them. Catabolism of visual purple occurs when the retina is exposed to light. The supply of this pigment may be exhausted if regeneration is retarded or if exposure to light is of such intensity that regeneration is not possible until illumination is removed or diminished.

A photochemical reaction has been described for this phenomenon and vitamin A has been implicated as the precursor of visual purple. It is inability to regenerate this substance that is one of the earliest manifestations of avitaminosis A. Mirsky [12] has described visual purple as a conjugate protein in which retinene is the prosthetic group. Exposure to light, he states, causes a denaturation of the protein, with loosening of bond uniting it to form the pigment group. Denaturation is reversed in the dark.

On exposure to light there is a movement of pigment from the outer epithelial layer to spaces between the rods, accompanied by

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[10] Adams, D.: Dark Adaptation. A Review of the Literature, Medical Research Council, Special Report Series, No. 127, London, His Majesty's Stationery Office, 1929.

[11] Sloan, L.: Instruments and Technics for the Clinical Testing of Light Sense; Arch. Oph., 21: 913, June 1939.

[12] Mirsky, A. E.: Visual Cycle and Protein Denaturation; Pros. Nat. Acad. Sci., 22: 147, Feb. 1936.

shortening of the rods and cones. These changes are dependent upon the intensity of light and are accompanied by electrical changes in the eye which are dependent upon the color, or wave lengths, of light used. With illumination of equal intensity, yellow rays give a larger current in the light adapted eye, and green in the dark adapted eye. This is one of two points for the possible explanation of my method.

Changes produced in the retina by light are dependent within lower limits, on the part exposed; however, if an intense light is used over a sufficient length of time, most of the retina will be exposed and fixation is no longer a factor.

When the retina is stimulated by light, a period elapses before sensation or the primary image is perceived and there is an interval of 0.07 to 0.16 seconds before sensation reaches its maximum. The greater the intensity the shorter the interval. This primary image is followed by a less intense image, which is not seen in the night blind. This image belongs to rod function, whereas the primary image was concerned with cones.

After this response, a tertiary image is formed which is of longer duration and lower intensity than the primary one, and as long as it persists, the retina will not respond to a similar stimulus of the same nature as the initial one, nor will recovery take place for the perception of color as long as it exists. Positive and negative phases of color images are dependent upon the quality and sequence of color stimulation. If the primary stimulus is red and no secondary stimulus is applied, the secondary and tertiary images are red and the reaction is positive. If the primary stimulus is red and is followed by a secondary stimulus of white, the secondary and tertiary images are green, the complementary color of the primary stimulus, and the reaction is negative. This is by virtue of the fact that the red portion of the white is excluded from the tertiary image of the initial stimulus.

The duration of this image with intensity varies with the stimulus. Stimuli varying in intensity from above moderate intensity to the point of causing drying of a normal cornea, and of one minute duration, will not change the duration of the normal tertiary image.

The perception of color is not possible until the rods and cones have returned to normal. Therefore, dark adaptation consists in removal of the tertiary image from the cones, thereby bringing them back to normalcy and in the regeneration of the visual purple of the rods. And since cone vision returns before red vision, as demonstrated by my method, the regeneration of visual purple is the most vital factor, and complete adaptation does not take place until this occurs. It is this recovery time that is noticeably prolonged in night blindness, not

in seconds as some believe, requiring logarithmic curves to demonstrate deviations from normal, but in minutes prolonged as much as five times the normal for age and varying with the degree present.

In night blindness the power of the eye to become dark adapted is absent, or present only to a markedly diminished degree. The photochromatic interval is not found. The eye does not pass through the same uncolored stage due to a deficiency of visual purple in the rods.

From perimetric studies we know that, with the size of the test object remaining constant and motionless and with the same intensity of illumination, definite fields may be outlined, of which green is the most central. Since it is this color that is the last to return to normal, it may be that there is some correlation between the two, which is further substantiated by the fact that with marked contraction of the visual fields night visual acuity is affected.

When intensity of illumination is increased suddenly, the amount of contraction of the visual field was demonstrated by Haycraft to be equal to the logarithm of the intensity of the light. However, as the retina becomes adapted to increased illumination of great intensity, the pupil relaxes and stabilizes. This is also true if a light of moderate intensity is used but stabilization does not take place for 8 to 10 minutes, and further increased intensity will cause greater constriction. On the basis of this, I believe we may ignore the size of the pupil if strong illumination is used for sufficient duration. In all cases of prolonged recovery time, or in variance with age, I have found no appreciable change in the reaction and size of the pupil.

It should be recalled that light, in order to produce a chemical change, must be absorbed. White light, because it consists of a number of rays of different wavelengths, is the best for bleaching.

By using a distance of 20 feet, factors affecting accommodation are ruled out. Errors in refraction were found to have no effect on the recovery time.

Lastly, it should be recalled that light sense is due to different degrees of intensity of illumination while color sense is due to illumination of varying wavelengths. Form sense is expressed in acuteness of vision and is entirely foveal. Light perception is dependent upon the size of the object viewed and we interpret change in light intensity as light difference.

FUNCTION OF THE TEST

1. It determines the length of time required for the eye to return to normal after exposure to illumination of intensity sufficient to impair vision temporarily.

2. It determines the recovery time limit that may be considered pathological or such duration as would make the individual dangerous to himself and others in operation of vehicles.

3. It demonstrates the effect of age on recovery time.

4. It demonstrates that light perception is not dependent upon visual acuity.

MECHANISM OF TEST

The mechanism of this test is based upon the physiological principles that have been outlined. The retina, after being exposed to intense illumination, becomes insensitive to minimum light, and a return to normal takes place through the visual fields by perception of color in varying degrees and intensities. The eye returns to normal only after green may be perceived or interpreted as such. To illustrate:

After intense exposure to white light and white light of a diminished intensity is again offered to the eye, it will be seen as a purple or lavender color, fading through to a color varying from red to rose and finally changing into a pale pink, orange, and then white. When red is offered after exposure to white, it will be seen as a grey-white, pink, and finally red. This is the first color to return to normal. White is the second color perception to return to normal. After exposure to white light, green will be perceived as white, grey-white, blue, and finally olive green. This is the last color to be perceived. Other colors vary in their recovery time, depending upon their wave lengths and relative positions in the visual fields.

Green has been chosen as an end point in this method, since it is the last color to return to normal and more closely represents physiological recovery.

In devising a method which could utilize all the physiological principles previously mentioned and which would still be practical, it was found that certain factors either had to be satisfied, standardized, or ruled out. Chief among these were to determine:

1. The ability of the individual being examined to properly interpret color and appreciate perceptions.

This ability is determined by having the examinee identify color by means of the Ishihara colored charts or the Eldridge-Green lamp. Obviously, if an examinee is unable to perceive and identify colors correctly, the test is discontinued.

2. Whether the factors of errors in refraction and accommodation would influence the recovery time.

By testing a number of hyperopes and myopes and those with errors in accommodation, without their correction, it was found that visual acuity was not a factor as recovery time was not influenced beyond the normal for their age group.

3. The effect of the size of the pupil and the effect of distance of the secondary illumination from the observer.

By experimental methods and observation of the involved physiological principles, it was found that if intense illumination of sufficient duration was used, the size of the pupil was not a factor, as stabilization was reached before recovery was made. The distance of 20 feet was determined to be the most accurate for standardization, inasmuch as accommodation was relaxed.

4. What could be considered a normal recovery time, a pathological recovery time, and the degree to which impairment could be present and still be compatible with night work.

The normal recovery time was determined by computing mean, high, and low of those with apparently normal night vision. Pathological recovery time was determined by wide variations from this normal as evidenced by degree of inability to accomplish tasks where vision was paramount.

5. The effects of age on recovery time and the normal for various age groups.

The effect of age was determined by comparison of age groups and analyzed.

6. A method of making the test objective rather than subjective.

The most effective method of making the test objective was found to be in the use of a colorimeter with a green solution for comparison; the concentration of the solution varying in the tubes but within comparable limits of the colorimeter. Before the test began, the examinee was directed to compare solutions and readings were recorded. This test was repeated after conclusion of the examination and if full recovery had not taken place, examinee would be unable to make readings comparable to the original ones.

7. The amount of intensity of illumination that is necessary to paralyze the cones and completely denaturize the rods and still not be injurious to the cornea.

The amount of intensity of illumination required was determined by experimental work and standardization was reached at the point of drying of the normal cornea.

METHOD OF THE TEST

1. Candidate is examined for color blindness and ability to interpret color by means of Ishihara charts or the Eldridge-Green lamp. This may be omitted on routine check-ups where previously determined as normal.

2. Individual is then directed to compare green solutions in colorimeter and readings recorded for each eye. The eyes are examined separately since they may differ in color perception.

3. A lamp that will give 250-foot candlepower of illumination at 5 cm. is focused on the eye to be examined. An ordinary spotlight that has a focusing lens and rheostat may be used for this exposure. Duration of exposure 1 minute. The other eye is covered by a patch that occludes all light.

4. Time is noted at end of exposure. After exposure, the examinee is directed to view a screen, 20 feet distant upon which is projected a white light which elicits 10-foot candles. He is directed to report the color changes which take place. A red light is then flashed on the screen and the color changes are reported. It is advisable to give this red light exposure before white has completely recovered, that is, before orange fades to white, in order to more closely observe

the early changes occurring in the red. The exposure to red is followed by exposure to green and the subjective changes are reported. It will be noted in some few individuals that slight variations in order of recovery will be present, but recovery has not been completed until all colors are recognized as projected.

If an instrument is not available for the projection of colors, an Eldridge-Green lamp may be used. Where no special equipment is obtainable, colored bulbs of equal intensity will give similar results if standardized with the normal individual. Where a large group is to be examined it will be found convenient to project simultaneously all three colors and observe recovery.

5. Time is observed and recorded for recovery to green.

6. The examinee then, as a final check, repeats the colorimeter test and the end point, that is, recovery of normal color vision, is reached when his colorimeter readings are the same as the readings on the first test.

Where a colorimeter is not available, lights may be alternated to confuse the individual and, inasmuch as end point may be wide and still normal, degree of variance is easily detected.

7. The procedure is repeated with the other eye.

RESULTS

1. Seventy-five cases were examined.

2. In the 20–30 year age group the lowest recovery was made in 2 minutes, mean 3.25 minutes, high 4 minutes. Normal recovery time in this age group is considered to be from 2 to 3.5 minutes. Recovery time exceeding 6.5 minutes is considered pathological.

3. In the 30–40 age group the lowest recovery was made in 3 minutes, mean 3.75 minutes, high 5 minutes. Normal recovery time in this age group is considered to be 3–4.5 minutes; that exceeding 8 minutes—pathological.

4. In the 40–50 age group the lowest recovery was made in 3.75 minutes, the mean 5 minutes, highest 6.5 minutes. Normal recovery time in this age group is considered to be 3.75–6.5 minutes; that exceeding 10.5 minutes—pathological.

5. In the 50–60 age group the lowest recovery was made in 5 minutes, the mean 6 minutes, highest 8 minutes. Normal recovery time in this age group is considered to be 5–8 minutes; that exceeding 12.5 minutes—pathological.

6. Difference in recovery time between the two eyes varied from 0 to 2 minutes. The mean was 0.75 minutes.

7. One examinee withdrew from examination after not making recovery in 13 minutes.

8. Maximum recovery observed was 15 minutes in an individual 35 years of age who is a very heavy drinker and has not driven at night for the past 5 years.

ADVANTAGES OF TEST

1. The difference between normal and pathological recovery time is of sufficient degree to eliminate errors in interpretation.
2. The size of the pupil is not a factor, thus eliminating special equipment that is expensive and requires careful supervision for accurate determination.
3. The test may be executed in a short time, thus permitting examination of a large number with minimum medical department personnel.
4. It is standardized so that it may be used by all flight surgeons and ophthalmologists.
5. It has a definite disqualifying point.
6. It requires no special preparation of examinee.
7. It is simple enough in detail that a trained technician can carry out the test and obtain reliable results.
8. It can be carried out aboard ship, in the office, or in the field with little or no special equipment.
9. It involves no pre-exposure in dark room.
10 Accommodation is not a factor.
11. Errors in refraction are not a factor.
12. It permits the return of examinee to routine duties upon the completion of the test as no mydriotics or miotics have been used.

IMPORTANCE OF TEST

The importance of the test in aviation is obvious. In the selection of candidates for flight duty, those with prolonged recovery time should be disqualified since their night flying would be hazardous. For similar reason it should be employed at the periodic examination of pilots.

It aids in the selection of those with hyperacute night vision for specialized training and service.

SUMMARY

A new method for testing night blindness is presented. Physiological principles involved have been discussed. Seventy-five cases have been analyzed and values are given for that which may be considered normal for different age groups. The advantages of the test have been outlined.

Acknowledgment.—Acknowledgment is made and appreciation expressed to Major John Hargreaves, Medical Corps, United States Army, Director, Depart-

ment of Ophthalmology, School of Aviation Medicine, Randolph Field, Tex., for his kindly suggestions and for making facilities available for the completion of this study.

ATEBRINE IN THE ERADICATION OF GIARDIA LAMBLIA

By Lieutenant Commander Julian Love, Medical Corps, United States Navy, and Lieutenant Commander Gordon B. Tayloe, Medical Corps, United States Navy

Recent studies indicate that 5 percent of all adults are infested with *Giardia lamblia* while in children from 11 to 15 percent are afflicted. It is said that this infestation is universally distributed throughout the world. In the United States a summarized report (1) from various authors showed that in 35,299 patients 2,620 or 7.4 percent of adults and children giardiasis was present. Many persons so affected apparently have no symptoms at all, while others have manifestations so vague that two schools of thought as to the pathogenicity of this parasite have developed. One maintains that the organism is nonpathogenic and is a harmless coincidental finding along with diarrheas and abdominal distresses of other etiology; the second contends that this parasite does cause pathosis in man with resultant symptoms, usually of diarrhea, abdominal distress, cramps, meteorism, and anorexia; and rarely prostration.

The treatment of this condition has long been unsatisfactory with all the anthelmintics and amebicides. The arsenicals have particularly been tried with inconstant results. Many forms of treatment used are more distressing than the symptoms of the infestation, and others are more hazardous to the patient than the disease itself, the prognosis of which for life is excellent. These include neoarsphenamine by vein and duodenal intubation, acetarsone, dihydranol, and others.

The successful parasiticidal action of atebrine in this type of infestation was first demonstrated by Galli-Valerio (2). He admitted atebrine in the usual dosage recommended for malaria which is 0.1 gm. (1 tablet) thrice daily for 5 days. Tecon (3) verified this, but feels that 0.1 gm. once daily for 2 to 3 days is sufficient. Morrison and Swalm (4) treated 10 patients with atebrine, and in 9 of these the infestation was eradicated and remained so for at least a year. Romano and his co-workers (5) reports that they had used many varieties of treatment without success until atebrine was employed, while Eusterman (6) states that he has become enthusiastic over the effectiveness of this drug in the eradication of *Giardia lamblia*.

The first two cases present interesting problems in differential diagnosis, and treatment by various amebicides. Moro's raw apple diet, and sulfanilamide were unsatisfactory. Atebrine as suggested by Galli-Valerio was then employed. In case 2 a second course was

required. Case 3 was apparently symptom free, but after the elimination of *Giardia lamblia* the mother stated that there was a noticeable general improvement in the child.

Case Reports

Case 1.—H. F. T., an enlisted man of 22 was admitted to the naval hospital, Brooklyn, N. Y., November 8, 1937, with complaints of weakness, cold sweats, abdominal cramps, and watery movements. He had six to eight stools for the past 2 days.

During the last 2 months he had noted hunger pains and an empty feeling in his abdomen several hours after meals, relieved by milk. Nausea and cramps were also noted the past 2 to 3 days. He had recently returned from duty in China. Otherwise, there was nothing of moment in his past or family history.

Physical examination revealed a young well-nourished white male of 22, not acutely ill. Physical findings were negative except that he flinched on pressure and was markedly tender over the appendiceal area. Abdomen was soft and no masses were noted. The clinical impressions were enterocolitis, acute, or appendicitis, acute. The surgical consultant did not believe the condition due to appendicitis.

Laboratory findings revealed a negative Kahn and urinalysis. The white blood count was 8,500 with 57 percent segments, 35 percent lymphs, 2 percent eosins, 5 percent monos. A check count was 6,900 with the same differential. Stool examinations were made and cysts of *Giardia lamblia* were found in great numbers on repeated examinations.

Treatment.—Carborsone in 0.25 gm. doses was given four times daily for 10 days, but though his clinical complaints disappeared, the cysts were found in the stool. On December 7, sulfanilamide in the usual course dosage was prescribed for a week, but the cysts persisted, and there were occasional attacks of diarrhea during which the number of cysts markedly increased. The raw apple diet likewise failed to eliminate the organism. In February 1938 atebrine in 0.1 gm. dosage was given thrice daily for 5 days, and the subsequent stools have been negative for cysts of *Giardia lamblia*, while the patient has remained asymptomatic for 8 months.

Case 2.—W. G., an enlisted man of 43, was admitted to the naval hospital Brooklyn, N. Y., with a tentative diagnosis of duodenal ulcer on December 14, 1937.

His complaint on admission was epigastric pain after meals. This condition had troubled him since January 1937. The pain came on 1 to 3 hours after meals and was relieved by taking soda. Food relief was variable. There had been no weight loss, melena, or hematemesis. His appetite had been good and bowels regular.

Except for duty in the Tropics and Orient there was nothing of importance uncovered in the past or family history.

Physical examination showed a well-developed and well-nourished white male of 43. Except for bilateral deafness due to otosclerosis, no abnormalties were noted.

Laboratory findings.—Urine and Kahn were negative. The white blood count was 11,400, 1 percent bands, 60 percent segments, 29 percent lymphs, 5 percent eosins, and 5 percent monos. Gastric analysis after histamin and alcohol stimulation showed a normal acid response. X-ray revealed no pathosis in the gastro-intestinal tract. Stools on admission showed numerous cysts

and vegetative forms of *Giardia lamblia* which persisted on repeated examinations.

TREATMENT.—The patient did not improve after two courses of carbarsone. Atebrine was then administered in 0.1 gm. doses three times daily for 5 days, and this course of treatment was repeated in 5 days because the stools remained positive after the first course. After completion of second course of atebrine his stools were negative for cysts and vegetative forms of *Giardia lamblia*. He was completely relieved of his symptoms and the stools have remained negative for 7 months.

Case 3.—A young girl, age 7, whose father is a Filipino and whose mother is a Panamanian was brought to the out-patient department on March 7, 1938, because of a lesion in her scalp which had caused a patchy alopecia. This was found to be due to trichophytosis. Because of her parentage and the fact that she had recently returned from a visit to Panama, stool examinations were ordered, and numerous cysts of *Giardia lamblia* were found. She had no complaints referable to her gastro-intestinal tract.

LABORATORY FINDINGS.—Kahn and urine were negative, and the white blood count was 9,400 with hemoglobin 85 percent. Differential of 49 percent segments and 51 percent lymphs.

TREATMENT.—On April 19 atebrine in 0.1 gm. dosage three times daily was prescribed for 5 days. Subsequent stool examinations were made in May 1938 and were found to be negative for *Giardia lamblia*. The mother volunteered the information that the child was noticeably and generally improved following the eradication of the infestation. There has been no recurrence during the ensuing months.

DISCUSSION

The value of atebrine in the therapeutics of malaria has been such that in a very short period it has gained an equal footing with time-honored quinine. As its action in malaria seemed to be that of a protozoacide, Galli-Valerio used it against *Giardia lamblia* which also is a unicellular organism.

The only complication caused by atebrine is a yellowish discoloration of the skin which has been proved to be merely a staining action due to its dye effect and not jaundice because of hepatic toxicity.

From the observation of this limited group of cases it would appear that *Giardia lamblia* infestation does cause clinical symptoms in some individuals so afflicted, and that atebrine is an effective agent for eradication. The dosage as recommended by Tecon would apparently be ineffective in some individuals as evidenced by the second case.

CONCLUSION

1. Three cases of *Giardia lamblia* infestation with apparent eradication following the administration of atebrine are reported.

NOTE.—Since the submission of this article, one of us (J. L.) has treated two additional patients with *Giardia lamblia* infection at Guantanamo, Cuba. Complete eradication was effected though one patient required a second course. The subjective improvement was particularly gratifying to the patient. To date, we have had no recurrences.

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DENTAL ABNORMALITIES IN RECRUITS

By Commander Walter Rehrauer, Dental Corps, United States Navy, and Lieutenant (Jr. Gr.) Karl Van Lear Berglund, Dental Corps, United States Navy

During the period from June 6, 1938, to September 15, 1939, examinations were made of the teeth of all recruits who reported aboard the U. S. S. *Saratoga* direct from the training stations for their first sea duty. The reason therefor was the desire to ascertain exactly how much dental treatment these recruits required. Many had been reporting to the dental office for the relief of pain soon after they arrived. The pain usually was caused by large carious areas in the approximal surfaces of teeth; sometimes by overhanging gingivo-approximal filling margins, and other abnormalties or pathology.

As most recruits are of the susceptible age, wherein dental caries progresses very rapidly, it was considered desirable to get an accurate record of the quantity and location of the carious areas, and the overhanging gingivo-approximal filling margins that might need correction. There was also a desire to ascertain the number of carious areas the average recruit has when he enlists.

Examinations of the teeth and mouth for dental caries, and oral abnormalities and pathology, are useful in direct proportion to the degree of accuracy with which they are made. This is the reason why it was decided to make the examinations as carefully and as accurately as human shortcomings would permit in the usual environment of a dental office afloat.

METHOD OF MAKING THE EXAMINATION

The procedure in each case was as follows:

1. One of us examined the man by clinical and visual means only, and recorded the findings in the usual dental record form used in the Navy. No reference was made to any other record at this time.

2. The other one of us examined the man in the same manner and checked the accuracy of the dental record which was made by the first one of us and made any corrections necessary.

3. A full-mouth series of bite-wing roentgenograms was made; three type 1 films for the anterior teeth and two type 3 films for the posterior teeth, a total of five films.

4. One of us read the bite-wing roentgenograms and he was checked by the other when the findings were recorded.

5. The dental record which was made at the training station was then procured and compared with the one made by us.

6. A special printed form was then used for recording the data desired. One of us checked the other when entries were made in this form. Our statistics were compiled from these special forms.

GEOGRAPHICAL SOURCE OF RECRUITS

The recruits who were examined represent a good cross section of the United States. They were born in, and enlisted from almost every state in the Union.

There were 359 recruits examined. The number received from the various training stations was as follows:

U. S. Naval Training Station, Newport, R. I	44
U. S. Naval Training Station, Great Lakes, Ill	119
U. S. Naval Training Station, Norfolk, Va	72
U. S. Naval Training Station, San Diego, Calif	101
U. S. Marine Corps Base, San Diego, Calif	23
Total	359

CARIOUS AREAS

In the dental records of these 359 recruits 1,419 carious areas and 35 useless roots were charted at the training stations where they received their recruit training. These carious areas and useless roots involved 1,278 teeth of 285 men. In 965 teeth of 237 men 1,109 fillings had been inserted at the training stations before they were transferred to sea duty; and 43 useless roots and teeth had been extracted. There was also 1 porcelain crown inserted. There remained 305 carious areas and 1 useless root involving 282 teeth, which were charted at the training stations, when these men reported aboard the U. S. S. *Saratoga.*

When these 359 recruits were examined by clinical or visual means in the U. S. S. *Saratoga*, an additional 1,288 carious areas and 1 useless root were found involving 1,160 teeth of 301 recruits.

In addition, 1,189 carious areas were found in 1,084 teeth of 276 of these men by means of bite-wing roentgenograms. Thus, a total of 2,477 carious areas and 1 useless root were found by clinical or visual means and with bite-wing roentgenograms in 2,058 teeth of 338 men in the U. S. S. *Saratoga.*

It is our opinion that the carious areas found by us did not occur primarily between the date that these men enlisted and the date on

which they were examined, but that these areas had their incidence before that time. The average period of service of these 359 recruits on the date on which they were examined by us was 6 months and 1 day. Therefore, when these 359 recruits enlisted, 350 of them had 3,896 areas of progressive caries and 36 useless roots involving 3,027 teeth—an average of about 11 carious areas per man. When they reported to the U. S. S. *Saratoga* for their first sea duty, 341 of the 359 men had 2,782 areas of progressive caries and 2 useless roots which involved 2,248 teeth—an average of about 7¾ carious areas per man.

Most of these progressive carious areas, which existed when these men were enlisted, could not have been detected by medical department personnel at the recruiting offices with the means available to them for making an examination of the teeth and the mouth.

OVERHANGING GINGIVO-APPROXIMAL FILLING MARGINS

Recruits often requested relief from pain caused by overhanging fillings which extended beyond the gingival margins of cavities into the approximal spaces and the inter-approximal gum tissue. These large masses of overhanging filling material caused severe gingivitis, hypertrophy of gingival gum tissue, destruction of inter-approximal bone and tissue crests, rapid resorption of the alveolar process, pockets between the teeth, caries in approximal surfaces of adjoining teeth, speedy recurrence of caries, and a general insanitary condition which was an ideal environment for Vincent's infection. The elimination of these overhanging filling margins is as important as the correction of carious areas and it is for this reason that a record was made of those fillings that showed overhanging masses large enough to indicate either attempted removal of the overhanging margins or replacement of the fillings. There were 139 of these overhanging fillings and 3 gold shell crowns with excessive gingival overhang found with the bite-wing roentgenograms. They involved 134 teeth in 87 of the 359 men.

EXCESSIVE RESORPTION OF ALVEOLAR CRESTS

Bite-wing roentgenograms are not intended for studying the alveolar crests in the approximal spaces between the teeth or the alveolar process that supports them. Nor can these films be used very well for diagnosing the degree of resorption of these tissues. The type 1 anterior film is of less value for this purpose than the type 3 posterior film.

However, while examining the bite-wing roentgenograms used in this study for locating carious areas and overhanging approximo-gingival filling margins, both of us observed that it is often possible to determine that extensive resorption does exist. We saw extensive

resorption of the alveolar crests and alveolar process, that affected numerous anterior and posterior teeth of some of these recruits to an extent that indicated early loss of these teeth. It was seldom detected when the clinical or visual examinations were made. Excessive resorption of the alveolar crests and alveolar process is usually associated with persons older than these men. The average age of these recruits when they were examined aboard the U. S. S. *Saratoga* was 19⅚ years. The fact that this condition is present in an advanced stage in men as young as these is therefore most interesting.

Because of the limitations of bite-wing roentgenograms when used for this purpose, it is not possible to state accurately to what degree and in exactly how many of the 359 recruits an excessive resorption of the alveolar crests and alveolar process exists. There were, however, 34 recruits in whom the excessive resorption was so far advanced that it was easily and unquestionably apparent in the bite-wing roentgenograms. If the regular 1¼- by 1⅝-inch full-mouth series had been made, we are convinced it would have been possible to report many more such cases. The plan of this study did not originally include the alveolar crests or alveolar process and the regular 1¼- by 1⅝-inch full-mouth series of roentgenograms were therefore not made. It is only because the condition appeared so prominently in the bite-wing roentgenograms made for these very young men that it is reported here.

POLICY IN MAKING THE EXAMINATIONS

The examinations of the teeth of these recruits were carefully made in a routine manner, and there was no desire to be ultraexact or critical. When the clinical or visual examinations were made, carious areas were not recorded unless they could be definitely entered with the usual types of explorers furnished in the supply table of the Medical Department.

No doubtful areas were recorded when the bite-wing roentgenograms were read. Some questionable areas, that had not been recorded, were filled when the recruits received routine dental treatment.

The carious areas that were recorded definitely appeared to need correcting during the first enlistment period.

Only the large overhanging gingivo-approximal filling margins, that clearly needed to be corrected, either by mechanical removal of the large overhanging masses or replacement of the fillings, were recorded. Many small overhanging margins were not recorded because there was a doubt as to whether or not they were causing damage. Some of these were also corrected later when carious areas in approximating surfaces of the adjoining teeth were filled.

The same policy applied to the cases in which an excessive resorption of the alveolar crests and alveolar process was reported.

It was found to be impossible to make a perfect examination for every recruit. Our efforts were limited to making a practical and useful record, in a routine manner. Discounts must be allowed for artificial illumination in the dental office, time limits, and the deficiencies of the persons making the examinations.

BITE-WING FILMS FOR DETECTING CARIOUS AREAS

We believe that many incipient carious areas and some more advanced areas, in approximal surfaces, that may require early correction, would have been found if the type 2, intermediate bite-wing films, for posterior regions, had been used instead of the type 3, large bite-wing films. Because of the varying degree of curvature of the dental arches and the diverse alignment of the teeth of different individuals, overlapping of shadows of mesial and distal surfaces of posterior teeth often occurs when only one exposure is made for each side with the type 3, large bite-wing film. This can be corrected to a great extent by making two exposures for each side with the type 2, intermediate bit-wing film.

SUMMARY

The following dental conditions were found in 359 recruits who were examined soon after they arrived direct from the training stations for their first sea duty:

(1) 1,454 carious areas and useless roots involving 1,278 teeth of 285 recruits were charted in dental records.
(2) 2,478 additional carious areas and useless roots involving 2,058 teeth of 338 recruits were found on examination.
(3) 139 overhanging gingivo-approximal filling margins in 134 teeth of 87 recruits.
(4) 34 recruits with excessive resorption of alveolar crests.
(5) 3 gold shell crowns with excessive gingival overhang in 3 recruits.
(6) 2 teeth with useless roots in 2 recruits.
(7) 40 recruits had 32 serviceable teeth.
(8) 260 recruits had 28 or more serviceable teeth.
(9) Average age of recruits was 19 years and 10 months and average length of service was 6 months and 1 day.

ELIMINATION OF ORAL SEPSIS

By Lieutenant C. W. Schantz, Dental Corps, United States Navy

The etiology of paradentosis remains unknown. The general trend has been to accept theories based on local and systemic factors as the cause. A vast amount of research is being done in this field, much

progress has been made, and undoubtedly the cause will eventually be determined.

The most important local etiologic factors in paradentosis which promote successful bacterial invasion include: (*a*) The malrelation of the teeth and their supporting tissues; (*b*) the character and the flow of the saliva; (*c*) dietary deficiency, and (*d*) the lack of proper oral hygiene. However, the disease may occur in the oral tissues where reasonable hygiene has been practiced. This has been explained as due to a systemic lowered resistance.

In the naval service the inflammatory and the suppurative types of paradentosis occur. The inflammatory type is often classed, in its early stages, as gingivitis and chronic Vincent's infection. The suppurative type is seen more frequently in the advanced-age group and then often when the ravages of the disease have advanced too far for corrective measures. The advanced type is seldom worthy of treatment if the welfare of the patient is to be considered. The loss of supporting bone precludes corrective treatment and ultimately results in the loss of the teeth. The common causes of paradentosis are of interest to us in the naval service because corrective conservative treatment in the early stages of the disease results in successful elimination of the disease.

The following case report is herewith introduced as evidence that early treatment and subsequent hygienic measures will produce lasting effects:

Case Report

R. H.—GM1c, United States Navy, age 40.

First impression was that of a chronic suppurative periodontitis, diagnosis undetermined. Patient gave history of early gingivitis and bleeding gums preceded by an acute Vincent's infection. Condition existed for a period of three years.

Examination.—Careful examination disclosed heavy calculary accumulations. The gingivae presented venule engorgement indicating marked inflammation. The interproximate papillae were blunted and slightly edematous. On pressure a pus exudate could be easily expressed from the gums. Marked gingival recession was also observed.

Mucous membrane.—Appeared anemic compared with the gingival tissues.

Occlusion.—Marked attrition molars.

Tongue.—Coated.

Palate.—Normal.

Tonsils.—Absent.

Pharynx.—Edematous.

Breath.—Fetor oris, foul.

Palpation.—Slight mobility of the teeth. Mandibular joint apparently normal. Lymphatic glands normal, except superficial group slightly tender.

Pulp test.—All teeth vital. (Pulp stones Nos. 1–2–3–14–15.)

Prognosis.—Favorable.

X-ray verification.—Full mouth x-ray examination revealed the following: There is extreme alveolar atrophy. This extends in more or less of a straight line including the entire upper and lower processes. Due to the loss of the well-defined alveolar ridge seen under normal circumstances there are many food impaction areas with definite periodontal pocket formations, especially in the posterior regions. The teeth show attrition previously referred to in the history. Pulp nodules are present in the molar teeth. There is no marked evidence of apical involvement.

DIAGNOSIS.—Paradentosis (suppurative).

OPERATION.—Gum resection.

GUM RESECTION

Gum resection, as a treatment for the elimination of infection and the production of a condition conducive to mouth hygiene is in itself not new to the profession, but it is in the opinion of this observer, a neglected one. The simplicity of the operation and the subsequent lack of discomfort to the patient should make the procedure a popular one and at the same time would divert much of the prosthodontists labor.

This operation is performed by passing a sawlike instrument between each tooth to be operated upon, cutting through the soft tissues to the surface of the bone at the bottom of the pocket, where a few horizontal strokes are made to level off the ragged bone edges. When this has been done there will be a series of vertical incisions of varying lengths on the bucco-lingual surfaces of the gums. These are united by a horizontal incision on the buccal and lingual surfaces and the entire gum tissue removed to the level of the bone loss. The root surfaces are then curetted (though this may be deferred to a later sitting if desired) and the exposed bone smoothed and cleaned of any remnants of soft tissue, after which the operative area is packed with a surgical cement. This is worked through between each tooth while soft, and moulded in such a way as to cover and protect the entire operative field, including the exposed root surfaces. Within half an hour this becomes hard and is allowed to remain in place for several days, after which it is removed and replaced by another pack. With an occasional repacking, the cement is kept in place for from 2 to 4 weeks, depending upon the amount of tissue removed. It serves the purpose of preventing the formation of other gum tissue through the organization of the blood clot, protects the wound during the period of healing, and the root surfaces against thermal shock. Without such protection, the operation of gum resection would not be feasible.

A good surgical cement that can be put up by any chemist is the following:

POWDER.—Equal parts: zinc oxide and powdered resin. To four parts of the above mixture add one part tannic acid. Measure by volume. Do not weigh it.

LIQUID.—Two parts eugenol. One part sweet almond oil (not bitter). Melt a little powdered resin into the eugenol, using a test tube, and heat very slowly. Pour into the almond oil and shake well.

As a rule there is very little post-operative pain in these cases. In operating, it is usually desirable to divide the mouth into six areas, the four molar and bicuspid regions and the incisal areas, and operate on each in turn. There are those, however, who recommend doing the necessary work in one operation. If root surfaces are found to be sensitive following the operation, these can usually be controlled by the application of silver nitrate or formaldehyde.

The objection urged against gum resection is that it is disfiguring. This is less so than would be expected, for the gum tissue slowly grows up from its new level until it occupies about half of its former position. Moreover, gum resection need not, as a rule, be practiced in the incisor regions. As pointed out, there are several other ways by which even deep pockets in these locations can be controlled.

Of all the operative procedures, gum resection is the most certain. The pocket is completely obliterated, and with it the possibility of its reinfection. When limited to the posterior part of the mouth it has few objections when measured by the satisfactory results that are obtained. And when compared with the possible loss of teeth by other and less effective methods, its value can hardly be overestimated.

CONCLUSION

It may be said that by the intelligent application of the different operative procedures now in use and proved by experience to be effective, paradentosis is as amenable to treatment and the results are as satisfactory as is the treatment of any other disease. Since this is true, there can no longer be any excuse for the unfortunate condition by which pyorrhoea heads the list as a cause of tooth loss. Prevention and early treatment should, however, be the goal toward which we should strive.

ACKNOWLEDGMENT.—The author desires to acknowledge the cooperation and advice of Dr. Arthur H. Merritt, New York City, and the assistance and interest of Lieutenant E. J. Shields (DC), United States Navy, who helped make this case a success.

LOCAL ANESTHESIA

ITS APPLICATION IN OPERATIVE DENTISTRY

By Lieutenant Glenn W. Berry, Dental Corps, United States Navy

Most dental operations are performed on structures, directly or indirectly, innervated by the terminal branches of sensory nerves, which are conceded to be most sensitive and highly responsive to pain.

Through a succession of dental experiences, a patient has recorded in his memory unpleasant associations with treatment procedure, es-

pecially that related with the dental engine. On countless occasions, patients have voluntarily expressed their views on dentistry, particularly in regard to this apparatus. The reason for this reaction in many cases may be due to insufficient regard for the patient's comfort especially in preparation of extremely sensitive cavities. The application of local anesthetics has been so simplified that we are now able to prepare cavities in all teeth without the resultant harmful psychologic experiences occasioned by actual pain.

Local anesthesia, widely used, has attained its rightful place among accepted dental treatment procedures. The responsibilities involved in its extensive use with almost complete safety and success, should never be minimized. Successful administration of local anesthetics involves careful application of its fundamental principles, namely: Scrupulous asepsis, thorough knowledge of the involved anatomic structures, and an exact technic.

The confidence of the patient should be gained beforehand in order that his excitement and fear may be allayed. He should be advised of the proposed procedure and of the sensations and after effects to be expected. A few moments spent in establishing such a relation may serve to add to the peace of mind of the patient as well as that of the operator.

Before local anesthesia may be efficiently employed, gentleness in syringe manipulation and the handling of the anesthetized tissue is especially important. The practice of boldly thrusting a hypodermic needle deeply into tissues not previously anesthetized should be discouraged. Careful insertion of the needle coupled with slow injection of the anesthetic solution with a sufficient time interval lapse for its effect will materially aid the patient's comfort.

There should be no hesitancy about blocking both right and left mandibular nerves at the same time. Although the lingual nerve is not deliberately anesthetized in cavity preparation, it has been my experience that anesthesia of the tongue occurs when the anesthetic solution is deposited in the pterygo-mandibular space.

In my experience, the infra-orbital injection has been very successful and, if the anatomy of the area is borne in mind, there are very seldom any postoperative sequelae associated with the injections. When cavity preparation, involving numerous teeth in either right or left maxilla is necessary profound anesthesia will be induced by means of two injections, the infra-orbital and the tuberosity. In cavity preparations involving only the bicuspids and incisors of both right and left maxillae, the infra-orbital injections are sufficient. In almost all cases the tuberosity injection will anesthetize the maxillary first as well as the second and third molars. Thus cavity preparation can be performed painlessly.

Experiences at various training stations has proven the practicability of using local anesthesia for cavity preparation. Among recruit patients, complete blocking of the entire maxillae and mandible may be indicated if all the necessary cavity preparations are to be performed at one sitting. Profound anesthesia can readily be induced in these areas by means of six injections *i. e.*, right and left inferior dental, tuberosity, and infra-orbital injections. Of course if the operator prefers, he can anesthetize only one-half of the mandible or maxillae, as indicated, having the patient return for subsequent appointments.

In a series of cases involving approximately 85 injections per month, there have been very few cases of toxic reaction, postoperative pain caused by injection, or tenderness. The patients have been very enthusiastic about this method of pain elimination. They prefer any unpleasant sensations they may experience as a result of the local anesthetic, to the pain involved in cavity preparation. Unfavorable reactions were, in practically every case, due to conditions such as apprehension, nervousness, improper technic, or vasoconstrictor agents rather than the procaine. The general systemic condition, naturally, must not be overlooked.

The following procedure is followed each morning: The dental technician prepares the necessary glass syringes and needles by boiling, places them in a sterile receptacle, and prepares a quantity of procaine solution.

If the injection has been made properly, anesthesia is usually induced very quickly, thereby eliminating delay in operative procedure. Pain is a sensation from which everyone wishes to escape. Yet we often forget that the purpose of the pulpal nerve tissue is to make the patient aware of the need of pulp protection against injury. The fact that this natural protection is absent under anesthesia, requires that additional care be exercised during operative procedures. In order to protect dental pulps the speed of the revolving bur should be diminished.

The arguments which are presented by some against the use of local anesthesia for cavity preparation are for the most part unfounded. If we are so unschooled and careless as to place dental restorations directly upon a pulp exposure or so foolish as not to respect the tissues upon which we operate, anesthesia should of course not be used.

During the past several months I have used local anesthesia routinely, in cavity preparations. I find that the use of local anesthesia permits me to perform better operative dentistry with less effort and with more comfort to the patient. No originality is claimed for this article, the primary object being to bring to the attention of the

dental officer the desirability and practicability of routine use of local anesthesia for operative dentistry in the Navy.

VISUAL DEFICIENCY

INCIDENCE AT THE UNITED STATES NAVAL ACADEMY AND MEASURES TAKEN FOR ITS REDUCTION

By Captain Reynolds Hayden, Medical Corps, United States Navy, and Commander Orville R. Goss, Medical Corps, United States Navy

Extensive experience at the Naval Academy in conducting physical examinations of candidates for admission and of midshipmen for promotion has demonstrated the fact that a great number of persons in this country, both service and civilian, understand neither the reasons for the present visual standards for admission to the Academy and for commission in the line of the Navy, nor the procedure in connection with eye examinations at the Academy. This includes ophthalmologists, many of whom do not understand why candidates having 20/20 or even 20/15 vision with each eye and such a low degree of myopia or myopic astigmatism as ¼ of a diopter should be rejected for admission to the Academy. Nor do they understand why midshipmen in the two lower classes having less than 20/20 vision with low refractive errors or those in the two upper classes having less than 15/20 vision should be required to leave the naval service; nor why midshipmen having slightly less than 20/20 vision caused by myopia should be refused a commission in the line of the Navy. This article is written in an endeavor to so explain this important matter that all concerned will understand. Furthermore, it is desired to report the findings at the Academy concerning various important ophthalmological facts in this connection, facts which are felt to have been conclusively demonstrated at the Academy but which are little known or appreciated elsewhere.

It is a matter of common knowledge that normal vision is a prime requisite for navigation and watch officers in the line of the Navy. They must be able to promptly distinguish other vessels, the coast, or navigational aids such as buoys, etc., in any weather and at any time. It should not be necessary for them to wear glasses in order to do this because, in addition to the danger of breakage or loss, glasses become fogged in bad weather with serious impairment of vision. The visual standards for the line of the Navy have always been based upon these facts.

As with all officers in the Navy, midshipmen are given annual physical examinations. Unless otherwise directed by the Navy Department in individual cases, passing these annual examinations is a prerequisite for promotion from a lower to a higher class at the Naval Academy as is the case with officers being promoted from a

lower to a higher rank. The annual physical examination of the graduating class is considered as their physical examination for commission. The visual standard required to be met by midshipmen at these annual physical examinations has always been the same as for a commission in the line of the Navy. Prior to 1907, this standard was "normal vision." For the period 1907–31 inclusive, the standard was 15/20 vision each eye unaided by lenses. Commencing with 1932, the visual standard for commission in the line of the Navy and for retention of midshipmen in the Academy was increased to 20/20 each eye unaided by lenses with the proviso that 18/20 vision in either eye would be accepted if refraction under a cycloplegic showed no myopia to be present.

For a number of years the comparatively high incidence of visual deficiencies which developed among midshipmen was a cause of serious concern to the Navy Department, to the Naval Academy authorities, and to the midshipmen, their parents, and Congressmen. The fact that so many midshipmen who had been admitted to the Naval Academy with supposedly normal or 20/20 vision developed visual deficiencies, sometimes as low as 8/20 or 10/20, within a year or two after admission was not only most distressing but resulted in the loss of much time and money by both the Government and the individual concerned. Before discussing the measures taken for the reduction in number of these cases of visual deficiency and in order that the reasons for the present visual standards at the Academy for the admission of candidates, retention as midshipmen, and commission in the Navy may be understood, a review of the background is necessary.

HISTORY OF METHODS OF HANDLING VISUAL DEFICIENCIES

Perusal of the history of annual physical examinations at the Naval Academy with special reference to cases of visual deficiency, shows a disturbing lack of coherency until 1939 when visual standards were decided upon which are logical and necessary from the Government viewpoint, fair to both the Government and the midshipmen, and presumably will be maintained. In previous years, notwithstanding very definite regulations concerning physical standards to be met by midshipmen, there were many variations of departmental policy from year to year in the cases of both individual midshipmen and groups of midshipmen. These variations were almost entirely in connection with visual deficiencies, which constituted a continual source of concern and embarrassment for the Navy Department and of disappointment or frustrated ambition for individuals.

Many remedial measures have been employed during the past 30 years in endeavor to prevent the development of myopia among mid-

shipmen. Surveys of the painting and lighting of midshipmen's rooms in Bancroft Hall were made from time to time in order that every advantage might be taken of advances in the quality of illumination furnished for study purposes. A common practice developed of giving cases of deficient vision complete rest of their eyes for as much as 2 weeks during the academic year before reexamination of their vision in order that they might have every possible aid in passing the Snellen test. Many cases found to have deficient vision at the annual physical examination in February or March were reexamined in the Bureau of Medicine and Surgery, Navy Department, and frequently passed there. Others were reexamined at the end of the academic year and, if then still deficient, were again reexamined in September after practically complete rest for their eyes all summer. In 1922 the date of beginning the annual physical examinations for midshipmen was changed from February to October on the grounds that the physical condition of midshipmen was at its best after their summer cruise and leave and that midshipmen who had visual deficiencies would have had ample opportunity to rest their eyes. Beginning in 1937, annual physical examinations of midshipmen were again commenced in February. This change was made because analysis of the results of 15 years of annual physical examinations in the fall showed that little, if anything, had been gained over the previous custom of commencing annual physical examinations in February. While it appeared somewhat advantageous to the midshipmen to have the examinations in the fall, evidence showed that commencing them in February was definitely for the better interests of the Government and not really detrimental to the midshipmen. Many cases of defective vision were continued at the Academy subject to visual findings at their next annual physical examination in the hope that their vision would improve. Some of these cases were so continued for 2 and even 3 years and then finally graduated but not commissioned. In addition to the desire of the Navy Department to retain as many midshipmen as possible, many decisions to continue cases of defective vision were made in an earnest endeavor to be absolutely fair to the individual concerned. Sometimes, midshipmen would obtain certificates from civilian eye specialists to the effect that their eyes were "normal." They would then use these certificates to bring pressure for continuance at the Academy.

In an endeavor to stabilize final decisions concerning cases of deficient vision, three recommendations were made to the Navy Department during the past 20 years that legislation be recommended to Congress placing physical fitness on the same basis as scholastic ability. In other words, midshipmen found to be physically dis-

qualified would be finally acted upon by the Academic Board of the Naval Academy and not referred to the Navy Department for final decision. These recommendations were all disapproved. In 1932 the Secretary of the Navy published a *Policy Regarding Physical Examinations at the Naval Academy*. This policy was absolutely fair to all concerned, both the Government and the midshipmen, and would have eliminated many perplexing cases of defective vision had it been enforced. Unfortunately, exceptions became so numerous that this policy was practically nullified until 1938 when all rejections for deficient vision in the fourth or freshman class were approved by the Navy Department.

Conditions concerning defective vision among midshipmen became more acute with increasing size of the Navy until, because of the number of younger line officers being retired because of defective vision, the Navy Department in 1931 directed that the visual standard for commission in the line of the Navy be increased to 20/20 each eye unaided by lenses, or 18/20 if the individual was shown by refraction to be hyperopic. In May 1932 the Department directed that this new visual standard first become effective for the class of 1933. This change was presumably made because of the considerably increased number of cases of visual deficiency found in the class of 1932 upon application of the new standard. While this new visual requirement would decrease the number of cases of defective vision among junior line officers, it had little bearing upon the incidence of defective vision among midshipmen.

During 1932–33 there was considerable criticism of the quality of illumination supplied the midshipmen for study and classroom purposes. Upon request of the senior medical officer, a board of nationally prominent illuminating engineers met at the Naval Academy in 1934 to consider this matter. Their report recommending extensive changes to improve the quality of illumination in the dormitory (study) rooms, classrooms, and laboratories for midshipmen was approved. The new lighting system for midshipmen's rooms was completed in November 1936 and for classrooms in the spring of 1937. As will be explained later, this improved illumination undoubtedly had a beneficial effect upon the maintenance of normal vision among midshipmen. Because of the changed visual standards for admission to the Academy which became effective the summer of 1937 however, the extent of this beneficial effect cannot be definitely determined.

In order that the importance of this problem may be appreciated, the total rejections for defective vision at the Naval Academy in the classes of 1927–39, inclusive, are shown in the following table. These percentages are averages for the entire time each class was at the Academy.

Percent rejected by year when the visual requirement for retention at the Academy and for commission was 15/20 each eye

Year	1927	1928	1929	1930	1931
Percent rejected	1.36	1.96	1.45	1.41	1.54

Percent rejected by year when the visual requirement for retention at the Academy and for commission was 20/20 each eye or 18/20 if hyperopic

Year	1932	1933	1934	1935	1936	1937	1938	1939
Percent rejected	5.21	4.14	9.63	7.18	5.06	6.78	7.67	12.75

From above figures it is readily seen that drastic measures were necessary to reduce the incidence of myopia at the Academy if the visual standard for the line of the Navy was to be maintained without a tremendous loss of midshipmen.

While the average incidence of defective vision at the Naval Academy for the period 1932–39 was only about one-fifth that among students in civilian colleges and while, because of their age and amount of study, it could not be entirely eliminated among midshipmen, the fact remained that it was extremely high from the governmental viewpoint because of the very considerable loss of time and money involved in educating a midshipman only to have him physically disqualified because of defective vision after 1 to 4 years at the Academy.

The following figures for the class of 1939, which may be considered as approximately applicable to all classes, show the practical futility of expecting any real results from the former policy of continuing cases of defective vision from year to year in the hope that their vision would so improve as to be satisfactory for a commission in the line.

CLASS OF 1939 AT GRADUATION

Number in class	581
Percent given revocable commissions, line or staff (18/20–20/20)	0.008
Percent given revocable commissions, staff only (15/20–17/20)	10.832
Percent leaving naval service because of defective vision (below 15/20)	8.09
Total percent having defective vision (below 20/20 each eye)	18.93
Percent rejected in previous years for defective vision but continued by Navy Department and now having 20/20 vision	1.37
Percent rejected in previous years for defective vision but continued by Navy Department and now having defective vision (below 20/20)	9.62
Percent of class which would have left the service because of defective vision (less than 15/20) had no cases previously rejected for defective vision been continued by the Navy Department	3.79

Percent of class which would have been given revocable commissions, line or staff (15/20–19/20 inclusive), had no cases previously rejected for defective vision been continued by the Navy Department	4.98
Total percent of class which would have had defective vision (below 20/20) had no cases previously rejected for defective vision been continued by the Navy Department	8.77

From these figures, it is seen that had all rejections of midshipmen in this class for defective vision during previous years been approved by the Navy Department, only 42.74 percent of the total cases actually having defective vision (below 20/20) at graduation would have been found. It is also seen that only 1.37 percent of previously rejected but continued cases of defective vision were found to have 20/20 vision at graduation. Such a small percentage of salvage is definitely not worth while.

LATE RESULTS OF PREVIOUS POLICIES CONCERNING VISUAL STANDARDS

The late results of previous policies concerning the enforcement of visual standards at the Naval Academy and for commission in the line of the Navy are well illustrated by a review of the visual findings at annual physical examinations in 1937–38–39 of all line officers below the rank of captain on duty at the Academy. These included members of all classes from 1916 to 1933 inclusive except the classes of 1920, '27, and '28, none of whom were then on duty at the Academy. All of these officers with the exception of those in the class of 1933 were commissioned under the old standard of 15/20 vision each eye. Because of the number and varying ranks of the officers concerned, they are considered to be a fair cross-section of the entire active line officer personnel of corresponding ranks in the Navy. These findings may, therefore, be considered a reasonable index of the existent vision of all active line officers of the Navy in the ranks of commander to lieutenant, junior grade, inclusive. Details are given only of those officers found on one of these annual physical examinations to have vision of 10/20 or less in each eye. See tables 1, 2, and 3.

TABLE 1.—*Visual findings of line officers on duty at the Naval Academy*

	1937	1938	1939
Total number examined	464	433	431
Percentage of number examined having defective vision (below 19/20 in 1 or both eyes)	20.47	23.09	34.33
Percentage having vision 15/20–18/20 inclusive in 1 or both eyes	10.13	11.31	12.99
Percentage having vision 11/20–14/20 inclusive in 1 or both eyes	4.53	4.85	9.74
Percentage having vision 10/20 or less in 1 or both eyes	5.81	6.92	11.60
Percent of total cases of defective vision found (below 19/20 in 1 or both eyes) who had defective vision at the Academy	64.21	67.00	56.08
Percent of cases now having 15/20–18/20 vision in 1 or both eyes who had defective vision at the Academy	57.44	57.14	48.21
Percent of cases now having 11/20–14/20 vision in 1 or both eyes who had defective vision at the Academy	66.66	76.19	47.61
Percent of cases now having 10/20 vision or less in 1 or both eyes who had defective vision at the Academy	74.07	76.66	72.00

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TABLE 2.—*Period when defective vision developed*

Present vision [1]	Before admission	At Naval Academy	After graduation	No record	Total
1937 EXAMINATIONS					
10/20 or below	[2] 1	19	7	0	27
11/20–14/20	0	14	7	0	21
15/20–18/20	[3] 2	25	19	1	47
Total	3	58	33	1	95
1938 EXAMINATIONS					
10/20 or below	[2] 1	22	6	1	30
11/20–14/20	0	16	5	0	21
15/20–18/20	0	28	18	3	49
Total	1	66	29	4	100
1939 EXAMINATIONS					
10/20 or below	0	36	12	2	50
11/20–14/20	0	20	20	2	42
15/20–18/20	0	27	26	3	56
Total	0	83	58	7	148

[1] Vision given is for one or both eyes.
[2] One case, vision on admission R. 14/20; L. 18/20. Waived by Navy Department.
[3] One case, vision on appointment R. 14/20; L. 20/20. Reexamined at U. S. Naval Dispensary, Washington, D. C., and passed. One case, vision on appointment R. 17/20; L. 17/20. Passed by Board of Medical Examiners, U. S. Naval Training Station, Newport, R. I.

TABLE 3.—*Records of vision at Naval Academy of officers now having vision of 10/20 or below, each eye*

Class	Number examined	Vision 1937–38–39		Record of vision as a midshipman									
				On admission		First year		Second year		Third year		Fourth year	
		R/20	L/20	R/20	L/20	R/20	L/20	R/20	L/20	R/20	L/20	R/20	L/20
1916	2	5	5	20	20	20	20	20	20	9	10	15	15
		10	10	20	20	18	20	17	20	15	20	15	18
1917	1	7	7	20	20	20	20	15	20	8	10	15	15
1918	2	5	6	20	20	20	20	20	20	20	17	20	20
		8	7	20	20	20	20	20	20	20	20	20	17
1919	2	6	8	20	20	20	20	18	20	17	20	20	20
		10	10	20	20	20	20	15	20	15	15	15	15
1921	1	8	10	20	20	20	20	15	15	10	16	15	15
1922	2	2½	2½	20	20	20	20	15	12	15	15	7	8
		10	10	20	20	20	20	14	14	14	10	15	15
1923	2	7	9	20	20	20	20	20	20	20	20	15	20
		10	10	20	20	20	20	20	20	15	20	18	18
1924	2	10	10	20	20	20	20	20	17	15	12	15	15
		10	10	20	20	20	20	15	20	18	20	20	20
1925	4	10	10	20	20	20	20	20	15	20	15	20	15
		9	10	20	20	20	20	15	17	16	20	17	20
		10	10	20	20	20	20	16	16	17	17	15	15
		9	6	20	20	20	20	20	20	20	20	20	15
1926	1	6	4	20	20	20	20	20	20	17	15	[1]	[1]
1929	1	10	9	20	20	20	20	15	17	20	15	15	15
1930	[2] 1	9	8	17	17	14	18	15	17	10	15	15	15
1931	8	10	10	20	20	20	20	15	15	15	15	16	17
		10	10	20	20	20	20	15	15	20	15	20	18
		7	7	20	20	20	20	20	15	15	15	15	17
		8	8	20	20	20	20	20	15	20	20	20	20
		10	10	20	20	20	20	15	15	20	15	20	18
		8	10	20	20	20	20	15	15	15	15	16	17
		10	10	20	20	20	20	15	15	15	15	17	16
		10	10	20	20	20	20	15	15	15	15	20	20
1932	8	7	7	20	20	20	20	15	15	15	15	15	15
		8	8	20	20	20	20	15	20	15	20	20	20
		8	8	20	20	20	20	20	15	20	20	20	18
		8	8	20	20	20	20	15	15	20	20	20	20
		8	8	20	20	20	20	20	20	16	20	15	18
		10	10	20	20	20	20	20	15	20	20	15	15
		8	8	20	20	20	20	20	20	20	20	15	20
		10	10	20	20	20	20	15	15	20	18	20	18
1933	1	10	8	20	20	20	20	16	14	18	16	20	20
Total	38												

[1] No record.

[2] See table 2.

Inasmuch as some of these officers were on duty at the Naval Academy for more than 1 year of the 3-year period discussed, a combination of the findings during this time would not present a true picture. The findings in 1938, however, are typical and are briefly recapitulated in the following summary to emphasize the salient points. The term "defective vision," as used below, refers to one or both eyes.

DEFECTIVE VISION IN OFFICERS EXAMINED IN 1938

(*a*) Number examined	433
(*b*) Percent having defective vision (18/20 or less)	23.09
(*c*) Percent of (*b*) who first showed defective vision (less than 20/20) at Academy	67.00
(*d*) Percent of (*a*) having vision of 14/20 or less	11.77
(*e*) Percent of (*d*) who first showed defective vision (less than 20/20) at Academy	76.47
(*f*) Percent of (*a*) having vision of 10/20 or less	6.92
(*g*) Percent of (*f*) who first showed defective vision (less than 20/20) at Academy	76.66

The fact that slightly more than three-fourths of all these officers now having vision of 14/20 or less and of those now having 10/20 vision or less first showed their defective vision while at the Naval Academy would certainly appear to indicate the advisability of the present high visual standard for midshipmen and its strict enforcement.

NEW VISUAL STANDARD FOR ADMISSION TO THE NAVAL ACADEMY

The Naval Medical Department had long realized that the chief cause of the high incidence of myopia apparently developing among midshipmen was the number of candidates having latent or low degrees of myopia who were admitted to the Naval Academy because they could manage to read 20/20 with each eye by the Snellen test on their entrance physical examination but whose myopia promptly increased under the strain of the hard study necessary at the Academy. It was evident that the proper time to eliminate these latent cases of myopia was at the physical examination for admission to the Academy. Therefore, in 1937 the Surgeon General of the Navy recommended to the Secretary of the Navy that the regulations for the admission of candidates into the Academy be changed to require that, as part of their physical examination for admission, each candidate must submit to refraction under a cycloplegic and that any degree of myopia or myopic astigmatism should be a cause for physical disqualification. This was approved by the Secretary and became effective with candidates for the class of 1941, examined the summer of 1937.

The present regulations on visual requirements for admission of candidates to the Naval Academy, effective in 1940, state as follows:

> **Each candidate on entrance to the Naval Academy must have normal or 20/20 vision in each eye and must submit to refraction under a cycloplegic. Any degree of myopia, myopic astigmatism, or more than two (2) diopters of hypermetropia, shall cause rejection of the candidate.**

The requirement that more than 2 diopters of hypermetropia would be disqualifying was added to the visual requirements because those with hypermetropia suffer from asthenopia ranging from simple discomfort in the eyes to severe ciliary spasm with marked headache after more or less close eyework. As these persons grow older, the hypermetropia becomes absolute with the result that their distance vision becomes defective. In order to have normal distance vision, the proper lens correction must be worn, and close work is impossible without the correction. When the presbyopic age is reached bifocals become necessary in order that distant vision and close work may be carried out.

The myope with a comparable refractive error never has good distance vision without the proper lens correction but when he becomes presbyopic, he can remove his glasses for close work and never needs bifocals.

In aviation, the presence of more than 1 diopter of hypermetropia is disqualifying because, as the aviator grows older, his vision will become less than 20/20 and further, since accommodation necessary to overcome the hypermetropic error is necessary at all times for good distance vision, eye strain and eventually some ciliary spasm will occur with resulting temporary impairment of distant vision which may result in crashes, especially upon landing.

In this connection, it may also be stated that the United States Military Academy does not accept candidates having more than 1.5 diopters of hypermetropia.

Before these new visual requirements were actually used, many expressed fear that they would be impractical because of the number of candidates who would be rejected. This fear was proved groundless, as shown by the following tables giving percent of rejections because of defective vision of candidates for the classes of 1927–1943 inclusive.

Percent of candidates rejected, by class, because of defective vision under old visual standard; 20/20 vision each eye, Snellen test

Class	1927	1928	1929	1930	1931	1932	1933	1934	1935	1936	1937	1938	1939	1940
Percent rejected	4.00	3.79	3.24	4.42	4.34	1.89	4.50	4.42	4.34	2.89	4.75	6.19	6.46	4.59

Percent of candidates rejected, by class, because of defective vision under new visual standard; 20/20 vision each eye by Snellen test and no evidence of myopia under cycloplegic

Class	1941	1942	1943
Percent rejected	13.08	6.59	9.43

The percent of rejection of candidates for the class of 1941 was naturally high because the new visual standard was not published until the early spring of 1937. While every effort was made to disseminate information regarding the new visual requirements as rapidly as possible, candidates that year had practically completed their preparatory work for the entrance scholastic examinations and the majority took the physical examinations for admission regardless of the refractive condition of their eyes because it was too late to do anything else. A considerable number of candidates for the classes of 1942 and 1943 however, had preliminary eye refractions done in order that they might not waste time and money in useless preparatory school work if they were visually disqualified under cycloplegic. The percent of rejection for these two classes was therefore considerably less than for the class of 1941. The approximately 50 percent increase in rejections for the class of 1943 over those for 1942 is felt to be due to the fact that, with candidates for the class of 1943, the use of atropine as a cycloplegic in cases having not more than ¼ diopter of myopia under homatropine was discontinued, and that a comparatively large number of candidates for this class had no preliminary refraction.

The results obtained following use of the new visual standard for admission to the Naval Academy have been most gratifying and fully up to expectations as shown by the following summary giving percent of rejections for defective vision during the first 2 years each class indicated was in the Academy.

Percent of rejection because of defective vision during first 2 years as midshipmen

Class	1938	1939	1940	1941
Percent rejected	6.06	10.17	9.01	[1] 4.95

[1] New visual standard.

PROCEDURE IN EXAMINATION OF EYES

The work of refracting the eyes of all candidates for the Naval Academy as part of their physical examination for admission has been somewhat onerous but most interesting because of the insight

which it gave into border-line cases of myopia and the probability of the development of myopia in candidates having emmetropic eyes or a low degree of hypermetropia. As the writers have been frequently asked how physical examinations are conducted at the Academy, especially as concerns vision, and how it is possible to refract so many cases in 1 day, a description of these examinations insofar as the visual requirements are concerned, will be given.

The present routine for entrance physical examinations at the Naval Academy is for 100 candidates to report at 8:30 a. m. on Monday, Tuesday, and Wednesday of each week during the examination period. For various reasons, less than this number report. The greatest number examined on any one day during the past 3 years was 87. In order to be able to refract candidates it is necessary to have four eye specialists as members of the medical examining board. Three additional eye specialists are therefore ordered to the Academy for temporary duty during the period, approximately 5 weeks, when the mass of candidates for each new class is examined. These examinations are conducted only on the first 3 days of the week because candidates are not sworn in until after their eyes have returned to normal or practically normal following administration of a cycloplegic. Usually therefore, candidates are sworn in on the third day, those examined Monday being sworn in on Wednesday, etc. While the medical department has no objection to examining candidates on Thursdays and Fridays, this is impractical because they cannot be admitted on Saturday or Sunday and it would cause the candidates unnecessary expense if they waited until the first of the next week. To many candidates, a board bill in Annapolis for 1 or 2 extra days is a matter for serious consideration. Some simply cannot afford it.

Upon reporting at sick quarters to take the entrance physical examination, the first 15 or 20 candidates are first examined for color vision; then the Snellen test; then the condition of the eyelids, conjunctiva, pupillary reflexes, etc. Administration of the cycloplegic (homatropine 4 percent) is then commenced immediately and continued while the candidate is receiving the remainder of his physical examination. The remainder of the candidates are given their physical examination in the regular routine, administration of the cycloplegic not being commenced until after completion of the rest of the examination. Between 10:30 and 11 a. m., the first group to receive their cycloplegic are ready to be refracted. In carrying out this method of administering the cycloplegic, it is of course necessary to have a record of the exact times of administration of the homatropine. This is done by notation on the record of physical examination, a rough copy of which each candidate carries with him throughout his examination. As refraction of the first group of candidates is completed,

those next to receive the cycloplegic are about ready for refraction, and are refracted as rapidly as possible. Each eye specialist has a separate room for refraction. Candidates found by one specialist to have myopia under cycloplegic are checked by two other eye specialists in order that there may be no doubt that myopia is present.

For the information of those who have expressed surprise at the number of candidates refracted daily during the entrance physical examinations at the Naval Academy and have made inquiry concerning the method followed, the following brief description is given together with examples of some border-line cases:

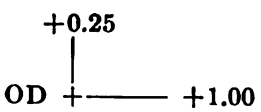
After the vision of each candidate is determined by the Snellen test, a cycloplegic consisting of a 4-percent solution of homatropine hydrobromide made up in a ¼ percent solution of cocaine hydrochloride, and ophthalmic tablets containing 1/50 grain each of homatropine and cocaine, is used. Experience at the Academy has shown that a 4-percent solution of homatropine is necessary in order to adequately overcome the spasm of accommodation so frequently present. The 2-percent solution commonly used for refracting eyes is often inadequate for this purpose.

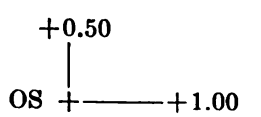
A drop of the solution is instilled in each eye and the patient is told to keep the eyes closed. Then, in 10 minutes, a tablet is placed in the lower conjunctival cul-de-sac of each eye followed again at 10-minute intervals with a drop of the solution for three doses so that there are five applications of the cyclophlegic to each eye. At the expiration of 1 hour from the first instillation of the cycloplegic, the candidate is taken into the refraction room for the completion of the examination.

The vision under cycloplegic is recorded, using the usual 20-foot distance from the Snellen test card, as 20/30 or whatever the reading may be. Retinoscopy at 1 meter distance is then carried out and the findings thus found are recorded. The candidate is then placed on the range and a lens equivalent to the retinoscopic findings, less 1 diopter for the meter distance, is placed over each eye. The objective findings by retinoscopy are the basis for the correction of all refractive errors found and not the subjective findings as obtained by use of the trial case alone. One eye is blanked off and the examinee is asked to read the smallest line he can see clearly on the test card. The refraction is considered completed if he reads 20/20 or better with each eye separately, provided there is a definite hypermetropia present which does not exceed 2 diopters. In low hypermetropic and myopic errors, the routine of the trial case is used in order to determine the best lens combination, and this is checked with the cross cylinders.

The following examples of the refraction in cases of some low myopic refractive errors are given. All these cases were rejected.

T. J. O'D.
Vision: OD 20/15; OS 20/15.
Vision under homatropine cycloplegic: OD 20/20–1; OS 20/15.
Retinoscopy:





Cycloplegic correction:

OD Minus 0.50 cyl. axis 30°=20/15

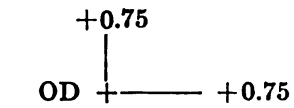
OS Minus 0.50 cyl. axis 150°=20/15

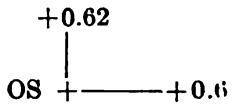
N. S. M.

Vision: OD 20/15; OS 20/15.

Vision under homatropine cycloplegic: OD 20/15–4; OS 20/20–1.

Retinoscopy:





Cycloplegic correction:

OD Minus 0.25 sphere=20/15

OS Minus 0.37 sphere=20/15

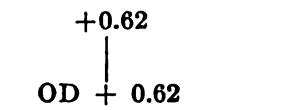
This case was also refracted under atropine, 1 percent solution, as cycloplegic and the same findings and corrections were found.

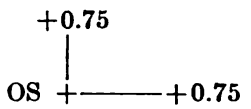
R. A. E.

Vision: OD 20/15; OS 20/15.

Vision under homatropine cycloplegic: OD 20/15; OS 20/15.

Retinoscopy:





Cycloplegic correction:

OD Minus 0.25 sphere=20/15

OS Minus 0.25 sphere=20/15

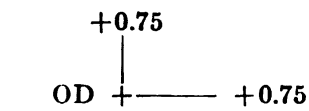
Refraction under 1 percent atropine cycloplegic was done in this case with the same findings.

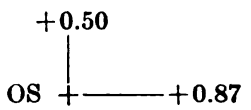
R. A. C.

Vision: OD 20/15; OS 20/15.

Vision under homatropine cycloplegic: OD 20/15; OS 20/20.

Retinoscopy:





Cycloplegic correction:

OD Minus 0.25 sphere=20/15

OS Minus 0.12 sphere combined with Minus 0.37 cyl. axis 180°=20/15.

The correction is usually made for 20/15 instead of 20/20 in these cases because these young men all have better than 20/20 vision when their visual acuity is first taken, with the exception of a few who have low hypermetropic astigmatic errors associated with some ciliary spasm. The candidates who cannot be corrected to better than 20/20 are not disqualified provided their visual acuity is 20/20 in either eye unaided by lenses.

Candidates are not examined for the phorias, but cases with an obvious squint are of course disqualified.

Upon completion of his physical examination, including refraction, each candidate reports to the president of the *Permanent Medical Examining Board* with his complete record of physical examination, including report of refractive findings. The candidate is then informed of his exact status, whether accepted, rejected, or to be reexamined, together with the reason or reasons for the action taken by the board. Some candidates, usually those having a comparatively high degree of hypermetropia, hyperopic astigmatism, or spasm of accommodation, do not read 20/20 with each eye on their first Snellen test but, under cycloplegic, are found to be definitely hyperopic. These are held for from 3 days to a week after their refraction in order that their eyes may definitely return to normal after the cycloplegic. They are advised not to use their eyes unnecessarily, not to go to the movies, *etc.*, and are reexamined by the Snellen test on as many successive days as may be necessary to give them every opportunity to pass the test. While an occasional candidate whose eyes are satisfactory on refraction fails to pass the Snellen test and is therefore rejected because of less than 20/20 vision, the vast majority who fail on their first examination pass the reexamination and are accepted.

All finally rejected candidates are informed that they may appear before a *Board of Medical Review* at the Naval Hospital, Annapolis, Md., for reexamination within 2 weeks of the date of rejection by the first board. This reexamination only pertains to the condition or conditions for which the candidate was rejected by the *Permanent Medical Examining Board.* It is a privilege and not required but assures the candidate of an impartial reexamination by a board composed of different medical officers than those who originally rejected him. The *Board of Medical Review* is provided in order that the candidate, his parents and friends, may be assured that he has been given every consideration and treated with absolute fairness. Candidates who have been rejected by the *Permanent Medical Examining Board* because of some degree of myopia or excessive hypermetropia found on refraction are directed to return to sick quarters at 8:30 a. m. the next day. They are then given sufficient additional homatropine to insure that their pupils are fully dilated and their accommodation adequately paralyzed. They are then sent to the *Board of Review* and promptly refracted by them. This is done in order to make it unnecessary for the candidate to take a cycloplegic twice. Subject to approval by the Navy Department, its decision is final. In case the candidate is accepted by the *Board of Review*, he is promptly sworn in as a midshipman.

At annual physical examinations of midshipmen, those who fail to pass the Snellen test are given every opportunity to do so on reexamination. Experience has shown that prolonged rest of the eyes

before reexamination is unnecessary. Normal eyes may not read 20/20 soon after excessive use, as in studying for examinations, but promptly return to normal with a moderate and reasonable amount of rest. In order to be fair to the midshipmen therefore, the annual physical examinations are not commenced until 10 days after completion of their bimonthly examinations in January. Those who fail to pass the Snellen test on first examination are advised regarding rest of their eyes, and are reexamined on the Monday following their first test. This gives a minimum of about 50 hours complete rest of their eyes before reexamination. The majority pass this reexamination. Those who do not are given every consideration before being finally rejected. While details vary with different individuals, all who cannot pass the Snellen test within a reasonable time later have their eyes refracted and are given a final Snellen test after the effects of the cycloplegic have worn off. If they then fail, they are sent before the *Board of Review* for reexamination before being finally rejected.

EXPERIENCE WITH ATROPINE AS A CYCLOPLEGIC

Probably our most interesting experience in connection with the refraction of the eyes of candidates concerned the use of atropine as a cycloplegic. Because recovery of the patient from the effects of homatropine is so much more prompt than from the more powerful cycloplegic atropine, and because homatropine is so generally and satisfactorily used as a cycloplegic, it was originally expected that only homatropine would be used for these examinations at the Naval Academy. In the case of one candidate, however, who was rejected in June 1937 because of myopia under homatropine cycloplegic (minus 0.25 diopter each eye), a civilian ophthalmologist stated that some cases showing such a low degree of myopia when refracted under homatropine would show either no myopia or a low degree of hypermetropia if refracted under atropine. The candidate in question actually refracted plus 0.25 diopter in each eye under atropine. In view of the fact that no statistics were available to indicate how the vision of such cases would stand up under the study regime at the Academy, in fairness to the candidate concerned, and in order that all elements of doubt with relation to border-line cases of myopia might be removed, the Bureau of Medicine and Surgery directed that, if they so desired, candidates refracting not more than ¼ diopter of myopia under homatropine should be again refracted with atropine as a cycloplegic and that such candidates showing no evidence of myopia under atropine should be accepted if otherwise physically qualified.

The consensus of opinion of the naval eye specialists on duty at the Naval Academy was that these candidates were poor risks insofar

as maintenance of 20/20 vision was concerned but, in accordance with the directions of the Bureau, atropine was used as a cycloplegic for 2 years with such candidates for the classes of 1941 and 1942. While complete results cannot be tabulated until 1941, the results to date are shown in the following summary and are believed to be the first statistics collected on this subject. The term defective vision, as used below, means less than 20/20 vision in one or both eyes.

RESULTS FROM USE OF ATROPINE AS A CYCLOPLEGIC

CLASS OF 1941

Number refracted under atropine	66
Percent accepted	65.15
Percent of acceptances found physically disqualified because of defective vision within 2 years	20.93

CLASS OF 1942

Number refracted under atropine	61
Percent accepted	63.93
Percent of acceptances found physically disqualified because of defective vision within 1 year	30.77

CLASSES OF 1941 AND 1942

Percent of all acceptances after atropine as a cycloplegic who developed defective vision within 2 years	25.60
Percent cases admitted after refraction under atropine formed of total cases in these classes rejected for defective vision	39.00

As a result of above findings, the Bureau of Medicine and Surgery decided that the percent of defective vision developing in cases admitted into the Naval Academy after refraction under atropine was so high as not to warrant continuance of this method of refraction. It was therefore directed that the use of atropine as a cycloplegic at the physical examinations for admission be discontinued. Candidates for the class of 1943 who showed a low degree of myopia under homatropine were therefore rejected without further refraction under atropine. This had considerable bearing on the increased number of candidates for the class of 1943 rejected because of deficient vision as compared with those for the class of 1942.

OBSERVATIONS ON REFRACTIVE FINDINGS ON PRELIMINARY AND ON ENTRANCE EXAMINATIONS

Following the numerous refractions of candidates at time of preliminary physical examinations made necessary as a result of the new visual standard for admission to the Naval Academy, it was found that many candidates who refracted PLANO or ¼ diopter of hypermetropia in one or both eyes when given their preliminary physical examination and were therefore qualified at that time, showed a low degree of

myopia at time of their entrance physical examination 6 to 15 months later and were therefore then rejected. Occasionally a candidate who refracted ½ diopter of hypermetropia on his preliminary physical examination would develop a low degree of myopia by the time he took his physical examination for admission and therefore be rejected. This change in refraction, while partially due to natural causes, was evidently caused largely by the intensive study these candidates performed during the intervening time. Practically speaking, an emmetropic eye is abnormal. Such eyes refract PLANO, showing neither myopia nor hypermetropia under cycloplegic. Those candidates therefore who refracted PLANO at time of their preliminary examination are considered to have been border-line cases definitely on their way toward myopia at that time. While experience at the Academy during the past 3 years has demonstrated to the medical officers on duty there the impossibility of predicting with any real degree of accuracy the probable future vision of any eye with any given refractive error, this experience has led them to consider all candidates showing less than 1 diopter of hypermetropia at time of admission to be doubtful cases insofar as maintenance of 20/20 vision during their 4 years at the Academy is concerned. Candidates refracting only ½ diopter of hypermetropia at time of admission to the Academy are considered to be definitely bad risks as regards maintenance of normal vision until graduation. To illustrate this, the cases of some candidates for the class of 1943, examined the summer of 1939, will be given.

Review of reports on file at the Naval Academy of refractive findings of 18 of these candidates passed on preliminary physical examination including eye refraction but rejected on entrance physical examination shows that all refracted either ¼ diopter of hyperopia or PLANO at time of their preliminary examination. Five enlisted candidates for the Academy were rejected physically upon their entrance physical examiation because of some degree of myopia under cycloplegic. These men had all been given preliminary physical examinations including eye refraction by competent naval medical officers during the summer of 1938. The records of two of these men given their preliminary examination at the Academy show that one refracted PLANO in each eye and the other refracted PLANO in one eye and plus ¼ diopter cylinder axis 90° in the other eye. The records of the preliminary examinations of the other three enlisted candidates were not forwarded to the Academy. Sixteen other candidates from civil life whose records of preliminary physical examinations including refraction were forwarded to the Academy from various naval hospitals refracted PLANO or ¼ diopter of hypermetropia in one or both eyes in 1938. Nineteen others were passed on preliminary refractions by naval medical officers but reports of refractive findings were not sent to the Academy. These were undoubtedly also border-line cases. At time of their physical examination for admission in June and July 1939, all of these candidates were found to have ¼ diopter or more of myopia in one or both eyes and were therefore rejected.

It is therefore evident that a reserve of preferably 1 diopter and at least ½ diopter of hypermetropia is necessary at time of preliminary refraction to be reasonably sure that the candidate will pass his entrance physical examination the next summer. A hyperopic reserve of not less than 1 diopter at time of admission to the Naval Academy is necessary if the candidate is to be reasonably sure of passing the physical examination for a commission in the line of the Navy at graduation 4 years later.

In view of the above, it has been recommended to the Bureau of Medicine and Surgery that no enlisted man be accepted as a candidate for the Naval Academy if he refracts PLANO in either eye at time of his preliminary physical examination. While this is a low requirement, it is desired to give the applicant every consideration. It has also been recommended that all naval hospitals and other naval activities where candidates are refracted as part of a preliminary physical examination for the Academy be instructed to warn any candidate found to refract PLANO or ¼ diopter of hypermetropia in either eye under cycloplegic that he is a border-line case and might develop a low degree of myopia by the following summer with consequent rejection on entrance physical examination.

Occasionally, the writers have been told by candidates that they had been informed by civilian ophthalmologists that they had a very small amount of myopia at time of a preliminary refraction, but that it was not serious or of no consequence and that, with proper care of the eyes or with proper treatment, their eyes should be all right when they took their physical examination for admission to the Naval Academy. Two outstanding examples of such cases were seen during the summer of 1939.

Both of these candidates were former midshipmen who had to leave the naval service in June 1939 because of defective vision and myopia (vision less than 20/20 with 1/2 diopter of myopia) in one or both eyes, found at annual physical examination in March of that year.

They stated that they were assured by civilian eye specialists that with proper treatment, muscle training, etc., their eyes should return to normal and that they should be able to pass the physical examination for admission to the Academy. They therefore obtained reappointments to the Academy and spent money for eye treatments only to be again rejected at entrance physical examination in August 1939 because of myopia under cycloplegic. One of these cases showed no improvement under treatment while the other actually had ¼ diopter more myopia than before commencing treatment.

While these civilian specialists were undoubtedly sincere in their advice, our experience shows that any real improvement in refractive errors in such cases is so extremely rare that the patient should be warned of this and not given false hopes.

PRESENT VISUAL STANDARDS

As previously stated, prior to 1939, the cases of all midshipmen found on annual physical examination to have defective vision were reported to the Navy Department for final decision as to their disposition. The departmental policy in regard to such cases had so varied from year to year in the past that all concerned were annually in a serious state of uncertainty as to what final decision would be made for the disposition of the various degrees of visual deficiency found physically disqualified at the Naval Academy. For years, the visual standard for midshipmen had been only a guide for the Academic authorities and did not necessarily govern final disposition of the cases involved. This situation was especially serious for the midshipmen concerned because they were left in a state of delightful uncertainty. Many members of the graduating class did not know until late spring whether to order their uniforms as officers or look for a job in civil life. Many underclassmen did not know whether or not to make arrangements to enter another college.

In 1937–38 the Navy Department followed a definite policy in this matter for 2 successive years. In general, members of the first or graduating class having vision of 11/20 to 19/20 inclusive in one or both eyes but whose refraction indicated that improvement in vision might be expected were given revocable commissions as ensign subject to physical examination at the end of 2 years to finally determine their fitness for commission in the line or staff corps. Members of the second, third, and fourth classes having vision of 15/20 to 19/20 inclusive in one or both eyes were retained at the Academy for another year to demonstrate whether their vision would improve or deteriorate and to determine definitely their fitness for further retention. In these classes, midshipmen having less than 15/20 vision in either eye were dropped.

In view of the above, in January 1939 the Superintendent of the Naval Academy recommended to the Navy Department that if, as appeared to be the case, a definite departmental policy had been established to give all graduates having vision of 18/20 or 19/20 in either eye, caused by myopia, revocable commissions in the line and those having vision of 15/20 to 17/20 inclusive revocable commissions in the staff corps, subject to reexamination at the end of 2 years to finally determine their status, such policy be officially announced and instructions issued the Academy to that effect. This would relieve both the Department and the Academy of considerable embarrassment and paper work while the individual midshipmen concerned would be relieved of a great deal of worry. This recommendation was approved by the Department and new instructions were issued incorporating the present visual standards for all classes at the

Academy. These standards, now in effect, are required to be met by midshipmen for retention in the Academy and for commission in the line or staff corps of the Navy. They are as follows:

FIRST CLASS (Graduating class)

(*a*) 18/20–20/20 (without myopia.) To be recommended for full-line commission.

(*b*) 18/20–20/20 (with myopia). To be recommended for a revocable commission for a period of 2 years, to be followed by a physical examination at the end of that time to determine their fitness for a regular commission in the line or staff of the Navy.

(*c*) 15/20–17/20. To be recommended for a revocable commission for a period of 2 years, to be followed by a physical examination at the end of that time to determine their fitness for a regular commission in a staff corps of the Navy.

Those members of the graduating class of the Academy who do not meet the requirements of (*a*), (*b*), or (*c*) above, will be brought before a board of medical survey with a view to their discharge from the naval service.

SECOND CLASS

Midshipmen qualifying in accordance with (*a*) and (*b*) above will be recommended for retention at the Academy. For those cases coming within classification (*c*) above, 15/20–17/20, special reports will be made to the Bureau of Medicine and Surgery for opinion prior to final action.

Those midshipmen found to have less than 15/20 should be brought before a board of medical survey with a view to their discharge from the naval service.

THIRD CLASS

The visual standard for each member of the third class at the Academy shall be 20/20 each eye in the presence of myopia or myopic astigmatism or 18/20 each eye without myopia or myopic astigmatism.

Those who fail to meet these standards shall be brought before a board of medical survey with a view to their discharge from the naval service.

FOURTH CLASS

The visual standards for each member of the fourth class at the Academy shall be 20/20 each eye in the presence of myopia or myopic astigmatism, or 18/20 each eye without myopia or myopic astigmatism.

Those who fail to meet these standards shall be brought before a board of medical survey with a view to their discharge from the naval service.

Summarizing, these new visual standards mean that members of the graduating class having vision of 15/20 to 19/20, inclusive, in one or both eyes, are passed by the medical examining board and immediately informed of their status instead of being rejected by the board, reported to the Navy Department, and having to wait 2 months or more before finally being informed of their status. Those having vision of 18/20 or 19/20 without myopia are recommended for full-line commission and have no more worries. Those having vision of

18/20 or 19/20 in one or both eyes and caused by myopia or myopic astigmatism are recommended for a revocable commission in the line or staff corps. At the end of 2 years after graduation they are given a physical reexamination. If at that time they have 20/20 vision in each eye, they are regularly commissioned in the line. If, however, they have 15/20 to 19/20 vision in one or both eyes, they are given a regular commission in a staff corps. Those found by the medical examining board to have 15/20 to 17/20 vision in one or both eyes, whether due to myopia or hypermetropia, are passed by the board and recommended for revocable commission in a staff corps. At the end of 2 years they are given a physical examination and, if they then have vision of 15/20 or better in each eye, are regularly commissioned in a staff corps. If they have less than 15/20 vision, they are required to leave the Regular Navy. Even though cases recommended at graduation for a revocable commission in the staff should read 20/20 with each eye at time of reexamination 2 years later, they are still not eligible for a commission in the line.

With the second class, those having vision of 18/20 or 19/20 in one or both eyes, with or without myopia, are automatically passed by the medical examining board and retained at the Academy. Those cases having 15/20 to 17/20 vision inclusive in one or both eyes are reported to the Bureau of Medicine and Surgery with the refractive findings in each case. These cases are reviewed in the Bureau, and those whose refractive findings indicate that they will be eligible for revocable commissions in a staff corps upon graduation are retained at the Academy for final determination of their status the following year. Those having less than 15/20 vision in either eye are dropped.

All cases in the third and fourth classes having vision of less than 20/20 in either eye if due to myopia or myopic astigmatism, or less than 18/20 in either eye without myopia, are dropped.

All cases having to leave the Naval Academy because of defective vision are allowed by the Navy Department to complete their current academic year. This enables them to graduate or to receive credits for a full year of academic work, as the case may be.

In view of the present visual standards for commission in the line and staff of the Navy, which have been decided by competent authorities to be necessary, these new visual requirements at the Naval Academy certainly appear to be entirely fair to both the Government and the midshipmen. As previously described, experience at the Academy over a period of years has amply demonstrated the futility of continuing cases of defective vision in the hope that they may eventually improve to such an extent as to be satisfactory for a commission. The Government has the legal right and should

establish adequate visual standards for the line and staff corps of the Navy.

It is only fair to the Government that it refuse to waste time and money on members of the two lower classes at the Academy who fail to meet the visual requirements during their first or second year at the Academy. It is only fair that such cases be dropped and others who meet the visual requirements should be appointed in their stead. It is really advantageous to the individual midshipman concerned to require him to promptly leave the Navy and thereby permit him to make an earlier start in some other profession.

As regards the two senior classes, the Government has already invested 50 or 75 percent of the total cost in time and money required to educate them as naval officers and it should endeavor to obtain a profitable return from this group. Graduates of the Academy with vision less than that required for the line but meeting staff requirements are excellent material for staff officers and it is for this reason that the Government commissions such midshipmen in a staff corps for which they may be qualified and thereby obtains an adequate return for its investment.

ESTIMATED FUTURE INCIDENCE OF VISUAL DEFICIENCY

While, as a result of the new visual standard for admission to the Naval Academy, the average annual incidence of visual deficiency in the classes of 1941–42 has been only 2.86 percent to date as compared with an average annual incidence of 8.18 percent for the classes of 1934–40 inclusive during their first 2 years, a low incidence of visual deficiency will necessarily continue to develop at the Academy. This is because of the average age of midshipmen and the refractive findings in their eyes at time of admission. The average age of midshipmen when admitted to the Academy is but slightly over 18 years and their eye development is therefore not yet completed.

Our experience is that those refracting less than ½ diopter of hypermetropia upon admission will almost certainly develop deficient vision while midshipmen and that those refracting less than 1 diopter of hypermetropia at time of admission are not good visual risks insofar as a commission in the line is concerned. Our advance estimate, based on average findings for college students generally and corrected for our selected material, anticipated an incidence of possibly 5 percent visual deficiency developing in each of these classes. The fact that, to date, only 2.86 percent has developed in the two classes admitted under the new standard is most gratifying. Nevertheless, until more statistics are available, an annual incidence of 3

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to 5 percent visual deficiency in each class must be considered normal expectancy.

In an endeavor to still further reduce the incidence of visual deficiency developing at the Naval Academy, recommendation has been made to replace the table lamps now used for study purposes in midshipmen's rooms by the recently developed fluorescent or daylight type of table lamp. While the present table lamps used by midshipmen for study were installed upon the approved recommendation of a board of illuminating engineers and were a very decided improvement over the lamps previously used, lights and lamps continue to improve from time to time as do other mechanical devices. Eighteen months' study of the newest illuminating fixtures plus 3 months' actual trial of new type table lamps have shown that the fluorescent type of lamp is apparently the best yet developed for study purposes. It provides a color of light that more nearly approaches the daylight ideal than any artificial light source previously developed, and the quality of illumination on the working surface is definitely improved.

While, with the present visually selected midshipmen, it would of course be more difficult to obtain appreciable results from improved quality of illumination than would be the case with a nonselected group, it is generally acknowledged that the quality of illumination is a prime factor in the conservation of vision. Any midshipman saved from developing defective vision is a distinct gain for both the Government and the individual concerned. The fact that 22 midshipmen from the visually selected group were forced to leave the naval service at the end of the last academic year because of deficient vision shows that there yet remains a definite field for betterment by improvement in the quality of illumination.

Endeavor was made to determine what decrease in the incidence of visual deficiency at the Naval Academy followed installation of the present table study lamps used by midshipmen. This was very difficult to evaluate statistically, however, because the new visual standard for admission was put into effect the summer of 1937 and because of other variable factors possibly affecting vision in previous classes. Figures for the class of 1940, however, appear to show that the vision of that class was definitely improved by installation of the present table lamps, completed in November 1936. This class entered the Academy during the summer of 1936 under the old visual standard for admission. It had a minimal amount of the old lighting, approximately 3 months. While the incidence of visual deficiency found in this class at its first annual physical examination in 1937 was approximately the same as for previous classes, the increase in visual deficiency noted at its annual physical examinations in 1938 and 1939 was slightly less than half that noted for the class of 1939 during its second and

third years at the Academy. As these two classes were admitted to the Academy under the same visual standard, and their life in the Academy was identical except for the improved quality of illumination provided the class of 1940, this would seem to indicate that improved quality of illumination was a decided factor in decreasing the incidence of visual deficiency in that class. It is therefore reasonable to expect that the improved quality of illumination furnished by the fluorescent type of lamp would still further decrease the incidence of visual deficiency at the Academy.

CONCLUSIONS

It is considered that the Naval Academy has definitely proven the necessity of midshipmen having a reserve of approximately 1 diopter of hypermetropia at time of admission to the Academy and of their meeting the present requirements for a commission if the present visual standard of the line of the Navy is to be maintained. The fact that this standard is not unduly high is attested by the facts that the British Navy requires all navigation and watch officers to have 20/20 vision and that figures above given of the results of recent annual physical examinations given members of the classes of 1931 and 1932 on duty at the Academy show that at least 8 members of each class, 1.8 percent of 1931 and 1.9 percent of 1932, now have vision of 10/20 or less in each eye. Furthermore, as previously stated, these all first developed defective vision (less than 20/20) while midshipmen. Figures are not available to determine the total number of cases with corresponding vision which may now exist in these two classes.

It is desired to invite the special attention of both naval and civilian ophthalmologists to the importance of this subject from the viewpoint of both the Government and the patient in addition to its interest ophthalmologically.

For the past 2 years, the Naval Academy authorities have been seriously concerned by the fact that a number of boys contemplating a naval career go to a local eye specialist, military or civilian, for preliminary eye examination, are told that their eyes are normal, spend time and money preparing themselves scholastically for the Academy, and are then rejected at their physical examination for admission to the Academy because of myopia under cycloplegic. This is not really the fault of the specialist concerned because the vast majority of ophthalmologists do not understand the situation at the Academy insofar as vision is concerned and are sincere in telling a boy having 20/20 vision and refracting PLANO or ¼ diopter of hypermetropia or even myopia that his eyes are normal. For the great majority of pursuits, certainly in civil life, such eyes are essentially normal. Naval visual requirements however are definitely different.

Attention is especially invited to the fact that the visual requirements for admission to the Naval Academy are dual. The candidate must not only read 20/20 each eye by the Snellen test, but, in addition, he must show **no evidence** of myopia under cycloplegic. This includes myopic astigmatism, simple or compound, and mixed astigmatism. In addition, beginning in 1940, the candidate may not have more than 2 diopters of hypermetropia under cycloplegic. This includes simple and compound hyperopic astigmatism.

In view of the facts above described showing the necessity of an adequate hyperopic reserve of approximately 1 diopter in candidates for the Naval Academy, ophthalmologists who may give preliminary eye refractions to candidates for the Academy are urged: (*a*) To use 4 percent homatropine as a cycloplegic, (*b*) to warn any candidate having less than ½ diopter of hypermetropia in each eye under cycloplegic that he is a border-line case who may develop a low degree of myopia by the time he takes his physical examination for admission to the Academy and therefore be rejected, and (*c*) to warn candidates having less than 1 diopter of hypermetropia on preliminary refraction that, while they may pass the entrance physical examination for the Academy, they stand no better than an even chance of meeting the visual requirements for a commission in the line of the Navy at graduation.

NOTES AND COMMENTS

ARTICLES OF SPECIAL MERIT, 1939

The Surgeon General takes this opportunity to express to all contributors his satisfaction with the excellence of their articles and his appreciation of their support of the Bureau's publication.

It has become an established practice for the Surgeon General to present letters of appreciation to authors who have contributed articles of outstanding merit to the *Naval Medical Bulletin.* For the calendar year 1939 the following authors have received letters of appreciation:

Lieutenant Commander Howard K. Gray (MC) U. S. N. R. and Lester R. Chauncey, M. D., Surgical Shock. January 1939.

Lieutenant Robert A. Bell (MC) U. S. N. Sulfanilamide and Chemotheraphy. January 1939.

Assistant Surgeon General D. G. Sutton, U. S. N. The Utilization of Psychiatry in the Armed Forces. April 1939.

Lieutenant J. J. Sapero (MC) U. S. N. and C. M. Johnson, Sc.D., protozoologist. Endameba Histolytica and Other Intestinal Parasites. April 1939.

K. F. Meyer, Ph. D., M. D. Univ. of California. Why Epidemics. April 1939.

Lieutenant Raymond A. Lowry (DC) U. S. N. Loss of Intermaxillary Distance. July 1939.

Commander John E. Porter (MC) U. S. N. and Lieutenant Paul Peterson (MC) U. S. N. Obstetrical Care of Dependents of Naval Personnel. July 1939.

Lieutenant Commander John H. Korb (MC) U. S. N. Perception Time and Nightblindness. July 1939.

Lieutenant A. R. Behnke (MC) U. S. N. and Lieutenant T. L. Willmon (MC) U. S. N. U. S. S. *Squalus*, Medical Aspects of the Rescue and Salvage Operations, and the Use of Oxygen in Deep Sea Diving. October 1939.

Walter M. Boothby, M. D., W. Randolph Lovelace, II, M. D., and Lieutenant Hubert H. Carroll (MC) U. S. N. Oxygen. October 1939.

Commander F. R. Hook (MC) U. S. N. Fractures of the Carpus. October 1939.

THE NAVY'S CONTRIBUTIONS TO MEDICINE

The Surgeon General desires to have available a comprehensive record of what the Navy has contributed to any and all branches of medicine, including military medicine and medical administration.

It is therefore requested that each officer send to the Bureau a list of all such contributions of which he has knowledge, together with dates and references to publications or reports whenever possible.

It is intended that the data shall be preserved in an historical file as source material for addresses, papers, biographies, etc. Hence, it is essential that the bibliography be complete and that all references be authenticated.

THE PUBLIC INTEREST IN HEALTH

The American Museum of Health reports an unprecedented popularity for its medical and public health exhibit at the New York World's Fair. During the 1939 season, 7.5 million visitors viewed this scientific exhibit. This number exceeds by two million the previous record of the Hygiene Museum of Dresden, made in 1911. The promotion of education in matters of public health is a national service of inestimable value and the American Museum of Health is to be congratulated for its contribution to this worthy cause. The fact that this exhibit was in competition with displays dramatizing industry, makes this success doubly significant. This exhibit will be enlarged and improved for the 1940 season and should continue this valuable educational program which has enjoyed such enthusiastic support.

NEW MEMBERS, AMERICAN COLLEGE OF SURGEONS

The following-named naval medical officers were nominated by the Bureau of Medicine and Surgery for fellowship in the American College of Surgeons and the Bureau has been informed that they were elected at the 1939 convocation of the College.

Lieutenant Commander J. P. Brady, (MC), U. S. Navy.
Lieutenant Commander James J. O'Connor, (MC), U. S. Navy.
Lieutenant Thomas G. Hays, (MC), U. S. Navy.
Lieutenant Jerry T. Miser, (MC), U. S. Navy.
Lieutenant William P. Stephens, (MC), U. S. Navy.
Lieutenant Clifford F. Storey, (MC), U. S. Navy.
Lieutenant Harold G. Young, (MC), U. S. Navy.

PHARMACIST'S MATES AWARDED NAVY CROSS

Benjamin T. Morris, pharmacist's mate, first class, attached to the U. S. S. *Falcon*, and Francis L. Westbrook, pharmacist's mate, first class, attached to the Experimental Diving Unit, Washington Navy Yard, Washington, D. C., were awarded the Navy Cross and advanced one grade in rating without examination, with the following citation:

> For extraordinary heroism in the line of his profession as a diver during the rescue and salvage operations following the sinking of the U. S. S. *Squalus* on 23 May 1939. His courage and devotion to duty in making repeated dangerous dives during the most difficult diving conditions characterizes conduct far above and beyond the call of duty.

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)

ENDOCRINOLOGY IN MODERN PRACTICE, by *William Wolf, M. D., M. S., Ph. D. Endocrinologist to the French Hospital; Attending Endocrinologist, Misericordia Hospital, New York City; Consulting Endocrinologist, New York University Dental School.* Second edition, completely revised, 1077 pages, illustrated. W. B. Saunders Co., Philadelphia and London, 1939. Price $10.00

Three years ago the first edition of this book appeared. At that time the author set for himself the task of presenting endocrinology in its clinical aspects free from technical and theoretical discussions. That he admirably succeeded is indicated by the popular reception that it received. But with a subject receiving as much study as this one is, both clinically and experimentally, it was obvious that if a text is to fulfill the purpose for which it was intended, frequent revision becomes a necessity. It was this need that lead to the complete revision of the present edition. Many of the chapters have been rearranged, new sections have been added where needed and others deleted in keeping with our present concepts. It is impossible to outline here all the changes, but of the new material, mention should be made of the sections on protamine zinc insulin, hypoglycemic states, the sympathetic and parasympathetic nervous systems in relation to hormonal and nonhormonal substances and the relationship of the vitamines in diseases of the ductless glands.

As in the first edition this one is well bound and well printed. It is excellently written, well illustrated and the subject matter concisely and logically presented with each chapter followed by a tabulated summary of the entire chapter contents. With the increasing realization that many nonendocrine disorders have an endocrinological aspect the author devotes a good portion of his book to their discussion following that of the endocrinopathies proper. In the section on diagnosis the method of history taking is outlined as well as the

method of examination from both the clinical and laboratory standpoints with a chapter on the interpretation of laboratory findings. The table on anthropometry is most useful. For ready reference two symptom indexes are presented, one alphabetical, the other regional, which suggest the possible cause of the disorder from the presenting symptoms. Also included is a chapter on laboratory procedures and a chapter listing the manufacturer, potency, dosage, etc., of the available commercial endocrine products.

Although of particular interest and value to the endocrinologist, this book is sufficiently wide in scope and provocative of thought that it can be unreservedly recommended to all practitioners of medicine. It will be found of special value to those in general practice as well as those who are called upon to examine and treat children or women.

THE PHYSIOLOGICAL BASIS OF MEDICAL PRACTICE, A University of Toronto Text in Applied Physiology by *Charles Herbert Best, M. A., M. D., D. Sc. (Lond.), F. R. S., F. R. C. P. (Canada), Professor and Head of Department of Physiology, Associate Director of the Connaught Laboratories, Research Associate in the Banting-Best Department of Medical Research, University of Toronto,* and *Normal Burke Taylor, M. D., F. R. S. (Canada), F. R. C. S. (Edin.), F. R. C. P. (Canada), M. R. C. S. (End.), L. R. C. P. (Lond.), Professor of Physiology, University of Toronto.* Second edition, 1,872 pages, The Williams & Wilkins Company, Baltimore. 1939, Price $10.00

This excellent work and others of its kind serve to emphasize a fundamental change in the approach of modern medicine to the study of disease. In the past century the study of disease was approached largely through pathological anatomy. Thus, we thought of disease as the picture was presented to us at the autopsy, the end result of a process or perhaps a number of processes. Today we are more and more directing our attention to the study of pathological physiology as we strive to solve clinical problems. In this work the authors have been wonderfully successful in enabling the student and the graduate clinician to bring physiological principles into intimate relation with problems of disease. The authors have surely succeeded in writing a book that "links the laboratory and the bedside." This edition of Best and Taylor continues to bring credit to medicine in Canada and to the University of Toronto.

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.* Third edition, 1939. Thoroughly revised. Illustrated with 218 engravings. 1,144 pages. Lea and Febiger, Philadelphia. Price $9.50

This third edition of an immensely popular work will be met with the same warm welcome that the previous editions have justified.

Here is a book that makes the practice of medicine a pleasure by helping the doctor understand the how and why of changes in physiology incident to disease processes. The section on the heart and circulation continues to be a most excellent one, and that on the alimentary tract deserves high praise for its simple and direct presentation of many phases of gastro-intestinal activity. Particularly may be mentioned motor and secretory phenomena. The section on vitamins and that on endocrines will be recognized as presenting the greatest changes in this edition for knowledge in these fields has been advancing most rapidly. The successful modern clinician has come to realize that he must understand the functional disturbances of patients rather than to catalog symptoms and make diagnoses on an empirical basis. He who follows disturbed physiology in its fluctuations is very apt to know what to do and when to do it.

PHYSIOLOGY OF EXERCISE, by *McCurdy, J. H.* and *Larson, L. A.* Third edition, revised, 349 pages, Lea & Febiger, Philadelphia, Pa. 1939, Price $3.75

An excellent reference on the subjects of general effects of exercise upon bodily functions, effects of special types of exercise upon bodily functions, and methods of indicating efficiency of bodily functions. It includes a comprehensive review and criticism of the various methods of determining efficiency of bodily functions, with comparative tables and scores. The McCurdy-Larson organic efficiency test, which is evaluated with relation to age and weight, is presented for comparative analysis. The exhaustive bibliography is a valuable inclusion.

PHYSICAL DIAGNOSIS, by *Cabot, Richard C.* and *Adams, F. Dennett.* Twelfth edition, 793 pages, 391 illus. William Wood & Co., Baltimore, Md., 1938. Price $5.00

This is the twelfth edition by this author. All of them have been interesting and instructive. This edition is even more valuable than previous ones as it really represents the opinion of most of the staff of the Massachusetts General Hospital, as well as that of the author. It is as near a complete treatise on physical diagnoses as can be incorporated in a single volume.

It is as valuable to the postgraduate student for a reference book as it is to the undergraduate who is delving for the first time into the intricacies of physical diagnoses. The print and style in which it is written fail to tire even after long periods of reading.

MARTINI'S PRINCIPLES AND PRACTICE OF PHYSICAL DIAGNOSIS, Edited by *Robert F. Loeb, M. D.*, from the authorized translation by *George J. Farber, M. D.* Second edition, 203 pages, J. B. Lippincott Co., 1938, Price $2.00.

This volume is condensed and attempts to cover the entire field of physical diagnoses. To accomplish this, only the most important

physical signs are listed with very little discussion. This would tend to make it very difficult for the undergraduate student to get a very clear idea of the disease he is studying and too elementary for the postgraduate student.

The section dealing with the circulatory system is very good and would serve very well for a quick reference as to the more important signs of the disease being studied.

CLINICAL DIAGNOSIS BY LABORATORY METHODS, A Working Manual of Clinical Pathology, by *James Campbell Todd, Ph. B., M. D., Late Professor of clinical pathology, University of Colorado, School of Medicine,* and *Arthur Hawley Sanford, A. M., M. D., Professor of clinical pathology, University of Minnesota (The Mayo Foundation); Head of Division on Clinical Laboratories, Mayo Clinic.* Ninth edition, thoroughly revised, with 368 illustrations, 29 in color, 841 pages. W. B. Saunders Co., Philadelphia and London, 1939. Price $6.00

This ninth edition is again an indispensable book for every active laboratory. It is not necessary to discuss the proven and well-known value of this work. It covers the field completely. It is hard to imagine a large or busy laboratory being able to keep house without it. In this edition the authors have brought this work up to date by dropping some obsolete methods and by adding other methods which though new, are well proven. These new methods represent worth-while advances in clinical pathology achieved in the last 4 years. Bodansky's methods for the determination of inorganic serum phosphate and phosphatase is given. Power and Wakefield's method for blood serum inorganic sulfates is also new. This method may prove to be a more accurate and delicate method of detecting minor degrees of early renal failure than has yet been offered. Quick's hippuric acid test for liver function is given, as well as the quantitative determination of sulfanilamide in blood and urine. In line with the United States Public Health campaign on venereal disease and the even wider use of serodiagnostic methods for syphilis, the Kline, Kahn, Hinton, Eagle, and Kolmer tests are given.

SYNOPSIS OF CLINICAL LABORATORY METHODS, by *W. E. Bray, B. A., M. D., Professor of clinical pathology, University of Virginia Hospital.* Fifty-one text illustrations, 17 color plates, 408 pages, second edition. The C. V. Mosby Co., St. Louis, 1938. Price, $4.50

The value of laboratory aids in clinical diagnosis need not be stressed. The work of any clinic or hospital can be evaluated very largely by an investigation of the laboratory. That is, clinical investigation cannot go far without the aid of clinical pathology. The pathologist and the clinician must work hand in hand. Bray's Synopsis of Clinical Laboratory Methods gives the essentials of

clinical pathology in 408 closely packed pages. The important new tests developed in the last 2 years are here. Descriptions are brief, but adequate. Unimportant details are necessarily eliminated. There are a few good color pictures and a number in black and white. The binding is waterproof. The size is that of a pocket manual. It will be found a valuable and compact synopsis of up-to-date laboratory methods.

EXPERIMENTAL PHARMACOLOGY AND MATERIA MEDICA, by *Dennis E. Jackson, Ph. D., M. D., F. I. C. A., Cincinnati, Ohio. Professor of pharmacology, materia medica, and therapeutics in the University of Cincinnati College of Medicine;* Formerly *Associate Professor of pharmacology, Washington University Medical School, St. Louis.* Second edition, 906 pages, with 892 illustrations including 55 color plates. The C. V. Mosby Co., St. Louis. 1939. Price $10.00

The book is divided into three parts. The first and largest section of 641 pages is made up of 217 experiments in which the pharmacology of important drugs is demonstrated in the laboratory upon man and experimental animals. For example, all important anesthetics are used in carefully detailed experiments in which their action on the cental nervous system is shown. Drugs which are important because of their action on the sympathetic nervous system are investigated in another series of experiments. Drugs acting principally upon the heart and circulatory system are studied in still another group of experiments. In this way pharmacodynamics in materia medica are brought home to the reader and experimenter. The second section is devoted to shop work and photography and the third to materia medica and prescription writing.

Throughout the whole book great care is evident. Nothing has been taken for granted, each step is proved, each action is demonstrated. As the author says, the chief foundation stone of pharmacology is physiology. The fact that the plan of the experiments and discussions have followed the physiological actions throughout will make it valuable to physiologists and even perhaps to clinicians, as well as to pharmacologists. The book is excellently illustrated, a point of great value in leaving a lasting impression on the student. Paper binding and printing are of the best.

CLINICAL PATHOLOGY AND TREATMENT OF THE DENTAL PULP AND PERIODONTAL TISSUES; by *Edgar D. Coolidge, B. S., M. S., D. D. S.; Professor of therapeutics, preventive dentistry, Loyola University, Chicago, Ill.;* formerly *Professor of materia medica, pharmacology and therapeutics of the School of Dentistry, University of Illinois.* Pages 461; illustrations 289. Publishers, Lea and Febiger, Philadelphia, Pa. Price $6.50

The subject matter of this text is a compilation and application of more recent original investigations by the author as well as others. Evidence of workers in related sciences is freely used with a resultant

closer association and understanding between these sciences and their practical clinical significance. Methods of clinical diagnosis and treatment here contained are not new but very well systematized. The question of microscopic sealing of root canals at their apical foramina is not considered, reliance being placed on roentgenographic evidence only. The histo-pathologic picture of favorable periapical tissue response to treatment as presented in the excellent photomicrographs is very convincing. However, correlating data to show under what circumstances these teeth were treated, or statistical information on the actual percentages of successes and failures, are lacking. Investigations of L. Bruket, T. K. Cook, R. F. Somer. M. C. Crawley, and others on the negative and positive teeth are apices of the roentgenographically negative and positive teeth are not mentioned. Thus nothing very illuminating on the indications and contraindications of treatment of pulpless teeth is advanced. Healing in the periapical regions via connective tissue with subsequent secondary cementum and bone formation as well as the inflammatory processes in the pulp and parodontium are beautifully represented by the author's as well as other investigators' photomicrographs. Among these are those showing experimental evidence supporting the evils of over medication, which phase of therapeutics is again stressed. The author is open-minded concerning medicaments used and describes the procedure in the use of those found to be most satisfactory. A brief rational review of some of the more common oral manifestations of systemic diseases and their treatment is found in this text.

The book is easy reading and the subject matter is presented in simple English, which fact, together with material found in it, makes it a good reference book.

The publishers, Lea and Febiger, are, as usual, to be commended on their excellent manner of publishing this text.

DENTAL ROENTGENOLOGY: by *LeRoy M. Ennis, D. D. S., Assistant Professor of roentgenology in the Thomas W. Evans Museum and Dental Institute, School of Dentistry, University of Pennsylvania; Instructor in dental roentgenology in the Graduate School of Medicine, University of Pennsylvania; Lieutenant Commander, U. S. Naval Reserve.* Third edition, revised, octavo, pp. 398; Ill. 789; Lea and Febiger, Philadelphia; 1939. Price, $6.50

The dental profession should welcome the third and revised edition of this comprehensive treatise on dental roentgenology. Forty-seven pages and 96 illustrations have been added to supplement the text. The introduction, as in the second edition, is concerned with the historical background of the roentgen ray, and also with the increasing importance of roentgenology in the field of progressive science. The first two chapters are devoted to a thorough discussion of the roent-

gen-ray tube, and of roentgen-ray dermatosis. Many new illustrations have been included in these chapters, and the newer, improved equipment is described. The third chapter is a comprehensive study of intra-oral and extra-oral technic, and should have particular appeal to those in the profession who do their own roentgenographic work. The technic is simplified by the addition of many excellent illustrations. In chapter four, the author discusses the roentgenographic examination of children. This is a phase of roentgenographic study that has been somewhat neglected in earlier texts on dental roentgenology, and Ennis is to be commended for the inclusion of this valuable material in his book. The following three chapters deal with methods of localization, dental roentgen-ray films, and routine examination of the oral cavity, respectively. The author avoids the use of highly scientific terminology in these chapters, and confines his remarks to the practical application of fundamental facts. Interesting features of chapter eight on normal anatomical landmarks of the teeth and jaws as seen in the roentgenogram are the study of the maxillary sinus, and the material on the blood and nerve supply of the jaws. The following chapter, the largest in the book, is devoted to dental pathology in relation to roentgenology. Here the author emphasizes the importance of correct interpretation of the roentgenogram, and accurate knowledge of pathology in diagnosis. All pathological manifestations of importance to the dental practitioner are reviewed, and abundantly illustrated. In conclusion, a short chapter on the localization of root canals and fistulous tracts by the use of lipiodol, is presented.

In the opinion of the reviewer, this publication covers a wide range of subject matter, is ably presented, accurately illustrated, and devoid of superfluous material which might be confusing. It should be a valuable source of reference and instruction to the practicing dentist.

The publishers, Lea and Febiger, are to be complimented for the excellent reproduction of roentgenograms, drawings, and other illustrations.

A Textbook of Practical Nursing, by *Kathryn Osmond Brownell, R. N., B. S., Director of the Young Women's Christian Association School of Practical Nursing, Central Branch, Brooklyn Young Women's Christian Association, Brooklyn, New York.* W. B. Saunders Co., Publishers, Philadelphia, Pa. Price $3.00

This book is well written in a clear and concise style, easily understood, and presents modern methods of general nursing procedure, and instructs the practical nurse in the fundamentals of hygiene, household management, cooking and foods, and simple nursing skills.

This book should be of value to both the practical nurse and the layman.

COMMUNICABLE DISEASES FOR NURSES, by *A. G. Bower, A. B., M. S., M. D., Head of Department of Communicable Diseases, and Clinical Professor of medicine, University of Southern California; Senior Consultant to the Department of Communicable Diseases, Los Angeles County Hospital,* and *E. B. Pilant, R. N., Director of nursing, Los Angeles County Hospital.* Fourth edition. W. B. Saunders Co., Philadelphia, Pa. Price $3.00

This is one of the best textbooks on the subject of communicable diseases for nurses which the reviewer has had an opportunity to read.

The book starts out with the definitions of important terms, immunity, and infection, and the value of the application of the principles of medical aseptic technique in the prevention of the spread of disease to and from the patient in the hospital, the institution, and the home.

Chapter five on public health control of communicable disease is well written and of the greatest importance to the nurse in the study and understanding of specific diseases in relation to modern clinical medicine.

Next, the authors present very clearly each disease, giving definition, etiology, pathology, symptoms, diagnosis, prognosis, treatment, nursing care, and management in a most interesting and instructive manner.

The book may serve as a combination text and reference not only for the student nurse but for the graduate as well. It contains many excellent illustrations. The glossary and index are thorough and adequate.

A MIRROR FOR SURGEONS. Selected Readings in Surgery. By *Sir D'Arcy Power, K. B. E., F. R. C. S., Consulting Surgeon to and Archivist at St. Bartholomew's Hospital, London.* Boston, Little, Brown and Co. 1939. Pp. 230. Price $2.00

For more than half a century the distinguished compiler's name has been known throughout the English-speaking world. Both his surgical and his biographical works have been of the highest order.

In the present volume he has chosen 22 of the great surgeons who have contributed to the advancement of the art and science of their specialty. There is a short sketch of each, then selections from his writings. Some of the names are little known, but there can be no doubt that the selections have been wisely made. The first name is that of John Arderne (1307–1380), the last is James Marion Sims (1813–1883). Other Americans are W. S. Halstead and Henry J. Bigelow. One Frenchman, Ambroise Paré, is included.

Here is a book for the surgeon with an historical trend. If you are planning a long cruise, or foreign shore duty, this is a grand book to take along.

THE SURGERY OF INJURY AND PLASTIC REPAIR. By *Samuel Fomon, Ph. D., M. D.* Williams and Wilkins Co., Baltimore, 1939. Pp. 1390. Ill. 925. $15.00

Here is a monumental work in which no stone has been left unturned that might conceivably be tied in with plastic surgery. The first 500 pages are devoted to detailed discussion of the general principles of surgery. The second part deals with regional surgery of the exterior head.

This work is stated to be the culmination of 25 years of experience in lecturing to post-graduate students and army surgeons. It is designed to serve the general practitioner, the surgeon and the specialist.

One cannot resist a feeling of awe and admiration for the man capable of the enormous industry required for the authorship of such a book. A single chapter has over 300 references. The author is deserving of congratulations for his intelligent selection and presentation of material.

NITROUS OXIDE-OXYGEN ANESTHESIA. McKesson-Clement Viewpoint and Technique. By *F. W. Clement, M. D., Director of anesthesia at Flower Hospital, Toledo, Ohio.* Lea and Febiger, Philadelphia. 1939. Pp. 268. Ill. 70. $4.00.

The author generously gives credit to the late Dr. E. I. McKesson for originating the technic of primary and secondary saturation, fractional rebreathing, and many other details of nitrous oxide-oxygen anesthesia. Whoever was responsible for developing the use of these agents surely deserves the gratitude of both surgeons and operative patients.

One who is interested in this form of anesthesia will find in Dr. Clement's book a complete, and authoritative discussion of all aspects of its use.

SURGERY OF THE EYE, by *Meyer Wiener, M D., Professor of clinical ophthalmology, Washington University School of Medicine, St. Louis,* and *Bennett G. Alvis, M. D., Assistant Professor of clinical ophthalmology, Washington University School of Medicine, St. Louis.* 396 Illustrations. W. B. Saunders Co., Philadelphia and London, 1939. Price $8.50.

In this book of only 445 pages we have a short surgical treatise on surgery of the eye which is apparently written for the busy practicing ophthalmologist as a handy reference book for surgical technic. It is well but tersely written and the detail of each operation is illustrated clearly and distinctly so that the descriptive text can be easily followed. The first five chapters are occupied with general considerations, preparation of the patient, anesthesia, preoperative preparation and postoperative care. The following thirteen chapters take up in

detail the different operative procedures in paracentesis, cataract, glaucoma, retina and sclera, intra-ocular foreign bodies, cornea, removal of the eye, conjunctiva, lids and socket, ptosis, muscles, and the tear apparatus.

DISEASES OF THE EAR, NOSE, AND THROAT, Principles and Practice of Otorhinolaryngology, by *Frances L. Lederer, B. SC., M.D., F. A. C. S., Professor and Head of the department of laryngology, rhinology, and otology, University of Illinois College of Medicine, Chicago; Chief of otolaryngological service, Research and Educational Hospital.* Second revised edition. Illustrated with 765 half-tone and line engravings on 463 figures and 16 full-page color plates. F. A. Davis Co., Philadelphia, Publishers 1939. Price $10.00.

In this second edition of this work, in less than a year, are correlated, plus the information of the original edition, corrections and additions of the latest developments to this specialty.

This edition is made up of five sections—section I, diseases of the ear; section II, diseases of the nose and sinuses; section III, diseases of the pharynx; section IV, diseases of the larynx, treachea, bronchi, and esophagus; section V, correlated considerations.

Each section is subdivided into chapters taking up specific parts of the designated sections. For example, section V is made up of material such as otolaryngological symptoms common to many diseases, action of cocaine, etc.; facial neuralgias, allergy, enlargements of the neck, disorders of speech, psychiatric aspects, legal medicine, temporal bone fractures, which is not ordinarily included in the average book on otorhinolaryngology, yet is of vital importance in such work.

The volume in general is well written, giving in detail anatomy, pathology, methods of examination, clinical findings, and treatment in such a manner that one can easily obtain the information desired.

This book is an excellent addition to the library of the student of otorhinolaryngology as well as the active practitioner of this specialty.

THE ETIOLOGY OF TRACHOMA, by *Louis A. Julianette, chairman of the trachoma commission, Washington University, St. Louis,* New York, The Commonwealth Fund. London, Humphrey Milford, Oxford University Press. 1938. Price $3.25.

This short volume on the etiology of trachoma is in reality a summary of the work done by the Trachoma Commission at Washington University as well as an excellent review of the etiology of trachoma. Dr. Julianette considers the epidemiology, causation, and infectivity with particular reference to the micro-organisms which are associated with trachoma, the inclusion bodies, and all phases of the laboratory study. This book is of value to ophthalmologists, bacteriologists, and

pathologists in that it coordinates their findings in a small, easily read, and interesting book. It is divided into 11 chapters: (1) trachoma and clinically similar diseases, (2) general considerations of epidemiology, (3) etiology, (4) infectivity, (5) the micro-organisms associated with trachoma, (6) the inclusion body of trachoma, (7) the relation of viruses to trachoma, (8) purification of the infectious agent, (9) cultivability of the infectious agent in tissue culture, (10) properties of the infectious agent, and (11) general discussion.

209001—40——10

THE DIVISION OF PREVENTIVE MEDICINE

Commander C. S. Stephenson, Medical Corps, United States Navy, in charge

FROZEN BREAD

By Lieutenant Martin V. Brown, Medical Corps, United States Navy

Commercial advertising has made the subject of frozen foods popular. The advantages of these frozen products are well known. Few people are aware of frozen bread, nevertheless it has been known for many years. With the habitation of the far north and other cold climates, the early settlers soon discovered that fresh bread was not one of their problems. The long cold winters made it unnecessary to bake bread more than one time during the entire winter. In more recent times various experiments have been conducted in regard to a more practical application of the known fact that freezing prevents the staling of baked products.

When the writer reported aboard the U. S. S. *Nitro*, it was discovered that bread was being stored in a refrigerated meat hold for long periods of time, with the result that fresh bread was available at all times, even on extended cruises. This method of preventing the staling of bread had been in use for several years, and it was decided to investigate. The ship was preparing for a trip to Manila and the plans were to purchase bread in Seattle in sufficient quantity to last for 3 months or until the ship returned to the States. In the construction of the U. S. S. *Nitro*, a very small space was allotted for the bake shop because no plans were made for carrying a considerable number of passengers, which is now the routine. In later years the common practice, on the regular run between the two coasts, was to buy bread only once on each coast. The time required for the round trip was approximately 3 months.

In this connection it is deemed advisable to describe the physical set-up which makes this practice possible in this particular ship. In the original construction of the *Nitro*, there were provided three large cargo spaces, or holds to be used for storing chilled and frozen foods. These spaces are well insulated and desired temperatures are maintained by an efficient refrigerating plant, resulting in more than adequate refrigerated space. Two holds are normally in use. One is kept at the optimum temperature of 2.6° C., for storing fresh vegetables, fruits, eggs, and other products which are advantageously kept

at this temperature, and the other is usually kept at −12° C., where frozen meat and bread are stored. The third hold is kept in reserve.

Insulated and sealed hatches, 3 by 3 feet, at the top of each hold, are the only means of entrance to these refrigerated spaces. The space is entered once a day (in the early morning when the external temperature is lowest) and the meat and bread are removed in less than 15 minutes. A temperature reading is made on an accurate (calibrated) mercury thermometer each time the hold is entered. In addition to this, temperature readings are made every 6 hours on a temperature panel in the brine tank room for comparison. The temperature varies but little and the elevation due to opening the hatch is from −12° C. to −11.5° C. When new supplies of bread and frozen meat are added the temperature elevation is from −12° to −10° C. The variation from day to day is from −11.5° to −14° C. On the whole the temperature is uniform and should be considered to be about −12° C. The bread is allowed to thaw for about 24 hours and is used the day after removal. For the uninitiated, the temperature of the ordinary household electric refrigerator varies between 4° C. and 6° C.

The bread used on board the *Nitro* and in this experiment is wheat or white commercial which is bought on Navy contract and calls for a one-pound sliced loaf of highest quality, individually wrapped in an impervious paper. It must be strictly fresh, packed in cardboard containers which can be handled by one man, and delivered within 8 to 12 hours after baking. The bread is removed from the cartons and stacked somewhat loosely in the hold. From inspection and temperature readings, we have assumed that our bread reaches −12° C. in about 2 to 4 hours after being placed in the freezer.

Our method of freezing is called "sharp freezing" in contradistinction to "quick freezing," which is the method used in the preparation of commercial frozen foods. Katz [1] found that the maximum rate of staling occurred at −2° C. to −3° C. and that the rate of staling decreases as the temperature is raised or lowered. Many experimenters have found that the freezing of bread has no effect on quality, that is, within certain limits. Alsberg [2] reports that a temperature of −6° C. delays the onset of staleness and a temperature of −10° C. to −20° C. prevents it altogether. Cathcart and Luber [3] report that at the present time the most effective and practical method of retarding the staling of bread is sharp freezing and that bread frozen at −22° C. remained good for 20 days. Bread frozen at −35° C. for 20 days was indistinguishable from fresh bread and at −35° it was salable, even after 110 days in the freezer.

[1] Bakers' Weekly, 81: 3–43 (1934).
[2] Wheat Studies Food Research Inst., 12: 244 (1936).
[3] Ind. Eng. Chem., 31: 362, March 1939.

In order to determine the difference in quality between fresh and frozen (freshly thawed) bread, it was necessary to form a table of terms used in describing the staling or staleness of bread. In this connection we borrowed from Cathcart and Luber a table of characteristics which make up bread quality. Each loaf of bread was weighed before being placed in the freezer, immediately upon removal, and after it had thawed for 6 hours. It was found that our measurements were not sufficiently accurate to determine the loss of moisture during the process, so the data were omitted and are not mentioned in our table. Cathcart, in his experiments, found that the type of wrapper (wax or cellophane) had very little effect on the taste or quality of bread. The chief commissary steward on the *Nitro* claims that certain types of wrappers produce an odor and taste to the bread not produced by others. The writer has not been able to verify his claims.

The results of our experience in preservation of bread by freezing are tabulated below showing the effect of duration of storage on the physical characteristics of the bread.

Preservation of bread by refrigeration

Number of sample	Days in freezer	Wrapper	Condition of crust	Break and shred	Texture	Aroma	Taste	Remarks
1	254	Wax	Dry and hard.	Even and tough.	Coarse	Gassy	Rancid	Aroma similar to freezer odors.
2	230	Cellophane	do	do	do	do	do	Do.
3	200	Wax	do	do	do	do	do	Do.
4	181	do	do	do	do	do	do	Do.
5	158	do	do	Very tough.	Very dry.	No odor	No taste	Tough crust.
6	147	do	do	do	Coarse	Pungent; gassy.	Rancid	Do.
7	137	Cellophane	do	Even and tough.	do	Gassy	do	Aroma like above.
8	102	Wax	do	do	do	do	do	Do.
9	95	do	do	do	do	do	do	Do.
10	83	do	do	do	do	do	do	Do.
11	65	do	do	do	do	do	do	Do.
12	42	do	do	do	do	do	do	Do.
13	35	do	Smoother	do	do	do	do	Do.
14	29	do	Smooth	Improved (irregular).	Better	No odor	Not much taste.	Very tough crust.
15	19	do	do	Fresh	Normal.	Fresh bread.	do	Fresh.
16	10	do	do	do	Fresh	do	do	Do.
17	5	do	do	do	do	do	Fresh	Do.

SUMMARY

In examining the bread as it was thawing, we noted that in the center of the loaf, as it was melting or softening, there was a definite line of demarcation and that just exterior to this line the bread was very dry and hard. In other words, the bread dried out so quickly after thawing that there appeared to be a definite thaw line. We

might add that thawing took place while the ship was in the Tropics and the humidity was 74°.

From a glance at the table it is evident that the length of time which bread may be kept at −12° C. is approximately 20 to 30 days. Bread is definitely not good if stored for longer periods at the above temperature.

In fighting ships, where there is always a demand for more space, this method is probably not practical in its present state. There are possibilities, in cargo ships, that this method may be used to advantage on extended cruises.

It is believed that further work is warranted and perhaps solid CO_2 might be advantageously employed to adapt this method to the needs of the naval service.

STATISTICS

MORBIDITY

Summary for the quarter ending Sept. 30, 1939

Average strength	Forces afloat 93,275		Forces ashore 50,864		Entire Navy 144,139	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	8,765	375.88	4,831	379.92	13,596	377.30
Disease only	7,517	322.36	4,198	330.14	11,715	325.10
Injuries and poisonings	1,248	53.52	633	49.78	1,881	52.20
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	84	3.60	31	2.44	115	3.19
(B)	1,145	49.10	992	78.01	2,137	59.30
Venereal diseases	2,725	116.86	615	48.36	3,340	92.69

DEATHS

During the quarter ending Sept. 30, 1939

Cause of death		Navy			Marine Corps		Nurse Corps	Total
Principal	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		10,466	2,268	111,204	1,404	18,358	439	144,139
Diseases								
Abscess, brain	Pneumonia, lobar			1				1
Abscess, brain	Mastoiditis, acute			1				1
Adenocarcinoma, rectum	None			1				1
Appendicitis, acute	Peritonitis, general, acute.	2						2
Coronary heart disease, arteriosclerotic.	None	1		1				2
Embolism, pulmonary	Bursitis, chronic, knee		1					1
Endocarditis, subacute, bacterial.	None					1		1
Glioma, brain	Pneumonia, broncho			1				1
Hypertension, arterial	None			1				1
Hypertensive heart disease.	Arteriosclerosis, general				1			1
Leukemia, acute (lymphatic type).	None		1	1				2
Mastoiditis, acute	Abscess, brain			1				1
Myocarditis, acute	Thrombosis, cerebral and arteriosclerosis, cerebral.			1				1
Myocarditis, chronic	None	1						1
Myocarditis, chronic	Pericarditis, chronic			1				1
Nephritis, acute	None	1						1
Pneumonia, lobar	None			1				1
Poliomyelitis, anterior, acute.	None			1				1
Septicemia	None			1				1
Thrombosis, coronary artery.	None	1		1		1		3
Thrombosis, coronary artery.	Arteriosclerosis, coronary artery.			1				1
Thrombosis, coronary artery.	Embolism, mesenteric artery.	1						1
Tuberculosis, pulmonary, acute general miliary.	None			1				1
Ulcer, stomach, perforated.	None			1				1
Total for diseases		7	2	17	1	2		29

Deaths—Continued

Cause of death		Navy			Marine Corps		Nurse Corps	Total
Principal	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Injuries and poisonings								
Decapitation	None			1				1
Drowning	None			6				6
Fracture, compound, skull.	None			2				2
Fracture, simple, skull	Intracranial injury			2				2
Fracture, simple, vertebra, cervical.	Pneumonia, broncho			1				1
Fracture, simple, vertebra, cervical.	Intracranial injury			1				1
Injuries, multiple, extreme.	None	5	4	10	1			20
Intracranial injury	None			3		1		4
Intraspinal injury	None	1						1
Rupture, traumatic, intestines.	Peritonitis, general, acute.			1				1
Rupture, traumatic, lung	Hemorrhage, internal, lung.			1				1
Wound, gunshot, chest	None	1				2		3
Wound, gunshot, head	None			1		1		2
Wound, gunshot, head	Psychosis, unclassified			1				1
Wounds, multiple	None	1						1
Poisoning, acute, carbon monoxide.	None	1						1
Poisoning, acute, sodium cyanide.	None			1				1
Total for injuries and poisonings		9	4	31	1	4		49
Grand total		16	6	48	2	6		78
Annual death rate per 1,000:								
All causes		6.12	10.58	1.73	5.70	1.31		2.16
Diseases only		2.68	3.53	.61	2.85	.44		.80
Drowning				.22				.17
Poisonings		.38		.04				.06
Other injuries		3.06	7.05	.86	2.85	.87		1.14

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for the quarter ending September 30, 1939

The following statistics were taken from sanitary reports submitted by naval training stations:

July, August, and September 1939	Naval Training Station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period	2,235	1,242	1,210	1,591
Recruits appearing before board of medical survey	23	0	17	(1)
Recruits recommended for discharge from the service	23	0	17	(1)
Recruits discharged by reason of medical survey	19	0	14	(1)
Recruits held over pending further observation	24	0	(1)	(1)
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	0	11	(1)	18

[1] Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess:		Fistula in ano	1
periapical	2	Fracture, simple, fourth and fifth lumbar vertebrae	1
periodontal	1	Goiter, simple	1
Ankylosis, spine	1	Gonococcus infection, urethra	1
Arthritis, chronic	3	Heart disease, congenital, septal defect	1
Asthma	4	Hernia:	
Blepharitis	1	inguinal, direct	1
Bursitis, chronic	1	inguinal, indirect	2
Calculus, ureter	1	Lupus erythematosus	1
Cardiac arrhythmia:		Malocclusion, teeth	1
premature contractions	1	Metatarsalgia	1
ventricular ectopic beats	1	Myopia	5
Caries, teeth	2	Nephritis, chronic	9
Chondritis	1	Neurosyphilis, serological	1
Choroiditis	1	Osteoma, right femur	1
Color blindness	3	Otitis, media, chronic	4
Constitutional psychopathic state:		Rupture, muscle, traumatic	1
emotional instability	1	Sinusitis, ethmoidal	1
inadequate personality	1	Sprain, joint	2
Deafness:		Somnambulism	1
bilateral	1	Strabismus	2
unilateral	3	Synovitis:	
Defective physical development	2	chronic	1
Deformity:		traumatic	1
acquired	6	Syphilis	1
congenital	6	Tuberculosis, pulmonary, chronic active, moderately advanced	1
Dementia praecox	2	Valvular heart disease, mitral insufficiency	3
Enuresis	14		
Epilepsy	2		
Flat foot	11		

VOLUME XXXVIII JULY 1940 NUMBER 3

United States Naval Medical Bulletin

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

Issued Quarterly by the Bureau of Medicine and Surgery

Navy Department

Washington, D. C.

VOL. XXXVIII JULY 1940 No. 3

UNITED STATES NAVAL MEDICAL BULLETIN

FOR THE INFORMATION OF
THE MEDICAL DEPARTMENT OF THE NAVY

DIVISION OF PUBLICATIONS
THE BUREAU OF MEDICINE AND SURGERY

THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY

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

Compiled and published under the authority of Naval Appropriation
Act for fiscal year 1940, approved May 25, 1939

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON: 1940

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

NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary.

Owing to 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, 1915, No. 1.
Volume X, 1916, No. 2.
Volume XI, 1917, No. 3.
Volume XII, 1918, Nos. 1 and 3.
Volume XXIV, 1926, Nos. 1 and 4.
Volume XXV, 1927, No. 1.
Volume XXVII, 1929, Nos. 3 and 4.
Volume XXVIII, 1930, No. 3.
Volume XXXIV, 1936, Nos. 1, 2, and 4.
Volume XXXV, 1937, No. 1.
Volume XXXVI, 1938, No. 1.

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

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means 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 Medical Department personnel, and reports from various sources, notes, and comments on topics of professional 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 professional interest.

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

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

ROSS T. MCINTIRE,
Surgeon General, United States Navy.

NOTICE TO CONTRIBUTORS

Contributions to the BULLETIN should be typewritten, double spaced, on plain paper, and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The BULLETIN endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnecessary changes in manuscript can be obviated if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All materials supplied for illustrations, if not original, should be accompanied by reference to the source and a statement as to whether or not reproduction has been authorized.

The BULLETIN intends to print only original articles, translations, in whole or in part, reviews, and reports and notices of Government or departmental activities, official announcements, etc. All original contributions are accepted on the assumption that they have not appeared previously and are not to be reprinted elsewhere without an understanding to that effect and that editorial privilege is granted to this Bureau in preparing all material submitted for publication.

EBEN E. SMITH, *Editor*,
Commander, Medical Corps, United States Navy.

U. S. NAVAL MEDICAL BULLETIN

VOL. XXXVIII JULY 1940 No. 3

SPECIAL ARTICLES

ROENTGEN PHOTOGRAPHY [1]

AN ECONOMICAL METHOD FOR GROUP SURVEY

By Captain W. Chambers, Medical Corps, United States Navy, and Commander C. F. Behrens, Medical Corps, United States Navy

Although a great deal of experimental work in roentgen photography has been done since Roentgen discovered the x-rays, it was not until recently that photographs of fluorescent images have had any great practical value. Improved fluorescent screens, fast lenses, and high-speed films today have opened new fields of application.

Early in 1939, Manoel de Abreu, Rio de Janeiro,[2] published the technic used during 7 years of research in mass thoracic survey work in Brazil, along lines suggested several years earlier by Cole, Leben, and others. Later Lindberg, Decatur, Ill.,[3] published suggested modifications of de Abreu's technic. Other reports of similar studies have appeared in the Lancet [4 5] and elsewhere. The reports referred to agree that for economical survey of large groups, roentgen photography has great practical value in the detection of early signs of tuberculous infiltration of the lungs.

A film size permitting large numbers of exposures per foot is naturally what is desired and thus the adequacy of 35 mm. film for diagnostic purposes is, of course, one of the most important considerations. It is not to be expected that the fine diagnostic detail of technically perfect 14- by 17-inch films will be duplicated. On the other hand, from published work of others, and from our own results, it is believed that very little significant detail will escape detection when the small size film is employed. (See fig. 2.)

Some work has been done by others with 4- by 5-inch films and with excellent results. These larger views, however, can only be obtained by the sacrifice of ease and efficiency of operation, and also of economy,

[1] From U. S. Naval Medical Center, Washington, D. C.

[2] de Abreu, Manoel: Process and apparatus for roentgen photography, Am. J. Roent., 41: 662, April 1939.

[3] Lindberg, D. O. N.: Suggested modifications of technique for roentgen photography, Am. J. Roent., 41: 867, May 1939.

[4] Hilton, G., Hart, P. D'Arcy, and Morland, A.: Tuberculosis in medical students, The Lancet, p. 263, Feb. 10, 1940.

[5] Dormer, B. A. and Collender, K. G.: Miniature radiography, The Lancet 1: 1309, June 10, 1939.

and with no substantial gain as to results. From a practical standpoint, the 35 mm. film is a most suitable size.

In order to study the matter in detail, plans were made late in 1939 at the Naval Medical Center to construct a roentgen photographic unit, there being none procurable commercially.

Following the general procedures reported by de Abreu and Lindberg, after trial-and-error experiments, a distance of 35 inches between fluoroscopic screen and camera (film) was selected. A light-proof, pyramidal-shaped box was constructed with a recent type fluoroscopic screen and lead glass at one end and a 35 mm. camera, with an F. 1.5 lens of 5 cm. focal length at the other. (See fig. 1.) The camera enclosure was lead-lined throughout except for an opening for the lens. Provision had to be made later to ground the camera. The lead glass covering for the fluoroscopic screen is probably not entirely essential as the lead shield for the camera and the camera lenses themselves protect the photographic film. However, the lead glass does afford protection to the screen and adds an additional safety factor relative to fog prevention by reducing secondary radiation in the region of the camera. The effect of a high-grade lead glass on exposure time we found to be very slight.

As to the technical x-ray factors, it has been found that slightly increased voltages over those usually employed, aid in producing contrast and keeping exposure time short. Our goal in this regard was a time of $\frac{1}{10}$ second. It was also desired to keep other factors within reasonable limits. Naturally, the more modest the capacity requirements, the less expensive the installation. A wide range of factors was experimented with, including 500 ma. current. Eventually the following was considered the most practical set of factors: Plus 5 kv. over the usual thickness of part figure from the standard charts for the 6-foot chest; 150 ma. current; $\frac{1}{10}$-second exposure time; lens opening of F. 1.5; 33 inches between tube and screen.

Other combinations of factors also give good results. Currents of 100 ma. or even less can be used. Voltages can be raised another 5 kv. or more. Exposure times even up to $\frac{1}{2}$ second give fair results, although just as in regular radiography of the chest, the longer exposures entail progressive loss of detail; this was verified by a few exposures extending up to 2 seconds made with 25 ma. currents. Thus very low capacity apparatus is not to be recommended. Toward the other extreme, some workers appear to feel that high-capacity apparatus and the use of rotating anode tubes are essential. Still another appears enthusiastic about ultra high-speed photographic equipment and has had a lens of F. 0.86 constructed. There can be no objection to high-capacity apparatus other than that of expense, but it is felt that when you attain the speed of $\frac{1}{10}$ second with apparatus of moderate capacity the point has been reached at which the law of

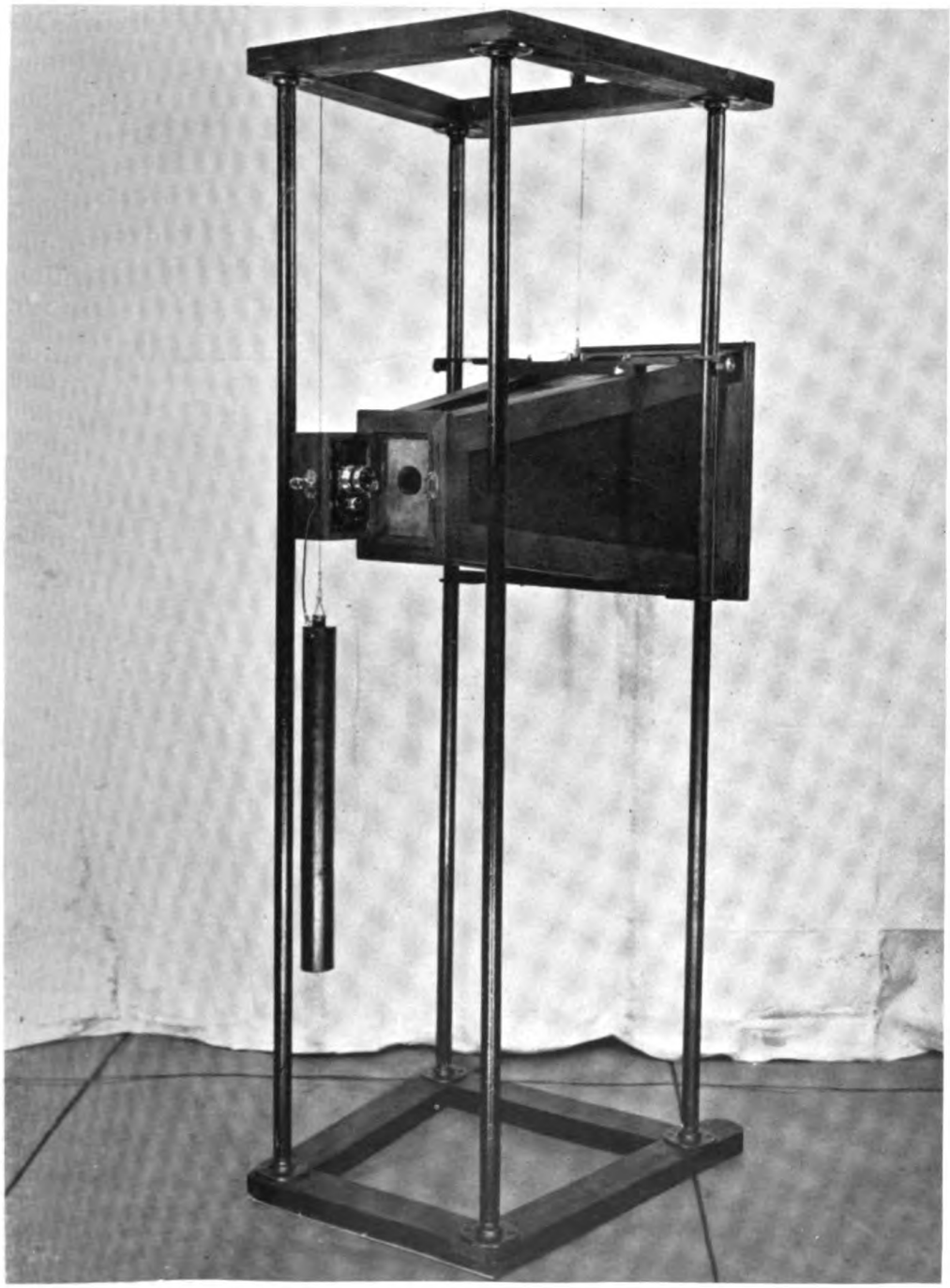

FIGURE 1.—ROENTGEN PHOTOGRAPH UNIT

FIGURE 2.—CONTACT PRINT FROM 35-MM. FILM.

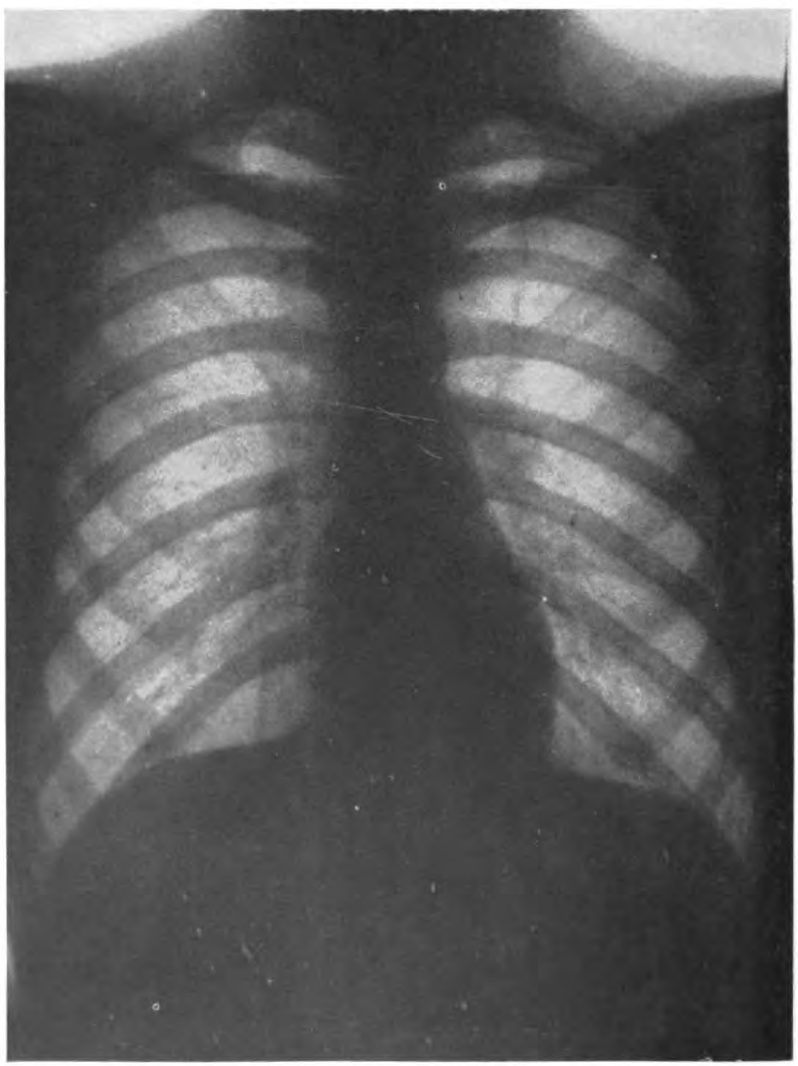

FIGURE 3.—PROJECTED PRINT FROM 35-MM. FILM

diminishing returns becomes the deciding factor. In other words, very little improvement in this type work will be obtained by further increases either in lens speed or x-ray capacity.

The 35 mm. chest films may be viewed over illuminated ground glass with an ordinary hand magnifying glass. An inexpensive viewing box is on the market today, which is quite satisfactory. Films may also be viewed by projection to any desired magnification. They stand enlargement very well even up to 10 by 12 inches and beyond, and excellent diagnostic detail is yielded. (See fig. 3.) General service use and the individual inclinations of roentgenologists will probably determine this detail.

Our belief, thus, is that the use of 35 mm. roentgen photographic units for chest surveys of service personnel is practicable, economical, and certain to be of immense value.

RECRUIT EXAMINATION

THE NEED FOR CHEST X-RAYS[1]

By Commander E. G. Brian, Medical Corps, United States Navy, and Lieutenant E. Ricen, Medical Corps, United States Navy

The detection of the minimal pulmonary parenchymal lesion caused by *Mycobacterium tuberculosis hominis* is often beset with many difficulties. Unfortunately, a great discrepancy exists between the physical findings elicited by examining the chest and the underlying pathology. It is not unusual for minimal or even moderately advanced tuberculous lesions to present so slight a variation from the normal chest as to be undetectable by the stethoscope. In connection with this, the authors recall a statement made by a prominent chest specialist to the effect that the greatest boon to the early detection and eradication of pulmonary tuberculosis would be to consign all stethoscopes to a huge fire and thereafter depend entirely upon the roentgen rays for the detection of all pulmonary lesions. This remark well illustrates the point in question. When such difficulties as these are encountered by the trained phthisiologist, it becomes more and more apparent that to the physician who has had no special training in the diagnosis of diseases of the chest, this problem becomes multiplied a thousandfold.

It is to the medical officer assigned to recruiting duty that the responsibility for detecting these minimal and moderately advanced lesions of pulmonary tuberculosis is relegated. The tremendous importance of eliminating the potentially tuberculous individual before he is accepted as a recruit cannot be overemphasized. The obvious and only solution to this problem lies in the routine roentgen-ray examination of the chest before final acceptance for enlistment.

[1] From the U. S. Naval Hospital, San Diego, Calif.

Prior to his hospitalization, the tuberculous individual acts as a source of infection to all with whom he comes in contact. With the crowded conditions which exist aboard ship this menace becomes a real one. Often these cases are kept aboard for a prolonged interval before their illness is discovered. By the time they are hospitalized they may represent far advanced cases. The exact number of cases which result from this continued exposure cannot of course be estimated but it is probably directly proportional to the time which has elapsed before the diagnosis has been made.

In addition, the problem is also a matter of public health involving the community and the home in which the individual lives. Following hospitalization of the tuberculous patient various social agencies must check to determine if his wife, children, and others with whom he has come in close contact are free of disease. This usually involves treatment and diagnosis by the medical officer on out-patient duty. Here again it is impossible to determine the number of individuals in the civil community who may have contracted the disease as a result of exposure to these individuals during their liberty ashore. Again there is the matter of the cost to Federal and local governments for the detection of pulmonary tuberculosis among those thus exposed. This cost though undeniably great cannot of course be estimated.

Prior to detection and hospitalization numerous sick days and decreased efficiency of the ship's organization may result. This is especially applicable to men in key positions.

Chest x-rays may also result in eliminating potential recruits who may be suffering from other diseases of the lungs, heart, and mediastinum. Among these we find: bronchitis, bronchiectasis, new growths of the lung, pneumoconiosis, silicosis, emphysema, mediastinitis, new growths of the mediastinum, valvular heart disease, pericarditis, aneurysm of the aorta, and congenital heart disease. The acceptance of a recruit with any of these clinical entities would of course, eventually involve cost in training, hospitalization, and benefits.

Aside from the monetary saving involved still another and most important consideration must be taken into account when considering the need for routine chest x-rays in the examination of recruits. In view of our present-day knowledge of the epidemiology of tuberculosis, gained by tireless and unending research, we should be grossly lacking in our duty to the medical profession and society as a whole, if we did not do our utmost in the fight against the eradication of tuberculosis. By early diagnosis many a young man may be shown the way toward an early cure and returned as a useful member of society, instead of being doomed to an early death as well as being a possible source of infection to numerous others. In this way, the detection of the minimal and moderately advanced lesions of pulmonary

tuberculosis, will result in untold value to the naval service itself as well as contributing largely toward our ultimate success in controlling this disease.

Case Reports

C. R. H., age 24 years. Reenlisted September 20, 1939, at a naval recruiting station. Transferred to the naval hospital October 4, 1939, from a destroyer with the diagnosis: *Diagnosis undetermined (pulmonary tuberculosis)*.

Sputum examination October 5, 1939, positive for *M. tuberculosis hominis*. Chest x-ray October 2, 1939, revealed a mottled infiltrative lesion in the right upper lobe, extending downward to the level of the sixth rib posteriorly. There appeared to be a cavity opposite the second interspace anteriorly, that measured approximately 3 cm. in diameter. Impression: Pulmonary tuberculosis, chronic, active, moderately advanced.

R. F. K., age 26 years, candidate for admission to Medical Corps, United States Navy. Routine chest film taken November 6, 1939, showed evidence of minimal lesions with pleural thickening and scars in both apexes. The process did not appear to be very active and the other lung fields were clear. The shadows of the heart and great vessels were within normal limits.

CONCLUSION

Routine chest roentgenograms taken on all recruits before final acceptance for enlistment would eventually result in a great monetary saving as well as contributing largely in the fight for the control and eradication of pulmonary tuberculosis.

RECRUIT SELECTION

By Lieutenant Robert A. Bell, Medical Corps, United States Navy

The importance of recruit selection has been repeatedly emphasized in this publication. In this connection it is desired to review some of the thought on the subject and to present a survey of the recruit rejections at the Parris Island Marine Recruit Depot during the recent period of Marine Corps expansion.

The newly enlisted personnel have, for the most part, accomplished their enlistments at recruiting stations. In actual practice, therefore, it is the physician assigned to the recruiting station who has the first opportunity to accept or reject applicants for enlistment. The importance of this duty was recognized by Cecha [1] who stated:

> The health and vigor of the Navy depend upon the good work of the recruiting station, that is to say, upon the medical officer, for according to his judgment, borderline cases are either accepted or rejected. However, quality should not be sacrificed to numbers, though there are times when the borderline cases, if enlisted, would improve the station's standing.

Poppen [2] considered it:

[1] Cecha, A. H., U. S. Nav. Med. Bull. 14: 371-372, July 1920.

[2] Poppen, J. R., Psychometric tests for recruiting stations, U. S. Nav. Med. Bull. 18: 14-25, January 1923.

Better to enlist one man with normal intelligence than a dozen who are simply hewers of wood and drawers of water.

In reporting on 1,000 applicants for enlistment, Templeton [3] accepted only 258, or roughly 25 percent. This ratio of acceptances approximates that for the entire service at that time, as is illustrated in table 1 which summarizes the statistics on recruiting for the calendar years 1934–37, as reported by the Bureau of Medicine and Surgery in Statistics of Diseases and Injuries in the United States Navy.

TABLE 1

Recruits	Navy				Marine Corps			
	1934	1935	1936	1937	1934	1935	1936	1937
Examined	88, 718	91, 449	79, 571	70, 614	16, 771	15, 290	13, 248	18, 630
Rejected	66, 419	67, 999	55, 282	46, 370	11, 603	11, 429	9, 068	12, 721

Templeton [3] considered one-half of those qualifying as excellent material, one-third as average, and the rest as acceptable. He states:

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

The major disqualifying defects which he listed as causes of rejection were, dental defects, 196, flat feet, 131, and defective vision, 73, with an additional 52 who were color blind. It is of interest that only eight had defective hearing. Marshal [4] stated:

The majority of our rejections are because of defective teeth, defective vision, flat feet, and underweight.

The leading causes for the rejections listed in table 1 were defective teeth, defective vision, flat feet, defective physical development, and underweight.

While the recruit is a potential unit of the service, it is anticipated that he will receive a detailed study at the training station [5] and that such conditions as the neuroses and the milder degrees of constitutional defect will be detected.[6] As Thomas and Agnew [7] recently pointed out, it is apparent that certain constitutional conditions may be present in an applicant for enlistment and that it is not possible to determine their existence during a physical examination at the recruiting station. When the recruits report at a training station,

[3] Templeton, H. D., U. S. Nav. Med. Bull. **34**: 22–27, January 1936.
[4] Marshal, L. B., U. S. Nav. Med. Bull. **24**:49–55, January 1926.
[5] Sutton, D. G., Utilization of psychiatry in armed forces, U. S. Nav. Med. Bull. **37**:262–273, April 1939.
[6] Jacoby, A. L., Treatment of military offenders, U. S. Nav. Med. Bull. **13**:229–236, April 1919.
[7] Thomas, G. E., and Agnew, W. J. C., Recruiting duty, U. S. Nav. Med Bull., **37**:276–279, April 1939.

they are given their first meal and marched to the detention barracks where they are required to take a shower bath and then present themselves for a rigid physical examination. This is most thorough and any physical conditions or defects which have been overlooked at the recruiting office are detected and recorded. If the disability is of a minor nature its presence is simply noted in the health record, but if it is more serious in nature and not necessarily a cause for rejection, a waiver is requested from the Bureau of Medicine and Surgery as provided for in existing instructions.[8] When such conditions are obvious, probable, or possible causes for rejection, the recruit is transferred to the hospital for further study and disposition.

During the period from July 1, 1939, to March 7, 1940, inclusive, 4,162 recruits were received at the Marine Corps recruit depot, Parris Island, S. C. Of this number 163 were surveyed before the completion of their training period for conditions existing prior to enlistment.

The following table indicates the diagnosis with which these cases were separated from the service:

23 Myopia.	1 Amputation traumatic, index finger.
3 Color blindness.	1 Deformity acquired, foot.
1 Trachoma.	1 Deformity congenital, knees.
1 Blepharitis.	3 Psychoneurosis, hysteria.
1 Conjunctivitis, follicular.	1 Psychoneurosis, situational.
1 Astigmatism, compound myopic.	1 Psychoneurosis, psychasthenia.
18 Absence acquired teeth.	1 Constitutional psychopathic state emotional instability.
16 Caries teeth.	
8 Malocclusion teeth.	1 Constitutional psychopathic inferiority, without psychosis.
1 Abscess periodontal.	
1 Extensive and numerous unsatisfactory restoration by fillings.	1 Constitutional psychopathic state, inadequate personality.
2 Abscess periapical.	2 Dementia praecox.
15 Syphilis.	1 Psychosis, unclassified.
1 Neurosyphilis, serological.	3 Epilepsy.
6 Enuresis.	1 Narcolepsy.
7 Otitis media, chronic.	1 Sclerosis, disseminated.
7 Deafness, bilateral.	1 Headache.
2 Deafness, unilateral.	4 Ulcer duodenum.
1 Otitis media chronic, bilateral.	2 Tuberculosis.
3 Defective physical development.	1 Calculus, kidney, bilateral.
3 Flat feet.	1 Bronchiectasis.
2 Pes cavus.	1 Pansinusitis.
1 Deformity acquired, wrist and hand.	1 Hernia inguinal, indirect.
1 Deformity acquired, arm.	1 Valvular heart disease, aortic and mitral.
1 Cicatrix, skin, leg and thigh.	
1 Deformity acquired, leg.	1 Splanchnoptosis.
1 Deformity acquired, leg and foot.	1 Hyperthyroidism.
1 Tenosynovitis, chronic, knee.	1 Varicocele.

All recruits in addition to receiving smallpox vaccination and typhoid inoculation receive a serological test for syphilis and blood

[8] Manual of the Medical Department, U. S. Navy, Par. 1407.

typing. In this way 15 cases were detected and discharged by reason of inaptitude [9] because of a positive serology for syphilis. No individual was recommended for discharge for such cause without first obtaining a serology recheck and careful history and physical examination in relation to a syphilitic background. All of the positive blood Kahn tests on these individuals were strongly positive and so no question of doubtful or weak positive tests arose.

This number of rejections may be compared with the 66 rejections among 5,578 recruits reported by Thomas and Agnew [7] and the rejections over the 5-year period 1925–29 inclusive reported for all naval training stations as summarized in table 2.

TABLE 2

Recruits	1925	1926	1927	1928	1929
Received	9,385	16,212	21,323	13,589	13,531
Appearing before a board of review:					
Number	688	842	937	611	709
Percent	7.33	5.19	4.39	5.50	5.24
Recommended for inaptitude discharge:					
Number	465	496	554	410	323
Percent	4.95	3.06	2.6	3.02	2.39

McDaniel [10] reported on the examination of 12,750 volunteers, passed by various regular naval recruiting stations and of these, 194 were recommended for survey because of the neurological examination. This included 76 luetics who were recommended for survey, before they had time to become a menace to themselves and to the service, and 86 who were recommended for survey because of failure on preliminary psychological tests.

It would seem that a number of the more obvious defects among this group of rejected recruits could have been noted at the recruiting stations. For instance one individual could neither read nor write and while no educational standard has been officially established for recruits, the regulations [11] require that a candidate shall be able to read and write and that he possess a reasonably quick and clear understanding. The most glaring examples, however, are in those new arrivals examined from 1 to 3 days after having been accepted at a recruiting station and found to have dry, inspissated wax lodged in an external auditory canal and hiding a perforated ear drum. On the other hand, certain constitutional inadequacies such as enuresis, epilepsy, neuroses and the constitutional psychopathic states, may be concealed or escape detection at the recruiting office. To help eliminate such cases Thomas and Agnew [7] have recommended closer

[9] Letter, Major Gen. Commandant to Commanding General, Marine Barracks, Parris Island, S. C., 1500-10/ACA-270 1. b., 8 August, 1939.

[10] McDaniel, F. H.: Report of the psychiatric division on recruits entering incoming detention camp, U. S. Nav. Med. Bull., Vol. 13: 854-858, October 1919.

[11] Manual of the Medical Department, U. S. Navy, Par. 1404 (a).

cooperation between the line officer in charge and the medical officer at the recruiting station. That the officer on recruiting duty will be immediately informed of the rejection of recruits selected by his office, the Bureau has directed that:

When recruits with less than 6 months' service are surveyed, an extra copy of the report will be prepared in each case and forwarded to the Bureau via the recruiting office where the man was enlisted for the medical officer's information and comment.[12]

Jacoby [6] has written:

Practical psychiatry has been defined as applied common sense. If the medical officer on recruiting duty will have a private interview with each applicant, directing his questions toward finding out what schooling the applicant has had, his occupation, the maximum wage he has earned, why he wants to enlist—in short, what he has done with his life—he will form an excellent idea of the applicant's fitness or unfitness. This interview must not take on the character of a perfunctory meeting, but must be done in such a way that the applicant feels that the doctor has a real personal interest in him. A very few minutes, not more than 5 in most cases, is all the time which needs to be consumed in this way.

It is recognized that these latent, quiescent, and hidden inadequacies and defects should be uncovered at the training station where the environmental conditions are more apt to force them into the open. Jacoby [6] considered the way a recruit adjusts himself to his environment is of far greater importance to the service than the way he may perform psychological tests. In considering the elimination of the epileptic, Bisch [13] states:

The men entering the naval service are young men and many are still in their adolescent period or have but recently emerged from it. A large number come from farming districts where emotional stresses and life's complexities are at a minimum. Very suddenly new and more exacting duties are thrust upon them; many for the first time have left their homes; their altered method of living and even thinking must needs appear cold and severe. Surely, we have here a very exacting test of an individual's ability to adjust himself and given an epileptic constitution—a tendency to faulty adjustment—it is small wonder that many cases are overwhelmed, that something breaks, and that the epilepsy at last appears in its more glaring symptoms. This, it should seem, would adequately explain the relatively large number of epileptics found among the recruits.

These remarks could well be applied to most of the constitutional inadequacies. Sutton [5] has observed:

The psychopath has more difficulty in adjusting to the service than has any other type of questionable individual. A recruit in this classification is not amenable to discipline and he ordinarily cannot comfortably be assimilated in any part of the organization without having an influence on morale. The constitutionally inferior and the medium-grade intellectual types are ordinarily acceptable if properly classified and utilized in the proper locations within the organization. Those found to be potentially psychotic, of course, should be eliminated together with the individual who is found exhibiting prodromal symptoms.

[12] Manual of the Medical Department, U. S. Navy, Par. 3423 (j) as changed in Circular Letter R.

[13] Bisch, L. E.: Eliminating the epileptic from the Navy, U. S. Nav. Med. Bull., Vol. 13: 5-16 January 1919.

In speaking thus, Sutton was apparently referring to wartime mobilization. It is questionable if the constitutionally inferior types are to be accepted during peacetime recruiting when a well-trained potential nucleus is our goal. These and related types require the closest appraisal for the machinery of modern warfare requires highly skilled personnel for operation and maintenance and the waging of war places a tremendous stress on combat personnel. Sutton [5] quotes a part of a memorandum submitted by the William Alanson White Psychiatic Foundation:

> Personalities already heavily burdened down by anxiety are apt to break down under this stress (warfare) and to be disabled by acute anxiety states, the so-called shell-shock, or by panic. The prevention of these acute disturbances is the more important because of their "psychic contagiousness," their tendency to spread through the unit, and rapidly to disable others whose feeling of personal security is threatened from within. This calls for immediate effective therapy or prompt evacuation of all affected personnel. As this is always costly to combat efficiency and often entirely impractical, precautionary measures are vital.

It is pertinent to this phase of the recruiting problem to quote from British experience:[14]

> It has been stated that over 20 percent of all the soldiers discharged for disability from the British Army during the Great War had one or another of the disorders grouped under the term "shell-shock," "neurasthenia," or "war neurosis."—The danger of enlisting such cases is in their power to spread their inefficiency amongst others, resulting in hysterical mass movement, loss of morale, and possibly panic. A large number of men who break down under the nervous strain of warfare do so from a fear of the unknown. The most suitable place—to eliminate those with nervous instability—is at the training depot.

This presentation of some of the thought and philosophy entering into recruit selection is submitted because of its wide applicability and repetitive value.

SPONTANEOUS PNEUMOTHORAX IN THE APPARENTLY HEALTHY

By Captain Paul Richmond, Medical Corps, United States Navy

During the past 2 years three cases of spontaneous idiopathic pneumothorax occurring in apparently healthy adults have been observed at the United States Naval Hospital, Bremerton, Wash. Although several articles in the recent medical literature adequately describe this condition and report series of cases it is believed that the relative frequency and the benign nature of the accident are not widely understood. Available text books on internal medicine and medical diagnosis mention principally the occurrence of the complication in cases of known active pulmonary tuberculosis, lung abscess, gangrene of the lung, septic infarction of the lung, bronchiectasis,

[14] Notes and comments: U. S. Nav. Med. Bull., Vol. 20: 463-466 (April) 1924. Referring to an article by C. R. Sylvester-Bradley, Lieut. Col., R. A. M. C. Some aspects of normality, with special reference to the selection of recruits, British Med. J., November 17, 1923.

empyema, hydatid disease, tumors, and other serious illnesses. Spontaneous pneumothorax occurring suddenly in otherwise apparently healthy persons is not clearly differentiated.

Recently published studies tend to show that the most frequent etiology is the rupture of an emphysematous vesicle or bulla. These blebs have been demonstrated around scar tissue or areas of collapse. Healed tuberculous foci or small broncho-pneumonic patches may leave such scars and collapsed areas. They are described also as congenital malformations. However, congenital cystic disease demonstrable by x-ray has only rarely been associated. Tearing of lung tissue from the pull on a plural adhesion has been suggested as a possible cause. Although rupture through an active subclinical tuberculous focus may be the cause it is possible by follow up x-ray and clinical findings to demonstrate tuberculous lesions in only a small percentage of cases.[1]

Complete recovery is usual. The lung expands in a few weeks and is normal to physical and x-ray examinations. However, recurrences sometimes occur, either on the same or opposite side, as adhesions do not tend to form. Effusion is rare.

No treatment is needed except bed rest and sedatives during the first few days. Dextrose solution has been injected into the pleural cavity to cause adhesions in recurrent cases. Aspiration has been recommended if symptoms are severe or bilateral. A previously reported bilateral case [2] associated with unrecognized active tuberculosis was so rapidly fatal after the collapse of the second side that aspiration could not be attempted. Although such termination has only rarely been recorded the occurrence suggests the advisability of early aspiration of all cases to safeguard the patients. The dangers of infection, pleural irritation with formation of effusion or possible damage to the expanding lung from the aspirating needle have seemed to outweigh that of sudden bilateral collapse.

The following cases are reported to emphasize the characteristic features of the benign condition. During the same period only one case was observed with previous suggestive symptoms and subsequent x-ray evidence of active pulmonary tuberculosis.

Case Reports

Case 1.—J. W. M. Ph. M. 2c. United States Navy. Age 29. Admitted December 18, 1935. No previous sypmtoms referable to chest. No recent illness. Onset December 15, 1935 while working. Had sudden severe pain in right chest, dizziness and dyspnoea. Symptoms improved rapidly and were slight by the time of admission. BP, 120/70; pulse, 70; no fever; no cough; WBC, 12,050; Kahn negative. Physical signs of right pneumothorax. X-ray showed partial

[1] Gordon, Ian, Lancet 2:178-181, July 25, 1936.
[2] Richmond, Paul, U. S. Nav. Med. Bull. 31: 369-370, Oct. 1933.

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collapse of the right lung. By January 6, 1936, x-ray showed complete expansion of right lung with no evidence of pathology. Patient discharged to duty January 17, 1936.

Case 2.—C. M. D. Sea. 2c. United States Navy. Age 24. Admitted September 22, 1936. No previous chest symptoms. Onset at 6:30 a. m. on day of admission. Sudden sharp pain occurred in left upper chest when he bent over to pick up something off the deck. Since then pain had been continuous but decreasing. Some dry cough and shortness of breath. Characteristic physical findings of left pneumothorax with some displacement of the heart to the right. X-ray showed complete collapse of the left lung with the mediastinum pushed over to the right. No fever; pulse, 100; respiration, 18; WBC, 8,150; Kahn negative. By October 14, 1936, x-ray showed the lung completely expanded and negative for pathology. Sedimentation rate on October 27, 1937, was 14 percent. Slight pain on deep inspiration persisted until November 9, 1936. X-ray on November 10, 1936, was negative for tuberculosis. Patient discharged to duty on November 17, 1936.

Case 3.—A. R. C. Electrician, United States Navy. Age 34. Admitted November 16, 1937. No previous sickness since influenza in 1918. Underweight but no recent loss of weight (Weight, 118 pounds; usual weight, 122; maximum weight, 124; height, 68⅛ inches). No previous cough or coryza. Felt well on retiring night before and on arising on day of admission. Sudden pain in right upper chest came on while on way to work. Stooping over partly relieved pain. Walking aggravated pain. Dyspnoea on exertion but no cough. Could not breathe comfortably lying down.

Physical findings of complete right pneumothorax with hyperresonance and increased respiratory sounds on the left. Heart sounds normal and heart not demonstrably displaced; rate 84 and regular; BP, 116/86; no cyanosis. No fever. Abdomen normal on palpation. Tendon reflexes very active. Profuse perspiration. Moderate apprehension. Hgb, 85 percent; WBC, 10,000; differential count normal. Sputum negative for A.F.B. Kahn test negative. X-ray showed 4/5 collapse of the right lung with mediastinal structures slightly displaced to left; no pleural exudate; and no lesion of either lung was visualized.

Symptoms gradually subsided with bed rest. No fever after admission. Slight nonproductive cough. Tuberculin skin test slightly positive on December 3, 1937. Weekly x-ray films showed gradual expansion. By December 20, 1937, right lung was completely expanded and normal to physical and x-ray examinations. Patient discharged to duty on December 22, 1937.

LUNG CYSTS

By Commander Frederick C. Greaves, Medical Corps, United States Navy

Cysts of the lung are comparatively rare, but reports of their occurrence are appearing more frequently. This is probably due to the increased use of the x-ray in searching for possible pathology within the chest, particularly in tuberculosis-prevention campaigns. The condition immediately suggests a developmental defect, and no doubt, most of them are congenital in origin. However, the possibility of their being acquired must not be overlooked. Inflammatory scarring is able to cause enough distortion to produce them, but in this event the original pulmonary inflammation would call attention to the lungs

and would overshadow the cystic condition. The congenital cysts will be found at all ages and when symptoms occur they will result from one of the following conditions:

1. Rupture of a cyst that communicates with the bronchial tree will produce pneumothorax, such as occurred in the case reported above.

2. Inflammation of the wall of the cyst. When this occurs exudation into the cavity of the cyst will result. If drainage through the bronchial tract is free and unobstructed, there will be a productive cough with little or no evidence of sepsis. If the drainage is partially obstructed and intermittent, there will be intermittent sepsis and the periodical raising of large amounts of foul sputum, depending upon the size of the cyst. If obstruction is complete, the signs will be those of a walled-off abscess in the lung until rupture occurs into a bronchus or into the pleural cavity.

3. The cyst walls themselves are composed of fibrous tissue of varying thickness and enclose spaces that contain air, fluid, or both. Hence, the presence of a cystic structure is at the expense of functioning lung tissue. Replacement of a sufficiently large amount of parenchymal pulmonary tissue to produce deficient aeration will result in dyspnoea and cyanosis. Multiple cysts of small and moderate size scattered throughout the lung tissue producing a honeycombed appearance or large thick-walled cysts replacing whole lobes will produce symptons of deficient aeration, cardiac hypertrophy, cyanosis, dyspnoea, and pulmonary osteo-arthropathies.

The treatment should be varied to meet the conditions present. Those cases discovered accidentally in which no symptoms have occurred should be left alone, but the patient should be warned that the condition exists, that rupture of a cyst may result in pneumothorax, and that increased sputum and varying degrees of sepsis will indicate that the cysts have become infected. However, the presence of uncomplicated cysts should not condemn the individual to a life of invalidism.

When infection occurs drainage must be established and maintained and an attempt should be made to clear up the infection by conservative measures and prevent a recurrence by strict attention to general health. These persons probably should be advised to live in a climate that is free from dampness and sudden changes of temperature. Those cases in which satisfactory drainage cannot be established through the bronchi or which resist conservative measures and continual sepsis is present will require more radical measures and lobectomy should be considered.

When insufficient aeration results from the presence of large or multiple cysts the treatment will resolve itself to outlining a program that will keep the patient comfortable with his diminished pulmonary capacity and safeguard him against acquiring pulmonary infection, plus the use of cardiac stimulants as needed.

The following case of lung cysts is reported because the condition is infrequently encountered and also because it may exist for a lifetime without seriously interfering with a normal existence.

Case Report

The subject of this report was a retired naval medical officer who died in September 1937 at the age of 70 from renal carcinoma. Since his retirement in 1896 he had practiced medicine until the onset of his fatal illness, and had been in good health with the exception of three attacks of spontaneous pneumothorax. The first occurred in 1904, the second in 1924, and the third in 1932. The episodes were similar in nature in that there was a sudden massive collapse of one lung. The clinical record fails to state whether it was the same lung in each case, but there can be no doubt that recovery was uneventful.

The patient visited many clinics where his case elicited considerable interest because the cysts were plainly visible in x-ray plates of the chest. He believed that he might have become infected with lung cysts during a tour of duty in the Philippines, but paragonimus was never demonstrated in the sputum and he enjoyed excellent health in the interval between episodes. When his death occurred the naval hospital in a neighboring city was informed that he had requested an autopsy to prove or disprove the parasitic etiology. This was done and the report is as follows:

Autopsy report: The body, which has been embalmed, is that of an emaciated man of about 70 years. Negative findings are omitted. The abdomen is markedly protuberant and the liver can be palpated below the umbilicus and in the left hypogastrium.

Heart: Is small, coronaries are visibly sclerotic but patent, and the aorta is markedly atheromatous.

Lungs: A few old dense adhesions are present in both pleural cavities, binding the lungs to the parietes. The left lung presents a thin-walled cyst 4 inches in diameter. It is located in the lower lobe and presents itself between the lung and the diaphragm posteriorly. Several smaller, very superficial cysts are present upon the surface of the left lung. The right lung presents about a dozen similar thin-walled, very superficial cysts, attached to the surface of the lower and middle lobes. The walls of these smaller cysts in both lungs are of tissue paper thinness, and resemble bubbles upon the surface. The walls of the cysts are resistant to trauma. Only one was ruptured in removing the organs. There is no evidence of any inflammation or other solidification in either lung.

Liver: Is tremendously enlarged, weighing approximately 4,000 grams, and is almost completely infiltrated with whitish metastatic tumor nodules.

Kidneys: The left kidney is completely enmeshed in a tumor mass that measures approximately 12 by 6 by 6 inches. This mass presents a nodular surface, areas of degeneration and infiltrates the surrounding tissues. The right kidney is similarly involved with these tumorlike nodules, but to a much less degree.

Ribs: Show metastatic involvement.

Microscopic report.—*Left kidney:* Blocks from numerous areas of the renal mass show a neoplastic process in which there is a definite attempt to reproduce renal tubules. Considerable degeneration and necrosis is present in the deeper portions of the mass. Near the periphery, however, cell outlines are clear and distinct and individual cells are seen to be relatively large and to be arranged in a distinct papillary pattern within the lumina of tubules. Loss of cellular polarity, hyperchromatism, and mitoses are prominent. There are no cystic areas anywhere. A narrow zone of compressed cortical tissue that is otherwise normal in appearance can be distinguished at the periphery in many of the sections. Nothing suggestive of adrenal tissue is noted. The neoplasm undoubtedly is one of renal origin.

Right kidney: The process in the tumor nodules of this kidney resembles that in the left with the exception that there is much less evidence of hyperchromatism

and loss of polarity. There is a much closer resemblance between the neoplastic cells and normal tubular epithelium, giving the impression that the process is one of primary hypertrophy undergoing malignant change, rather than a metastatic one from the opposite kidney.

Liver: The structure of the tumor nodules resembles that seen in the left kidney and has become so extensive that very little hepatic tissue remains. That which does remain is markedly degenerated.

Sections of the metastatic nodules in the ribs show a process similar to that of the left kidney.

Sections of the cystic lungs show the cyst walls to be thin and fibrous. There is no evidence of inflammation, neoplasm or parasitic infiltration or infestation.

ANATOMIC AND MICROSCOPIC DIAGNOSIS.—

1. Pulmonary cysts, multiple (congenital).
2. Papillary carcinoma, both kidneys.
3. Metastases, liver, ribs, and abdominal nodes.

SUMMARY

1. A brief description of the pathogenesis of the symptoms associated with lung cysts and the treatment of these symptoms that seem indicated are given.

2. The history and autopsy findings of a case of multiple lung cysts of long standing are reported.

HEMATURIA

ASSOCIATED WITH ACUTE UPPER RESPIRATORY INFECTION [1]

By Commander William H. Funk, Medical Corps, United States Navy

This hospital serves the adjacent naval training station where several hundred recruits are in training. These men remain here for 3 months so the personnel is constantly changing. Young adults from the ages of 18 to 25 are sent here from their homes on the farms, small towns, and large cities and begin their life in the Navy. Here they contract the upper respiratory infections so common in civilian life.

All upper respiratory infections severe enough to give elevated temperature for more than 1 day are admitted to this hospital. Therefore, there has been an opportunity to observe closely the course of these cases and especially to watch for and observe any renal manifestations.

The average case manifested rhinitis, sore throat or cough with headache and general malaise as the presenting symptoms. Elevation of temperature lasted from 1 to 5 days. Routine treatment consisted of bed rest, nasal sprays, gargles, inhalations and medication such as aspirin and cough mixtures, both expectorant and sedative. Patients were on forced fluid intake and liquid diet as long as there

[1] From U. S. Naval Hospital, Great Lakes, Ill. Thesis submitted to the Faculty 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 for graduate work in internal medicine.

was any elevation of temperature. On the average, patients remained in bed for 2 to 3 days after their temperature became normal. They were then up and about the ward for several days, then out of doors with gradually increasing activity. The average case was hospitalized 10 to 14 days before returning to duty to participate in the regular life of the training station.

Military or naval hospitalization is of necessity of much longer duration than in civilian life. In the service, the entire period of convalescence must be spent in the hospital. When a man is discharged from the hospital he must be entirely well and fit for duty, no matter how strenuous or arduous this may be. Our duration of hospitalization, therefore, includes the time spent to complete the entire convalescence.

All cases had a routine, complete urine analysis on admission and at least one other urine examination while up and about prior to discharge. All urines showing red blood cells on sediment examination were checked for occult blood by the orthotoluidine test. It was found that it required two to three red blood cells per high-power field to give a positive occult blood test.

The cases showing urinary changes, as well as many of the others, were given daily urinary examinations, and on many cases before discharge concentration tests, intravenous 15 minute phenolsulphonphthalein, and urea nitrogen blood determinations were carried out. Careful clinical observations, including blood pressure estimations were made on every patient showing any urinary abnormality.

During 22 consecutive months of 1935, 1936, and 1937, 343 cases of acute upper respiratory infection were observed in the medical service. These comprised cases of catarrhal fever acute (common cold) and acute tonsillitis. No cases of scarlet fever or the other communicable diseases are included.

An account of these cases with series number, hospital case number, patient's initial, age, urinary findings in regard to albumin, casts, blood, occult blood test, blood pressure, duration of fever in days, and additional tests of renal function is presented in table 1. The cases showing hematuria are indicated by an asterisk and are reported in more detail below. Of these 343 cases, 90 showed albumin in varying amounts, 7 showed casts, and 36 showed hematuria.

TABLE 1

Identification				Routine urine examination										
Series No.	Hospital No.	Initial	Age	Albumin	Casts	RBC	Occult blood	Number days fever	Blood pressure	Number of urine examinations	Urine concentration	P. S. P. test 15 min.	Blood urea nitrogen	Hematuria, days duration
1	S-10	S	56	1+	0	0		1		1				
2	S-4	V	58	0	0	0		3		2				
3*	137	S	34	0	0	+		3		1				
4	145	H	19	+0	0	0		1	105/70	1				
5	150	J	18	+0	0	0		4	105/78	2				
6	185	T	34	0	0	0		0	142/90	1				
7	279	H	23	0	0	0		2	130/70	1				
8	303	M	17	0	0	0		6	94/70	3				
9	281	U	18	0	0	0		0	112/72	1				
10	396	K	19	0	0	0		2		1				
11	15	V	34	0	0	0		3		1				
12	17	R	20	0	0	0		3	106/60	2				
13	18	P	19	0	0	0		2		1				
14	53	S	22	0	0	0		0	108/76	1				
15	64	H	20	0	0	0		2	112/78	1				
16	116	S	23	0	0	0		2	112/64	1				
17	59	K	22	0	0	0		0	118/60	1				
18*	S-32	J	23	2+	+	+	1+	2	112/62	7	1.012			7
19	S-70	K	20	0	0	0		2	116/70	1				
20	S-5	F	43	1+	0	0		2	120/80	1				
21	652	T	22	0	0	0		0	110/68	1				
22	635	H	20	0	0	0		0	132/76	1				
23	629	S	21	0	0	0		2	128/60	1				
24	627	C	21	0	0	0		2	110/74	1				
25	610	Z	18	0	0	0		6	120/70	1				
26	607	W	35	0	0	0		1		1				
27	606	S	29	0	0	0		2		1				
28	603	R	33	0	0	0		3		1				
29	599	N	22	0	0	0		1		1				
30	598	A	22	0	0	0		2	110/70	1				
31*	583	C	22	1+	0	+		0	125/78	8	1.023			1
32	576	B	29	0	0	0		4	120/70	1				
33	380	H	22	0	0	0		1		1				
34*	461	R	24	2+	+	2+		15	148/60	13	1.028	11	18	20
35	464	H	19	0	0	0		0	122/86	1				
36	446	S	18	1+	+	0		1		2				
37*	S-29	H	34	1+	0	1-2+		3	110/74	1	1.030			1
38	775	B	21	0	0	0		5	108/70	2				
39	771	S	18	0	0	0		0		1				
40	770	L	19	0	0	0		2	120/70	2				
41	720	S	27	0	0	0		2	102/60	1				
42	718	D	19	0	0	0		0		2				
43*	717	J	18	2+	0	12-11		5	120/70	7	1.013			4
44	716	E	22	0	0	0		4		2				
45	705	M	24	0	0	0		0	120/62	2				
46	690	G	26	1+	0	0		5	116/74	3				
47	682	M	18	0	0	0		2	112/64	2				
48*	681	N	18	3+	+	7-8+		6	117/60	4	1.020			2
49	681	M	18	0	0	0		2	118/58	3				
50	680	G	21	0	0	0		1	115/65	2				
51	677	W	17	0	0	0		1	106/60	2				
52	674	H	18	0	0	0		0	118/72	2				
53	673	G	18	0	0	0		0		2				
54	672	T	18	0	0	0		2	110/70	1				
55	670	E	17	0	0	0		2	118/76	2				
56	671	S	17	0	0	0		1	110/70	2				
57	776	G	21	0	0	0		2	110/62	2				
58	777	R	18	0	0	0		4	118/66	2				
59*	792	A	21	+	0	+		3	142/72	2	1.021			1
60	793	B	19	0	0	0		3	104/74	2				
61	794	D	20	0	0	0		2	130/55	2				
62	799	B	18	0	0	0		0	100/62	2				
63*	800	C	19	0	0	+		2	114/72	2	1.020			2
64	801	D	18	+	+	0		2	120/60	3				
65	802	S	20	0	0	0		2	110/66	1				
66*	809	D	23	2+	0	3-6+		3	125/70	4	1.030			5
67	810	L	21	+	0	0		0	118/68	3				
68*	813	N	19	+	0	+		2		3	1.028			2
69	814	P	21	+	0	0		2	116/72	2				
70	762	F	23	+	0	0		0	120/80	2				
71*	818	A	23	+	0	Rare+		0	120/60	4	1.027			6
72	819	E	19	0	0	0		1	140/80	4				
73	S-38	M	24	0	0	0		2	124/74	1				

TABLE 1—Continued

Identification				Routine urine examination				Number days fever	Blood pressure	Number of urine examinations	Urine concentration	P. S. P. test 15 min.	Blood urea nitrogen	Hematuria, days duration
Series No.	Hospital No.	Initial	Age	Albumen	Casts	RBC	Occult blood							
74	821	G	18	0	0	0		4	120/68	2				
75*	822	H	19	+	0	Rare+		2	134/68	3	1.031			2
76*	823	H	17	+	0	1-2+		6	75/55	4	1.025			2
77*	820	G	18	+	0	1-3+		2	120/60	4	1.025			6
78	S-68	H	19	0	0	0		2	134/74	2				
79*	825	R	18	+	0	Rare+		3	104/72	3	1.016			2
80	826	S	18	0	0	0		3	110/72	3				
81*	827	S	18	1+	0	4+	2+	1	104/64	7	1.023			6
82	827	S	18	+	0	0		3	104/64	5				
83	828	R	19	0	0	0		2	128/72	2				
84	832	C	17	0	0	0		3	120/70	2				
85	840	C	18	0	0	0		2	120/54	2				
86*	841	E	18	+	0	Rare		1	130/66	2	1.015			1
87	842	P	19	0	0	0		3	118/58	2				
88	844	S	17	+	0	0		1	132/60	2				
89	845	S	18	0	0	0		3	100/60	2				
90	846	T	19	0	0	0		4	118/70	2				
91	847	V	18	0	0	0		1	130/58	2				
92	848	P	21	0	0	0		3	120/62	2				
93	858	E	21	0	0	0		0	108/70	3				
94	859	W	20	0	0	0		1	120/80	2				
95	860	W	18	0	0	0		1	120/60	2				
96	863	C	23	0	0	0		2	116/70	2				
97	864	D	17	0	0	0		4	108/56	3				
98	865	K	18	+	0	0		6	110/66	2				
99	866	N	19	0	0	0		3	110/60	3				
100	425	E	21	0	0	0		2	120/72	2				
101	896	W	17	0	0	0		3	106/66	2				
102	862	J	22	+	+	0		1	106/76	4				
103	891	B	32	0	0	0		0	114/64	2				
104	889	C	17	0	0	0		2	116/70	2				
105	887	P	35	+	0	0		3	110/70	2				
106	883	H	18	0	0	0		2	112/64	2				
107	882	H	17	0	0	0		0	110/68	2				
108	873	L	18	0	0	0		2		2				
109	871	S	19	0	0	0		2	120/72	2				
110	870	N	19	0	0	0		3	154/72	2				
111	869	R	21	0	0	0		2	112/76	2				
112	868	P	17	0	0	0		2	124/66	2				
113	867	P	18	0	0	0		2	100/50	2				
114	S-67	A	20	0	0	0		1	116/72	2				
115*	679	B	17	+	0	Rare+	Neg.	1	106/66	3	1.015			5
116	S-16	B	23	0	0	0		2	132/84	2				
117	944	E	20	2+	0	0		5	118/80	7				
118	943	A	20	0	0	0		3	118/65	2				
119	940	D	18	0	0	0		4	124/74	3				
120	931	H	19	0	0	0		3	110/58	3				
121	904	C	20	0	0	0		2		2				
122	900	J	18	0	0	0		0	116/50	2				
123*	566	M	18	+	0	Rare +	Neg.	3	110/50	3	1.030			2
124	949	G	21	0	0	0		3	108/62	2				
125	954	W	22	0	0	0		1	120/60	2				
126	961	H	18	0	0	0		1	138/82	2				
127	962	J	21	0	0	0		2	117/68	2				
128	963	F	21	0	0	0		2	124/82	2				
129	966	N	21	0	0	0		1	115/70	3				
130	983	H	20	+	0	0		3	125/78	2				
131	988	S	18	0	0	0		2	116/60	2				
132	989	S	21	0	0	0		0	110/65	2				
133	990	S	20	0	0	0		1	118/64	2				
134	991	P	18	+	0	0		2	130/70	2				
135	993	M	18	0	0	0		2	108/48	2				
136	1001	R	18	0	0	0		0	128/70	1				
137	1003	T	17	0	0	0		1	128/60	2				
138	975	W	23	0	0	0		3	110/70	1				
139	1018	J	18	+	0	0		2	108/60	2				
140	1040	C	20	+	0	0		4	138/68	2				
141	1386	C	19	+	0	0		3	120/70	6				
142	1419	G	20	0	0	0		2	130/70	1				
143	1445	W	21	+	0	0		3	120/70	3				
144	1463	R	54	0	0	0		2	120/80	2				
145	1469	J	19	0	0	0		0	120/80	2				
146	1471	G	18	0	0	0		0	120/70	1				
147	1471	G	18	+	0	0		2	120/70	2				

TABLE 1—Continued

Identification				Routine urine examination				Number days fever	Blood pressure	Number of urine examinations	Urine concentration	P. S. P. test 15 min.	Blood urea nitrogen	Hematuria, days duration
Series No.	Hospital No.	Initial	Age	Albumin	Casts	RBC	Occult blood							
148	1480	R	19	0	0	0		2		6				
149*	1497	D	18	4+	0	2–4		1	116/84	8	1.024	34	16	1
150	1498	N	17	2+	0	0		1		5				
151	1507	L	17	0	0	0		1		5				
152	1508	A	17	0	0	0		4		5				
153	1506	G	17	0	0	0		0		4				
154*	1505	M	24	+	0	Rare		2		6	1.015			1
155*	1500	T	32	+	0	B–10 U		5	106/60	9	1.025	31	14	2
156	1499	C	19	0	0	0		2		5				
157	1444	S	17	0	0	0		2	116/70	5				
158	1365	H	21	+	0	0		0		3				
159	S–29	H	34	0	0	0		3		2				
160	S–8	P	36	0	0	0		1	110/80	1				
161	S–22	D	29	0	0	0		2		2				
162	1364	S	19	0	0	0		0	110/60	4				
163	1363	O	22	2+	0	0		0	120/70	6				
164	1363	O	22	0	0	0		4	120/70	4				
165	1362	W	17	0	0	0		2	130/70	6				
166	1361	N	20	0	0	0		2	130/70	7				
167	1355	B	18	0	0	0		0	120/64	5				
168	1355	B	18	0	0	0		3	120/64	4				
169	1350	M	21	0	0	0		2	130/70	8				
170	1342	D	18	0	0	0		1	116/74	6				
171	1294	T	19	0	0	0		3	130/80	6				
172	1271	D	20	0	0	0		2	115/70	7				
173	1307	H	24	0	0	0		1		5				
174	1276	H	18	0	0	0		3	120/70	5				
175	1261	C	21	0	0	0		0	110/70	4				
176	1260	B	18	0	0	0		0		4				
177*	1249	D	17	+	0	+		2	142/74	15	1.025	30	11	4
178	1211	R	21	0	0	0		0	142/70	4				
179	1248	N	17	0	0	0		2	110/70	1				
180	958	M	21	+	0	0		3		1				
181	1054	M	17	0	0	0		0	108/68	1				
182	1509	G	21	0	0	0		2		3				
183*	1510	M	21	2+	0	Few	+	3		9	1.030	26		1
184	1363	O	23	+	0	0		0	120/70	6				
185	1363	O	23	0	0	0		4	120/70	3				
186	1511	S	17	0	0	0		2		3				
187	1513	S	18	+	0	0		2		3				
188	1514	L	18	0	0	0		2		1				
189*	1515	N	17	+	0	4–6	+	3	110/70	7	1.025	46		1
190	1516	M	18	0	0	0		2		3				
191	1517	G	18	0	0	0		1		2				
192	1518	I	17	0	0	0		1		2				
193*	1519	G	17	+	0	1–2	Neg.	4		2	1.020			
194	1520	V	17	0	0	0		2		2				
195	1521	D	18	0	0	0		3		2				
196	1522	V	35	0	0	0		2		3				
197*	1523	K	19	3+	0	1–2		4		10	1.024	20		2
198	1524	W	17	0	0	0		4		4				
199	265	F	35	0	0	0		1		1				
200	15	V	35	0	0	0		0		1				
201	1525	E	18	0	0	0		1		1				
202	1526	W	19	0	0	0		3		1				
203	1527	P	17	0	0	0		2		2				
204	1528	H	20	0	0	0		2		1				
205	1529	V	18	+	0	0		2		3				
206	1530	B	21	0	0	0		2		1				
207	1531	S	22	0	0	0		3		1				
208	1532	L	17	+	0	0		3		2				
209	1533	M	22	0	0	0		2		1				
210	1534	H	18	0	0	0		1		1				
211	1535	F	37	0	0	0		0		3				
212	1536	W	20	0	0	0		0		1				
213	1537	D	18	0	0	0		1		1				
214	1538	H	19	0	0	0		1		1				
215	1539	A	20	0	0	0		2		1				
216	1540	F	17	0	0	0		3		1				
217	1541	H	20	+	0	0		4		3				
218	1542	W	19	0	0	0		1		1				
219	1543	N	18	0	0	0		4		2				
220	1544	P	19	0	0	0		1		2				

TABLE 1—Continued

Identification				Routine urine examination				Number days fever	Blood pressure	Number of urine examinations	Urine concentration	P. S. P. test 15 min.	Blood urea nitrogen	Hematuria, days duration
Series No.	Hospital No.	Initial	Age	Albumen	Casts	RBC	Occult blood							
221	1545	Y	17	0	0	0		2		1				
222	1546	F	22	0	0	0		4		1				
223	1547	S	20	0	0	0		1		1				
224	1548	W	18	0	0	0		1		1				
225	1549	O	17	0	0	0		2		1				
226	1315	D	17	0	0	0		3	120/70	4				
227	1244	O	19	+	0	0		2	126/80	3				
228	1550	J	22	0	0	0		1		1				
229	1471	G	18	0	0	0		0		1				
230	1471	G	18	+	0	0		2	120/70	2				
231	1551	S	19	0	0	0		0		1				
232	1552	C	20	0	0	0		3		3				
233	1553	O	20	0	0	0		3		3				
234*	1554	R	17	+	0	2–3	1+	3		3	1.022			1
235*	1555	G	17	+	0	4+	4+	0		7	1.024	16		1
236	1556	F	24	+	0	0		2		3				
237	1557	W	33	0	0	0		2		1				
238	1558	S	18	0	0	0		1		3				
239	1559	C	22	0	0	0		3		3				
240	1560	S	37	+	0	0		1		4				
241	1561	E	17	0	0	0		3		3				
242	1562	D	20	+	0	0		3		4				
243	1564	B	19	0	0	0		2	120/70	1				
244	1564	B	19	+	0	0		0	120/70	3				
245	S–69	S	20	0	0	0		2		1				
246	S–49	S	25	+	0	0		0		1				
247	1569	C	23	0	0	0		1		3				
248	1570	F	24	0	0	0		0		2				
249	1571	G	20	0	0	0		1		3				
250	1572	M	18	0	0	0		1		3				
251	1573	M	18	0	0	0		1		3				
252	1574	M	19	0	0	0		1		3				
253	1575	B	37	0	0	0		4	120/80	2				
254	1576	P	21	0	0	0		0	124/84	3				
255	1576	P	21	0	0	0		0	124/84	2				
256	1577	W	17	0	0	0		1		3				
257	1548	W	18	0	0	0		1		3				
258	1579	B	17	0	0	0		3		3				
259	1580	C	19	0	0	0		4		4				
260	1581	J	17	0	0	0		1		3				
261	1582	M	17	0	0	0		1		3				
262	1583	U	20	0	0	0		2		3				
263	1584	O	19	0	0	0		2		3				
264	1585	H	17	0	0	0		3		3				
265	1586	W	18	0	0	0		2		2				
266	1587	S	21	0	0	0		0		3				
267	1588	P	20	0	0	0		2		3				
268	1589	B	21	0	0	0		0		2				
269	1590	M	19	0	0	0		2		3				
270	1591	G	17	0	0	0		5		4				
271	1592	F	20	0	0	0		4		3				
272	1593	H	17	+	0	0		4		3				
273	1594	B	17	0	0	0		2		3				
274	1595	B	18	0	0	0		0		3				
275	1479	B	20	0	0	0		3		1				
276	1671	K	18	+	0	0		4		1				
277	1669	H	20	0	0	0		2	110/70	2				
278	1668	P	20	0	0	0		2	112/70	1				
279	1667	A	20	0	0	0		2	140/70	2				
280*	1802	B	20	0	0	Rare	Neg.	2	116/80	2	1.018			1
281	1004	H	19	+	0	0		2	120/70	2				
282	1799	M	18	+	0	0		2	120/70	2				
283	1798	B	19	0	0	0		2	120/70	2				
284	1798	C	18	0	0	0		0	120/70	2				
285	1755	O	18	0	0	0		2	106/60	2				
286	1726	S	19	+	0	0		0	110/64	2				
287	1710	T	18	0	0	0		2	120/80	2				
288*	1512	S	22	+	0	2–3	+	2	110/50	10	1.022	21		1
289	1699	L	18	+	0	0		1	110/72	3				
290	1695	M	18	+	0	0		2	140/74	3				
291	1803	V	17	0	0	0		1	110/70	4				
292	1840	S	18	0	0	0		5	94/54	4				
293	1844	S	17	+	0	0		1	130/60	4				

TABLE 1—Continued

Identification				Routine urine examination				Number days fever	Blood pressure	Number of urine examinations	Urine concentration	P. S. P. test 15 min.	Blood urea nitrogen	Hematuria, days duration
Series No.	Hospital No.	Initial	Age	Albumin	Casts	RBC	Occult blood							
294	1845	W	19	0	0	0		4	140/70	2				
295	1658	B	19	0	0	0		3	100/68	2				
296	1658	B	19	0	0	0		0	112/60	2				
297	1852	H	19	0	0	0		2	120/70	2				
298	1853	H	19	0	0	0		3	150/50	2				
299	1564	B	19	0	0	0		1	120/70	2				
300	1854	B	17	0	0	0		0	120/70	2				
301	1866	W	17	0	0	0		1	110/70	2				
302	1883	H	18	+	0	0		2	90/60	1				
303	1884	Z	18	0	0	0		0	112/70	2				
304	1884	Z	18	0	0	0		3	112/70	2				
305	1886	M	17	0	0	0		1		2				
306	1905	R	18	0	0	0		1	110/70	2				
307	1906	D	22	0	0	0		1	100/70	2				
308	1908	M	18	0	0	0		3	120/60	2				
309	1920	H	18	0	0	0		2	112/60	2				
310	1921	H	21	0	0	0		1	112/60	3				
311	1922	K	19	0	0	0		0		2				
312	1923	E	21	0	0	0		0	106/60	2				
313*	1947	C	17	+	0	Rare	Neg	3	110/70	12	1.025	37		6
314	1964	H	17	0	0	0		2	110/70	2				
315	1965	R	18	0	0	0		2	110/70	2				
316	1966	O	18	+	0	0		3		3				
317	1563	C	22	0	0	0		0	130/70	2				
318	2003	T	20	0	0	0		3		3				
319	2005	S	20	0	0	0		3		2				
320	1982	J	20	0	0	0		0	112/80	2				
321	2007	A	18	0	0	0		1		2				
322*	S-79	H	20	+	0	Rare	Neg.	4	130/90	4	1.019			1
323	2024	H	18	0	0	0		3	120/68	2				
324*	2033	D	21	+	0	Rare	+	4	112/70	3	1.015			1
325	2034	C	19	0	0	0		0	124/70	3				
326	2035	B	22	0	0	0		2	160/70	2				
327	2038	W	17	0	0	0		3	120/70	2				
328	2040	M	21	+	0	0		2	130/70	3				
329	2041	S	18	+	0	0		4	110/60	2				
330	2045	J	20	0	0	0		0	130/70	2				
331	2046	R	18	0	0	0		0	120/68	2				
332	2047	M	17	+	0	0		3	110/60	2				
333	2049	R	17	0	0	0		0	110/72	2				
334	2050	B	17	+	0	0		2		2				
335	2051	F	18	+	Rare	0		5	108/54	6				
336*	2052	M	18	+	0	Rare	Neg.	2	110/70	8	1.021			1
337	2070	S	18	+	0	0		3	100/58	2				
338	2081	P	17	0	0	0		1	104/70	2				
339	2086	R	18	0	0	0		1	112/72	2				
340	2096	R	22	0	0	0		2	100/60	4				
341	2097	R	17	0	0	0		4	180/106	2				
342	2099	A	21	0	0	0		4	108/70	2				
343	2109	J	20	0	0	0		4	135/60	2				

CASE REPORTS

Series No. 3.—Was admitted with the complaint of sore throat, chills, and general malaise. Physical examination showed injected pharynx and post-nasal discharge. There was elevation of temperature for 4 days. Admission urine showed rare red blood cells. Patient was discharged well at the end of 5 days. This man was admitted to the hospital 1½ years later for an orthopedic condition at which time the urine was negative.

Series No. 18.—Was admitted with the complaint of sore throat beginning 3 days previously. Physical examination showed surgically absent tonsils, hyperemic pharynx with cervical, and tonsillar adenopathy. There was elevation of temperature for 2 days. Admission urine showed 2-plus albumin, red blood cells

20 to 30 per high-power field, with positive occult blood test. The urine became negative 8 days later. Slight discomfort in the region of both kidneys was noticed during the height of the renal involvement.

Series No. 31.—Was admitted with the complaint of fever during convalescence from an acute rhinitis of 1 week's duration. Physical examination showed only mild rhinitis. There was no elevation of temperature. Admission urine showed 1-plus albumin with a rare red blood cell. Daily urines, thereafter, were negative. No symptoms referable to kidney involvement were noticed. Upon discharge, the patient was able to concentrate his urine to 1.023. Blood pressure was within normal limits throughout his hospital stay.

Series No. 34.—Was admitted with the complaint of rhinitis of 2 weeks' duration, while awaiting operation for an inguinal hernia. Physical examination showed mild, acute rhinitis. Admission urine was negative. The patient recovered from his acute rhinitis and was sent to the surgical service for operation without another urinary check up. Eight days after operation his urine showed 1-plus albumin, rare finely granular casts, and a rare red blood cell. Four days later the blood urea nitrogen was 18 and the 15-minute phenolsulphonphthalein was 6 percent. After 4 days the urine was negative and remained so until discharged.

Series No. 43.—Was admitted with the complaints of headache, fever, and general malaise. There had been an acute rhinitis for 5 days preceding admission. Physical examination showed chronically diseased tonsils, hyperemic pharynx, and tonsillar adenopathy. The admission urine was negative, but with an exacerbation of his sore throat 8 days later, the urine showed 1-plus albumin with a rare red blood cell. The urine became negative after 1 day and the patient underwent a tonsillectomy before leaving the hospital. The urine remained negative during the remainder of his hospital stay.

Series No. 48.—Was admitted with the complaint of having had rhinitis off and on for the past 2 months. The immediate symptoms were weakness, backache, and sore throat. Physical examination showed a pin point erythematous rash over the trunk, hyperemic pharynx, acute rhinitis, surgically absent tonsils, with cervical and tonsillar adenopathy. Admission urine showed 3-plus albumin, rare finely granular casts, and 5 to 7 red blood cells per high-power field. There was elevation of temperature for 5 days. The urine became negative after 5 days. The Schultz-Charlton blanching test for scarlet fever was negative and the patient did not desquamate. Patient was discharged with a negative urine. This patient was readmitted 2 months later with an acute rhinitis with elevation of temperature for 2 days. Daily urines were negative during this last period of hospitalization.

Series No. 59.—Was admitted complaining of weakness, headache, and elevation of temperature for 2 days. Physical examination showed rhinitis, chronically diseased tonsils, and hyperemic pharynx. Admission urine showed a trace of albumin and an occasional red blood cell. The urine became negative after 2 days and remained negative. There was some elevation of temperature for 3 days.

Series No. 63.—Was admitted complaining of general malaise and headache which had its onset 1 week previously, with a slight sore throat which had become progressively worse. Physical examination showed a slight rhinitis, injected tonsils and pharynx. Admission urine showed a rare red blood cell. Urines after this were negative. There was elevation of temperature for 2 days.

Series No. 66.—Was admitted complaining of headache, fever, and general malaise with onset 3 days previously. Physical examination showed rhinitis,

surgically absent tonsils, injected pharynx with tonsillar adenopathy. Admission urine showed 2-plus albumin with 3 to 5 red blood cells per high-power field. The urine became negative after 3 days and remained negative. Elevation of temperature lasted for 4 days.

Series No. 68.—Was admitted complaining of headache, fever, and general malaise which had lasted for 1 day. Physical examination showed rhinitis, surgically absent tonsils, and hyperemic pharynx. Admission urine showed 1-plus albumin with an occasional red blood cell. Three days later the urine became negative. There was elevation of temperature for 3 days. This patient developed a complication, acute nonvenereal epididymitis, from which he completely recovered in a few days.

Series No. 71.—Was admitted complaining of fever, chills, and general malaise with onset the day prior to admission. Physical examination showed rhinitis, hyperemic pharynx, and some cervical adenopathy. Admission urine showed 1-plus albumin with a rare red blood cell. These rare red blood cells persisted in the urine for 6 days, after this the urine became negative and remained so. There was no elevation of temperature during hospitalization.

Series No. 75.—Was admitted complaining of fever, headache, and general malaise. Onset was 2 days previous, with a head cold. Physical examination showed rhinitis, hyperemic pharynx with cervical adenopathy. Admission urine showed a trace of albumin with a rare red blood cell. The urine 2 days later was negative and subsequent urines remained so. There was elevation of temperature for 2 days.

Series No. 76.—Was admitted complaining of sore throat, preceded by an acute sore throat for which hospitalization had not been required. Physical examination showed rhinitis, surgically absent tonsils, hyperemic pharynx with tonsillar and cervical adenopathy and persistent moist rales at both bases. Admission urine showed 1-plus albumin with 1 to 2 red blood cells per high-power field. All urines after this were negative. There was elevation of temperature for 5 days and afterward there were occasional elevations of temperature through the course of complications of acute maxillary sinusitis, serofibrinous pleurisy, and chronic nontuberculous pneumonitis.

Series No. 77.—Was admitted complaining of headache and general malaise with onset 2 days previously. Physical examination showed rhinitis, hypertrophied tonsils with hyperemic pharynx. Admission urine showed 1-plus albumin with 1 to 3 red blood cells per high-power field. Urine became negative 4 days later and remained negative. Patient showed ability to concentrate the urine to 1.025 prior to discharge. There was elevation of temperature for 1 day.

Series No. 79.—Was admitted complaining of fever and general malaise with onset the day before. Physical examination showed rhinitis, surgically absent tonsils, hyperemic pharynx with tonsillar adenopathy. Admission urine showed a trace of albumin with a rare red blood cell. Two days later the urine was negative and remained so until discharged. There was elevation of temperature for 3 days.

Series No. 81.—Was admitted complaining of fever, headache, and general malaise with a history of acute rhinitis for 3 or 4 days previously. Physical examination showed acute rhinitis, chronically diseased tonsils, and injected pharynx. Admission urine showed 1-plus albumin with 1 to 2 red blood cells per high-power field. The red blood cells in the urine increased in amount until they were too numerous to count and the occult blood test of the urine was 2 plus. Six days later the urine became negative and remained so. There was temperature only on the day of admission. There were no symptoms at any time referable to the

kidneys. This patient returned to the hospital 1 month later for tonsillectomy, at which time the urine was negative and the patient was able to concentrate his urine to 1.028.

Series No. 86.—Was admitted complaining of fever, chills, and general malaise with onset the day prior to admission. Physical examination showed rhinitis, surgically absent tonsils, injected pharynx with cervical adenopathy. Admission urine was negative but the urine 5 days later showed a trace of albumin and a rare red blood cell. There was elevation of temperature for 1 day and the urine was negative on discharge from the hospital 2 days later.

Series No. 115.—Was admitted complaining of sore throat of 1 day's duration. This patient was hospitalized for 23 days for mumps 1 month previously, at which time admission urine showed 1-plus albumin with 3 to 4 red blood cells per high-power field. No further urinary studies were made during this prior hospitlization. Physical examination on this admission showed rhinitis, chronically diseased tonsils with tonsillar adenopathy. Admission urine showed 1-plus albumin with a rare red blood cell. Subsequent urines were negative and patient had a tonsillectomy at a later date. There was elevation of temperature for 1 day.

Series No. 123.—Was admitted complaining of sore throat. Physical examination showed a mild, acute follicular tonsillitis. Admission urine showed a trace of albumin and a rare red blood cell with a negative occult blood test. The urine 2 days and 4 days later was negative. There was elevation of temperature for 2 days.

Series No. 149.—Was admitted complaining of cough and general malaise of 4 days' duration. Physical examination showed rhinitis, chronically diseased tonsils with cervical adenopathy. Admission urine showed 3-plus albumin with 2 to 4 red blood cells per high-power field. Daily urines thereafter were negative. There was elevation of temperature for 1 day. Examination before discharge showed blood urea nitrogen 16, ability to concentrate to 1.025, and 15-minute phenolsulphonphthalein of 34 percent.

Series No. 154.—Was admitted complaining of rhinitis and general malaise of 4 days' duration. Physical examination showed rhinitis, surgically absent tonsils, hyperemic pharynx with tonsillar and cervical adenopathy. Admission urine showed 1-plus albumin with rare red blood cells. Subsequent daily urines were negative and the red blood cells in the urine lasted for 1 day. There was elevation of temperature for 2 days.

Series No. 155.—Was admitted complaining of chills, fever, and general malaise with onset on the day of admission. Physical examination showed rhinitis, tonsils surgically absent, and hyperemic pharynx. Admission urine showed a trace of albumin with 8 to 10 red blood cells per high-power field. The following day a rare red blood cell was seen with negative occult blood test. Daily urines thereafter were negative. There was elevation of temperature for 4 days. Prior to discharge the blood urea nitrogen was 14, the 15-minute phenolsulphonphthalein was 31 percent, and there was ability to concentrate the urine to 1.025.

Series No. 183.—Was admitted complaining of headache and fever with onset the previous day. Physical examination showed slight rhinitis, chronically diseased tonsils with cervical and tonsillar adenopathy. Admission urine showed 3-plus albumin, a few red blood cells with positive occult blood test, and rare coarsely granular cast. The following day the urine became negative and remained so until discharged. There was slight elevation of temperature for 2 days. Prior to discharge 15-minute phenosulphonphthalein was 26 percent and

there was ability to concentrate the urine to 1.030. This patient returned for tonsillectomy 1 month later, at which time the urine was negative.

Series No. 189.—Was admitted complaining of fever, chills, and general malaise with sudden onset on the day of admission. Physical examination showed slight rhinitis, hyperemic pharynx with tonsillar and cervical adenopathy. Admission urine showed 1-plus albumin with 4 to 6 red blood cells per high-power field and positive occult blood test. The day after the urine was negative and daily urines until discharge remained negative. There was elevation of temperature for 2 days. On discharge the 15-minute phenosulphonphthalein was 46 percent, and there was ability to concentrate the urine to 1.025.

Series No. 193.—Was admitted complaining of headache, chest cold, and general malaise with onset 1 day before. Physical examination showed rhinitis, hyperemic pharynx with tonsillar adenopathy. Admission urine showed 2-plus albumin with no red blood cells seen. There was a secondary rise of temperature 1 week later with urine showing 2-plus albumin and 1 to 2 red blood cells per high-power field with negative occult blood test. Urines on the following days were negative. This patient returned for tonsillectomy 3 weeks later at which time the urine was negative.

Series No. 197.—Was admitted complaining of fever and headache, with onset the day prior to admission. Physical examination showed rhinitis, hyperemic pharynx with cervical and tonsillar adenopathy. Admission urine showed 3-plus albumin with 1 to 2 red blood cells per high-power field. The urine became negative 2 days later and remained so. There was elevation of temperature for 4 days. Prior to discharge, the 15-minute phenosulphonphthalein was 20 percent, and there was ability to concentrate the urine to 1.024.

Series No. 234.—Was admitted complaining of sore throat and fever, with onset the day prior to admission. Physical examination showed hyperemic pharynx. Admission urine showed a trace of albumin with 2 to 3 red blood cells per high-power field and 1-plus occult blood test. The urine 2 days later was negative and remained so until discharged. There was elevation of temperature for 3 days.

Series No. 235.—Was admitted complaining of headache and general malaise with onset 3 days previously. Physical examination showed slight rhinitis and hyperemic pharynx. Admission urine showed 1-plus albumin with an occasional coarsely granular cast and red blood cells too numerous to count with 4-plus occult blood test. Two days later the urine became negative and remained so until discharged. There was no temperature during hospitalization. There were no symptoms referable to the kidneys. Prior to discharge the 15-minute phenolsulphonphthalein was 16 percent and there was ability to concentrate the urine to 1.027.

Series No. 280.—Was admitted complaining of sore throat and cough of 2-days' duration and increasing severity. Physical examination showed rhinitis, hyperemic pharynx with cervical and tonsillar adenopathy. Admission urine was negative but 2 days later showed plus-minus albumin with rare red blood cells with negative occult blood test. Upon discharge the urine was negative.

Series No. 288.—Was admitted complaining of cough and pain in the chest with onset the day prior to admission. Physical examination showed slight rhinitis. Admission urine showed 3-plus albumin with red blood cells 2 to 3 per high-power field with positive occult blood test. Two days later the urine was negative and remained so until discharged. There was elevation of temperature for 3 days. Before he was discharged from the hospital, 15-minute phenolsulphonphthalein test was 21 percent and there was ability to concentrate the urine to 1.025.

Series No. 313.—Was admitted complaining of chest pain and cough with onset the day previously. Physical examination showed slight rhinitis, tonsils surgically absent, pharynx hyperemic with cervical adenopathy. Admission urine was negative but 5 days later the urine showed plus-minus albumin with rare red blood cells and negative occult blood test. Daily urines continued to show rare red blood cells for 5 days and after that were negative. There was elevation of temperature for 3 days. Prior to discharge, the 15-minute phenolsulphonphthalein was 37 percent and there was ability to concentrate the urine to 1.025.

Series No. 322.—Was admitted complaining of sore throat, with onset the day before. Physical examination showed slight rhinitis, follicular tonsillitis with tonsillar adenopathy. Admission urine was negative but 9 days later the urine showed plus-minus albumin with rare red blood cells and negative occult blood test. All urines after this were negative. There was elevation of temperature for 3 days.

Series No. 324.—Was admitted complaining of sore throat and chest cold of 2-days' duration. Physical examination showed slight rhinitis, hyperemic tonsils and pharynx with cervical and tonsillar adenopathy. Admission urine showed 1-plus albumin with 2 red blood cells per high-power field and faintly positive occult blood test. The urine 4 days later was negative and remained so until discharged. There was elevation of temperature for 2 days.

Series No. 336.—Was admitted complaining of a head cold which had been present for 2 weeks. Physical examination showed rhinitis, surgically absent tonsils, and hyperemic pharynx with cervical and tonsillar adenopathy. Admission urine showed plus-minus albumin with rare red blood cells with negative occult blood test. The following day the urine became negative and remained so up to discharge from the hospital. There was elevation of temperature for 2 days.

Out of 343 cases of acute upper respiratory infection 90 showed albumin and 36 showed hematuria at one time or another as manifestations of renal involvement. All of these cases of hematuria were microscopic. None of these cases showed the edema, smoky urine or clinical picture in any way typical of diffuse glomerulonephritis. The great majority of these cases of hematuria showed evidence of renal involvement in their admission urine, at the time that symptoms and elevation of temperature were most pronounced. All cases had negative urines before being returned to duty and many of these had additional tests of kidney function such as intravenous phenolsulphonphthalein, concentration tests and estimations of blood urea nitrogen which also were negative. After discharge these cases were all under medical supervision and upon any manifestations of complications or sequels would have been returned to the hospital for further treatment. Many of these cases were returned to the hospital for routine tonsillectomy later at which time all urine examinations were negative. In 11 of these cases of hematuria, the tonsils had been previously cleanly removed, but infection, elevation of temperature, signs and symptoms and renal involvement were the same as in the patient not tonsillectomized.

Several of these cases of hematuria have some unusual features which should be commented upon.

Series No. 34.—After convalescing from an upper respiratory infection had an inguinal hernia repaired without a urinary check up just prior to operation. After operation albumin, rare granular casts and slight hematuria were present and lasted for several days. There were no clinical signs of diffuse glomerulonephritis. Undoubtedly, the patient should not have been operated upon at that time and urinary examination prior to operation might have disclosed some abnormal findings.

Series No. 43.—Had an acute rhinitis with no urinary involvement but an exacerbation 8 days later with a sore throat showed some hematuria.

Series No. 48.—Had a diffuse pin point erythematous rash with his upper respiratory infection associatied with hematuria. This patient was carefully observed for scarlet fever, but Schultz-Charlton blanching tests were negative and there was no desquamation.

Series No. 68.—Showed a slight hematuria upon admission. As a complication he developed a mild, acute nonvenereal epididymitis. During this complication his urine was negative and remained so until his discharge from the hospital.

It is believed that these cases of microscopic hematuria which for the most part appeared during an acute upper respiratory infection represent cases of focal nephritis as distinguished from acute diffuse glomerulonephritis. The following references from the literature are quoted:

Boyd [2] mentions a benign hemorrhagic nephritis associated with tonsillar infection, but this was characterized by a clinical gross hematuria.

McCann [3] would classify the cases reported in this paper as glomerulonephritis because of the finding of red blood cells in the urine.

V. Bies [4] mentions that hemorrhagic nephritis is a rare complication of angina faucium, but a frequent complication of scarlet fever. He also mentions that the nephritis of angina occurs at the onset of the primary disease or within a week rather than later as mentioned in most texts.

Geldrich [5] quotes Volard's definition of focal nephritis as an intra-infectious, metastatic inflammatory disseminated injury of the glomeruli.

Lyttle [6] says that renal involvement with upper respiratory infection is less frequent in children who have had their tonsils removed so that this operation may prevent some cases of glomerulonephritis.

Snoke [7] mentions that glomerulonephritis is a serious disease and that after an initial attack the renal lesion may persist for many years. He believes that the best way to check on this is quantitative examinations of properly concentrated urine. Glomerulonephritis cannot be proved to be healed until repeated quantitative examinations of a concentrated urine over a period of at least 1 year have been normal.

[2] Boyd, William: The Pathology of Internal Diseases, 1935, Kimpton, London.
[3] McCann, W. S.: Penna. Med. Journ. 37: 199, Dec. 1933.
[4] V. Bies: Ugesk. f. Laeger, 96: 1, January 1934.
[5] Geldrich: Jahrbuch fur Kinderheilkunde, 141, January 1934.
[6] Lyttle, J. D.: Penna. Med. Journ. 37: 877, Aug. 1934.
[7] Snoke: Am. Journ. of Dis. of Children, 53: 673.

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McElroy [8] observes that diffuse glomerulonephritis, supposedly of toxic origin, as a rule develops 2 to 3 weeks after the occurrence of the causative disease (scarlet fever or angina) while the focal glomerulonephritis, supposedly of infectious origin, develops along with the causative infection. Focal glomerulonephritis begins suddenly and simultaneously with the causative infection. This causes no general symptoms though there may be slight difficulty in voiding or lumbar pain. The distinguishing symptom is the general bloody appearance of the urine. On physical examination no edema is found, the blood pressure is not increased and there is no evidence of cardiac hypertrophy. The urine generally shows a bloody appearance, much albumin due to the blood present, and many white blood cells. Bacteria are often present. The functional kidney tests are usually good and there is usually no retention of urea in the blood. The diagnostic feature of acute focal glomerulonephritis is the presence of hematuria with the absence of increased blood pressure and cardiac hypertrophy. It may sometimes be difficult to differentiate between a subsiding acute, diffuse glomerulonephritis and acute, focal glomerulonephritis. The prognosis of focal nephritis is good.

Fishberg [9] says that focal nephritis is a manifestation of infection which occurs at the height of infection and often streptococci are found in the urine. It is generally due to a streptococcus. Diagnosis is made on the occurrence of a hematuria during the course of acute infectious disease. Impairment of renal function is rare. The diagnosis is often difficult. Prognosis is excellent. Diseased tonsils should be removed after the acute infection has subsided, often the hematuria persists until the tonsils are removed. Differential diagnosis is macroscopic or microscopic hematuria occurring during an acute infection. Edema, hypertension or impairment of renal function mean diffuse nephritis and not the focal type. If hematuria appears after acute infection has subsided, it is more likely to be a diffuse nephritis. Focal nephritis is more closely related to hematogenous abscess of the kidney than to diffuse glomerulonephritis. Fishberg would call it focal glomerulonephritis, due to direct action of the micro-organisms on the renal structures.

In the various classifications of nephritis given by Cecil,[10] the following mention focal nephritis: Volard and Fahr, Elwyn, nonembolic focal glomerulonephritis; Bennett, focal glomerulonephritis; Fishberg, focal glomerulonephritis; Mosenthal, focal infectious glomerulonephritis; Christian mentions hemorrhagic nephritis but does not seem to consider it the same as focal nephritis.

CONCLUSION

Out of 343 cases of acute upper respiratory infection, 36 cases of microscopic hematuria were observed. With this hematuria, there was no associated edema, no hypertension and tests of renal function were essentially normal. In a few cases there was some lowering of the 15-minute phenolsulphonphthalein test. These tests were the only deviation from normal and should probably have been repeated in the few cases where they were below the accepted normal of 25 percent in the first 15 minutes. In none of these cases could a definite diagnosis of diffuse glomerulonephritis be made, and certainly the vast majority of the cases showing hematuria could, without question, be classified as focal nephritis. In a few, it might be necessary to

[8] McElroy: Diseases of Kidney in Tices Practice of Med., W. F. Prior Co.
[9] Fishberg, Arthur M.: Hypertension and Nephritis, Phila. Lea & Febiger, 4 ed. 1939.
[10] Cecil, R. LaF.: Textbook of Medicine, 4th ed. Saunders, 1937.

continue the examinations for a period of 1 year at least to absolutely rule out a mild diffuse glomerulonephritis. All had negative urines upon discharge and up to the present, none have been returned to the hospital for renal complications. The prognosis, as stated in the references in the literature, appears to be excellent.

SUMMARY

1. Of cases of acute upper respiratory infection 10.4 percent showed a microscopic transient hematuria, the average duration of which was 1 to 2 days. The maximum duration was 8 days.

2. Renal involvement as shown by hematuria was present at the height of constitutional involvement and disappeared in most cases very promptly upon subsidence of constitutional symptoms.

3. Such cases need careful clinical observation for edema, hypertension and tests of renal function to differentiate diffuse glomerulonephritis.

4. The majority of these cases if not all could be classified as focal nephritis with an excellent prognosis.

5. Focal nephritis is probably much more common than is supposed with cases of acute upper respiratory infection and its occurrence justifies the prolonged period of convalescence with hospitalization given all these cases of acute upper respiratory infection.

ACTINOMYCOSIS OF LUNGS AND LIVER

REPORT OF CASE

By Commander R. W. Hutchinson, Medical Corps, United States Navy, and Lieutenant J. L. Zundell, Medical Corps, United States Navy

This case is reported because of the relative rarity of the disease, the unusual combination of organs involved, and because it presented a diagnostic problem which remained unsolved up to the time of its fatal termination.

Actinomycosis is a chronic suppurative disease produced by a number of species of the genus *Actinomyces*. *Actinomyces bovis* is apparently the most common offender in man as well as in cattle. It predominantly attacks males in the third and fourth decades of life. The essential lesion is the so-called mycetone consisting of an area of central necrosis surrounded by a zone densely infiltrated by neutrophilic leucocytes, wandering histiocytes, newly formed blood vessels and varying amounts of fibrous stroma. The ray fungus can be found both in the liquefied pus and in the abscess wall. Extension is believed to be due to wandering of macrophages which have engulfed the organism. Erosion of blood vessels may result in pyemia and widespread metastases. Four regions of the body are predominantly

involved: (*a*) Head and neck; (*b*) abdomen; (*c*) thorax; and (*d*) skin. The organism gains access to the body by inhalation or ingestion, or through puncture wounds of the skin or mucous membranes. According to Lord, *Actinomyces* can live as saprophytes in the mouth and at a favorable time assume pathogenic qualities, invading neighboring tissues.

Case Report

Case 774-38.—G. A. M., white, married male, 52 years old, Veterans' Administration patient, occupation, lineman for an elevated railroad, was admitted to the Naval Hospital, Chelsea, Mass., on June 14, 1938, for treatment of pleuritis.

Chief complaints.—Pain in left shoulder and entire left side of chest 1½ weeks; pain left upper quadrant of abdomen 1½ weeks; anorexia and loss of 45 pounds weight the past 6 months; nocturia 3–4 times the past 6 months; night sweats.

Present illness.—In November 1937 he caught cold and developed pain in the right side of his chest which was made worse by coughing and deep breathing. He returned to work before recovery was complete and had a recurrence of these symptoms. He improved then under the care of his physician but has since had no appetite and has lost 45 pounds weight in the last 6 months. He continued to work although he had some pain in his left chest and frequent night sweats and moderate cough for the last 6 months. During the last month, his sputum was blood tinged on occasions. About 1 week prior to admission, the pain spread from his left chest to his left shoulder and became very severe. His bowels were irregular for the last 6 months and he had been taking laxatives frequently. Considerable pain occurred in the left upper quadrant while straining to defecate. Ingestion of excessive quantities of water probably accounted for the nocturia and polyuria.

Past history.—Patient was born in Massachusetts in 1886. He had the usual childhood diseases with good recovery and had influenza and scarlet fever a few years ago.

Family history.—Father died aged 79 years from "shock and kidney trouble." Mother died aged 76 years from "heart trouble." Two brothers and five sisters are living and well. Two brothers died, one of typhoid fever, and one of cause unknown. No history of cancer or tuberculosis.

Marital.—Wife living and well. Two children living and well.

Physical examination.—Patient is a well-developed and moderately obese male, 52 years of age. He is mentally clear and cooperative. Temperature 98.8°; pulse, 96; respiration 21; blood pressure, 135/75; weight, 185 pounds; height, 68 inches.

Head: No abnormalities.

Eyes: Pupils round, regular and equal. React normally.

Nose: No obstruction.

Mouth: Tonsils moderately inflamed. Teeth in fair condition.

Neck: Thyroid not enlarged. No palpable adenopathy.

Chest: Symmetrical.

Lungs: Excursions limited on left side. Area of dullness with decreased fremitus and decreased breath sounds over the left base posteriorly.

Heart: Size, shape, and sounds normal. No murmurs.

Abdomen: Marked tenderness under left costal margin in the anterior axillary line. Pressure here caused pain in the left shoulder also.

Extremities and reflexes: Normal, no tenderness in left shoulder.

Clinical course.—June 20, 1938, WBC, 19,100; segmented, 68; bands, 11; lymphs, 18; and monos, 3. Temperature has varied from normal to 101.2°. Urine

contains trace of albumin. Pain has subsided somewhat with baking and salicylates.

June 27: Temperature approaching normal. Blood Kahn negative. Sedimentation rate 28 mm. Very little pain in chest or shoulder.

July 5: Left chest tapped. No fluid.

July 11: Left chest tapped. Needle seemed to strike a firm mass. No fluid.

July 18: RBC, 3,410,000; Hgb, 75; WBC, 18,600; segmented 54; bands 11; monos, 2; eosins, 1. No acid fast bacillis in sputum, predominating organism being a grampositive diplococcus.

July 21: Has developed a firm small subcutaneous mass in left posterior axillary line which seems to be attached to the eighth rib.

July 22: Mass in left chest incised and contained thick yellow pus. Abscess cavity had a thick wall. Smear and culture of pus failed to reveal any organism.

August 15: Continues to run a low-grade fever. WBC, 27,000 with a shift to the left. There is still dullness, with absent breath sounds at the left base. Little cough or expectoration. No pain in chest or abdomen. Incised wound in left chest drains thin yellow pus.

August 16: Tapped in ninth left interspace post. No fluid.

August 20: Tapped in tenth left interspace post. No fluid.

August 28: Began complaining of severe pain in right upper quadrant. Definite tenderness and rigidity in right upper quadrant over gall bladder area.

August 29: Laparotomy performed. Three masses noted on liver which appear to be early abscesses. No fluid aspirated from masses.

September 16: Liver abscess aspirated 25 cc. of thick pus removed. Culture negative.

September 23: Developed abscess on right elbow. Pus aspirated and smear and culture are negative. WBC, 23,000. No fungi present in pus.

October 17: Pus continues to drain from right elbow. Culture and smear continue to be negative. Patient rapidly losing weight.

November 8: Specimen of sputum negative for fungi.

November 12: Guinea pig inoculated with pus from right elbow, but on December 10 showed no pathology. Blood culture negative.

December 29: Patient became progressively worse and toward the last was irrational and very dyspneic. He expired at 9:45 a. m.

Throughout his illness his temperature varied from normal to 101–102° and he ran a leukocytosis with a shift to the left. Blood cultures were consistently negative and sputa were negative for fungi on several occasions. Kahn negative.

Pathology.—Autopsy revealed numerous small abscesses measuring up to 2 cm. in diameter and containing thick green oily pus throughout all lobes of both lungs. The lungs were densely adherent to chest wall by fibrous adhesions. The abscess walls were unusually thick measuring up to 1 cm. and having the appearance of malignant tissue. There was a sinus tract in the ninth left interspace at the site of the repeated thoracenteses. The liver contained two large abscesses in the right lobe, the largest measuring 15 cm. diameter, filled with thick green oily pus which when washed out left a honeycombed cavity crossed by numerous white septal bands. The wall of the abscess was yellowish gray, measured 3 cm. in thickness and grossly resembled a neoplastic process rather than a live abscess. The rest of the autopsy findings revealed nothing remarkable. Histopathological examination of the lungs and liver revealed typical ray fungi in the walls of the abscesses.

DISCUSSION

From a clinical standpoint, several features make this case unusual and interesting. No satisfactory history of exposure or contact with animals, grain, or any other host of this fungus was elicited. The organism was never found in the pus from any of the lesions on repeated examination. Actinomycosis infection is sufficiently rare that it does not appear in the foreground as a probable diagnosis. Actinomycosis involving the lungs and liver is particularly rare.

The typical picture of a pulmonary actinomycosis (1) is that of a veillike shadow obscuring the base of one lung probably due to a peripleuritis. There is no fluid curve present and the lung is visible through this shadow. The chest wall is somewhat contracted with drawing together of the ribs and narrowing of the pulmonary field. The shadow is uniform but usually more opaque toward the axilla and fades away toward the mediastinum. This bizarre picture has the appearance of atelectasis without displacement of the mediastinum and is pathognomonic of peripleuritis of which actinomycosis is the commonest cause. Involvement of the ribs usually occurs but is not readily demonstrated. A study of the radiograph of the chest taken soon after the patient was admitted to this hospital (fig. 4) illustrates these features and is quite characteristic of this disease.

From the roentgenological viewpoint, the case departs from the characteristic picture in that the later radiographs of the chest (fig. 5) present an appearance not usually seen in this condition. A marked change has taken place throughout both lung fields. There are present many shadows which suggest infiltration involving both lungs throughout. These shadows are not characteristic enough to make their etiology clear. They do not have the appearance of a tuberculous infiltration exactly nor do they resemble any carcinomatous metastases. They are widespread and so suggest a blood-stream dissemination. A review of the literature fails to show any such picture in the actinomycosis cases presented.

The autopsy report explains these shadows. The many shadows are due to multiple abscess formation. All of those opened were found to be full of pus and were not of any very great size.

A case of actinomycosis of the lung such as here presented produces an early characteristic picture of a peripleuritis at the base of one lung with narrowing of the lung field but without the displacement of the mediastinum seen in atelectasis. Widespread dissemination of the disease in the lungs may lead to the formation of multiple miliary abscesses which somewhat resemble carcinoma metastases and tuberculosis.

Two other cases of actinomycosis, identical in the early stages, were studied at a large civilian hospital and were found to have a veillike shadow at the base of one lung exactly like our case (2).

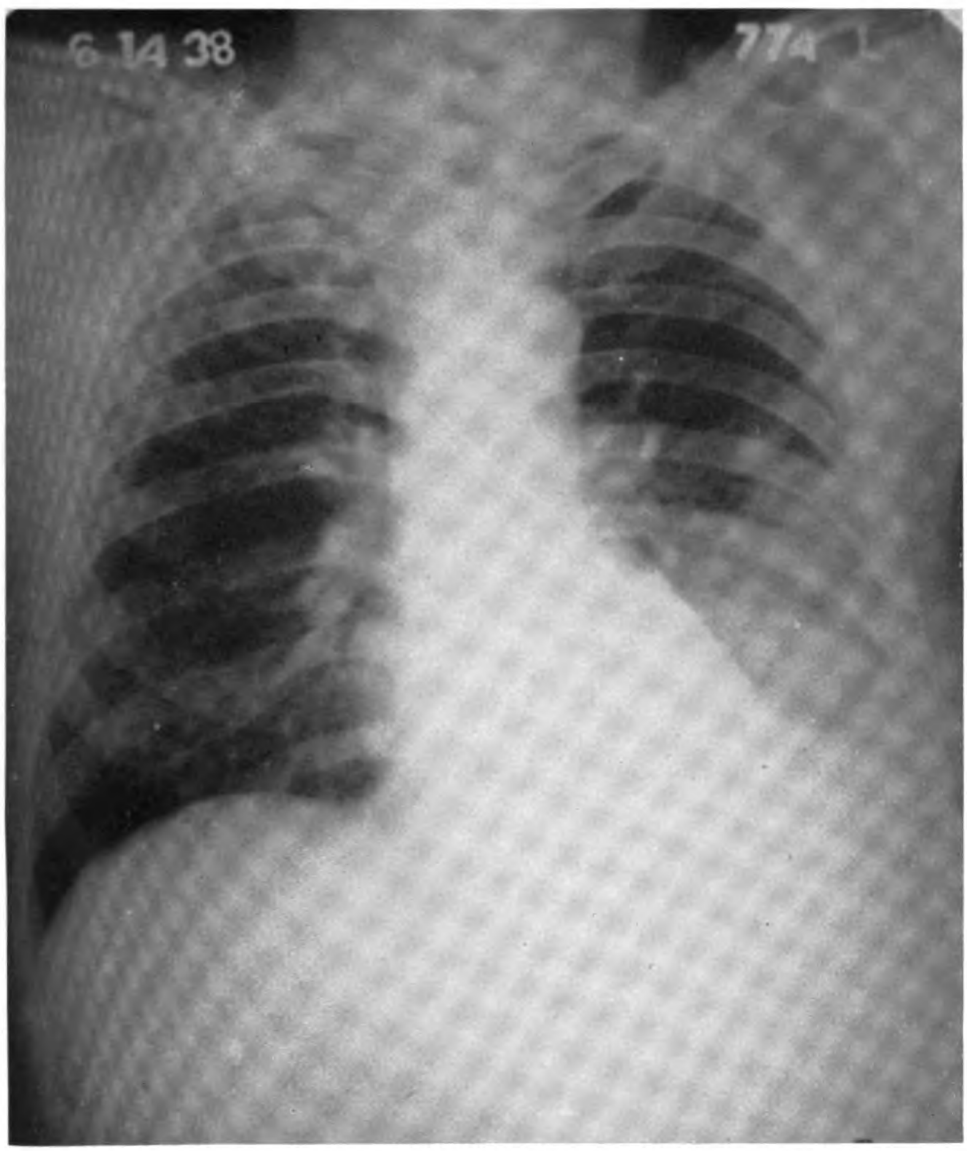

FIGURE 4.—CHEST ROENTGENOGRAM TAKEN ON ADMISSION ILLUSTRATING CHARACTERISTIC FEATURES OF ACTINOMYCOSIS.

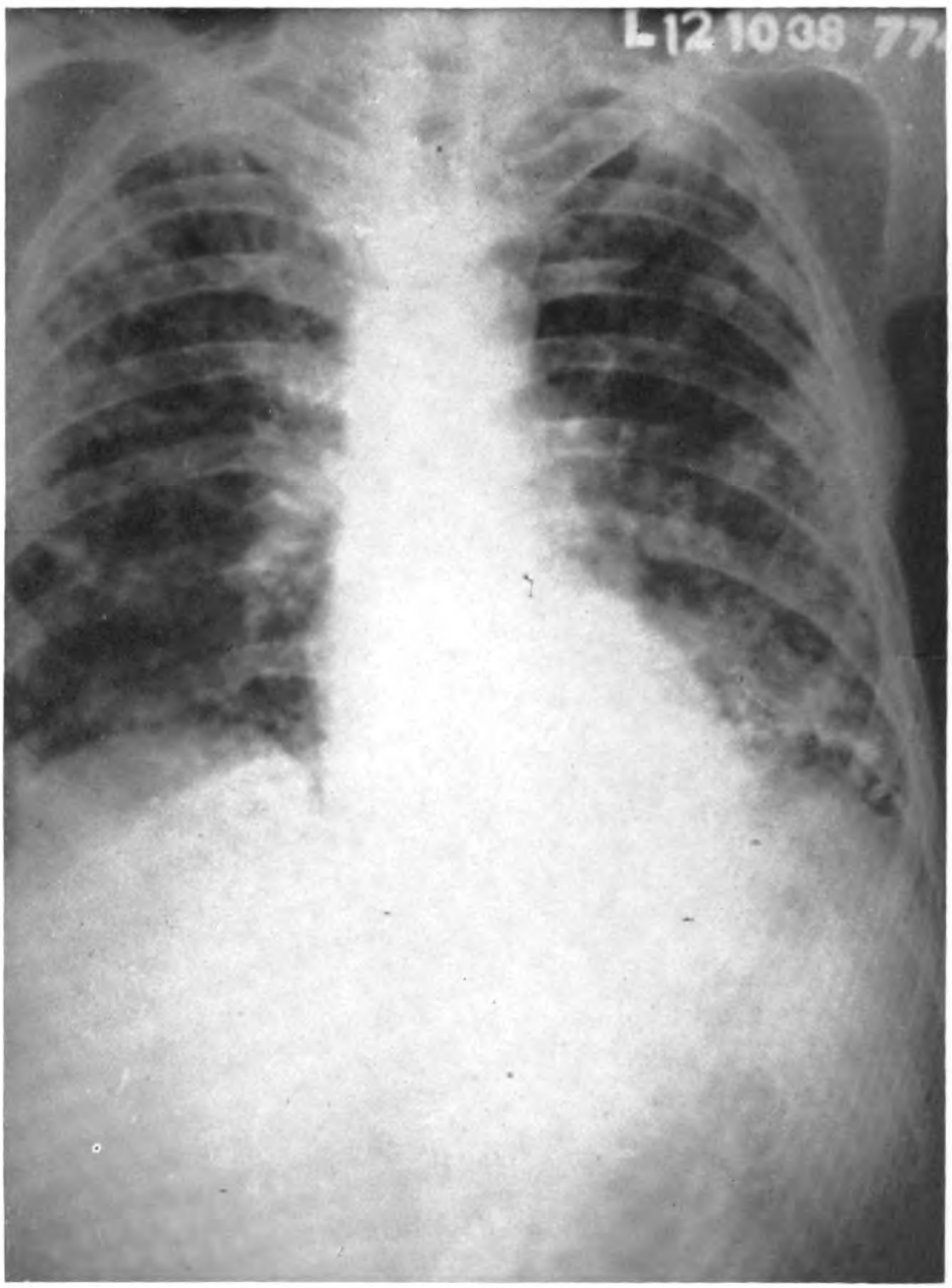

FIGURE 5.—CHEST ROENTGENOGRAM, TAKEN AT A LATER PERIOD, ILLUSTRATING UNUSUAL FINDINGS.

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CARCINOMA OF THE LUNG [1]

A CLINICAL STUDY

By Lieutenant (Jr. Gr.) Justin J. Stein, Medical Corps, United States Naval Reserve

Sufficient data, both clinical and pathological, have been obtained in recent years to justify the conclusion that there is an actual increase in carcinoma of the lung.

The incidence of primary carcinoma of the lung has been reported from 2.6 percent to as high as 19.3 percent of all carcinomas. Moersch and Bowing [2] state that few cases were seen at the Mayo Clinic prior to 1925. However, from 1925 to 1935 approximately 250 cases were diagnosed. Koletsky [3] reported that in the period from 1927 to 1937, at the Cleveland City Hospital, 1,064 cases of malignant neoplasms of all types were found and that of this number, 9.4 percent were primary carcinomas of the lung. Mattick [4] in 1938 reported diagnosis of only 4 primary carcinomas of the lung between 1917 and 1921 while 48 were diagnosed between 1932 and 1936. A study of the incidence of this type of tumor at the Hines Hospital reveals that 7 cases were seen in 1931 representing 1.7 percent of carcinomas of the lung as compared with all carcinomas; by contrast, 57 cases or 7.3 percent were seen in 1937. Although such factors as the more general use of bronchoscopy and other diagnostic aids and the present active tumor program may account for some of this increase, there is apparently a definite increase as well.

SEX, AGE, AND RACE

A review of 768 cases reported by various authors from the literature shows that 85 percent of the cases occur in males and 15 percent in females.

The majority of cases occur in the age-interval between 40 and 60 years. Approximately 60 percent or more of all cases reported are found in this age group. Only 9 percent of the 135 cases reported by Arkin and Wagner [5] were from 21 to 40 years of age. In a series of 140

[1] From the tumor and surgical service, Edward Hines Jr. Hospital, Hines, Ill. Published with the permission of the Medical Director of the Veterans' Administration, who assumes no responsibility for the opinions expressed or the conclusions drawn by the author.

[2] Moersch, H. J. and Bowing, H.H.. Primary carcinoma of the bronchus treated successfully with surgical diathermy, Ann. Surg. 102: 989, Dec. 1935.

[3] Koletsky, Simon: Primary carcinoma of the lung, Arch. Int. Med. 62: 636, Oct. 1938.

[4] Mattick, W. L.: Primary bronchus carcinoma, radiology, 30: 471, June 1938.

[5] Arkin, A., and Wagner, D. H.: Primary carcinoma of the lung, J. A. M. A. 106: 587, Feb. 1936.

cases reported by Vinson [6] the youngest was 20 and the oldest 75 years of age.

There has been no evidence submitted as yet to indicate that the incidence is higher in the colored race. Practically all reports concerning this show the same percentage of cases occurring in the colored race corresponding to the ratio of colored to white in that particular locality.

SYMPTOMS

The majority of articles list cough as the first symptom followed shortly afterward by chest pain and hemoptysis. Generally speaking, the hemoptysis is not marked and consists of blood streaked sputum. In a series of 164 cases recently reported by Stein and Joslin [7] the following symptoms were noted in the order as listed: Cough, loss of weight, chest pain, dyspnea, weakness, expectoration with or without hemoptysis, gastro-intestinal discomfort, hoarseness, night sweats, anorexia, heart palpitation, dysphagia, orthopnea, and pain other than in chest. Loss of weight is a late symptom and occurs in the majority of cases. The three most outstanding early symptoms are, first, cough with or without expectoration, second, chest pain, and third, dyspnea. Anorexia and night sweats occur in only a small percent of the cases and late in the disease as compared with tuberculosis in which condition they are present relatively early and in a large percentage of the cases.

All symptoms produced are the result of complications. The obstruction of the bronchus will cause atelectasis, which in turn causes pain. In the atelectatic part of the lung infection may develop such as bronchiectasis and abscess formation. If a blood vessel is injured in the bronchial mucosa as a result of ulceration, hemorrhage or blood streaked sputum will result. The inflammatory condition about the tumor will cause chills, fever, and leukocytosis in many cases.

In most cases the onset of symptoms is usually insidious. In approximately one-third of our cases the onset was described as being a "heavy cold," "flu," "pleurisy," "chills and fever," or as "pneumonia." Therefore, fully one-third of the cases will describe the onset similar to that of an acute respiratory infection. By contrast, in many cases, the tuberculous patient consults the physician because of a "generally run-down" feeling, in addition to anorexia, and fatigue on slight exertion. In four of our cases there was no history referable to the chest.

All individuals 35 or more years of age presenting complaints referable to the chest should be thoroughly examined in order to rule out a primary carcinoma of the lung.

[6] Vinson, P. P.: Primary malignant disease of tracheobronchial tree, J. A. M. A. **107**: 258, July 1936.

[7] Stein, J. J., and Joslin, H. L.: Carcinoma of the bronchus, Surg. Gynec. and Obst. **66**: 902, May 1938.

PATHOLOGICAL AND HISTOLOGICAL FINDINGS

The tumors may be classified pathologically as follows: (*a*) hilus or main bronchus type, circumscribed or diffuse, (*b*) smaller bronchi, circumscribed or diffuse, (*c*) diffuse type in which the point of origin cannot be determined, and (*d*) the so-called superior pulmonary sulcus type.

Approximately 70 percent of the cases occur in the right lung. There has been no satisfactory explanation to account for the greater number of cases on the right side. Also more than 75 percent involve a first order bronchus. Occasionally a case is seen which involves the trachea, carina, and first-order bronchus. A very small percentage of the cases are so grossly diffuse that it is difficult to determine the point of origin in the bronchus. Statistics vary as to the lobes involved. In some series the upper lobe will be more commonly involved than the lower lobe. Only a few cases are reported as occurring in the right middle lobe.

All carcinomas of the lung probably take their origin from the bronchial epithelium. The mucous glands in the wall of the bronchi apparently arise from the bronchial epithelium. The cells of the less differentiated basal layer of the bronchial epithelium have the capacity to produce squamous epithelium, columnar epithelium, or undifferentiated round or spindle cells. In the series of cases reported by Vinson [6] 76 were squamous-cell carcinomas, 59 were adenocarcinomas, 3 were of the undifferentiated carcinomas type, and 2 cases were not accurately classified. The 74 necropsy cases reported by Arkin and Wagner [5] were as follows: 21 were adenocarcinoma, 18 were squamous-cell carcinoma, and 31 were undifferentiated round or spindle-cell carcinomas. In the 100 autopsy cases reported by Koletsky [3] there were 35 small-cell carcinomas, 40 squamous-cell carcinomas, 22 adenocarcinomas, and 3 tumors which were classified as carcinomas, simplex

METASTATIC LESIONS

The most common sites of metastases to organs from carcinoma of the lung are liver, adrenals, contralateral lung, kidney, pleura, brain, heart or pericardium, and bones. In the lymphatic system the tracheobronchial, retroperitoneal, cervical, and the mesenteric nodes are the ones most commonly involved. Because of the frequency of metastasis to the brain, all patients having intracranial symptoms should have a chest roentgen film. The bones most frequently involved are ribs, vertebrae, skull, pelvis, and the femur.

SO-CALLED SUPERIOR PULMONARY SULCUS TUMORS

Many articles have appeared in recent months regarding the sulcus tumors which were first described by Pancoast in 1924 [8] and again in 1932.[9] The characteristic syndrome produced by tumors of this type is unilateral pain in the shoulder girdle radiating down the upper extremity, Horner's syndrome, weakness of the muscles of the upper extremity, homogeneous shadow of increased density situated in the extreme apex of the lung seen in the roentgenographic film. Also evidence of rib destruction and often infiltration into the adjacent vertebrae is noted. Pancoast was of the opinion that this was a special type of tumor and that it probably did not take origin from the lungs, pleura, ribs, or mediastinum.

The preponderance of evidence has shown that the majority of malignant tumors located in the region of the thoracic inlet are carcinomas of the terminal bronchioles of the lung. The signs and symptoms produced are the result of pressure on or infiltration of the inferior cervical sympathetics and the brachial plexus on the involved side. This author [10] recently reported 15 so-called superior pulmonary sulcus tumors in 14 of which the site of origin was definitely considered to be the terminal bronchioles of the lung. This was histologically confirmed in 12 of the 13 cases in which biopsy material was available. Six were squamous-cell carcinomas, four adenocarcinomas, one osteogenic sarcoma (metastatic from the thigh), and one undifferentiated carcinoma with areas simulating an adenocarcinoma. Because of the symptoms, patients having apical lung tumors are generally given a diagnosis of tuberculosis, neuritis, or arthritis before a tumor is suspected. Any tumor, whether primary or metastatic, located in the thoracid inlet and pulmonary apex may produce the characteristic symptoms and signs of a Pancoast tumor solely because of its anatomic location. A series of 47 cases of this type from the literature reveals 45, or 95 percent, to be present in males.

ROENTGENOLOGICAL FINDINGS

In the majority of cases a definite diagnosis can be made from the roentgen examination alone. However, wherever feasible, bronchoscopic examination should be done unless the tumor is of the apical type. The typical roentgenographic findings are that of a unilateral density in the hilum with its apex directed toward the periphery of the lung. If the bronchus is stenosed, only the shadow of the atelectatic lung may be seen. The lateral border of the density may blend in with the veil-like shadow of the atelectatic lung. The ribs are

[8] Pancoast, H. K.: Importance of careful roentgen-ray investigations of apical chest tumors, J. A. M. A. 83: 1407, Nov. 1924.

[9] Pancoast, H. K.: Superior pulmonary sulcus tumor, J. A. M. A. 99: 1391 Oct. 1932.

[10] Stein, Justin J.: Apical lung tumors, J. A. M. A. 111: 1612, Oct. 1938.

narrowed on the affected side, the trachea pulled to that side, and the diaphragm elevated. Enlarged mediastinal lymph nodes should be looked for and also the presence of metastasis in the ribs, spine, or bones of the shoulder girdle. Radiating strand-like shadows extending from the root in a fan-like manner toward the periphery of the lung is characteristic. Soon after the atelectasis has occurred, evidences of infection can be seen about the tumor with the exception of the apical lung tumors. After the injection of iodized oil, irregularity of the bronchial wall may be noted in both the stenosing and non-stenosing types. Tumors occurring in the smaller bronchi may have the appearance of an unresolved pneumonia. A regular postero-anterior roentgen film, a Potter-Bucky postero-anterior film, and a lateral film should be made in all cases of suspected carcinoma of the lung, in order that the lesion may be properly studied. Metastatic lesions in the lung seldom cause pulmonary symptoms, are usually multiple, rarely cause bronchostenosis, and are not usually associated with secondary infection.

ASSOCIATION OF TUBERCULOSIS AND TUMOR

In a very small percentage of cases both tuberculosis and carcinoma will be found in the same lung. However, carcinoma formation from the metaplastic epithelium of an old tuberculous cavity is rarely found. It is believed the presence of the two different types of lesions is merely a coincidence.

TREATMENT

The majority of cases when seen are inoperable and are therefore generally treated by irradiation. Irradiation therapy will produce a palliative effect, such as reduction of cough, lessening of pulmonary infections about the tumor, and general clinical improvement with prolongation of life. Following irradiation the infection about the tumor is markedly diminished and the clinical improvement is probably explained on this basis. In none of our cases was there any marked diminution in the size of the tumor following irradiation. It is difficult to believe that primary carcinoma of the lung can be cured by irradiation since the tumors are of epithelial structure, deeply situated, and surrounded by lung tissue. If large doses of irradiation are given the danger of pneumonitis is always present, which may be fatal. In the cases of so-called superior pulmonary sulcus tumors which we have treated by irradiation, there has been no relief of symptoms produced. As has been previously mentioned, these tumors are not secondarily infected.

Regarding the question of pulmonary fibrosis following irradiation of the lung, we have given up to 5,000 and 6,000 roentgen units in

fractionated doses through two or three portals in many cases without producing pulmonary fibrosis. At post mortem many of the irradiated lungs have been examined and no appreciable evidence of fibrosis noted. If, however, heavy doses are given through one or two portals, it is quite possible that fibrosis may be produced. If the patient complains of increase in cough or expectorates blood while receiving irradiation, the treatments are discontinued because of the danger of producing a pneumonitis.

At the present time surgery offers the only hope for a cure. Surgical extirpation of a centrally located growth must include removal of the entire lung and hilum, as well as the adjacent mediastinal lymph nodes. Lobectomy may be sufficient if the lesion is peripherally located. In spite of the high operative mortality, cases which are considered to be operable should be explored since total extirpation offers the best chance for a permanent cure. It is unfortunate that so few of the cases are operable when seen by the physician.

The so-called superior pulmonary sulcus type of tumor is usually inoperable even when the lesion appears to be quite small. These tumors apparently invade the mediastinum and brachial plexus at an early stage in their development.

SUMMARY

1. There is a definite increase in the incidence of primary carcinoma of the lung.

2. Approximately 85 percent of the cases occur in males and more than 60 percent of the cases between the ages 40 to 60. Ninety-five percent of the so-called superior pulmonary sulcus tumors occur in males.

3. All individuals 35 or more years of age presenting complaints referable to the chest should be thoroughly examined in order to rule out a primary carcinoma of the lung.

4. The majority of cases can be diagnosed from the roentgenographic examination.

5. Although definite palliative results are obtained by irradiation in these cases, surgical extirpation of the growth offers the only hope for a permanent cure.

TUMORS OF THE VERMIFORM APPENDIX

By Lieutenant Clifford F. Storey, Medical Corps, United States Navy

INTRODUCTION

Primary tumors of the vermiform appendix, although uncommon, are not as rare as they were once thought to be. The apparent increase in incidence is no doubt due in large measure to the increasing

frequency of careful routine microscopic tissue studies of operative and autopsy specimens. Most of these tumors are benign. Primary malignancy of the appendix is rare. A fairly wide variety of primary new growths, including carcinoids, adenocarcinoma, mucoceles, sarcomas, polyps, fibromas, myomas, myxomas, hemangiomas, neuromas, and possibly others have been observed in the appendix and reported in the medical literature.

Malignant metastatic tumors, infrequent in the appendix, usually represent direct extension by continuity or contiguity from adjacent structures, especially the cecum or ileocecal valve (see case 3) and rarely from distant locations as illustrated by case 4 reported herein which originated in the lung.

In common with other viscera, the appendix is also susceptible to involvement by various specific granulomas, such as tuberculosis and syphilis. While not neoplasms in the strict sense, these lesions do give rise to tumors of the appendix, hence they rate at least a mention in any consideration of this subject.

Rosenblatt and Robertson,[1] Montgomery and Johnson,[2] and others have emphasized the confusion and statistical inaccuracy arising from the failure of many authors to make a distinct differentiation between true gelatinous adenocarcinoma of the appendix and the benign so-called "carcinoid" lesion. The fact that the two conditions represent separate and distinct clinico-pathological entities is now widely accepted, but some authors [3 4 5] apparently continue to regard them as different manifestations of the same pathological process. A further confusing factor is the simultaneous occurrence of both lesions in the same appendix, as was doubtless the situation in the case cited by Neuman.[4]

Appendiceal tumors usually give rise to few or no symptoms and those which are most frequently present ordinarily fall into that vague and indefinite group of complaints commonly attributed to chronic appendicitis. They occasionally present the symptoms of subacute abdominal disease, at times associated with the findings suggestive of partial, rarely merging into those of complete, intestinal obstruction. It is most exceptional for the patient to exhibit the characteristic picture of acute appendicitis, and perforation of the appendix in cases with tumors of that organ has been reported with extreme infrequency. Usually these lesions are unexpectedly discovered during operative or autopsy procedures, or, even more frequently, their presence is not

[1] Rosenblatt, M. D., and Robertson, T. D.: Carcinoid and carcinoma of appendix. Northwest Medicine, 35: 103, Mar. 1936.

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suspected until it is revealed in the course of routine histopathological tissue examinations.

It is the purpose of this paper to review briefly the subject of tumors of the appendix and to add to the literature six previously unpublished cases illustrative of different varieties of these interesting lesions. One of these cases is considered of special interest because of the wide variation in its clinical behavior from that usually described for similar tumors (case 1).

HISTORICAL

Carcinoma of the appendix, according to Eltinge,[6] was first observed by Merling, who reported a case in 1838, followed in order by Prus (1 case, 1855); Rokitansky (4 cases, 1867); Kalaczed (1 case, 1875); Leichenstern (3 cases, 1876); and Berhoff (1 case,1880). Berger,[7] however, is generally credited with having reported the first authenticated case in the year of 1882. By 1906, 68 years after the first report and 24 years following publication of the first verified case, only 42 such lesions had been cited in the literature and of these 28 were by American authors. Since that time, routine pathological tissue studies have become a more common practice and the number of reported cases has increased proportionately so that by 1931, 360 cases were recorded in the literature. The Cumulative Index Medicus lists numerous reports under the heading of cancer of the appendix since that time, but without a careful and detailed study of each individual case report it is impossible to state with certainty how many of these cases represent true carcinoma and how many were carcinoids. Such an investigation of a number of these articles shows that at least 9 out of 10 cases reported under the heading of cancer of the appendix are in reality carcinoids and makes it apparent that the lack of a precise distinction between the 2 tumors has fostered a growing misconception as to the frequency of genuine carcinoma in this site.

CARCINOIDS

Carcinoids of the appendix, also called benign cancer, schirrous or basal cell cancer, pseudocarcinoma, chronic hyperplastic lymphangitis, and argentaffine or chromaffin tumor, has been thoroughly discussed by St. George,[8] who describes the work which led up to the recognition of their origin and histological classification. He cites the work of Oberndoefer,[9] who in 1907 suggested the term *carcinoid* to identify certain appendiceal neoplasms, an apt designation which

[6] Eltinge, A. W.: Primary carcinoma of vermiform appendix. Ann. Surg. 37: 549, 1903.

[7] Berger: Quoted by Selinger (19) and others.

[8] St. George, A. V.: Carcinoids of appendix. Am. J. Clin. Path. 4: 297, May 1934.

[9] Oberndoefer, S.: Handbuch der Speziellen Pathologischen Anatomis und Histologie, F. Henke und O. Lubarsch, Verlag von Juliun Springer, Berlin, 1929. Band iv, Teil 3, pp. 905–911. Quoted by Norment (5), Waugh and Findley (29) and others.

signifies their similarity to carcinomas yet differentiates it from them. Gossett and Masson,[10] using silver impregnation methods, studied these tumors and concluded that they represent hyperplasias of certain cells of a chromaffin nature. Masson[11] noted a connection between these cells and fibers from the plexus of Meissner and advanced the theory that they were of neurogenic origin. This theory was proved by Danish,[12] who showed by exacting studies that these chromaffin cells reduced ammoniacal silver and arose in the primary anlage of the sympathetic ganglia from undifferentiated sympathetic embryonal elements. Using various technics, he established the fact that these cells, rich in lipoids, represent a hyperplasia or possibly a regeneration following inflammation of the argentaffine cells normally found in the appendix and its histological counterparts elsewhere in the gastro-intestinal tract. These silver reducing cells (Kultschitsky's cells) are normally present in the depths of the crypts of Leiberkuhn's glands. Danish's findings have been amply confirmed by St. George,[8] Hasegawa,[13] Forbus,[14] Norment,[5] and others yet there remains some difference of opinion as to the mode of origin of these tumors. Cooke,[15] summarizing the evidence presented up to 1931, concluded that there are a number of unproved theories in regard to the histogenesis of carcinoid tumors and that as yet a final opinion has not yet been reached. Collins et al.[16] agree with this conclusion and have recently summarized the various theories as to the origin of gastro-intestinal carcinoids. As Masson[11] states, these cells apparently pile up in the nerve fibers, finally rupture their sheaths and infiltrate the tissues of the mucosa, then that of the submucosa, and may even invade the muscularis. Grossly the resultant tumor is small, ordinarily so minute, in fact, that the tumefaction is frequently not macroscopically evident. There is characteristically an orange or yellow ring at the base of the submucosa due to the high lipoid content of the tumor cells. The appendiceal lumen is almost invariably obliterated, but the overlying mucosa is rarely if ever ulcerated. The lesion may occur singly or in multiples. Microscopically, groups or nests of rounded, spheroidal, or polygonal cells are seen, frequently being arranged in strands or bundles which are separated by bands of fibrous and muscular stroma. The nuclei are hyperchromatic but there is no marked variation in the size or shape of the individual cells and mitotic figures are rare. The cytoplasm contains acidophilic granules with a marked ability to reduce silver salts. True anaplastic ele-

[10] Gosset and Masson: Quoted by Norment (5).
[11] Masson P.: Significance of muscular "stroma" of argentaffin tumors. Am. J. Path. 6: 499, Sept. 1930.
[12] Danish. Quoted by St. George (8).
[13] Hasegawa. Quoted by St. George (8).
[14] Forbus. Quoted by St. George (8).
[15] Cooke, H. H.: Carcinoid tumors of small intestine. Arch. Surg. 22: 568, Apr. 1931
[16] Collins, D. C., Collins, F. K., and Andrews, V. L.: Ulcerating carcinoid tumor of Meckel's diverticulum. Am. J. Surg. 40: 454, May 1938.

ments are not present. (See fig. 9.) Approximately 90 percent of these tumors are located at or near the appendiceal tip and this portion of the organ may present a grossly bulbous appearance. Based on their histological picture and growth behavior, these tumors must be considered benign. Metastasis probably never occurs in the true sense. Recurrence after removal is unusual and when it does occur, is commonly of local character only. Ewing [3] states that while other gastro-intestinal carcinoids are frequently malignant, those of the appendix are practically always benign. In the words of Boyd,[17] this tumor has never caused the death of a patient. Reports of recurrences or distant spread can probably be explained on the basis of the original presence of a primary mixed lesion or the failure to distinguish between adenocarcinoma and carcinoid.

ADENOCARCINOMA

In contradistinction to carcinoids, true gelatinous adenocarcinoma of the appendix is a genuinely malignant lesion, exhibiting all the characteristics of malignancy shown by cancer occurring elsewhere in the body. Its location is usually at the base of the appendix and Masson [11] feels that it finds its origin, not really in the appendix, but in the adjacent cecum or ileocecal valve. Commonly quite small, these tumors may attain considerable size. One reported case attained the size of a grapefruit and several cases on record have been sufficiently large to give rise to obstructive symptoms. Microscopically the picture simulates that of other gastro-intestinal cancer, particularly carcinoma of the cecum. Neoplastic epithelial cells of the cylindrical, columnar, or goblet type showing considerable variation in size and shape, hyperchromatic nuclei and fairly numerous mitotic figures comprise the main tumor mass. The cells are arranged in more or less adenomatous form but the gland like structures are commonly extremely ill-defined. (See figs. 6, 7, and 8.) Widespread mucoid degeneration accompanies these lesions accounting for the gelatinous character of the tumors. (See figs. 7 and 8.) All coats of the appendix may be invaded. Ulceration of the mucosa is a common finding and even the serosa may be similarly affected. It is usually stated that these cancers are of low malignancy with metastasis late and rarely extensive but cases leading to generalized carcinomatosis have been cited and extremely rapid spread can take place. (See case 1.)

INCIDENCE: Due to the confusion regarding the differentiation of appendiceal carcinomata and carcinoids, or rather the failure to distinguish between them, statistics relative to the incidence of either or both are bound to be misleading. Norment [5] studied 45,000 appendices histologically and found 67 lesions which he designated as car-

[17] Boyd, Wm.: Surgical Pathology. Second edi. W. B. Saunders & Co., Phila., Pa. 1929, pp. 378-381.

cinoma. However, it appears probable that 7 of his cases were true adenocarcinoma and the remainder carcinoids. If this assumption be correct, his studies would reveal an incidence of carcinoma of 0.00156 percent of appendices examined and of carcinoid, 0.0135 percent. St. George [8] reports 7 carcinoids encountered in 9,108 appendices examined at Bellevue Hospital between 1916 and 1932, an incidence of 0.077 percent. Two carcinoids were found in 18,700 autopsy specimens. He admits that probably more would have been discovered had a more painstaking search been made. Gnassi [18] reports 5 carcinoids among 4,224 appendices. Selinger [19] examined 45,302 specimens at the Postgraduate Hospital, New York City and found 34 examples of malignant disease of the appendix, an incidence of 0.075 percent of all specimens, but 0.35 percent of appendices examined. Reiman [20] reported 13,151 surgical appendices of which 17, or about 0.13 percent, were reported as cancerous. In another series of 8,038 appendices studied microscopically 17, or about 0.49 percent, were diagnosed as malignant. MacCarty and McGrath [21] examined 5,000 appendices and reported 1 in 225 carcinomatous. McWilliams [22], on the other hand, studied 15,481 tumors, of which 7,878 were carcinomas, yet none of these were of appendiceal origin. It must again be emphasized that the reports cited, with rare exceptions, make no differentiation between adenocarcinoma and carcinoid. Kelly and Hurdon [23] found 2 instances of carcinoma of the appendix among 343 cases of gastro-intestinal cancer discovered among 40,738 autopsies at the Vienna General Hospital. Frauenthal and Graussman [24] report 1 case of true gelatinous adenocarcinoma from 1,390 appendices studied histologically, an incidence of 0.07 percent. During the same study 53 gastro-intestinal cancers were observed. Hence in this series cancer of the appendix comprised 1.8 percent of digestive tract malignancy. This ratio is somewhat low and contrasts with that of Kelly and Hurdon [23], namely 1 to 172. Even this figure is rather high, the usually stated average being about in the proportion of 1 to 250 or less than 0.5 percent. One gains the impression from a fairly comprehensive review of the literature that of all appendices subjected to routine microscopic study about 1 in 400 (0.25 percent) will show a cancer-like neoplasm of which 10 percent or less will be true adenocarcinoma (about 1 in 4,000 appendices) and the remainder carcinoids.

[18] Gnassi, A. M.: Chromargentaffine tumors of appendix. Am. J. Surg. 40: 470, May 1938.
[19] Selinger, J.: Primary carcinoma of vermiform appendix. Ann. of Surg. 89: 276, Feb. 1929.
[20] Reiman: Quoted by Frauenthal and Grausman (24).
[21] McCarty, W. C. and McGrath, B. F.: Frequency of carcinoma of appendix. Annals of Surgery, 59: 673 1914.
[22] McWilliams, C. A.: Primary carcinoma of vermiform appendix. Amer. Jour. Med. Sc. 135: 822, 1908
[23] Kelly H. A. and Hurdon, E.: The Appendix and Its Diseases. W. B. Saunders & Co. Phila., Pa. 1905.
[24] Frauenthal, M. and Grausman, R. I.: Primary carcinoma in vermiform appendix. Am. Jour. Surg. 19: 118, Jan. 1933.

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Age incidence: The age incidence of these lesions shows the widest variations. The youngest patient was that of MacCarty and MacGrath [21] who was 5 years of age, while Kudo [25] reports a case in a patient of 92. Kelly and Hurdon [23] found these tumors most frequently in the third decade. In the Bellevue series the average age was 34½ years. Selinger [19] found the average age of his cases to be 34 years but since this coincided almost exactly with the average of those who did not have cancer he concluded that the age factor was of absolutely no importance. Here again it is difficult to ascertain whether the reported cases represent true malignant lesions of the appendix or so-called benign cancer. Thomas [26] reports a typical adenocarcinoma of the appendix in a boy of 14 years, probably the youngest authentic case on record. Published statistics incline one to conclude that carcinoids are more common in the third and fourth decades of life when obliterative and inflammatory disease of the appendix is more often encountered, while there is nothing to indicate that the age incidence of appendiceal adenocarcinoma differs in any material degree from that of other gastro-intestinal cancer. Ewing [3] gives the average age of onset of this variety of malignancy as between 40 and 60 years.

Sex distribution: Considering adenocarcinoma and carcinoids together, one would infer from available statistics that they are more common in females than in males in the proportion of about 3 to 1. The figures vary from 82.4 percent in females as found by Selinger [19] to 57 percent reported by McWilliams.[22] Deaver [27] alone found more in males, 62 percent. In summary, it may be said that approximately 75 percent of reported cases have been discovered in women patients. However, as Frauenthal and Graussman [24] and others have pointed out, these figures should not be accepted without reservation, as it must be borne in mind that the discovery of these lesions often comes as a matter of surprise in the course of routine pathological examination of appendices removed as an incidental measure at the time of abdominal surgery for some unrelated condition, and women are subjected to far more laparotomies than are male patients. It appears quite probable that parallel cases studied would reveal no significant preponderance in either sex.

Etiology: The true cause of adenocarcinoma and carcinoids of the appendix is unknown. The following theories as to cause have been cited by Van Alstine [28]: (*a*) Embryonal rests within the appendix; (*b*) infection, the appendix being a vestigal organ is very susceptible to infection; and (*c*) nutritional, due to a relatively poor blood supply,

[25] Kudo. Quoted by Frauenthal and Grausman (24).
[26] Thomas, A. O'D.: British Medical Journal, 1: 680, 1923.
[27] Deaver, J. B.: Quoted by Frauenthal and Grausman (24).
[28] Van Alstine, G. S.: Primary carcinoma of appendix. Ill. Med. Journ. 49: 469, June 1926.

to which Waugh and Findley [29] add: (*d*) Chronic irritation, whether mechanical, inflammatory, or neoplastic. In summation, one may say that these are interesting theories but have no practical application or clinical importance, for here as elsewhere, the real etiology of cancer remains unknown.

SYMPTOMATOLOGY AND DIAGNOSIS: Neither carcinoids nor adenocarcinoma of the appendix give rise to characteristic symptoms. Indeed they may not, and frequently do not, give rise to any at all. Various attempts have been made from time to time by different authors to point out distinguishing features of each but these suggestions possess little practical merit and the fact remains that appendiceal tumors give rise to no pathognomonic signs or symptoms and their discovery continues to be a matter of pure chance. Even when they give rise to obstructive symptoms as in the case reported by Leonardo [30] the pre-operative diagnosis can be nothing more than a suspicion. Vance [31] agrees with the statement that has been repeatedly made that cancer of the appendix has never been correctly diagnosed before operation and well summarizes this phase of the problem in his conclusion:

> After carefully weighing all the evidence at hand, one can reach no conclusion nor can one make a definite positive statement regarding a single symptom or chain of symptoms that would assist in making a diagnosis of cancer of the appendix. Even if a tumor in this region were palpated, to say that it is a tumor of the appendix would not be much more than a mere guess.

TREATMENT: The treatment of carcinoids and adenocarcinoma of the appendix is excision. This has usually already been accomplished before the diagnosis is established by the pathologist. Due to their usual if not universal benign character, simple appendectomy ordinarily suffices for the complete cure of carcinoids.

The treatment of gelatinous adenocarcinoma of the appendix merits, I believe, further thought and consideration. The truly malignant nature of this lesion leads one to wonder whether appendectomy alone constitutes sufficiently adequate surgical attack to produce a reasonable expectation of a permanent eradication of the disease process. Case 1 clearly illustrates the advisability of examining sections taken from the extreme margin of the base of a proved malignant appendix. If microscopic study of these sections shows that the cancer has spread by continuity to the adjacent cecum (if indeed it did not primarily arise there as Masson [11] suggested), it is obviously essential to re-enter the abdomen and endeavor to remove the cancerous tissue left behind.

[29] Waugh, T. R. and Findley, D.: Mucocele with peritoneal transplantation in adenocarcinoma of appendix. Am. Journ. Surg. 37: 518, Sept. 1937.

[30] Leonardo, R. A.: Primary carcinoma of appendix vs. carcinoid. Am. Journ. Surg. 22: 290, Nov. 1933.

[31] Vance, C. A.: Primary carcinoma of vermiform appendix. Amer. J. Surg. 24:854, June 1934.

In the light of our present knowledge and prevailing opinions regarding this condition, there are several factors that suggest the feasibility of such a procedure, namely: (*a*) This particular malignancy is generally considered to be of a low grade; (*b*) its habit is toward late metastasis with either early or distant spread a relative rarity; (*c*) the second operation need not be an emergency procedure, thus allowing time for preparation of the patient so as to minimize the operative risk. The fact that the histological character and location of these tumors render them unfavorable for irradiation therapy lends further weight in favor of more radical surgery.

At the second operation, resection of the cecum, the lower one-third or one-half of the ascending colon, 8 to 12 inches of terminal ileum, together with the contiguous omentum and all accessible tributary lymph glands, and followed by ileo-colostomy (see case 3) should furnish fair assurance of complete eradication of the cancer. Similar measures have been suggested by Loe [32], Neuman [4], and others. Wide excision of the adjacent abdominal wall to forestall implantation metastases has also been mentioned.

Were one ever so alert or fortunate as to suspect the true nature of the lesion at the operating table, a frozen section would be in order and a report of adenocarcinoma, conditions being favorable, would indicate the immediate performance of the extensive operation suggested, thereby obviating the necessity of a second operation and reducing the chances of a catastrophic outcome as was experienced in one of my cases. (See case 1.) Any patient presenting a definitely suspicious appendix should at least have a careful examination of the cecum, ileocecal valve, terminal ileum, mesentery, omentum, and tributary lymph glands and the possibility of appendiceal cancer given distinct and direct consideration.

MUCOCELE

The term *mucocele* was coined by Fere [33] to describe certain tumors of the appendix, usually benign, which are characterized by occlusion of the lumen at some point with a collection of mucoid material distal to that site and a resultant enlargement or ballooning out of this portion of the organ. The lesion was first described by Virchow [34] in 1836 who ascribed it to colloid degeneration. Rokitansky [35] verified the lesion as a pathological entity in 1842.

Although encountered infrequently, the condition is not rare. Dodge [36] in 1916 collected 116 cases from the literature and since

[32] Loe, A. O.: Carcinoma of appendix, Northwest Med. 26:60, Feb. 1927.

[33] Fere. Quoted by Norment (5).

[34] Virchow. Quoted by Norment (5).

[35] Rokitansky. Quoted by Weaver (37) from Ranzi, Egon. Uber Mukokelen der Appendix. Wien. med. Wchnschr. 76:643, 1926.

[36] Dodge, G. E.: Cystic dilatation of vermiform appendix. Ann. Surg. 37:549, 1903.

that time many additional ones have been reported. Weaver [37] collected 256 cases reported up to 1937 and added 7 cases of his own found in a study of 6,225 appendices. Castle [38] found mucocele in 0.2 percent of appendices at autopsy, while Kelly and Hurdon [23] report its presence in 0.42 percent of surgical cases. Norment [5] states that he found 35 in the 45,000 appendices which he examined. The average incidence seems to be about 0.2 percent.

The basic cause of these tumors appears to be some type of obstruction of the lumen of the appendix. Norment [5] states that this may result from: (*a*) A normal involutional or obliterative process occurring at some point between a secretory area and the base of the appendix; (*b*) general proliferative and ulcerative inflammatory changes; and (*c*) extrinsic causes, such as kinking or pressure from a neighboring mass. To these causes I would add: (*d*) The possibility of fecaliths or other foreign body obstructions with secondary reaction within the appendix playing a role. In any case, the process is initiated by closure of the lumen and this is followed by the retention of secretions, to which the products of degeneration are later added.

Waugh and Findley [29] describe two types of changes which may take place following occlusion. According to these workers, there may be simply an accumulation of viscid fluid which dilates the lumen, stretching, flattening, and compressing the mucosa, rendering it atrophic. The resultant picture they would designate *hydrops* of the appendix. On the other hand they describe cases in which the normal mucosa "has been transformed into a very hyperplastic and even papillary adenomatous type of active secreting surface." Thick viscid secretion far in excess of the normal amount is poured out and the walls become thick, fibrous, and quite vascular. They would reserve the designation *mucocele* for this second type, which possesses many features similar to a papillary cystadenoma of the ovary. The contained secretion is not true mucin, but pseudo-mucin.[5 29 37]

Mucoceles may attain astonishing size. Probably the largest on record is the case of Neuman, said to have been as large as a man's head. Kelly [39] mentions one 30 centimeters long, the size and shape of a banana. Case 5 reported below was as large as a grapefruit. (See fig. 10.)

This condition is probably never diagnosed correctly prior to operation. It presents no characteristic symptoms. The sex incidence appears to be of no significance, though 61 percent of Norment's [5] cases were in males. No age can claim exemption from the lesion, it having been observed in a patient as young as 5 and as old as 80 years.

[37] Weaver, C. H.: Mucocele of appendix with pseudomucinous degeneration. Am. J. Surg. 36:523, May 1937.

[38] Castle. Quoted by Waugh and Findley (29).

[39] Kelly, J. A.: Cystic dilatation of vermiform appendix. Ann. Surg. 49:524, 1909.

However, it appears to be reasonable to assume that it would be more common in middle and later life when obliterative and involutional changes are normally taking place, adhesions leading to kinking more common, and tumors giving rise to extrinsic pressure more frequently found. Generally speaking, this expectation has been borne out by experience. When palpated (as these tumors often may be) the preoperative diagnosis is almost invariably that of some pelvic condition such as ovarian cyst, chronic tubal disease, pedunculated fibroid, etc., if the patient happens to be a female. (See case 5.) Given a male patient, one familiar with the condition would be much more prone to give mucocele some thought, but even in this situation primary consideration is usually devoted to other lesions which may give rise to a pelvic or right lower abdominal mass.

These tumors have been known to rupture from intrinsic pressure and set up secondary foci on the peritoneum overlying neighboring viscera. They have been pointed out by Heatley [40] as one of the two causes of pseudomyxoma peritonaei, and he, contrary to the general opinion, regards them as potentially malignant. Most authorities feel that these tumors and such secondary implantations as occur are benign in their behavior and growth propensities. It appears probable that such degree of malignancy as pseudomyxoma peritonaei may possess rests in the fact that it may tend to cause the death of a patient because of its bulk alone and not in the sense that it is truly cancerous. Waugh and Findley [29] report an extremely rare case of mucocele associated with true adenocarcinoma of the appendix.

Since mucoceles *per se* are usually entirely nonmalignant, excision by simple appendectomy ordinarily constitutes adequate therapy. Due to the possibility of peritoneal implantations occurring if the mucocele ruptures during removal, especial care should be taken to avoid this accident at the time of surgery. Weaver [37] and others have suggested postoperative x-ray irradiation as a prophylactic measure in cases of rupture with spilling of the pseudomucinous contents of these tumors prior to or during surgery.

SARCOMA

Sarcoma is occasionally seen in the appendix but its appearance at this site is rare. It may be in the form of lymphosarcoma, myxosarcoma, myosarcoma, and possibly others. The first case was reported by Glazebrook [41] in 1895. By 1932, 32 cases had appeared in the literature, and since that time a few additional authentic cases have been recorded. The diagnosis is made only by the pathologist and the treatment is excision. The radical operation proposed for gelatinous adenocarcinoma would doubtless be indicated in this condition

[40] Heatley, T. F.: Hemorrhagic mucocele of appendix. J. A. M. A. 112:1935, May 1939.
[41] Glazebrook. Quoted by Ewing (3), Norment (5), and others.

as well. Carnelli [42] advises a similar procedure. The lesion is said to resemble a specific granuloma microscopically, hence lesions presenting this appearance might well be subjected to rapid histological study while the patient is still on the table. Frozen section study of the involved tissue would clarify the diagnosis and simplify the surgical treatment. A high *index of suspicion* on the part of surgeons will lead to the detection of an occasional case of appendiceal malignancy at the time of operation.

NEUROMAS

Plexiform neuromas of the vermiform appendix are said to be quite common. According to Ewing [3] the rule in neurofibromatosis is for the nerve fibers to play a passive role and gradually undergo atrophy, but he states that there are rare forms of overgrowths of nerve trunks and ganglia in which the nerve elements take part in the hyperplasia. Fein, Hanan, and Seidler [43] have recently published an extensive review of this subject in which they quote from the work of Verocay, Ewing, Masson, Hosoi, and others. From their findings it appears probable that neuromatosis forms the basic pathology in a high percentage of cases operated on for chronic appendicitis.

These lesions are most commonly of a microscopic character only, being found in small atrophic appendices with obliterated lumens, but tumors of very large proportions have been reported, including Oberndoefer's case in which the appendix was 16 cm. in length. Three cases have been reported in which the growth had broken through the muscularis mucosae and appeared in the subserosa. Hosoi [44] in 1933 studied 195 cases of neuromatous growths of the appendix, of which 69.2 percent were found associated with complete obliteration of the lumen. Conversely, this investigator was able to find evidence of neuromatosis in 82 percent of appendices with obliterated lumina. He describes the microscopic pathology as consisting of:

> Loosely anastamosing strands of nonmedullated nerve fibers, or as islands of compact interlacing bundles of spindle-shaped cells, always located central to the muscularis mucosae. If the neuromatous tumor mass was large enough, the muscularis mucosae was closely apposed to it along the periphery, in the manner of a capsule. Rarely the tumor mass broke through the muscularis mucosae. These neuromatous growths contained a variable mixture of argentaffin cells and lymphocytes.

Some connection between this appendiceal condition and von Recklinghausen's disease (generalized neurofibromatosis) as manifested on the surface of the body is conjectured and awaits further

[42] Carnelli, R.: Anatomico-pathologico-clinical studies of three rare diseases of vermiform process. Arch Italdi Chir. **30**: 158, 1931.

[43] Fein, M. J., Hanan, J. T. and Seidler, V. B.: Plexiform neuroma of appendix. Am. J. Surg. **39**: 27, Jan. 1938.

[44] Hosoi, K.: Neuromatosis of vermiform appendix. Arch. Path. **16**: 500, Oct. 1933.

study. The diagnosis of this tumor is made only on pathological examination of the excised appendix.

RARE BENIGN LESIONS

INTRA-APPENDICEAL POLYPS have been mentioned by van den Bergh.[45] They are exceedingly uncommon and of no clinical inportance. Removal of the appendix likewise removes the polyp and of course they do not recur. These lesions are already cured before they are diagnosed.

FIBROMATA, MYOMATA, and MYXOMATA can and occasionally do occur. They are exceptionally rare. Norment [5] reports two myxomata in his series. He describes the cells as presenting a stellate appearance with a tendency toward branching cytoplasmic projections. Rare multinucleated cells were seen. No section showed a definite line encapsulation.

NAEVUS HEMANGIOMA: One has been reported. It presents the same histopathological picture as a similar growth appearing elsewhere in the body.

These rare benign lesions are of interest chiefly as clinical curiosities. They represent accidental discoveries by the pathologist and their practical importance is nil.

METASTATIC LESIONS

But little has been written relative to involvement of the appendix by metastatic malignant growths. Barring direct extension from adjacent bowel of gastro-intestinal carcinoma, such an occurrence is apparently quite rare. The standard works on pathology mention the subject only briefly if at all. The literature contains few reports directing attention to this group of secondary appendiceal tumors.

There is no valid reason why malignant new-growths which spread via the blood stream cannot become implanted in the appendix following the lodging of tumor cells in the end arteries of that organ. That such an occurrence does take place is illustrated by case 4. Extension by way of the lymphatics is another possibility and malignancy of the appendix secondary to a primary neoplasm of the ovaries is not unknown.[46] Implantation by contiguity from a similar site is also occasionally seen. Endometriosis of the appendix, an implantation phenomena, has been observed in a few cases, Seelig.[47] Direct extension to the appendix by continuity from a cancer of the lower ascending colon, ileocecal valve, cecum, or terminal ileum would be an almost normal expectancy, and as a matter of fact such appeni-

[45] van den Bergh. Quoted by Norment (5).

[46] Cabot case 21092: Papillary cystadenocarcinoma of ovary with invasion to appendix. New England J. Med. 212: 397, Feb. 28, 1935.

[47] Seelig, M. G.: Endometrial adenoma (implantation) in vermiform appendix. Am. J. Obst. & Gynec. 11: 461, April 1926.

ceal invasion is not uncommonly associated with those conditions. (See case 3.)

SPECIFIC GRANULOMAE

This class of neoplasms forms a relatively unimportant group of appendiceal tumors. When seen in this organ they are usually primary elsewhere with the appendix secondarily involved, and they are unimportant in the sense that the appendiceal lesion is overshadowed by the primary focus. Tuberculosis, syphilis, coccidoides, actinomycosis, etc., can affect the appendix.

TUBERCULOSIS of the appendix is not exceptionally rare. It was first described by Corbin [48] in 1837. Carson,[49] in 1936, collected 125 cases from the literature and reported 1 of his own. Drissen and Zollinger [50] in 1935 reported a series of 5,149 appendices examined at the Peter Bent Brigham Hospital in the preceding 20 years in 16 of which (0.3 percent) tuberculosis was found. Slaffens [51] found tuberculous affection of the appendix in 2 of 40 cases of pulmonary tuberculosis in which appendectomies were performed.

The modes of infection are by: (*a*) Aspiration; (*b*) deglutition; and (*c*) ritual circumcision. The tissue reaction may be either acute inflammatory in character or by the formation of tubercules. Carson[49] tabulates the method of distribution of the bacilli in the body as follows: (*a*) Mechanically along with air, food, secretions; (*b*) by growth through the tissue with the production of lesions; (*c*) by way of the lymphatic channels; and (*d*) by the blood stream.

The diagnosis of tuberculosis of the appendix and other specific granulomas is ordinarily made only by miscrosopic study. Actinomycosis might be recognized microscopically by the characteristic yellowish granules, while luetic gumma could be suspected in an appendiceal tumor found in a known tertiary luetic.

The surgical treatment consists of complete extirpation of all affected tissues. The follow-up therapy varies with the causative agent. These lesions, especially tuberculosis, should be seriously considered in cases developing fistulous tracts following appendectomy.

CASE REPORTS

Case No. 1.—Mrs. J. W. J., white female, age 37, housewife. Admitted February 2, 1939.

CHIEF COMPLAINT: Excruciating pain in the lower abdomen, slightly more intense in the right side than elsewhere. Past history: Usual childhood diseases with complete recovery without complications or sequels. Five years ago suffered with moderate discomfort in both lower abdominal quadrants in the adnexal regions and was told by her physician that she had small bilateral ovarian

[48] Corbin, M.: M. Gaz. Med. de Par. 5:639, 1837. Quoted by Carson (49).
[49] Carson, W. J.: Tuberculosis of appendix. Am. J. Surg. 34: 379, Nov. 1936.
[50] Drissen, E. M. and Zollinger, R.: Acute tuberculous appendicitis. Ann. Surg. 101: 740, Feb. 1935.
[51] Slaffens, L. F.: Minn. Med. 16: 743, Dec. 1933.

cysts. These symptoms soon subsided and have not recurred. Obstetrical and gynecological history: Menarche at 13; interval 28–30 days; duration 4–5 days; flow moderate; no dysmenorrhea. Gravida, 1. Para, 1. Voluntary sterility since spontaneous birth of full term infant 15 years ago. Family history: Irrelevant. No history of malignancy. Present illness: Patient first seen at 6 p. m. at which time she was tossing about in bed, frequently sitting upright and almost constantly changing her position in an effort to find one that would afford her some relief from the agonizing pain. She was moaning and intermittently crying with pain and her body was covered with cold, clammy perspiration. She described the pain as almost unbearable in degree and of a constant boring character with occasional knife-like pains of even greater intensity. The pain was generalized over the entire abdomen, considerably more severe in the lower half than in the upper, and slightly greater in the right lower quadrant than the left. The fulminating pain in the right lower quadrant extended upward into the right flank to an area corresponding to the right kidney.

The patient stated that she had been quite well until this morning. When she arose at 6:30 a. m. she had no desire for breakfast. She drank one cup of black coffee which gave rise to a feeling of nausea but did not result in vomiting. Around 8 a. m. she first noted generalized abdominal pain of a mild degree, slightly greater in the lower abdomen than elsewhere but by no means definitely localized. Her sensation of discomfort was similar to that experienced 5 years ago when troubled with the ovarian disorder and she did not consider her condition serious. By noon the pain had shown a gradual but steady increase in degree and there was a vague sense of nausea but no vomiting. The patient took nothing by mouth except small sips of water and no medication. The pains increased progressively until attaining the violent character present on admission. There had been slight frequency of urination since onset of the illness, but no pain, burning, hematuria, or other urinary signs or symptoms. The pain did not radiate to the urethra or vulva. Upon close questioning the patient recalled that 5 days ago she had suffered with fairly severe generalized abdominal pain which was completely relieved following 1 ounce of milk of magnesia and a liquid diet for 1 day.

PHYSICAL EXAMINATION: Well developed, somewhat obese white female, tossing about in bed, crying, and apparently in acute distress. Her height was stated to be 62 inches and weight 169 pounds. She appeared to be somwhat older than her stated age of 37 years. Her skin was clear and covered with cold, clammy perspiration. T, 99.4°; P, 90; R, 22.

Head and neck: Essentially negative.

Chest: The lung fields were clear. Respiration slightly increased in rate and shallow.

Heart: Normal. B. P. 124/76.

Abdomen: Very obese. Abdominal respiratory excursions sharply limited. The abdomen was exquisitely tender over the lower two-thirds and quite sensitive in the upper third. As well as could be determined through a very thick layer of adipose tissue, the abdomen was rigid throughout, the rigidity in the lower half apparently boardlike, whereas in the upper portion perhaps somewhat less muscle spasm was present. No organs or masses were palpable. Very little difference in tenderness or rigidity could be detected in comparing the right lower quadrant with the left, although the exquisite tenderness extended higher in the right flank than the left. Percussion of the abdomen gave rise to a tympanitic note and on auscultation considerable gurgling could be heard.

External genitalia: Negative, no vaginal discharge.

Pelvis: Examination was difficult and on the whole rather unsatisfactory due to the intense pain, the thick layer of fat present in the lower abdomen, and the

marked lower abdominal rigidity. The cervix was normal to palpation; the vaginal canal not unusual; no bulging in either of the fornices. It was felt that the uterus was approximately normal in size and position. The adnexae could not be palpated. There was marked tenderness on palpation high in the fornices, no difference noticeable in comparing the two sides.

Rectal: Negative except for marked tenderness.

Joints and extremities: Normal.

Reflexes: Active and equal.

LABORATORY FINDINGS: Urinalysis (catheterized)—entirely negative. WBC, 15,300; polys, 91, monos, 1; lymphs, 8. Urinalysis (second specimen, catheterized), positive for acetone and diacetic acid, otherwise negative.

DISCUSSION: The patient presented an interesting diagnostic problem. Barring the possibility of a renal calculus, it was apparent that the case was one of an acute surgical abdomen but beyond that the diagnosis was by no means clear. The pain was considerably greater than that seen in the ordinary acute inflammatory lesion, such as appendicitis, being more typical of that commonly observed in cases of ureteral or biliary calculi or acute hemorrhagic pancreatitis. Ruptured peptic ulcer was considered as was purulent cholecystitis with necrosis or gangrene of the gall bladder, but both seemed improbable on the basis of a negative antecedent history, the sudden onset of the present attack, and the predominance of the physical findings in the lower abdomen. Intussusception and volvulus were considered. In view of the past history of ovarian cysts, an ovarian cyst with twisted pedicle, gangrene, rupture, and peritonitis was thought of and could not be excluded on the basis of the unsatisfactory pelvic examination. Ruptured tubal pregnancy was not impossible but there had been no menstrual irregularity and the patient did not present the picture of shock. The excruciating character of the pain, its presence high in the right flank, and the urinary frequency led to the consideration of renal calculus but this diagnosis seemed improbable on the basis of two negative urine specimens, the noncolicky character of the pain, absence of the typical radiation, and its widespread diffusion.

On the basis of elimination acute appendicitis, possibly ruptured, and probably with considerable peritoneal reaction, was deemed the likeliest diagnostic possibility and she was taken to surgery.

OPERATIVE RECORD: Abdomen opened by midline incision. The peritoneal cavity contained a great deal of cloudy yellowish fluid in which flakes of pus and debris could be seen floating. The left ovary contained a small simple follicular cyst and the right ovary was normal in appearance. Both tubes were slightly enlarged and thickened. The uterus was normal in appearance, size, shape, position, consistency, and mobility. The appendix was enormous, the size and shape of a large sausage (approximately 2½ by 10 cms.). There was a small round perforation near its base through which purulent material was dripping. The appendix was lying against the lateral abdominal wall, largely anterior to the hollow viscera, and was covered by a thick grayish fibrinous exudate. The great omentum was placed over the organ and was very thick, hard, and indurated, as was the mesoappendix. The cecum at the appendiceal base was likewise thickened, red, and indurated. This portion of the cecum was elevated in cone-shaped fashion and its consistency approached a cartilaginous hardness, although it showed no blanching. No enlarged glands were palpable. All visible peritoneal surfaces showed the signs of acute inflammation.

An appendectomy and partial cecectomy was done, the involved portion of the cecum constituting approximately one-third of that structure being excised between intestinal clamps along with the appendix. The excised portion was edematous, reddened, and thickened by a brawny type of induration but it did not appear malignant. The cecum was closed by two superimposed rows of

intestinal sutures of fine black silk, being inverted by each row. No tissue that appeared in any way involved was left behind. One rubber dam covered gauze drain was inserted into the right iliac fossa and brought out through the lower angle of the incision.

POSTOPERATIVE DIAGNOSIS: Appendicitis, acute, perforated, and peritonitis, acute, spreading.

PATHOLOGICAL REPORT: *Gross*: The appendix is extremely large, measuring 8 cm. long and 1.8 cm. in diameter. It is pale, whitish, and firm, and has a grayish fibrinous exudate on the surface. A small perforation is present near the base around which the wall is gangrenous. The lumen of the proximal two-thirds is filled with solid mucoid grayish colored structure; the lumen of the distal third is cystic and contains gelatinous fluid material. The submucosa does not have the characteristic encircling yellow ring of a carcinoid tumor.

Microscopic: All layers of the appendix, including the serosa are permeated with various-sized groups and masses of neoplastic epithelial cells in more or less adenomatous arrangement, and showing a widespread mucoid degeneration. Karyokinesis is frequent and many of the cells present a signet ring appearance. This appears to be primary adenocarcinoma arising from the mucous membrane. The tissue around the perforation is necrotic. It and all adjacent structures are heavily infiltrated with pus cells. The more common tumor of the appendix, the benign carcinoid (argentaffinoma) has been considered.

Diagnosis: Adenocarcinoma, appendix, perforated. (See figs. 6 and 7.)

CLINICAL COURSE: The patients postoperative course was stormy. She developed a generalized peritonitis and for days wavered between life and death but the process subsided and by the twelfth day she seemed well on the road to recovery. There was a moderate purulent discharge from the wound at the site from which the drain had been removed, but her TPR were normal, she was eating a general diet, and her bowels were moving normally once daily.

On the afternoon of the twelfth day, during a severe coughing spell, her incision broke open in the lower third and the patient suffered a partial evisceration. At the time of repair of the disrupted abdominal incision and replacement of the evisceration the condition of viscera in the ileocecal valve region was observed. The terminal ileum, ileocecal valve area, the remaining portion of cecum and the lower ascending colon all appeared perfectly normal except for residual signs of the preexisting peritonitis. The intestines were soft and pliable and no evidence of recurrent or metastatic malignancy was discernible.

Following this operation convalescence was relatively uneventful except for continued drainage from the lower angle of the incision. The patient was discharged to home on the thirty-second postoperative day in fairly good condition and irrigations of the abdominal wound continued at home, using azochloramid solution as the irrigating solution.

After the diagnosis of primary adenocarcinoma of the appendix had been made by the pathologist he was requested to make some sections from the extreme margin of the excised portion of cecum. This was done and microscopic study showed an invasion of the cecum by the tumor cells, which, while not marked, definitely established the fact that all the malignant tissue had not been removed.

By May 11, 1939, the patient had regained her strength, her weight had picked up, blood picture was excellent, general condition good, and she was considered in suitable condition to undergo surgery of an extensive character. Thorough x-ray studies had failed to reveal evidence of metastasis and she was symptom free. Our intention at this time was to resect a fairly lengthy portion of the terminal ileum (8–10 inches), the remainder of the cecum, and most of the ascending colon, together with the mesentery and all accessible tributary lymph glands

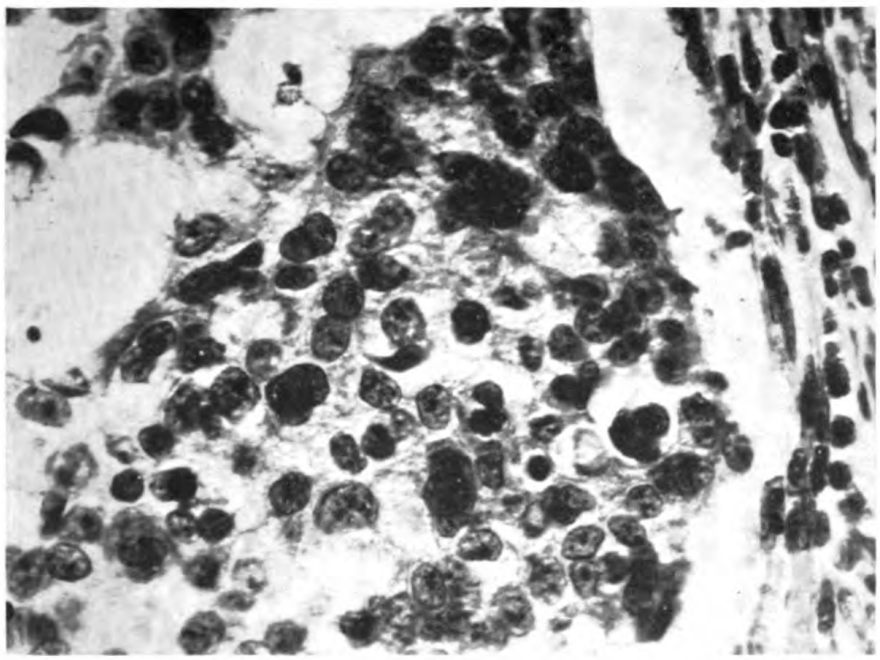

FIGURE 6—CASE 1.—PRIMARY ADENOCARCINOMA OF THE APPENDIX. CELLS SHOW MARKED VARIATION IN SIZE, SHAPE, AND STAINING CHARACTERISTICS WITH CONSIDERABLE HYPERCHROMIA. AREAS OF COLLOID DEGENERATION CAN BE SEEN. COMPARE WITH FIGURE 9, CASE 2. (X 1,200.)

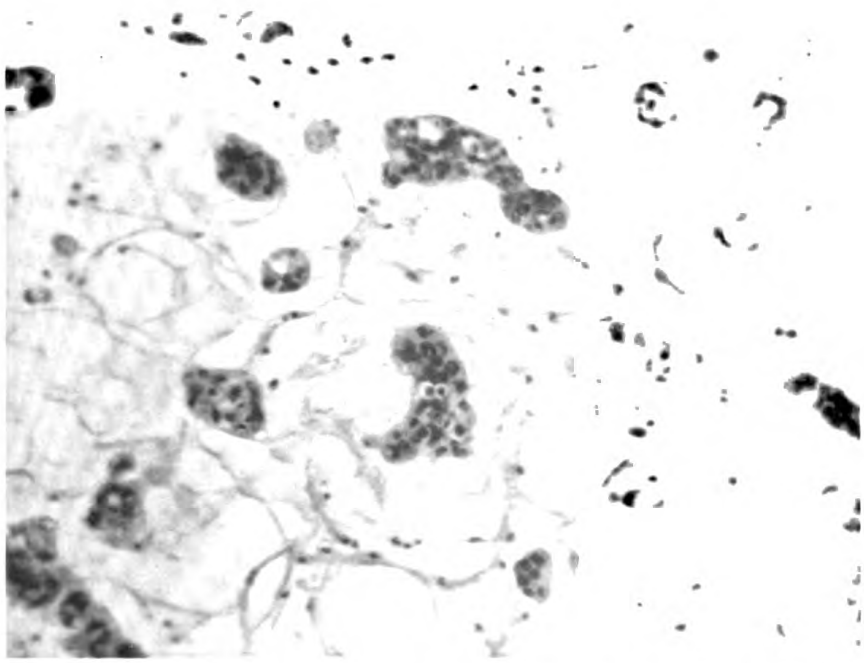

FIGURE 7—CASE 1.—SECTION FROM PRIMARY APPENDICEAL ADENOCARCINOMA SHOWING MARKED COLLOID DEGENERATION AND AREAS OR NESTS OF TUMOR CELLS. THIS FEATURE OF THESE TUMORS GIVES RISE TO THEIR GELATINOUS CHARACTER. (X 400.)

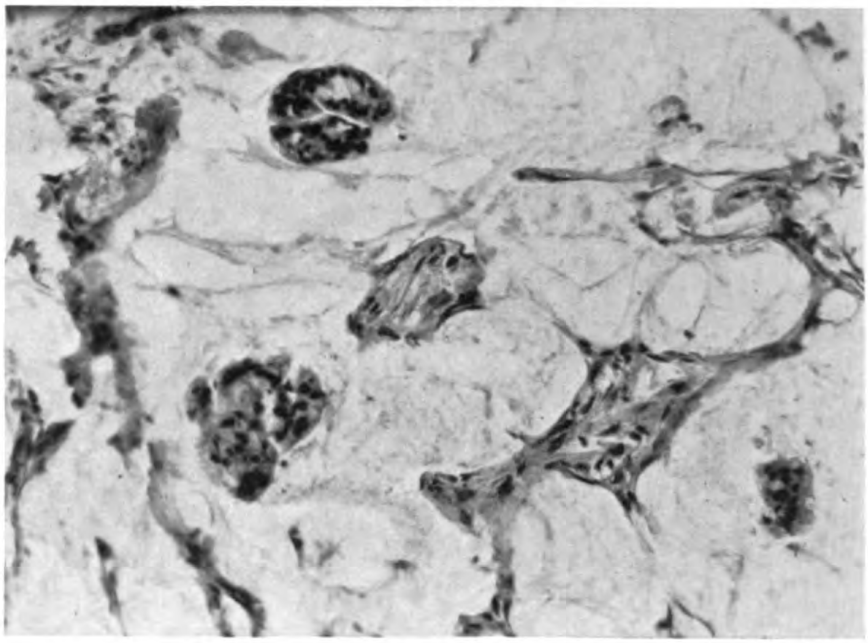

FIGURE 8—CASE 1.—SECTION FROM METASTATIC NODULE OF GELATINOUS ADENOCARCINOMA SHOWING EXTENSIVE COLLOID DEGENERATION. COMPARE WITH FIGURE 7 FROM PRIMARY TUMOR. (X 400.)

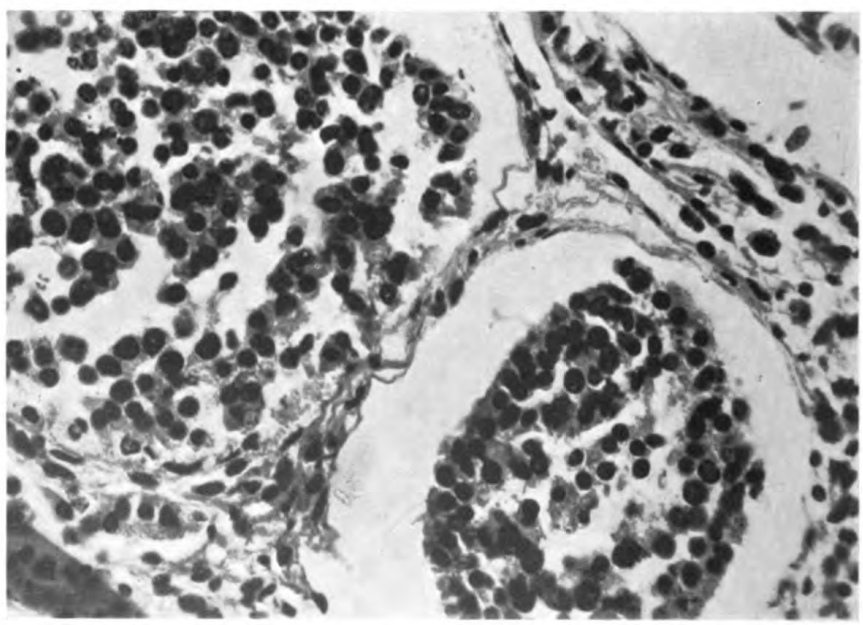

FIGURE 9.—CASE 2.—PHOTOMICROGRAPH EXHIBITING THE CHARACTERISTIC CYTOLOGIC DETAILS OF A TYPICAL CARCINOID TUMOR OF THE APPENDIX. SECTION SOMEWHAT MORE HEAVILY STAINED THAN IDEAL. CONTRAST WITH FIGURE 6, CASE 1. (X 1,000.)

and to reestablish the intestinal continuity by anastamosing the ileum with the transverse colon.

OPERATION: On the above date, under spinal anesthesia, the abdomen was entered through a lower right rectus incision and inspection of the abdominal viscera showed that the carcinoma had spread like wildfire since the last operation (February 14, 1939). All visible hollow viscera and parietal peritoneum were studded and literally covered by metastatic lesions varying in size from a millet seed to a large bean. The mesenteric glands were universally enlarged and hard, some being the size of walnuts or hen's eggs and of ligneous consistency. The cecum, lower colon, and 2 to 3 inches of terminal ileum were as hard as wood, very blanched in appearance, and resembled cartilage to a remarkable degree. Strangely enough, there were no palpable liver metastases.

The widespread dissemination of the malignancy made complete extirpation utterly impossible and the intended operation was abandoned. Enterocolostomy as a palliative measure was considered but not performed because it was felt that the ultimate benefits to be expected were outweighed by the immediate operative risk. After removing a small metastatic nodule from the surface of the ileum the abdomen was closed without further surgery and no drainage was used.

PATHOLOGICAL REPORT of the metastatic lesion: *Gross:* The specimen consists of a small firm nodule of white tissue 2 mm. in diameter.

Microscopic: Microscopic examination reveals adenocarcinomatous tissue with widespread mucoid degeneration giving the characteristic signet ring appearance to the individual cells. Considerable mucus is present. The picture follows rather closely that of the original tumor.

Diagnosis: Adenocarcinoma, metastatic. (See fig. 8.)

CLINICAL COURSE: Her incision healed by primary intention and she was discharged on the tenth day. Since that time her course has been steadily downhill. She suffers with constant severe lower abdominal pain, more intense on the right side, and just recently has developed alternating constipation and diarrhea. Appetite and sleep are poor, there is a steady loss of weight, and a fatal termination appears to be rapidly approaching. On July 10, 1939, patient returned to the Mainland in order to be with her family. When last heard from she was failing rapidly.

COMMENT: This case, illustrative of typical primary gelatinous adenocarcinoma of the appendix presents several interesting and unusual features:

1. Pain of such a fulminating character is seldom seen in appendiceal lesions and the pain was not abated although the appendix had ruptured.
2. Perforation of an appendix resulting from carcinomatous degeneration has rarely if ever been reported.
3. The absence of distant metastasis and lack of involvement of the adjacent lymph glands at the time of the primary operation, despite very extensive involvement of the appendix, are considered characteristic.
4. The site of the initial lesion (near the base) was typical.
5. The pathological picture was clear-cut.
6. The findings at the time of the evisceration were those to be expected, *i. e.*, no apparent spread and no visible or palpable recurrent lesion.
7. The picture at the time of the final operation was decidedly unexpected and contrary to the general experience with similar lesions.
8. It appears probable that an extensive resection at a much earlier date might have salvaged this patient.

Case 2.—L. T., white male, age 28, clerk. Admitted October 8, 1936, complaining of pain in the right lower abdominal quadrant. He gave a history of intermittent pain in this area for 6 weeks. The pain would come on suddenly

and was quite severe for a few hours and then would subside to return in 24 to 36 hours. The pain was centered over McBurney's point but also was noted in the right inguinal region. He complained of feeling tired and weak, loss of appetite and constipation, with slight nausea at times but no vomiting.

He stated that he had had considerable "gas on the stomach" during the past few monlths. Past history essentially negative. Mother died of carcinoma. Several bood relatives died of tuberculosis. Family history of asthma.

The physical examination was entirely negative except for tenderness over McBurney's point and in the right inguinal region. No rigidity present. Urinalysis revealed nothing abnormal and the white blood count was within normal limits.

The preoperative diagnosis was appendicitis, chronic, with acute flare-ups.

Under avertin-ether anesthesia the abdomen was opened by a McBurney incision. The appendix was found to be completely retrocecal in location and the cecum in turn was looped up behind the ascending colon. It was bound down in its entire length by fairly dense adhesions. The tip of the appendix contained a hard nodule the size of a marble. Examination revealed on enlarged glands in the mesentery, and the cecum, ascending colon, ileocecal valve area, and terminal ileum showed nothing of a suspicious character. The appendix was excised between clamps and the stump inverted by a purse string suture of black silk. Abdomen closed in layers in the usual manner without drainage.

Convalescence was nonmorbid and uneventful and the patient was discharged on the tenth postoperative day at which time he was free of symptoms and his incision well healed. He has remained free of symptoms and in excellent health since that time.

PATHOLOGICAL REPORT: *Gross:* The appendix is 10 cm. long and the tip is markedly dilated and composed of a tumor 2 by 3 cm. in size. The tumor cuts with increased resistance and the surface is granular and white. The remainder of the appendix appears normal.

Microscopic: The tumor of the appendix is well limited by the wall of the muscular coat. The tumor itself is composed of a diffuse growth of cells, some of which are arranged in strands or bundles. The cells are round, spheroidal, or polygonal in shape and show some variation in size and staining characteristics. The cellular growth is dense and the stroma sparse. No undue cellular activity is noted.

Diagnosis: Carcinoid of the appendix. (See fig. 9.)

COMMENT: This case is considered typical of appendiceal carcinoid. Both the gross and microscopic picture were characteristic. It was a benign tumor from the pathological standpoint and the clinical course has been in accord with this finding.

Case 3.—M. D. T. Patient was admitted to the hospital on April 20, 1938, with a chief complaint of pain in the abdomen of 6 months' duration. The past history was unimportant. One sister had died of malignancy of the gastro-intestinal tract. The patient stated that about 6 months previously he had experienced a dull pain in the right lower abdominal quadrant which radiated to the left side. He also noted a mass in the region complained of. He suffered with constipation at this time. The attack lasted about 10 days. During the past 6 months patient has had six similar attacks, each lasting about the same length of time. With the last two attacks there has been some dysuria. There had been occasional nausea but at no time did he vomit. He complained of considerable loss of strength since onset of his illness and of tiring easily.

Physical examination was essentially negative except for the abdomen. There was a palpable mass in the right lower quadrant 10 by 4 cm. in size extending

toward the midline. The mass was firm, more or less fixed, and palpation along the lateral border produced slight pain, otherwise it was not tender.

The positive laboratory findings consisted of 4 plus occult blood in the stools, trace of albumin in the urine, and a blood sedimentation rate of 28 mm. in 60 minutes. The blood picture was within normal limits except for a mild leucocytosis and all other findings were negative. X-ray of the colon following a barium enema showed a definite filling defect in the region of the ileocecal valve. The patient was put on a regime calculated to build him up and on June 1, 1938, an exploratory laparotomy was performed. The preoperative diagnosis was appendiceal abscess, with an infectious granuloma, such as tuberculosis, and bowel malignancy given some consideration.

OPERATIVE FINDINGS: Through a transverse incision just beneath the umbilicus and extending to the right side a large mass was exposed at the head of the cecum. Omental adhesions fixed the mass firmly in the right pelvis. The distal ileum was edematous and inflamed. A hard irregular shaped lesion obstructed the ileocecal valve and obliterated the appendiceal fossa. The mass looked and felt definitely malignant. The process involved the terminal ileum, the ileocecal valve, the cecum, and appendix. The appendix was so involved in the tumor mass that it was impossible to be certain where the primary site of the malignancy was located. Eight inches of the terminal ileum, the ileocecal valve, the cecum, appendix, and the ascending colon up to the hepatic flexure were resected and an ileocolostomy done, uniting the ileum with the transverse colon. Marked glandular enlargement studded most of the mesentery as high as the right colic artery and approached the root of the mesentery. The liver was normal to palpation and no other metastatic lesions noted.

PATHOLOGICAL EXAMINATION: *Gross:* Specimen consists of a section of the ileum with the cecum attached. There is a large friable papillomatous mass projecting into the lumen of the ileum and cecum and extending partially around the outside surface. The mesenteric glands are enlarged. On cut section the mass is white, friable, and semitranslucent. There are numerous necrotic areas throughout. On cut section the glands are similar in appearance.

Microscopic: Sections of the lesion involving the cecum and terminal ileum show a papillary mass projecting from the inner wall of the gut. The mass is made up mostly of a proliferation of typical mucosal glands, the epithelium of which is several cells thick. The nuclei are hyperchromic and show many mitotic figures. Glandular structures of similar nature invade the entire thickness of the intestinal wall and have grown into a fungating mass projecting from the outer surface of the gut wall. There is considerable production of mucous and secondary mucoid degeneration. Examination of a number of the nodules in the mesentery show most of them to be composed of fat. There, however, are lymph glands showing a marked dilation of the sinuses which are filled with serum. There is some proliferation of lymphocytes and some of the germinal centers are destroyed. No tumor cells were seen in any of them.

Pathological diagnosis: Adenocarcinoma, cecum, grade 4, with secondary extension.

Unfortunately for the purposes of this case report, the appendix *per se* was not examined pathologically. However, from a gross description of that organ obtained from the operating surgeon, it is a reasonable certainty that an identical pathological picture would have been found in that organ had it been individually studied microscopically.

POSTOPERATIVE COURSE was uneventful and after a period of sick leave he was subjected to a complete study and no evidence of metastasis found and he was returned to active duty. At the present time he remains symptom free and in excellent general health.

COMMENT: While the primary site of this lesion cannot be definitely determined, all odds favor the cecum as the point of origin. It illustrates:

1. A slowly growing tumor (palpable mass present 6 months, probably present a great deal longer).
2. Extension by continuity of tissue.
3. Absence of distant metastasis or early spread.
4. Apparent cure of long-standing malignancy by radical extirpation.

Adenocarcinoma primary in the appendix, being a histopathological counterpart of the tumor here reported, could be expected to behave in similar fashion.

Case 4.—S. H. L., Korean male, laborer, age 60. Admitted to the hospital on September 23, 1938, in a stuporous state. Although oriented as to time and place he showed a marked retardation of speech and thought, making it difficult to obtain a history. According to friends he had been in poor health for 6 months and had shown increasing loss of weight and weakness.

Physical examination was essentially negative except for poor nourishment, weakness, stupor, and an increase of the left knee jerk as compared with the right.

The laboratory findings showed a 4 plus Wasserman and Kahn, normal blood picture and urinalysis. Spinal fluid: Cell count 3, globulin negative, sugar negative, Wasserman positive, mastic curve 455000. X-ray chest: Tuberculous lesion, left apex, low grade. Both hila thickened. A shadow of the left hilum which could be one of many things, *i. e.* Hodgkins' disease, lues, malignancy.

He became progressively worse following admission, lapsing into unconsciousness 2 days later, followed by urinary incontinence, moist rales in chest, rising temperature and pulse rate, and vomiting and expired on the tenth hospital day.

AUTOPSY SUMMARY: *Heart and aorta*—atherosclerosis 2 plus, no gross evidence of syphilis.

Lungs.—A firm tumor mass, fairly well circumscribed, about 5 cm. in diameter, involving and arising from the left upper main bronchus at its bifurcation close to the hilum.

Spleen and pancreas showed nothing of note.

Adrenals.—Right adrenal presents two metastatic tumor nodules, the largest 1 cm. in diameter.

Kidneys.—Nothing of note.

Appendix average size, midportion greater in diameter (slightly over 1 cm.), filled with white tumor tissue with complete obliteration of the lumen. The tumor extended from a point 1 cm. from the cecal junction to within slightly more than 1 cm. from the tip, a distance of 4 cms.

Brain.—Widespread, diffuse, metastatic involvement, both cerebral hemispheres, the pons, medulla, and cerebellum being studded with circumscribed nodules, the largest about 4 cm. in diameter, many with soft gray, degenerated centers.

MICROSCOPIC: *Primary lesion in the lung:* The tumor can be seen arising from the mucosa of one of the larger bronchi. It takes the form of a very cellular growth, the cells oval to spindle in shape, growing in solid masses with a tendency here and there toward alveolar arrangemennt. No attempt at gland formation is observed. Many of the nuclei are elongate, suggesting an "oat cell" carcinoma.

Right adrenal: A metastatic nodule is present with partial destruction of the cortex. The morphology is identical with that of the primary tumor, the cells being round to oval in shape with elongated nuclei and growing in solid masses.

Liver: Sections show metastatic tumor involvement. The morphology of the tumor is entirely similar to that of the primary lesion.

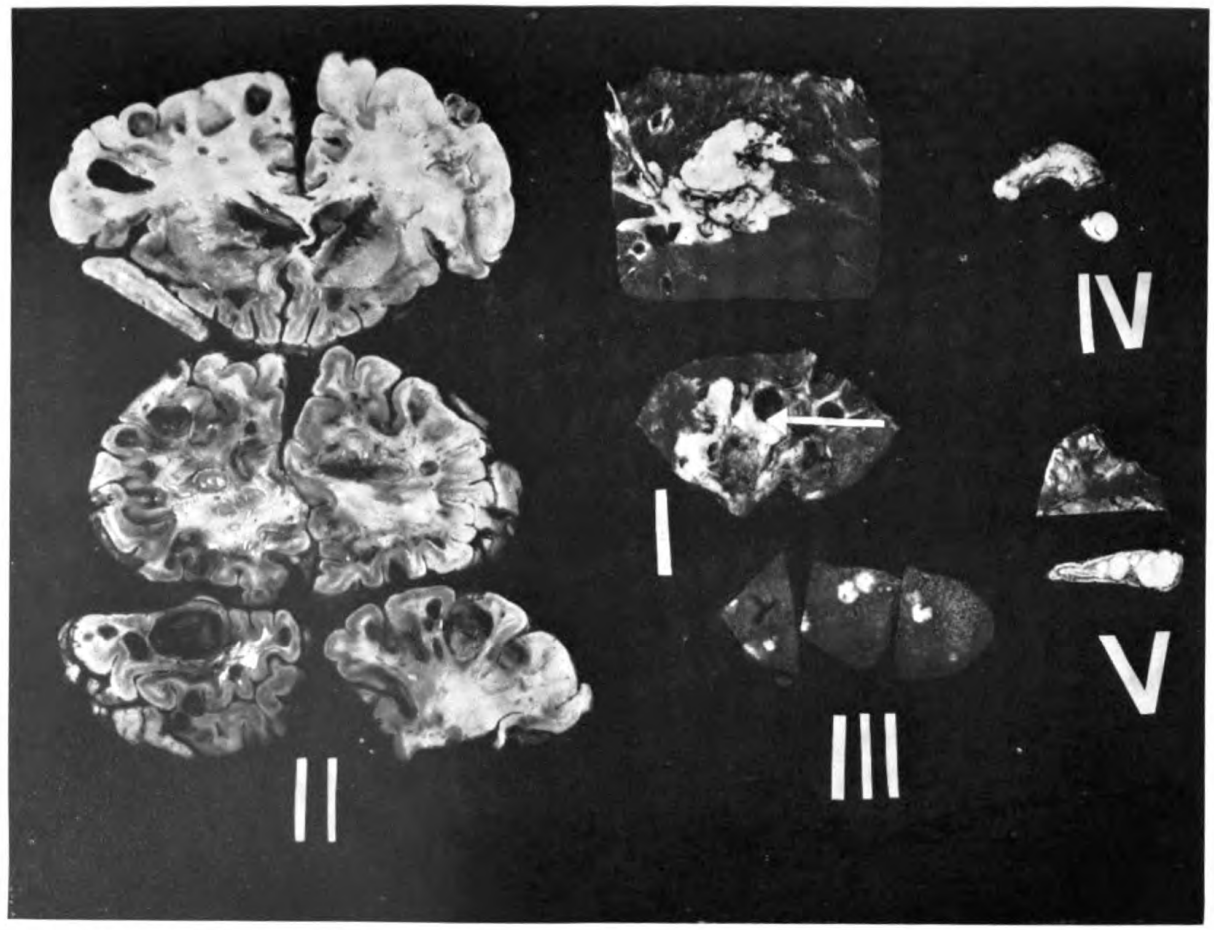

FIGURE 10—CASE 4.—PRIMARY BRONCHOGENIC CARCINOMA WITH VARIOUS DISTANT METASTASES. ARROW INDICATES PRIMARY LESION IN MAIN BRONCHUS. I—LUNGS. II—BRAIN. III—LIVER. IV—APPENDIX. V—ADRENAL. THE METASTATIC NODULES ARE GROSSLY APPARENT IN ALL SECTIONS. (X ⅕.)

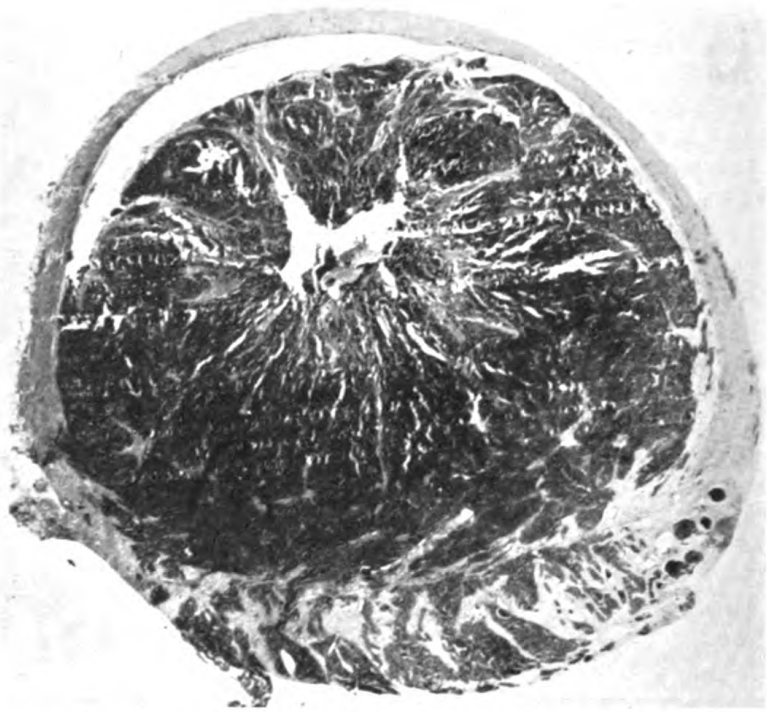

FIGURE 11—CASE 4.—SECTION THROUGH METASTATIC BRONCHOGENIC NODULE IN APPENDIX. THE ENTIRE DARK AREA (LOWER HALF OF THE SECTION) IS COMPOSED OF TUMOR CELLS. (X 20.)

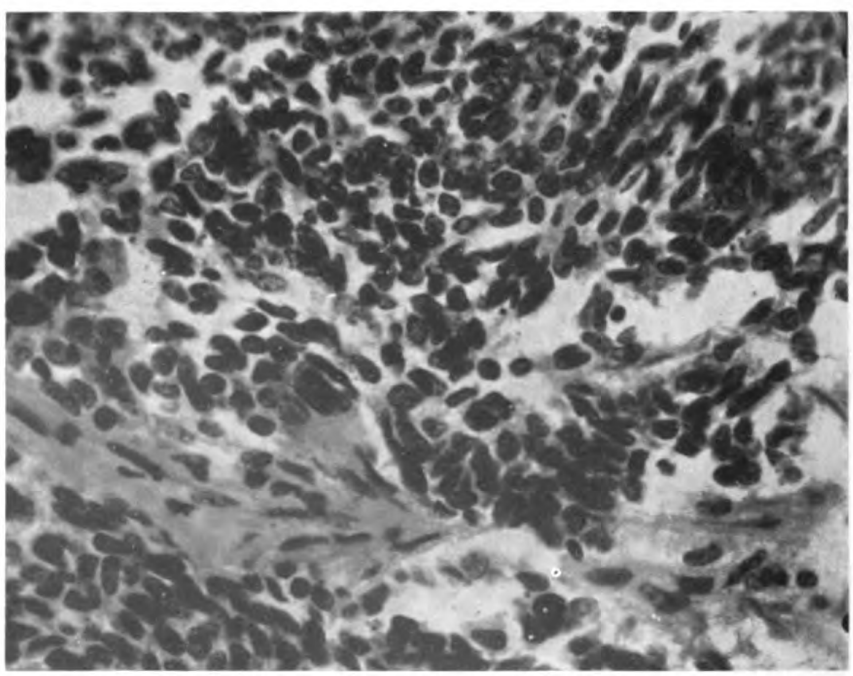

FIGURE 12—CASE 4.—HIGH MAGNIFICATION PHOTOMICROGRAPH OF METASTATIC APPENDICEAL BRONCHOGENIC CARCINOMA. THE MALIGNANT CHARACTERISTICS ARE EVIDENT. "OAT CELL" TYPE OF NEOPLASTIC CELL PREDOMINATES. (X 1000.)

Appendix: The mucosa is completely destroyed and the lumen obliterated by tumor tissue. The muscle is thin but is preserved in its entirety. A number of tumor emboli can be seen lying in the capillaries of the serous layer. As in the previous sections, the cells are oval in shape and growing in solid masses.

Brain: Several well-defined metastatic lesions are observed, the morphology of the cells being similar to those already described.

DIAGNOSIS (1) "Oat cell" carcinoma arising at the bifurcation of the upper left main bronchus.

(2) Metastasis to: (*a*) Brain—cerebrum, cerebellum, and brain stem; (*b*) liver; (*c*) righ adrenal; (*d*) vermiform appendix.

(3) Slight atherosclerosis of the aorta. (See figs. 10, 11, and 12.)

COMMENT: This case clearly illustrates secondary involvement of the appendix by a metastatic malignancy from a distant site. The mode of spread was via the blood stream.

Case 5.—A. W., housewife, age 47. Patient was admitted to the hospital on March 28, 1939, complaining of a sensation of discomfort in the right lower abdomen and pelvis. She first noticed this feeling some months previously but at no time had she had severe pain, nausea, or vomiting. There was no abdominal tenderness or rigidity. She described her symptoms as merely an uncomfortable feeling, a feeling of fullness without definite pain. Her past history was essentially negative. There was no history of either appendicitis or pelvic disorders. She was in the midst of the climacteric when seen. The family history gave no pertinent information.

Physical examination showed nothing unusual except for the abdomen and pelvis. Low in the right lower abdominal quadrant there was a palpable tumor mass which was smooth, round, and regular in outline, of about the size of a grapefruit. It was freely moveable and was only very slightly tender. The abdomen showed no rigidity and no pain was present. On pelvic examination the tumor could be felt in the right adnexal region and presented the features described above. The uterus and left adnexal area showed nothing abnormal.

Laboratory findings were all within normal limits.

The preoperative diagnosis was right ovarian cyst, with old tubal disease and pedunculated fibroid considered as possibilities.

LAPAROTOMY was performed on March 29, the abdomen being entered through a low midline incision. The uterus and both tubes and ovaries were normal. The vermiform appendix lay partly in the abdomen and partly in the right pelvis. It was constricted at a point about 2 cm. from the base and distal to this site was enormously dilated so that it presented as a smooth globular mass of cystic nature the size of a large grapefruit. It was not adherent to the surrounding organs and a careful examination of the cecum, ileocecal valve, ascending colon, terminal ileum, mesentery, and tributary lymph glands revealed nothing of a pathological nature. The stump was ligated and the appendix excised, the stump not being inverted, and the mass removed *in toto*. The abdomen was closed in layers in the usual manner without drainage.

CONVALESCENCE was nonmorbid and uneventful and the patient was discharged on the tenth postoperative day with her incision well healed. She has remained symptom free to the present time.

POSTOPERATIVE DIAGNOSIS: Appendiceal tumor, probably mucocele.

PATHOLOGICAL EXAMINATION: *Gross:* The specimen consists of a cystic tumor which measures 9 cm. in diameter. The appendix is attached to this and communicates with it in its midportion. The total length of the appendix is 5.5 cms. The proximal end is 1 cm. in diameter and contains mucoid material. The main portion is dilated, measuring 2 cms. in diameter and is entirely walled off

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from the small proximal portion. The cystic mass which communicates with the appendix contains 300 cc. of thick mucoid material. The cyst is unilocular and thin walled.

Microscopic: A section taken through the proximal portion of the appendix shows the wall greatly thickened and the lumen filled with mucinous material. A rather marked chronic inflammatory reaction is present, the wall thickened by fibrous tissue and infiltrated by leukocytes. The cyst wall is thin and is composed entirely of connective tissue. No epithelial elements appear in any of the sections and there is no evidence of neoplastic change.

Diagnosis: Mucocele of the appendix. (See figs. 13 and 14.)

COMMENT: This is a typical example of a mucocele of the appendix and illustrates:

1. The absence of acute symptoms.
2. The benign character of the lesion.
3. The simplicity of treatment.
4. The tendency to confusion in the diagnosis in female patients.
5. The characteristic gross and microscopic picture.

Case 6.—C. M., white female, housewife, age 23 years. Patient first seen on October 11, 1937, complaining of a constant dull ache in her right lower abdomen since the birth of her last child 1 year previous. She also complained of irregular menses, the periods recurring at intervals varying from 2 to 6 weeks but tending to come early.

Examination revealed moderate tenderness without rigidity over the right lower quadrant, old bilateral cervical lacerations, tenderness high in both fornices and the right ovary enlarged and tender.

LAPAROTOMY was done on November 13, and the vermiform appendix and an enlarged cystic right ovary removed.

PATHOLOGICAL REPORT: *Gross.*—The specimen consists of a markedly cystic ovary measuring 5 cm. in diameter. All the cysts contain clear fluid except one which contains clotted blood. They measure up to 1 cm. in diameter. The appendix is 7 cm. long. The surface is smooth and shiny.

Microscopic—Ovary.—All the cysts are folicular in type except one which contains old blood surrounded by luteal tissue. Three small tubercles are noted around the edge of a corpus albicans, one of which contains a giant cell of the Langhan's type.

Appendix.—All sections show a tuberculous destruction of the lymph follicles, with many small tubercles and the presence of many giant cells typically of the Langhan's variety.

Diagnosis.—Tuberculosis, ovary; tuberculosis, appendix. (See fig. 15.)

POSTOPERATIVE CONVALESCENCE was uneventful and nonmorbid and patient has remained in good health since. Upon the completion of her convalescence she was very thoroughly studied from the standpoint of tuberculosis but no other foci were found. These studies have been repeated at intervals of 6 months since and have included stereoscopic chest x-rays, physical and radiographic examination of the lymph glandular system, blood sedimentation rates, TPR recordings, investigation of the urinary system with pyelograms and guinea pig inoculations, and study of the skeletal system but all findings continue to be negative for evidence suggestive of a tuberculous lesion.

COMMENT: This case represents an example of a specific granuloma involving the appendix, namely, tuberculosis, whether primarily or secondarily cannot be determined. The evidence favors primary appendiceal involvement, as the appendix was much more extensively invaded than was the ovary, suggesting that it was the first involved.

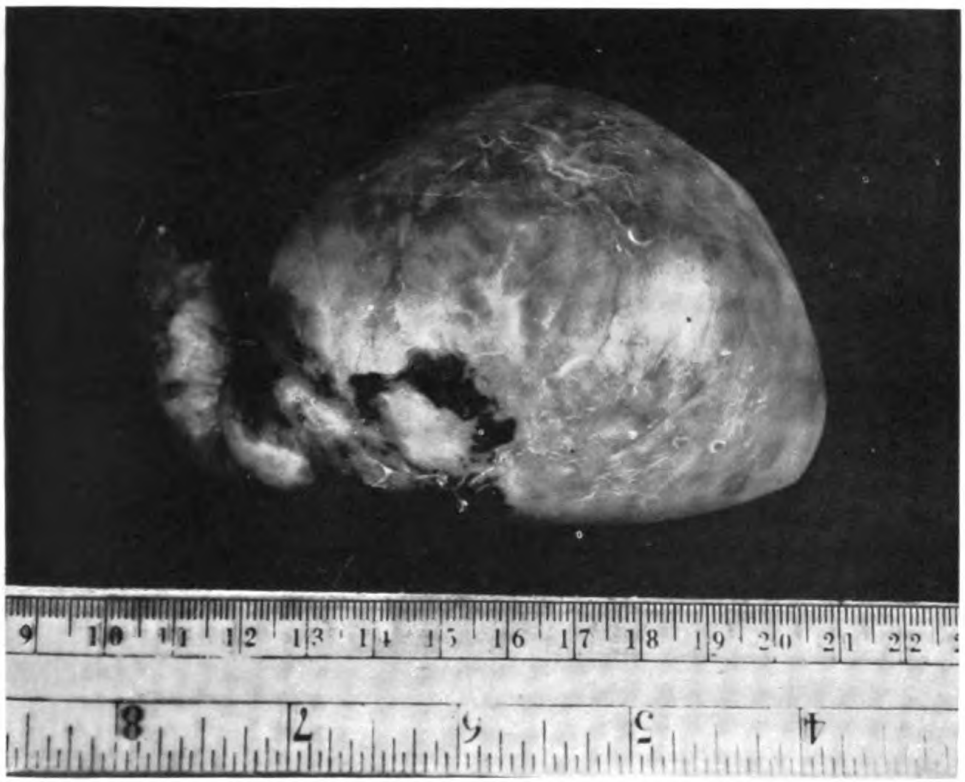

FIGURE 13—CASE 5.—TYPICAL MUCOCELE OF THE APPENDIX. THE TUMOR CONTAINED 300 CC. OF VISCID FLUID. NOTE POINT OF CONSTRICTION TO THE LEFT.

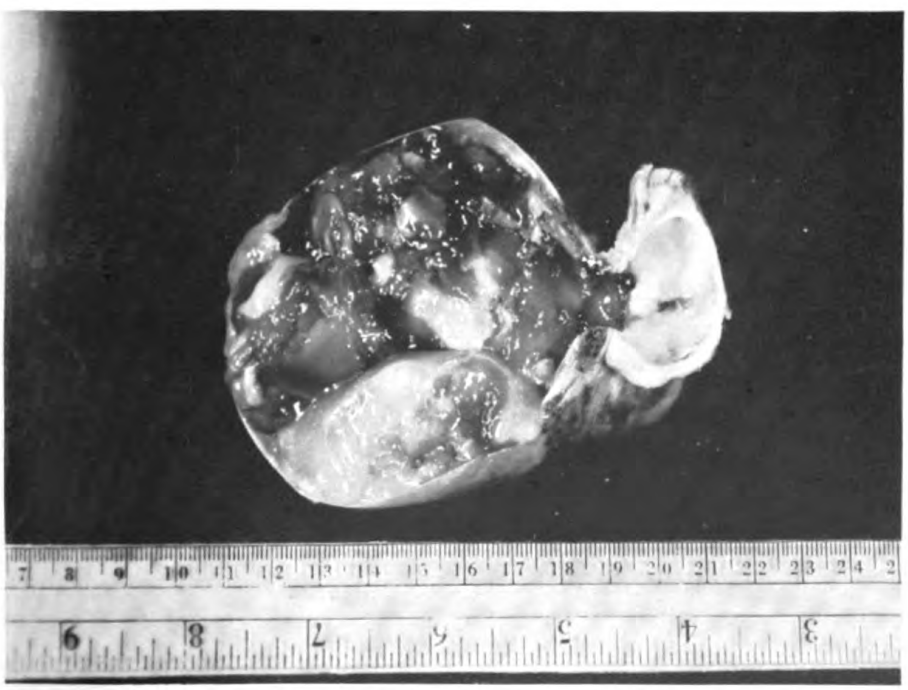

FIGURE 14—CASE 5.—MUCOCELE OPENED BY INCISION FROM TIP TO BASE. NOTE POINT OF CONSTRICTION PROXIMAL TO BASE (RIGHT) AND THE DIFFERENCE IN THE CHARACTER OF THE LINING PROXIMAL AND DISTAL TO THIS POINT.

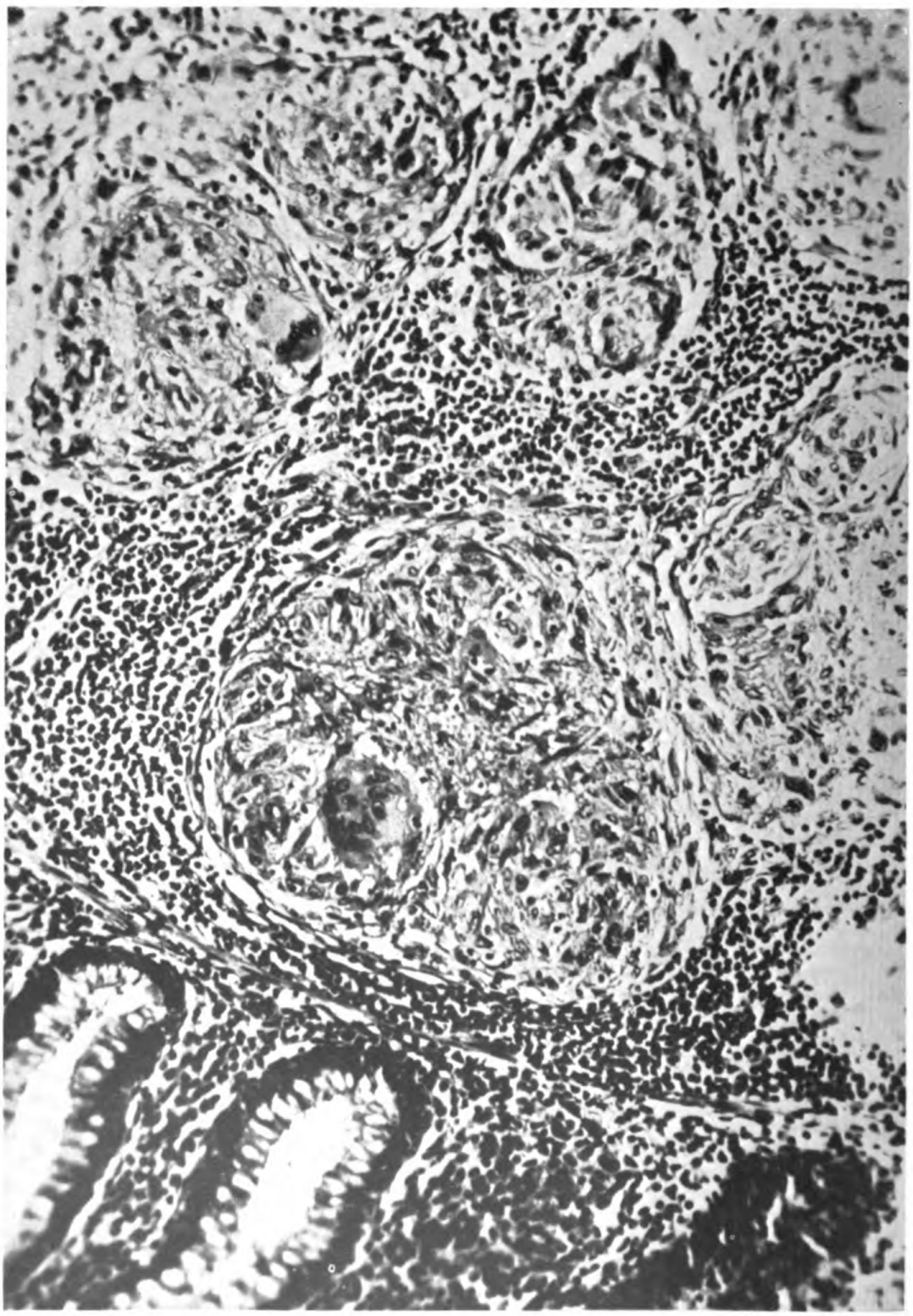

FIGURE 15—CASE 6.—CLASSICAL EXAMPLE OF TUBERCULOSIS OF THE APPENDIX. PRIMARY TUBERCLES WITH SURROUNDING ZONE OF EPITHELOID CELLS AND CONTAINING FOREIGN BODY GIANT CELLS OF THE LANGHANS TYPE ARE SHOWN. APPENDICEAL GLANDS ARE SEEN IN THE LOWER LEFT. (X 400.)

SUMMARY

A brief review of appendiceal tumors is presented. Cases representing examples of the various types of these lesions are reported. The lack of statistical accuracy in the failure to distinguish clearly between adenocarcinoma and carcinoids of the appendix is emphasized. Some suggestions as to therapy are advanced.

CONCLUSIONS

1. Tumors of the appendix are not as rare as they are popularly believed to be. Appendiceal adenocarcinoma, however, is a genuinely rare lesion.

2. Distinct differentiation should be made between the two cancer-like appendiceal neoplasms not only for the sake of scientific accuracy but also because a precise distinction is important from the standpoint of prognosis and treatment.

3. The diagnosis of a tumor of the appendix cannot be made prior to operation and is most often made only by the pathologist.

4. Simple appendectomy constitutes adequate therapy for a vast majority of tumors of the appendix. Radical surgery merits consideration when the lesion is proved malignant.

5. Frozen sections with immediate histopathological examination during operation should prove of value in appendixes presenting a definitely suspicious appearance.

6. Since fairly early and widespread metastasis can occur from an appendiceal carcinoma, radical surgery should be resorted to as soon as conditions permit.

7. As it is frequently impossible to detect or suspect the presence of tumors of the appendix by gross inspection at the time of operation, and as neoplasms of this organ, occasionally malignant, are not infrequent, it should be axiomatic to remove all appendices at the time of laparotomy unless there are definite contraindications.

NOTE.—I wish to thank Dr. I. B. Tilden for making all the photomicrographs shown in the illustrations, for the pathological description of cases 2, 4, and 5, and for the privilege of reporting case 4; Commander M. D. Willcutts (M. C.), United States Navy, for permission to report case 3; Commander N. Roberts (M. C.), United States Navy, for allowing me to abstract cases 2 and 6; Dr. C. E. Fronk for permitting me to use case 5; and Lieutenant W. M. Silliphant (M. C.), United States Navy, who made the pathological studies of case 1.

BRAIN ABSCESS

CLINICAL OBSERVATIONS ON TWENTY PROVEN CASES[1]

By Lieutenant Commander Gerald W. Smith, Medical Corps, United States Navy[2]

The diagnosis of brain abscess is frequently difficult, especially in the case that is devoid of any history or physical finding of an infectious process on examination. The purpose of this paper is to discuss the symptomatology of brain abscess and to review the clinical observations in 20 proven cases from Dr. Temple Fay's neurosurgical records.

Emphasis is placed on the importance of the leukocyte count, spinal fluid cell content, and the erythrocyte sedimentation rate as aids in differentiating brain abscess from brain tumor, and also on the value of these laboratory tests for determining the propitious time for surgical intervention.

It is extremely important to determine if a patient has a brain abscess, and once knowing it, it is equally important to make a correct neurological localization of the lesion, for satisfactory treatment cannot be instituted otherwise. Satisfactory treatment depends on accurate timing of the surgical intervention, for if done prematurely or too late in the course of the disease, the outcome is usually fatal to the patient. Frequently it is almost impossible to differentiate clinically the brain abscess from the brain tumor case and every possible clinical and laboratory aid must be used in order to come to a correct diagnosis.

In patients showing cerebral symptons that have developed subsequent to an inflammatory process in the middle ear, mastoid cells, nasal accessory sinuses, intrathoracic cavity, skull, or osteomyelitic processes elsewhere in the body, it is not usually difficult to make the diagnosis of brain abscess. However, the diagnosis of brain abscess is often hard to make in patients having some focalizing neurological signs with some evidence of increase in intracranial pressure but without noticeable evidence of infection in the nervous system or elsewhere in the body. It is usually these cases which are wrongly diagnosed and operated for brain tumor. Coleman states that it is often difficult to determine whether the cerebral mass is tumor or abscess, especially if in the frontal lobe, and cites three cases where an unnecessary osteoplastic flap was made on patients with erroneous diagnosis of brain abscess.[3] Grant and others have enucleated a cerebral mass only to learn afterward that the mass was a brain abscess.[4]

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[1] Presented before the staff meeting, Temple University School of Medicine and Temple University Hospital, January 12, 1940.

[2] On assignment, for instruction, to the department of neurology and neurosurgery, Temple University School of Medicine, clinic of Dr. Temple Fay, Temple University Hospital, Philadelphia, Penn.

[3] Coleman, C. C.: Brain abscess, a review of 28 cases with comment on the ophthalmologic observations. J. A. M. A. 95: 568, Aug. 1930.

[4] Yaskin, J. C., Grant, F. C., and Groff, R. A.: Brain abscess of undetermined etiology. Ann. Surg. 107: 492, April 1938.

Eighteen of the twenty brain abscess cases in this group came to operation. Two of the group died before operation was performed and the diagnosis was confirmed by autopsy.

In most instances the primary cause of brain abscess is quite obvious for there is a definite history of some inflammation in the neighboring parts or elsewhere in the body. However, in two of the cases in this group, no primary infection was ascertained by careful history, physical examination, and x-ray study. Six cases of the group had primary infections in the middle ear or mastoid cells; 6 followed accessory nasal sinus infection; 3 followed head injuries, 2 were subsequent to primary pulmonary infection and 1 developed secondary to a carcinoma of the scalp.

The most frequent and important cause of brain abscess is by direct extension from disease of the middle ear, mastoid cells or of the accessory nasal sinuses. Evans reports a series of 186 cases of brain abscess wherein 121 had their primary source of infection in these regions.[5] The infection readily passes to the sigmiod sinus from the roof of the mastoid antrum and produces an infective thrombosis and by retrograde propogation, infection is carried into the substance of the cortex. Macewen found that the infective agents may be transmitted through the lymph and blood channels without producing an area of meningitis and that the infections of the cerebellum are most likely to be caused from infection extending from the mastoid cells, while abscess in the temporal lobe is usually caused by infection extending from the roof of the tympanic cavity.[6] It has been noted by Coleman that a brain abscess seldom occurs subsequent to an acute process in the middle ear or mastoid cells but only in the chronic infections of these regions.[7]

Head trauma may be followed by brain abscess and it is not necessary that the trauma be severe enough to produce a skull fracture. The trauma may be slight and the appearance of symptoms of brain abscess may occur weeks or months afterwards.

Eighteen patients of the group are male, 2 are female. In a group of 27 brain abscess cases listed by Alpers, it is noted that only 5 are female.[8] Other writers have noted the high rate of incidence in the male though Evans in his large group found the incidence about equal in both sexes.[5]

Brain abscess following injury or operation frequently is ushered in by symptoms of rigor, high fever, delirium and signs of increase in intracranial pressure. More often, however, its mode of onset is insidious. Superimposed on some already existing infection there

[5] Evans, W.: Pathology and etiology of brain abscess. Lancet 1: 1231, June 1931.
[6] Macewen, Wm.: Pyogenic Infective Diseases of the Brain and Spinal Cord, Macmillan Co., New York, 1893.
[7] Coleman, C. C.: Brain abscess resulting from aural and sinus infection. Radiology 21: 59, July 1933.
[8] Alpers, B. J.: Abscess of brain. Arch. Otolaryng. 29: 199, Feb. 1939.

may appear symptoms of headache, mental dullness, irritability, restlessness and a low grade septic fever. During this period of invasion of the brain by the bacteria, a localized cerebritis or encephalitis is developing with perhaps more or less meningeal irritation depending on the site and extent of the disease process. During this stage, as pointed out by Woltman, there may be a throwing off of polymorphonuclear cells into the spinal fluid.[9]

Globus and Horn show that in the early stages of suppuration in the brain, the beginning of capsule formation may be discerned. It is characterized by proliferation of blood vessels and migration of lymphocytic elements into the zones surrounding the suppurative focus. This is shortly followed by the metamorphosis of lymphocytes into fibroblasts and by progressive organization of the changing extravasated elements, with the result that a distinct protective wall is formed, separating the intact brain from the abscess.[10] It is noted that the time for formation of the capsule around the abscess varies considerably. For instance, Homen observed reparative changes beginning on the fifth to seventh day in experimental brain abscess.[11] Westphal saw evidence of capsule after 10 and 17 days.[12] Eagleton noted a capsule formation in a brain abscess after 17 days.[13] Huguein saw no capsule at 32 days, a delicate membrane at 53 days and a thick capsule at 83 days.[14] But Kaplan on the other hand stated that it takes 6 weeks for formation of a capsule from the time of development of the abscess.[15] Alpers has shown that the extent of the encapsulation depends upon the virulence of the invading organism and upon the resistance of the patient.[7] Sometimes no capsule is formed after infection by very virulent organisms and the result being a rapidly spreading encephalitis. It has long been recognized that operation before encapsulation of the abscess has taken place invariably produces unfavorable results.

As encapsulation of the brain abscess progresses the temperature is lowered, the headache diminishes in severity and tends to become more unilateral in character, the leukocytosis is less pronounced, the polymorphonuclear cells in the spinal fluid are reduced in number and may be superseded by lymphocytes.[9] During the latent stage of the inflammatory process, surgical intervention is indicated and the results of drainage of the abscess at this stage are good.

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[9] Woltman, H. W.: Spinal fluid cell count and encapsulation of brain abscess; attempt to correlate these factors and to determine the optimal time for drainage. J. A. M. A. **100**: 720, Mar. 1933.

[10] Globus, J. H. and Horn, W. L.: Inherent healing properties of abscess of the brain. Arch. Otolaryng. **16**: 603, Nov. 1932.

[11] Homen, E. H.: Experimentelle und Patholische beitrage zur kenntnes der Hernabszesse. Arb. a. d. path. Inst. d. Univ. Helsingfors. **1**: 1, 1913.

[12] Westphal, A.: Ueber Gehernabscesse. Arch. f. psychiat. **33**: 206, 1900.

[13] Eagleton, W. P.: Brain Abscess. Macmillan Co., New York, 1922.

[14] Huguenin, G.: Encephalitis und hernabszess in von ziemssen, Handbuch der speziellen pathologie und therapie, Leipzig. F. C. W. Vogel, **2**: 331, 1876.

[15] Kaplan, A.: Abscess of the brain. Arch. Otolaryng. **21**: 385, Apr. 1935.

In some instances the encapsulated mass continues to enlarge and the neurological signs presented are identical to those found in brain tumor. If the ventricular system is encroached upon, signs of increase in intracranial pressure such as rapid choking of the optic disks, slow pulse, vomiting, rise in blood pressure with an increasing pulse pressure, dulling of the sensorium and stupor will ensue. Rupture of the abscess into the ventricle or through the cortex may occur producing a terminal meningitis. These eventualities may occur if drainage of the abscess has been delayed too long. However, Grant points out that many more mistakes are made by operating too soon than by operating too late for in his series of 19 cases operated before the capsule was formed there was a 100 percent mortality, while in 30 of his cases operated after encapsulation, there was only 33.3 percent mortality.[16]

The symptoms of brain abscess may be grouped into the general and the focal, the former being due to the increased intracranial pressure and toxic factors, while the focal neurological signs depend upon the site and extent of the cerebral lesion and differ in no way from those found in any other cerebral lesion.

Headache was the most common sympton found in this group of cases. It was present in every case and in 12 instances out of 20, it was unilateral and on the side of the lesion. The headache usually begins as an early morning complaint and lasts for an hour or more. Gradually it increases in severity and in duration until it becomes constant. It is a congestive type of headache, increased with straining, coughing, and on stooping over. Percussion of the head frequently accentuates the headache and elicits tenderness over the area involved.

Nausea and vomiting occurred in 11 patients in the group. It was not the typical projectile type of vomiting so commonly ascribed to increase in intracranial pressure though projectile vomiting did occur in the 3 children of this group.

A subnormal temperature was present in only 3 patients of the group. Three had normal temperature, 9 had a low-grade septic temperature between 99° and 101° F., and in 5 cases the temperature ranged from 101° to 106° F.

A slow pulse rate has long been known to be suggestive of brain abscess (Gower,[17] Oppenheim,[18] Bailey,[19]). This was noted in 8 patients in the group but it was not constant. It is advisable to chart the TPR. q.2 hr., for occasionally the pulse becomes commensurately slow and unless frequent readings are taken this observation will be missed.

[16] Grant, F. C.: Mortality from abscess of the brain, J. A. M. A. 99: 550, Aug. 1937.
[17] Gowers, W. R.: A manual of diseases of the nervous system, 2: 482, Phila. 1893.
[18] Oppenheim, H.: Lehrbuch der nervenkrankheiten kargeu, 2: 1359, Berlin 1923.
[19] Bailey, P.: Intracranial Tumors. C. S. Thomas, p. 411, 1923.

Choking of the optic disks was present in 14 cases of the group. The choking was usually bilateral and it was not noted that the greater degree of choking occurred on the side of the lesion. Coleman found that choking of the disk occurred in about the same frequency in brain abscess as in tumor. He did not find any relationship between the size of the abscess and the degree of choking.[8] Frequent ophthalmoscopic examinations were made and accurate measurement of the degree of disk choking determined as advocated by Lillie who has pointed out that a rapidly developing choked disk indicates a changing cerebral process.[20]

Mental dulling and drowziness were present in all the patients in the group except those who were definitely stuporous or unconscious. The stupor and mental dullness seemed out of proportion to the degree of increase in the intracranial pressure in these patients, due probably to the toxic factor associated with the increased intracranial pressure. Kennedy has remarked about this disproportionate drowziness in these cases as compared with brain tumor cases.[21]

Eight of the group had generalized convulsions at some time before operation. One patient who was suspected of having a brain abscess subsequent to a pneumococcic type VIII meningitis from which he had recovered, had Jacksonian type of convulsions which aided in the neurological localization of the process. The convulsion was preceded by an aura described as a burning, stinging sensation in the region of his anus. Subsequently a tonic-clonic convulsion would start in the left buttock which extended to the left lower extremity, to the left upper extremity and to the left side of the face. The lesion was localized in the posterior portion of the right paracentral lobule for this is the area for sensory innervation in the cortex of the contralateral anal region. A trephine opening over the area by Dr. Fay uncovered the abscess which was located just below the cortex and proved to be the size of a large walnut.

In the presence of considerable increase in intracranial pressure, focal neurological signs are frequently hard to evaluate and hence the accurate localization of the lesion is difficult. All of the patients in this group showed either mental dullness or stupor which masked, to some extent, the focal neurological signs. Fay has stressed the proper interpretation of intracranial pressure and volume relationships in management of cases with high intracranial pressure.[22] Very often after 24 hours of dehydration the patients became more alert and the focal neurological signs became crystallized, enabling correct localization. As so often is the case, operation must be performed before the

[20] Lillie, W. I.: Clinical significance of choked discs produced by abscess of the brain. Surg. Gynec. & Obst. 47: 405, Sept. 1928.

[21] Kennedy, Foster: Diagnosis of abscess of the brain. Arch. Neurol. & Psychiat. 22: 627, 1929.

[22] Fay, Temple: Treatment of acute and chronic cases of cerebral trauma by methods of dehydration. Ann. Surg. 101: 76, Jan. 1935.

optimal time because of the increasing intracranial pressure. By controlling this intracerebral mechanism, one is often able to delay surgical intervention long enough to enable further encapsulation of the abscess.

TABLE 1.—*Location of brain abscess in the group of cases reviewed*

Site	Right	Left	Total
Frontal lobe	4	2	6
Parietal lobe	1	1	2
Occipital lobe	1	1	2
Temporal lobe	1	5	6
Cerebellar hemisphere	1	2	3
Multiple			1
			20

All the patients in this group had some neurological findings of localizing value. (See table 1.) Of the 6 patients with frontal lobe abscess, 4 had definite personality change, such as indifference, apathy, untidiness and carelessness in their personal habits, while 2 presented the Witzelsucht reaction characterized by unusual facetiousness. The pupils were unequal and larger on the homolateral side of the lesion in 3 instances and smaller on the lesion's homolateral side in 1. In 15 patients in the group, pyramidal tract signs such as positive Hoffman and Babinski reactions, exaggeration of the deep reflexes, suppression of the abdominal reflex and impairment in voluntary muscular control, were observed resulting in a hemiparesis in 12 cases and a hemiphlegia in 3 instances. Sixth nerve involvement causing a weakness of the external ocular muscle and internal squint was present in 4 patients but this finding could not be considered of localizing importance because of the accompanying increase in intracranial pressure. Aphasia occurred in 4 cases. A contralateral homonomous hemianopsia was demonstrated in 1 patient with a temporal lobe lesion. This important localizing sign requires considerable cooperation on the patient's part and as most of these patients are usually mentally dull or stuporous its presence often could not be demonstrated. The 2 patients with abscess of their cerebellum showed the classical cerebellar syndrome of asynergia, dysmetria, hypotonia with suppression of deep reflexes on the lesion's homolateral side. The results of the Barany test aided in localization of the lesion.

Roentgenological examination of the skull was made in all but 3 patients of the group. The pineal gland was found to be calcified in 5 instances and in 4 cases, measurement showed a shifting from its normal position in the brain. In 6 cases ventriculogram was done and in 8, encephalogram was performed. These procedures when well timed cause no untoward effects and offer considerable aid in localizing the lesion.

The laboratory observations are interesting. In only 8 cases of the group were erythrocyte sedimentation rates determined. (See table 2.) It was not found to be normal in a single instance. In 2 cases the sedimentation rate ranged from 15 to 20 mm. per 60 minutes; in 4 cases it was between 20 and 25 mm. per 60 minutes and in 2 cases it was found to be between 30 and 35 mm. per 60 minutes. This observation of a consistently high sedimentation rate in all the patients examined is considered significant and one more lead in differentiating the brain abscess from the brain tumor in the obscure case. It is our routine here to determine the erythrocyte sedimentation rate on every patient having a suspected intracranial space taking lesion.

TABLE 2.—*Sedimentation rate, millimeters per 60 minutes, in 8 of the cases reviewed*

Case	1	2	3	4	5	6	7	8
Millimeters	17	20	20	30	15	32	21	24

A leukocytosis was present in every case in the group except one. The white cell count was between 9,000 and 12,000 per cu. mm. in 6 cases; from 12,000 to 15,000 in 3; from 15,000 to 20,000 in 7; from 20,000 to 25,000 in 3; and in 1 instance it was above 25,000. It is noted that in the patients with a white cell count above 15,000, 75 percent terminated fatally, whereas cases in the group having a white blood count below 15,000 the mortality was only 13 percent.

Spinal puncture was done on 14 patients in the group. Four cases showed a spinal fluid cell count of 5 to 10 per cu. mm.; 2 were between 10 and 20; 1 was between 40 and 50; and 5 ranged from 50 to 100. It is noted that only 1 patient with a spinal fluid cell count of over 50 recovered. The spinal fluid did not show xanthochromia in any instance. In our experience the presence of xanthochromia points toward brain tumor rather than brain abscess. The spinal fluid pressure ranged from 15 to 60 mm. of hg. in 11 cases. Three patients of the group had normal spinal fluid pressure.

COMMENT

In this group of cases, along with the general and focal neurological signs present, one or more laboratory findings of an infectious process was present in every instance: (*a*) An increase in cells in the spinal fluid; (*b*) an increase in the number of polymorphonuclear neutrophile cells in the blood; (*c*) or an increase in the erythrocyte sedimentation rate. It is pointed out that in every case where a brain tumor is suspected, attention must be given to these three laboratory findings for in some cases of brain abscess no lead from the history or physical

examination as much as even suggests that the lesion is abscess rather than tumor. The rapidity of the growth of the intracranial lesion gives very little help in differentiating the abscess from the tumor, for a hemorrhage into a glioma will produce a rapid development of focal and general neurological symptoms similar in many respects to that found in a rapidly expanding abscess. As Foster Kennedy points out:

> In the absence of etiological factors pointing to a source from which abscess formation could take origin the diagnosis of brain abscess is almost impossible.[21]

For this reason every possible study must be made to aid in differentiating abscess from tumor and we feel that the sedimentation rate determination is of value and that its importance is overlooked on many neurosurgical services.

No extensive report on the erythrocyte sedimentation test in brain abscess could be found in the literature. Cutler's experience with the sedimentation test in over 5,000 patients during a 6-year period emphasizes that the test is not specific for any particular disease, that the mechanism is obscure, and that the phenomenon depends on the amount of cellular destruction going on in the body. In his list of diseases giving abnormal sedimentation rates he mentions localized suppuration such as mastoiditis.[23]

Operative procedures should be avoided if possible while the leukocyte count exceeds 15,000 in the blood and 50 in the spinal fluid and while the temperature is above 101° F., for these observations indicate the presence of a diffuse cerebritis or meningeal involvement. Surgical drainage at this stage causes a dissemination of the infection.

The correct timing of the operation is probably of more importance than the actual method used. Whether one uses the repeated aspirations as recommended by Dandy,[24] King's [25] method in which unroofing and herniation are the principal features, Kahn's [26] decompression for migration, Coleman's [27] subtemporal decompression synchronous with aspiration to prevent the ill effects of secondary intracranial pressure, or the conventional tubular drainage, depends largely upon the individual case. In any type of operative procedure the correct pre-operative neurological localization of the lesion is imperative and the prevention of any trauma to the area surrounding the abscess during the operation is of most importance. It is well to remember Frazier's remark that "an abscess in the making is not a surgical lesion" [28] and delay operation until the clinical signs indicate that

[23] Cutler, J. W.: The practical application of the blood sedimentation in general medicine; observations based upon approx. 5,000 patients over a period of 6 years. Am. J. Med. Sc. 183: 643, May 1932.

[24] Dandy, W. E.: Treatment of chronic abscess of the brain by tapping. J. A. M. A. 87: 1477, Oct. 1926.

[25] King, J. E. J.: Treatment of brain abscess by unroofing and temporary herniation of the abscess cavity with the avoidance of the usual drainage methods. Surg. Gynec. & Obst. 39: 554, Nov. 1924.

[26] Kahn, E. A.: Treatment of encapsulated brain abscess. J. A. M. A. 108: 87, Jan. 1937.

[27] Coleman, C. C.: Treatment of abscess of the brain. Arch. Surg. 18: 100, Jan. 1929.

[28] Frazier, C.: How shall we treat brain abscess? Surg. Gynec. & Obst. 57: 122, July 1933.

encapsulation of the abscess has occurred. This time varies in individual cases but the consensus of opinion is that it requires from 4 to 6 weeks after the onset of the cerebral symptoms.

SUMMARY

1. The symptomatology of 20 proven cases of brain abscess is reviewed.

2. Six cases of the group had primary infections in the middle ear or mastoid cells; 6 followed accessory nasal sinus infection; 3 followed head injuries; 2 were subsequent to primary pulmonary infection and 1 was secondary to carcinoma of the scalp. In 2 cases no primary infection could be found.

3. Headache was present in every case and in 12 instances it was unilateral and on the side of the lesion. Nausea and vomiting occurred in 11 cases. Subnormal temperature was present in 3 cases; 3 had normal temperature; and in 14 instances the temperature was elevated. Eight patients showed a slow pulse rate. Choking of the optic disks occurred in 14 instances. All the group had mental dullness or stupor. Sufficient focal neurological signs were present in all the cases to make accurate localization. Sedimentation rates were increased in 8 cases on which the test was done. Nineteen of the group had some degree of leukocytosis and the spinal fluid cell count was increased in 10 out of the 14 cases that had lumbar taps performed.

4. Attention is called to the value of a careful analysis of the temperature curve, white blood count, spinal fluid cell content and sedimentation rate as an aid in diagnosis of the obscure brain abscess case.

5. Surgical drainage of brain abscess when the white blood count is above 15,000, spinal fluid cell count is above 50, or when the temperature is over 101° F., in our experience is exceedingly dangerous and should be avoided if possible.

OPHTHALMOLOGICAL FACTS, FADS, AND FALLACIES

By Lieutenant R. C. Boyden, Medical Corps, United States Navy

Too many synthetic ocular symptoms are accepted by the medical profession without scientific investigation. Irrational treatment with a great variety of medication and corrective lenses, predicated solely on symptomatology, is too frequently prescribed, often with harmful results.

The following observations are the result of the writer's findings and opinions only. It has been noted that, in the Navy, the number and variety of complaints of an ocular nature have been on the increase in the past several years. This may be due to any one or combinations of the following factors:

1. Misinformation circulated by retailers of glasses whose profits vary from 3 to 22 dollars on each sale, and health articles pertaining to ocular symptoms due to so-called eye strain.
2. Exaggerated belief in the value of glasses for the relief of symptoms.
3. Neurotic fixation or inadequate personality.
4. Definite malingering and desire to excite sympathy.
5. Reluctance to make cruises because of family or other ties ashore.

The term *synthetic symptoms* has been used to characterize the complaints voiced by patients in which no pathology can be demonstrated. The examinations used to rule out pathology were complete and included all of those well known to those doing ophthalmological work. The symptoms most frequently encountered were:

1. Headache: (*a*) Usually bitemporal, and cast in the same mould with the headaches of neurosis or hysteria.
(*b*) Occurs after long or short periods of reading.
2. Burning sensation in the eyes: Following periods of reading or movies.
3. Blurring of vision: Following short periods of reading or study.
4. Poor vision: Usually unilateral.
5. Sensitivity to light.
6. Postorbital pain.
7. "Pulling sensation".
8. Muscular twitching of lids.
9. Diplopia.
10. Astigmatism: A popular expression among the laity and occasioning a garrulous recital of previous examinations and opinions of unscientific advisors.
11. "Eye-strain": A general symptom complex embracing any one or several of the above symptoms.
12. Blindness: (*a*) Unilateral or bilateral.
(*b*) Sudden onset.

REASONS FOR THIS ARTICLE

1. The increasing frequency with which young healthy men recently accepted in the naval service with normal vision are appearing in naval ophthalmological clinics with synthetic symptoms and routine requests for refraction.

2. The incessant drain on the medical officer's time, averaging 1 hour and 15 minutes per patient, in ruling out possible pathology in these individuals, and the resultant loss of time available for examination and treatment of those who present themselves with genuine symptoms based on organic lesions.

3. The hope that these observations will impress the readers with the value of careful preliminary examinations. Without leveling criticism against the medical officers afloat or on independent duty, it is believed that much time will be saved for all concerned, in the cases presenting synthetic symptoms, if the ophthalmoscopic and other preliminary examinations are carefully done. Such cases should be scuttled in their incipiency and not referred to the clinics ashore.

Merely signing a slip for refraction tends to impress on the patient's mind the feeling that he really must have a serious ocular disturbance or the medical officer would never have referred his case to others.

4. An attempt to assist in diminishing the inevitable loss of man working hours in the naval service incurred while these patients are ashore awaiting special examinations.

DISCUSSION OF SYMPTOMS

HEADACHE: This is the most commonly encountered synthetic symptom. It is usually bitemporal, dull and throbbing, lacking regularity in onset, and appears following slight or excessive use of the eyes.

It is significant that these headaches sometimes appear when the eyes are being used for distance, when all the eye muscles and the accommodation processes are at rest.

The patients commonly, when questioned by the medical officer, exhibit a characteristic anxious frown. They delineate the picture of dejection, and not realizing that there may be other causes than ocular for their headache, they unconsciously simulate the ancient Chinese medicine man and locate their pathology near the headache. The majority, after a few minutes of simple explanation, gladly drop their temporary ocular neurosis, built up by suggestion, and occasionally intensified by the busy medical officer who does not take time to explain possible reasons for symptoms, but merely signs a request for refraction. Many of them seen at the clinic are pleased to be assured that their vision is normal and that the purchase of glasses would be a needless expense. This assurance could be given by the doctor afloat after a few simple examinations.

Occasionally, these headaches are complained of by patients with neurotic, hysterical, or inadequate tendencies, seeking a fixation symptom as an outlet for unpleasant situations or duties. In these cases a satisfactory solution is more difficult, but frequently obtained by more emphatic suggestive therapy.

BURNING SENSATION: This may be, and frequently is, associated with the third of the common triad, blurring of vision. The patient states that the burning and blurring occur as a result of reading or typing. The vision is practically always normal. It seems strange that complaints like these are relatively infrequent among our book reviewers, civilian stenographers, or laboratory workers who do close work over long periods. Here again, through suggestion, the patient is led to believe that these symptoms must be due to refractive errors. My experience has been that retinoscopy rarely reveals refractive errors in these cases. I have found, however, that many of these patients have been using various types of "eye-drops" over some

period of time, and it is my impression that, disregarding the synthetic symptoms, the genuine may be due to ill-advised medication.

Poor vision: This symptom must be investigated by one of several well-known methods designed to detect deception. It is frequently encountered among those who wish to get out of the service before their enlistment expires. It lends itself to those who have not the mental stature to exhibit more complicated symptoms.

Frequently, inability to read test charts properly with one or the other eye is the complaint in those with normal vision. The use of simple tests, such as crossed cylinders, plus and minus 2.50 D at right angles on both eyes and singly neutralizing them, reveals normal vision in the supposedly "bad" eye, without the patient's knowledge. This deception is also exposed by the use of the Maddox rod on the good eye and a double prism exactly centered on the "poor eye."

It is true, that vision is seldom equal in otherwise normal eyes, but as a rule the inequality is more frequently on the plus side than the minus. For example, the subject can, and frequently does, read below the 20/20 line with one eye. Occasionally the vision will be 20/20 in one eye and 16/20 in the other. In any event 20/20 is an arbitrary standard. Here the slight variation from this arbitrary standard does not necessitate the prescribing of lenses for the relief of eyestrain, for true eyestrain, if such an entity exists, is caused by fatigue of the muscles of convergence, and slight refractive errors do not overwork these muscles. As a safeguard against stress, strain, or injury the eye is provided with a reserve capacity of over 50 percent for ordinary desk work. Many cases have come to my attention in which marked myopic or hyperopic astigmatism were discovered, with no history of any of the usual symptoms of eyestrain.

In contrast to this, consider the frequent complaints of young healthy men in the naval service, who insist that they have headaches, burning and blurring of vision after only 15 minutes of reading. It is difficult for me to believe that these young people with good accommodation and normal vision in each eye can be the victims of eye strain; and the inconvenience of glasses for these small physiological errors is greater than the relief obtained. The only one who profits is the dispensing optician. It is physiologically wrong to add corrective lenses to eyes with normal vision, inasmuch as these lenses act as crutches to the eye muscles, and eventually produce true asthenopia.

The word, *astigmatism* is one of the widely known medical terms, which, like sinusitis and others, has been grasped by many, as the solution of their difficulties. The majority, of course, have no knowledge of the true nature of this defect. We know that it is hereditary or congenital, and caused by slight differences in opposing diameters of the eyeball, which produce from childhood, minute distortion of all images cast on the retina. We also know that slight astigmatism is

physiologic, and it is rare to find a truly nonastigmatic eye. This distortion, unless it be excessive, is corrected in the occipital visual cortex as the child matures, and few with small astigmatic errors ever become aware of it unless they are informed by their doctor. I believe it is a mistake to inform patients that they have astigmatism, unless excessive, as it is one expression with which they are familiar. They use it fluently, meaninglessly, and glibly hold it responsible for their synthetic symptoms.

In summation, it may be said, then, that the inconvenience and discomfort produced by lenses prescribed for small refractive errors is greater than the perceptible improvement in vision. Before leaving the subject of poor vision, a word about styes might be useful.

Many parents think that a crop of styes in their children is the result of refractive errors, and point to their neighbors' children who were cured by glasses. I believe that styes are similar in origin to furuncles on other parts of the body, namely, irritation plus infection, and the glasses, by preventing children from rubbing their eyes and introducing infection, cause remission of the infection.

Sensitivity to light: This symptom brings up a matter about which there has been some discussion in the past few years, namely, tinted lenses. It is well known, that spectral rays exert a deleterious effect on various eye structures, when they are allowed access in excessive amounts. More specifically, the rays of the infrared and ultraviolet bands produce changes, primarily in the lens, and secondarily in the retina. Practically, however, the danger from these rays is very slight, and it is only in cases of those engaged in certain industries, such as glass blowers and foundry workers, welders, etc., that protection from them by tinted lenses is necessary.

The visible spectrum exerts, so far as we know, no damage to the eye structures. The eye has a perfect physiological shutter system, which controls to a nicety the amount of light rays allowed to fall on the retina. This shutter system is actuated by two sets of muscles, receiving their stimuli from the amount of light admitted. In placing tinted lenses in front of the eye, shutting out one or more portions of the visible spectrum, these muscles become atrophied, due to disuse; the visual system becomes increasingly sensitive to light, and increasingly unable to cope with this light. The subject becomes aware of the need of tinted lenses more frequently, and with deeper tints. In short, tinted lenses act as crutches to the shutter system of the eye, with resulting distrophy of the system. I believe that the use of tinted lenses for road glare on long trips, for sun glare on the water or beaches and occasionally snow glare is indicated, and in few other conditions.

The lighter tints recommended by various dispensers are of little value in eliminating glare from the visible spectrum, and with the

exceptions noted above, are not necessary for the elimination of harmful rays. They merely add several dollars to the cost of the original prescription. The more deeply tinted lenses, usually found on counters of variety and drug stores, are made of blown ripple glass and are more harmful than useful. I believe that the unscientific and injudicious prescribing of tinted lenses among naval personnel is frequently useless, always expensive, and too frequently deleterious.

POSTORBITAL PAIN: This is a fairly frequent complaint among subjects whose vision, media and eye grounds are **normal**. It is one of the more common symptoms in those in whom psychogenic difficulties are suspected. Ordinarily psychotherapy is indicated, and a cure is brought about by assurance that the eyes and surrounding structures are entirely normal. This assurance must, of course, be preceded by examination, including ophthalmoscopy.

PULLING SENSATION: This, unless heterophorias can be shown to exist, is usually synthetic and of neurogenic or psychogenic origin.

MUSCULAR TWITCHING OF THE LIDS OR FLOATING OBJECTS IN THE LINE OF VISION: These are comparatively infrequent, and it is significant that the patient frequently cannot exhibit them at the time of examination, but can minutely describe them as they occurred previous to examination. They are truly synthetic in origin.

DIPLOPIA: This has been utilized twice in the writers' experience as a means of almost acquiring a medical survey. In both cases there was a history of a blow on the orbit with a demonstrable hyperphoria. The extremely slow convalescence aroused suspicion of malingering. Examination, using phorometer and Maddox rod, and other simple tests revealed that the residual diplopia was largely psychic in origin.

BLINDNESS: This symptom, excluding pathological causes such as intraocular hemorrhages, which are easily demonstrated, and providing the blindness has occurred after maturity, is usually hysteroid in nature, sudden in onset, and has an apparently intolerable situation as the immediate etiological factor. If the blindness is unilateral, its detection is the matter of a few simple tests, such as a strong plus sphere for the good eye and a weak plus or minus for the bad eye, crossed plus and minus cylinders, tinted lenses and letters, the pin cushion test, etc. Bilateral blindness is more difficult to detect and requires close observation of the patient unknown to him, thorough ophthalmological examination, simple tests which will suggest themselves to the examiner and finally suggestive therapy or hypnotism. Two such cases of bilateral blindness have come under the writer's observation and both were cured in this manner.

DELETERIOUS MEDICATION: This article would not be complete without a few words concerning the use of various eye washes. The term *eye wash* is at once physical and mental. It is true that eye

washes flush away various debris from the conjunctival sac. They also flush away forebodings of possible disaster from the minds of patient and doctor alike. They engender a sense of false security and the unexpressed hope that all will go well.

The belief that all eye conditions can be relieved or cured by the use of *drops* is widespread. The solutions most frequently prescribed contain boric acid or one of the silver salts such as silvol. Actually, these are only very mild antiseptics and this mildness is further attenuated by immediate tear dilution. The resultant action, then, is not only useless, but may become harmful if these drugs are used to excess because the introduction into the conjunctival sac of various solutions changes the pH of the tears and causes them to become irritating through loss of their isotonic qualities.

The dangers of argyria and its permanent muddy sclera must be considered. One such case has recently come under the writer's observation.

The long continued use of various ointments on the lids may be harmful, also on account of the variation in pH of the tears. The various proprietary eyewashes, usually expensive, have no especial virtues and rely on camphor water and adrenalin for their bleaching and sparkling qualities.

Frequently the busy medical officer when approached by a patient complaining of burning eyes, blurring, etc., will, after a hasty examination with negative findings, prescribe a solution of boric acid or silvol. The patient's symptoms are relieved in a day or two. If they reappear at some future time the patient this time does not wait in line at sick bay, but procures a supply of one of the solutions and treats himself. He may continue this for months or even years.

Careful examination at the initial visit, accompanied by a few words of explanation, and a suggestion of the use of warm compresses at night, will go far in obviating this self treatment. The false sense of security, founded on the use of eye drops, has often caused serious eye diseases to go untreated for lengthy periods.

It is my belief that solutions of boric acid should be used only for their mechanical cleansing effects, and that other antiseptics, such as silvol, are of value only in certain purulent infections of the eye, and should not be prescribed indiscriminately for all eye conditions.

SUMMARY

Many common ocular symptoms have no organic basis and therefore can be termed *synthetic symptoms*.

This synthesis can be broken down into its components by careful examination and a few simple tests.

As a consequence, the patient can be relieved of worry and needless expense, the outpatient load in the ophthalmological clinics can be

markedly diminished, and finally, a saving in time lost and pensions paid will accrue to the naval service.

EYESTRAIN

By Commander E. C. Ebert, Medical Corps, United States Navy

The diagnosis of eyestrain is made too frequently by our medical officers and causes not only unnecessary expense to the individual, but also the wearing of ophthalmic lenses tends to handicap him. The patient feels that he cannot carry on his duties when lenses are lost or broken. These patients, who actually possess good vision, become a liability instead of an asset to the service.

The busy medical officer at times uses it for quick disposal of patients. The pharmacist's mate uses it because it sounds professional.

This preconceived diagnosis is an easy method of disposing of a mentally troubled patient with a fixation without studying his emotional conflicts or getting an insight to his troubles.

A thoughtless diagnosis of eyestrain and a prescription for very weak ophthalmic lenses usually satisfies the patient for about a month and then he goes to the next oculist or optometrist for a change of lense and he is well on his way to make the rounds of Navy sick bays. Eighty percent of eye consultations for vision among the young men come under this group. An old but true saying is that among these patients, "We produce more neurotics than we cure."

Commercial advertising of ophthalmic lenses contributes a large percent of these cases. Regardless of discount promised on naval prescriptions, this writer has noted that naval personnel pay uniformly 400 to 800 percent above distributor's price on naval prescriptions. Obviously, such a profit may act as a motive that is detrimental to the patient's interest.

A careful study of these cases over a period of years with many cycloplegic refractions shows that young men from 18 to 30, in the Navy, have an average accommodation of 9 to 10 diopters. The chart illustrated in figure 16 was plotted on this basis. The 18-year group has an average of 11 to 12 diopters, or about 1 diopter higher than reported by Thorington. This minor variation is probably accounted for by the elimination of recruits with weak or defective vision. Those with excellent vision are the only ones who are selected for the service.

The following vague and indefinite complaints are usually used by patients who become eyestrain conscious:

1. Headache.
2. Burning of eyes.
3. Watering of eyes.
4. Eyestrain when reading.
5. Light bothers their eyes.
6. Sleepiness when reading.
7. Astigmatism.

Inability to see well is a rare complaint.

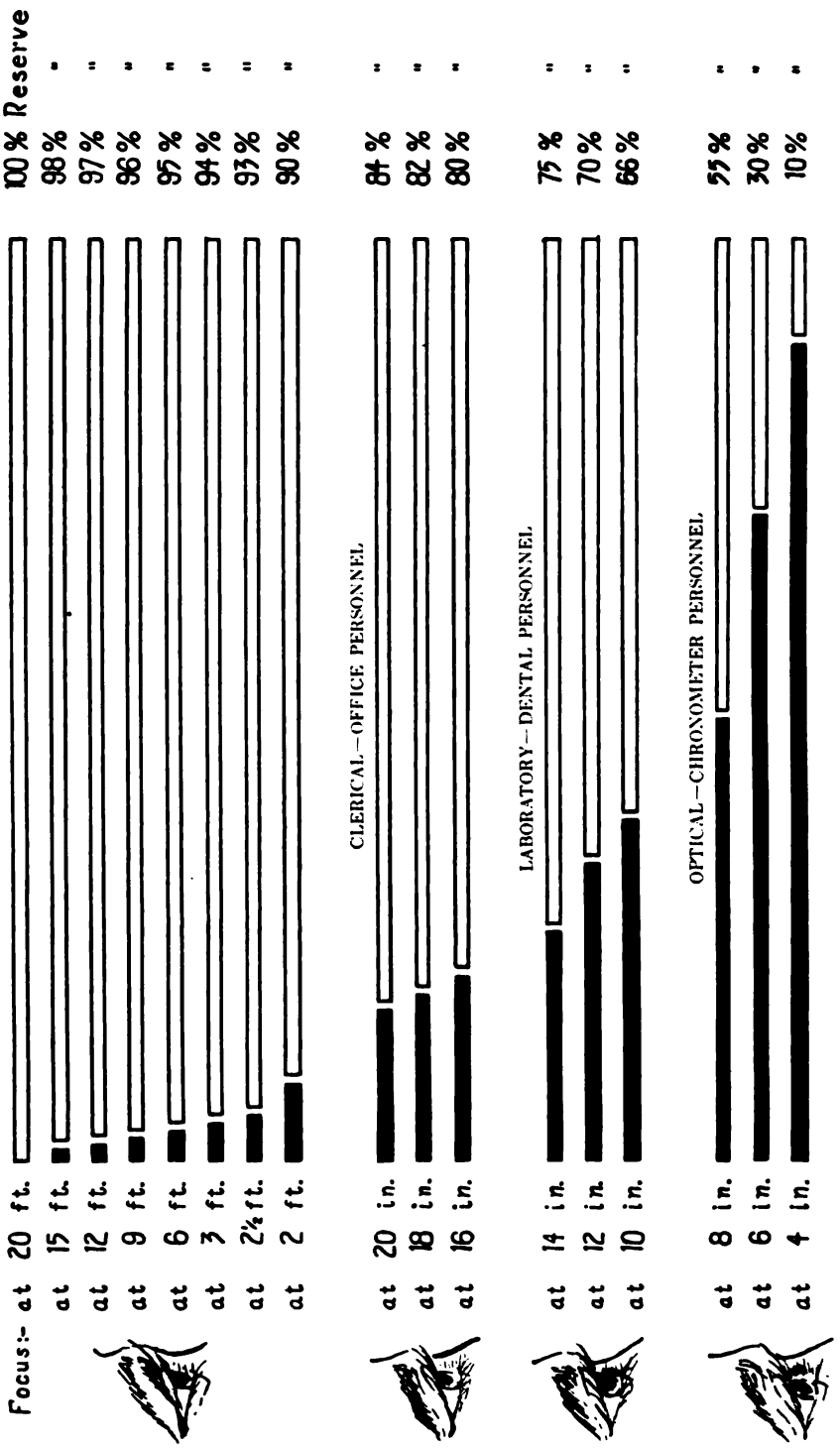

FIGURE 16.—Plotted chart illustrating active vision utilized daily by naval personnel in comparison with actual reserve.

SUGGESTIONS

Patients should be discouraged from becoming introspective relative to ocular fatigue. To accomplish this, eyestrain should not be suggested to the patient since this diagnosis is rarely indicated.

Introspective patients with eyestrain conflicts can usually be relieved of their worries by a few minutes rational explanation that the average young person rarely uses more than one-tenth of his total visual capacity during the day as noted by plotted chart. This is using 1 diopter of accommodation at 3 feet.

INDICATIONS FOR OPHTHALMIC LENSES

Colorless lenses:

1. Presbyopia (old age).
2. Myopia (nearsightedness).
3. Abnormal astigmatism.
4. After cataract operation

Tinted lenses:

1. Strong beach glare.
2. Strong sand glare.
3. Auto or searchlight glare.
4. Acetylene torch glare.

TREATMENT OF CONCOMITANT STRABISMUS

By O. W. Cole, M. D., Long Beach, Calif.

Concomitant squint is frequently met with in the outpatient service of the naval dispensaries. During the past 2½ years the writer has treated more than 100 such cases. The ages of these patients varied from less than 1 year in the youngest, to 46 in the oldest. In addition, lower degrees of strabismus are occasionally met with in active naval personnel. The treatment of strabismus cases is not only interesting, but it is also very gratifying as the results which may be obtained with the proper treatment are almost uniformly good and often appear almost miraculous. The changes wrought in the individual, from cosmetic and psychological standpoints, are most striking and the families of such patients are very appreciative.

The best results are naturally obtained when treatment is instituted early. Unfortunately, however, only a small percentage of these patients come under treatment at an early stage. The usual story is one of prejudice on the part of the patient and the patient's family, toward treatment. Usually this prejudice is the result of poor advice which the family has received from a doctor who has examined the patient before; and frequently it is an ophthalmologist who has given this damaging advice. Often they have been told that an operation on the eye will cause the eye to become blind, and might even cause a loss of sight in both eyes. This usually discourages the patient from seeking any further relief, and makes him fear any treatment which may be suggested for the condition.

The treatment of strabismus is divided roughly into: (*a*) Orthoptic training and (*b*) surgical treatment. In the majority of cases best end results are naturally obtained when these two forms of treatment are combined. Strabismus is either monocular or alternating, that is, either one eye deviates constantly while the other fixes, or else the two eyes alternate in fixation. The patients with alternate strabismus usually have no fusion sense whatsoever and consequently orthoptic training will accomplish little or nothing in these cases. Such patients may be operated on as soon as the degree of squint has remained constant for a period of 6 months, and the patient is old enough to stand a 20-minute general anesthetic. The object in these cases is the attainment of a perfect cosmetic result and the prevention of the development of an inferiority complex by the patient. In general, it is in monocular strabismus that orthoptic training finds its greatest use. In such cases the first thing which must be done is to prevent the patient from developing an amblyopia of the deviating eye. This is accomplished by occlusion of the fixing eye for a period of 6 hours or more daily, thus forcing the patient to see with the eye which tends to deviate. The patient is encouraged to use the open eye, particularly for near work, during the period in which the sound eye is covered. If the patient is old enough to cooperate, fusion exercises should be begun. Orthoptic training must be continued faithfully for a long period of time if success is to be attained.

The number of operations which have, in the past, been proposed for the correction of strabismus attest to the inadequacy of most of these procedures. In general, however, they consist of shortening or advancing one ocular muscle, or of lengthening or receding the opposing muscle.

The shortening operations which are now in general use consist of:

1. ADVANCEMENT OF THE INSERTION of an ocular muscle finds its greatest value in the treatment of a postoperative divergence which has developed following an operation for convergent squint, in which the internal rectus has been tenotomized or receded too far. In other cases it has several objectionable features which render it inferior to certain other methods of shortening. It is a more difficult procedure than other methods, and the occasional operator will have few if any cases in which it cannot be replaced by a safer, more exact, and less difficult form of operation.

2. RESECTION OF A PORTION OF THE TENDON and suture of the cut ends of the muscle tendon and tendon stump is only occasionally used and is not a safe procedure to use, as the sutures may slip and a tenotomy of the muscle results instead of the shortening which was originally sought.

3. TENDON TUCKING is a safe procedure since the tendon is not cut, but is merely folded upon itself and sutured in this position. It has certain objectionable features however, the principal of which are: (*a*) Partial failure, due to overstretching of muscle fibers or slipping of the tuck because of insecurely tied sutures; and (*b*) prolonged tumefaction over the tucking area.

The writer uses tendon tucking occasionally to shorten the external rectus in very high degrees of convergent strabismus in adult patients.

4. THE O'CONNOR CINCH OPERATION seems to be the answer to all the requirements of a good tendon shortening operation. It consists of dividing the tendon into several strands, usually 4 to 7, then lacing a bundle consisting of 4 to 10 strands of dermal suture about the strands of tendon and pulling on the bundle of dermal, thereby transferring the loops from the dermal to the tendon strands, as shown in the accompanying diagram (fig. 17):

This operation may be used in all cases where a shortening is desired. It has many advantages. It is a simple and easy procedure and can be performed in as little time as any other type of shortening operation. The amount of shortening obtained is definite, as there are no sutures which may slip. The tendon is not cut. The insertion of the tendon is not altered in any way whatsoever. This operation is also adjustable within wide limits as to the amount of shortening desired. If too much shortening is obtained, some of the dermal strands may be removed, and the amount of shortening is thereby decreased. Certain objections have been raised to the cinch operation, chiefly of which are: (*a*) Premature escape of the dermal sutures before the tendon has had time to heal in the shortened position. In approximately 60 cases in which the writer has used this operation this accident has never occurred. (*b*) The danger of infection. The writer has never had a case of infection following the use of this procedure and does not believe that any infection which may occur in these cases would be due to the type of operation chosen, but rather to either injudicious operation upon a patient

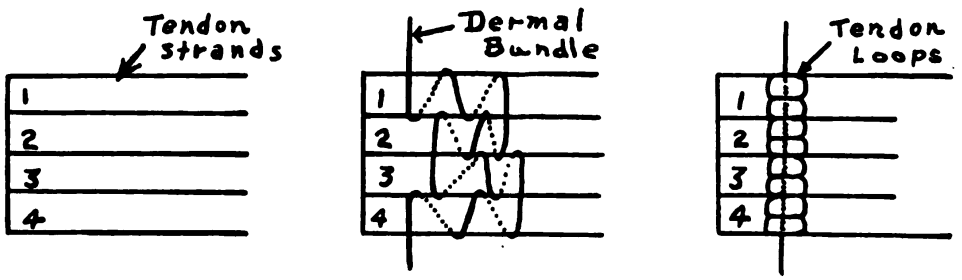

FIGURE 17.—The O'Connor cinch operation.

whose conjunctival flora already harbors an infection, or to improper sterile operative technic.

The lengthening operations comprise:

1. COMPLETE TENOTOMY consists of cutting the tendon of a muscle near its insertion and allowing it to retract. This procedure, which was once widely used, is beyond a doubt the most unsatisfactory and most dangerous of all the operations which have been used in treating strabismus. There is not a single feature to support the use of a complete tenotomy in the treatment of concomitant squint, and it is mentioned only to be strongly condemned.

2. PARTIAL OR GUARDED TENOTOMY is a secure and useful procedure, but has certain definite limitations. In the manner in which it is frequently used it is quite haphazard and gives practically no effect. However, if carefully performed, it does not need to be so. Of the many forms of partial tenotomy, perhaps the best is the central tenotomy in which all of the fibers of the tendon are severed except the very few fibers which are left intact at the upper and lower borders of the tendon. The tendon and anterior portion of the muscle fibers must first have been carefully freed from the adhesions to the underlying sclera. Although the amount of correction which may be obtained is limited, it is often sufficient when used in connection with a shortening operation of the opposing rectus muscle.

3. RECESSION OPERATION consists of cutting the tendon near its insertion and resuturing it to the sclera, several millimeters behind the original insertion area. The Jameson and Peters recession procedures are the most satisfactory forms of this operation. If too great a recession is done, an enophthalmos may result and on the other hand if an ocular muscle is shortened too much, an exophthalmos results. In order to avoid these undesirable occurrences, it is usually advisable to combine a shortening of one ocular muscle with a lengthening of its antagonist if the degree of squint which it is desired to correct is very marked.

The accompanying photographs (fig. 18), taken of patients before and after operation, clearly illustrate the cosmetic changes which may be attained. Frequently the patient's entire facial expression is changed, due not only to the straightened eye, but largely to the improved psychological outlook. The improvement in the patient's personality is often even more striking than the improvement in appearance. After surgical correction of the angle of squint, the orthoptic training should be continued under careful supervision with a final view to establishment of single binocular vision in all cases where it can possibly be obtained.

MALIGNANT MELANOMA OF THE ORBIT

WITH CASE REPORT

By Lieutenant Commander Fred Harbert, Medical Corps, United States Navy

About one-third of all melanomas are primary in the choroid but melanoma of the orbit is a comparatively rare disease. The choroid is the favorite site but the ciliary body may also be the point of origin. Fuchs distinguishes 4 stages:

1. Before glaucoma.
2. Glaucomatous.
3. Extra-ocular growth.
4. General dissemination.

The above is not necessarily the order of occurrence for cases are seen in which generalization may occur with apparently insignificant primary lesions. Other cases resemble the one reported in that there is marked extra-ocular growth without metastasis. Excision in the intra-ocular stage results in cure in 60 to 80 percent of cases and local recurrence is very rare. After extra-ocular extension, the prognosis rapidly becomes worse. Metastasis occurs via the blood stream to the liver, brain, stomach, lungs, lymph nodes, bones, and other tissues in the order named frequency. The age incidence is chiefly between 40 and 50 years, but a few cases have occurred in infants.

The cellular structure of melanomas varies. Some are nonpigmented, others may show pigmentation in some organs and not in others. The distribution of pigment even in the same organ often varies markedly. Primary melanomas must be distinguished from

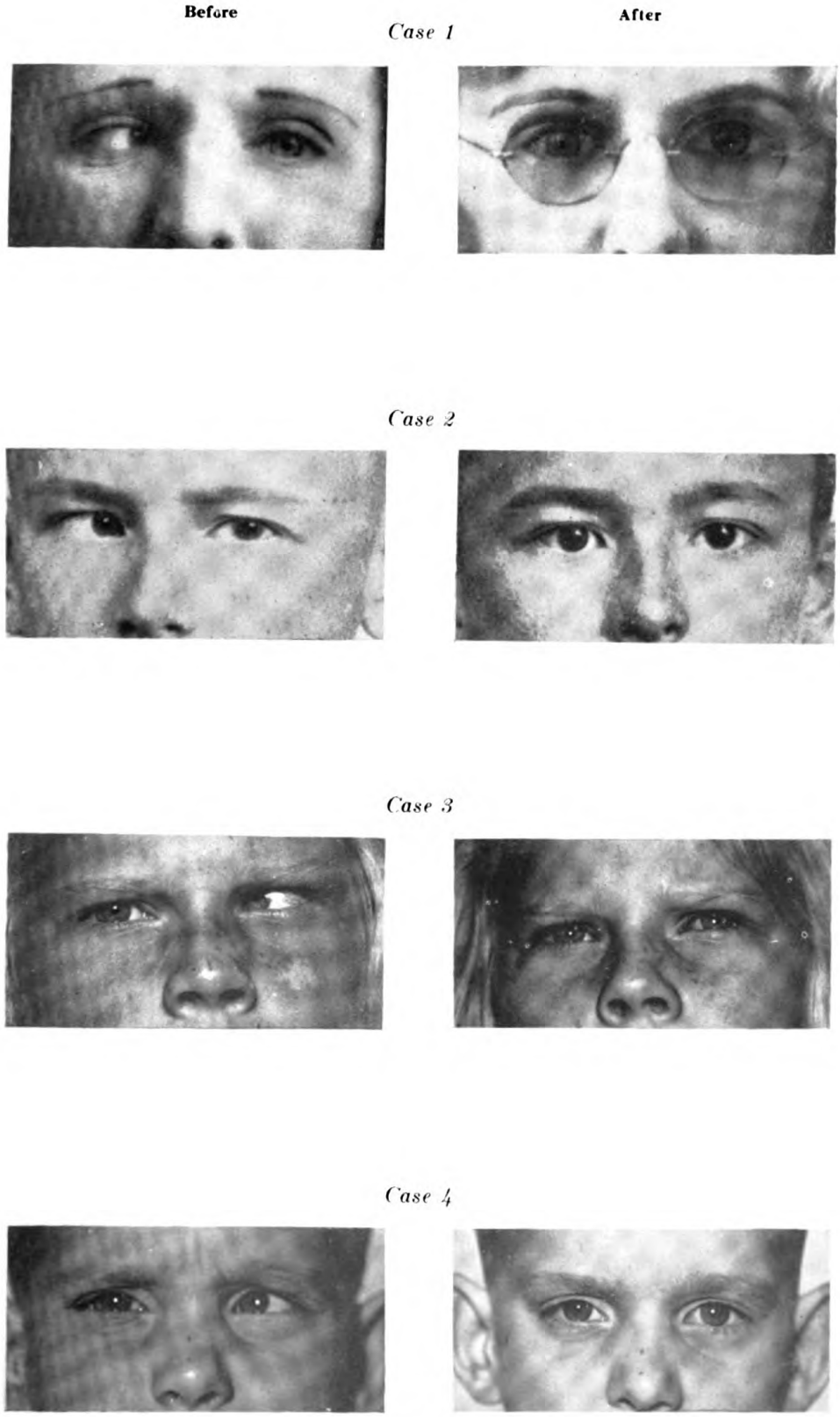

FIGURE 18.—ILLUSTRATING RESULTS OF OPERATIVE TREATMENT OF CONCOMITANT STRABISMUS.

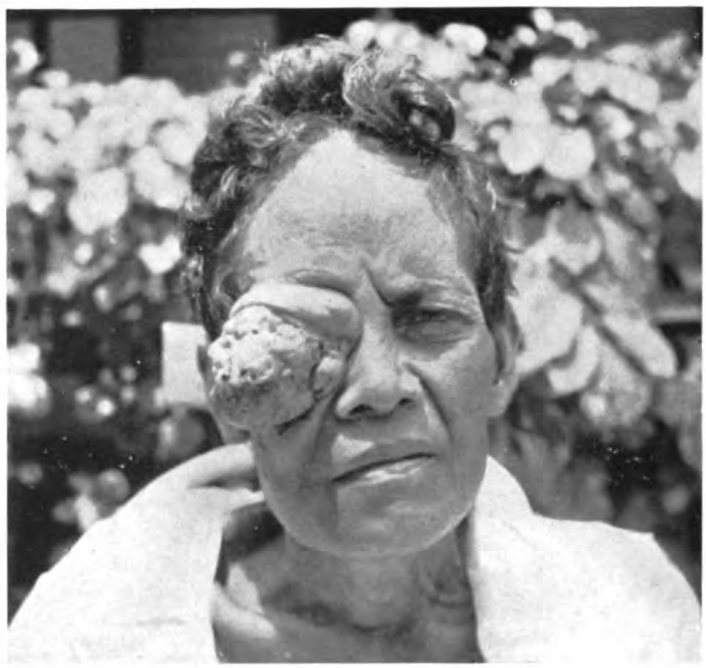

FIGURE 19.—FUNGATING TUMOR MASS

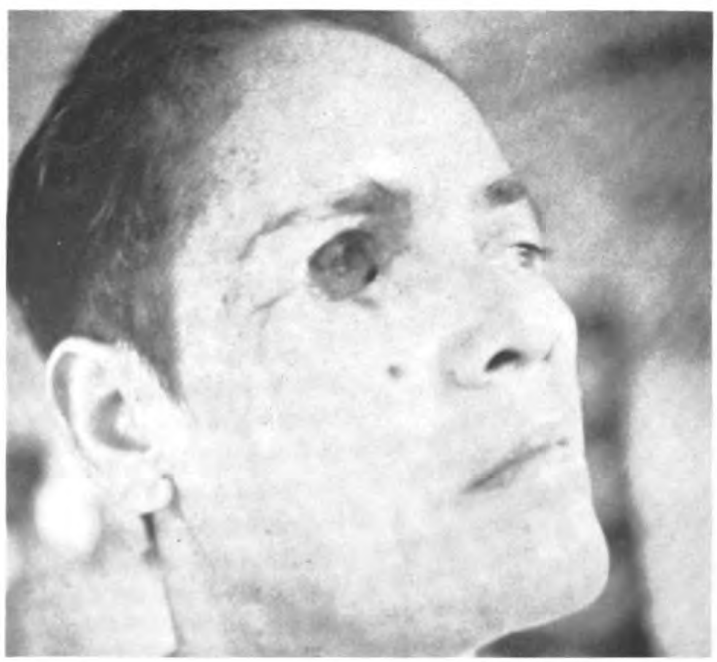

FIGURE 20.—ORBITAL DEFECT FOLLOWING REMOVAL OF TUMOR.

tumors showing secondary pigmentation. Two types of cells are recognized, namely, the spindle cell, resembling sarcoma, and the large polyhedral cell with a tendency to alveolar arrangement. The latter are sometimes concentrically arranged about blood vessels which led to the name of perithelioma or endothelioma.

The structure of the tumor in the following case report indicates its origin from the uveal tract. The marked local growth without evidence of metastasis indicates that surgery may be curative even in advanced cases.

Case Report

M., Samoan, female, age 42, admitted to the Samoan Hospital, December 9, 1936, complaining of a growth in the right orbit.

Present illness: The neoplasm was first noticed as a protuberance in the pupillary region of the right eye 6 months previously and was accompanied by severe and almost constant pain in the right side of the head and neck. Later the growth ruptured the eyeball and continued to grow outward. Since then the pain had not been so severe. As the tumor enlarged she steadily lost weight and felt feverish. She denied any previous illness or injury.

Physical examination: The patient was a native female Samoan who appeared considerably older than stated age because of well-marked emaciation. The right eye was entirely replaced by a large fungating mass of new growth and granulation tissue which invaded both lids and protruded from the cheek. (See fig. 19.) The mass bled easily when touched and there were several sinuses exuding pus. There was no cervical or other adenopathy and no masses were palpable in the abdomen. Physical examination was otherwise essentially negative except for pallor of the mucous membranes indicating anemia. X-ray of the chest and long bones showed no evidence of metastases.

Clinical course: December 12, 1936. The temperature ranged from normal to 100 degrees daily. Blood findings: RBC, 2,890,000; Hgb, 60 percent; WBC, 11,800, juveniles, 1 percent; bands, 8 percent; segmented, 70 percent; lymph, 13 percent; eosins, 8 percent. The Kahn test was 4 plus, which is the usual finding in nearly all native adults due to yaws infection. The sedimentation index showed a rapid drop to 26 mm. in 60 minutes. She was placed on antiyaws treatment and continuous hot boric acid compresses to reduce secondary infection.

Operation: Exenteration of the right orbit was performed under chloroform anesthesia December 15. A preliminary external canthotomy was performed to facilitate dissection of the large growth. The line of cleavage between periosteum and bony wall of the orbit was located in the canthotomy wound and a blunt dissector introduced. An incision was then made all around the orbital margin to the bone. By swinging the dissector in an arc, the orbital contents with periosteum attached were completely freed from the bony orbit except for the attachments at the apex which were severed by scissors. During the dissection the orbital plate of the ethmoid bone was inadvertently pierced by the dissector. The ciliary margins of both lids, all of the palpebral conjunctiva, and portions of the skin of the lids showing infiltration with neoplasm were excised. Portions of remaining healthy skin were laid into the orbit to help epithelialize the bony defect. Careful inspection showed no evidence of residual new growth. The apex of the orbit containing fat was cauterized by the actual cautery as an additional precaution. The wound was packed with gauze.

December 22. The patient reacted well to the anesthetic and operation. Her temperature fluctuated between normal and 101 degrees daily. When the packing was removed the wound was clean except for an eschar at the apex of the orbit.

December 26. The temperature was normal all day for the first time since her admission to the hospital. The orbit was irrigated daily with permanganate solution and lightly packed with gauze. Hot compresses were employed to facilitate separation of the eschar.

December 30. A small slough was seen at the site of the accidental perforation of the lamina papyracea of the ethmoid. Irrigating fluid entered the nose.

January 5, 1937. Patient was gaining weight and strength on cod-liver oil and full diet. The orbit showed healthy granulations at the base of the eschar.

January 9. Patient's weight was 104 pounds. Chloramine irrigations were used daily to stimulate granulations.

January 16. The temperature, pulse, and respiration continued normal. The palpebral skin flaps were found to be firmly attached to the bony wall of the orbit.

January 23. Granulations from the apex of the orbit were slowly spreading along the bony wall of the orbit. Areas of new epithelium were also spreading from the transplanted palpebral skin. After separation of the slough from the ethmoid cells there was a temporary localized ethmoiditis. The exposed ethmoid cells were now clean and beginning to fill in with granulations.

January 30. Granulations nearly covered the entire surface of the bony orbit which were not epithelialized. Exuberant granulations were reduced by touching with silver nitrate sticks.

February 2. Thiersch skin grafts from the right thigh were placed on the cleansed granulations of the orbit. The blood picture was now RBC, 3,510,000; Hgb, 70 percent; WBC, 8,100.

February 16. About two-thirds of the grafts had taken. The exposed ethmoid cells were clean and the fistula into the nose appeared to be closing spontaneously.

March 11. Her weight was now 123 pounds. The orbital defect was almost epithelialized but there is still annoying scab formation. (See fig. 20.) X-rays of the chest and long bones showed no evidence of metastases.

PATHOLOGICAL FINDINGS, reported by the pathologist, United States Naval Medical School, Washington, D. C.:

Gross specimen consists of a white, fungating mass which measures 7.5 by 4.5 by 3.5 cm. On cut section it is white throughout, rather firm and has a somewhat granular appearance. In the center of the mass is a cavity lined by a black membrane which appears to be choroid of the eye. No other remnants of eye could be found.

Microscopical section through various areas of the white part of the orbital mass shows large, clear, round and polyhedral cells in sheets and grouped in alveolar or pseudo-peritheliomatous arrangement. Cells show many mitotic figures. There are strands of connective tissue running throughout the section, and many pus cells. No pigment is present. Sections from near the center of the specimen show the same structure, but there is considerable melanin present. Sections from around the cavity show a line of flattened cells loaded with pigment. There is no evidence of a retina. Around one edge is considerable relatively acellular connective tissue and few bundles of striated muscle fibers.

Diagnosis: Malignant melanoma of the orbit.

A communication from Samoa dated September 19, 1939, reports this patient is well and free from symptoms.

CHRONIC THYROTOXIC MYOPATHY [1]

REPORT OF A CASE COMPLICATED WITH DIABETES MELLITUS

By Lieutenant (Jr. Gr.) Michael V. MacKenzie, Medical Corps, United States Navy

INTRODUCTION

Generalized muscular weakness and the occasional atrophy of individual skeletal muscles are not uncommon findings in hyperthyroidism. Means [2] points out the frequency with which atrophy of the temporal and interosseous muscles will be found if the patient is carefully inspected. The muscular disturbances which may occur in hyperthyroidism have been summarized recently by Brain [3] under the term "the thyrotoxic myopathies." His classification is as follows:

1. Acute thyrotoxic myopathy: A rare form of bulbar palsy usually fatal in a week or two.
2. Chronic thyrotoxic myopathy: A progressive muscular atrophy of insidious development with little or no bulbar involvement. The wasting is most evident in the pelvic girdle and thighs and is symmetrical. Fibrillary twitching may be seen but is rarely as conspicuous as in true progressive muscular atrophy. The tendon reflexes may be diminished or lost.
3. Thyrotoxic periodic paralysis: A rare condition which is probably identical with familial periodic paralysis. The attacks last from several hours to 2 to 3 days.

The second of these conditions, chronic thyrotoxic myopathy, is extremely rare or at least infrequently reported. In 1934, Ayer, Means, and Lerman [4] published what they believed to be the fifth description of an actual case. Searching the literature since then with reasonable care, I have been able to find only two additional cases reported.[3] It is in conjunction with these that Brain's classification of the thyrotoxic myopathies appears.

The main features of all the reported cases are consistent with one another and with the case I shall presently describe. So striking was the degree of generalized muscular atrophy in the majority of them that the coexistent hyperthyroidism was at first either overlooked or thought to be of only incidental significance. Indeed, in one instance [4] the wisdom of a thyroidectomy was debated, because it was thought that the muscular atrophy, as an unrelated disease, had a poor prognosis. The clear-cut relation between the two conditions was not appreciated until the disappearance of the myopathy following operation. The specific curative effect of thyroidectomy on the skeletal musculature is illustrated in two of the cases [3] by remarkable before and after photographs.

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[1] From the U. S. Naval Hospital, Philadelphia, Penna.

[2] Means, J. H.: The Thyroid and its Diseases. J. B. Lippincott Co., 1937. p. 319.

[3] Starling, H. J., Darke, C. S., Hunt, B. W., and Brain, W. R.: Two cases of Graves' disease with muscular atrophy. Guy's Hosp. Rep. 88: 117, Jan. 1938.

[4] Ayer, J. B., Means, J. H., and Lerman, J.: Simulation of progressive muscular atrophy by exophthalmic goitre. Endocrinology, 18: 701, 1934.

The following case is reported as being possibly the eighth recorded instance of chronic thyrotoxic myopathy. The fact that diabetes mellitus is a complicating factor in this case is of additional interest.

Case Report

A 46-year-old white male was admitted to the naval hospital August 24, 1938, complaining of loss of weight and strength of 8 months' duration.

Past history: In 1929 he had experienced similar but less severe symptoms and was found to be suffering from moderately severe diabetes. This condition was easily controlled by a diet and 5 to 10 units of insulin daily. He recovered completely and continued in excellent health until January 1938. At that time, following an acute upper respiratory infection, weight loss and asthenia again set in, and within 3 months he had lost 30 pounds. His physician was unable to find either a cause for his condition or a change in his diabetic status, and the patient entered a local hospital for study. He remained there for 5 months and underwent a complete diagnostic work-up, which included two basal metabolism determinations. He continued to lose weight and strength; his diabetes became more difficult to control and when he was discharged, several days before his admission here, no diagnosis had been made and he was taking 40 units of protamine insulin daily together with an unlimited diet.

When admitted here, he had lost a total of 47 pounds during the 8 months of his illness. His appetite had remained excellent throughout. Diarrhea, dyspnoea on mild exertion, cough, tremor, nervousness, palpitation, and excessive perspiration were symptoms which he definitely recalled, but which were overshadowed in his mind by extreme weakness and fatigue. He had remarked a visible loss of muscle substance accompanying this asthenia and weight loss but did not recall any single location in which a loss of strength or substance first began. Hoarseness and difficulty in swallowing had developed within the few weeks before admission. On more than one occasion he either saw or felt localized twitching in the muscles of his arms and legs.

Physical findings: The general appearance of the patient was arresting. Observed from across the room he appeared cadaverous and cachectic; his bony framework was so prominent that he resembled a skeleton over which a covering of parchment-like skin had been drawn taut. A more intimate inspection of the patient, together with an appreciation of his alert, somewhat agile, bright-eyed and apprehensive demeanor, precluded ordinary cachexia as the explanation of his wasted appearance. His subcutaneous tissue was firm and apparently of normal amount. The skin overlying his extremities was of normal elasticity; over the thoracic cage it was drawn with the tightness of a drum-head and was extremely resistant to attempts to pinch it into a fold. In this region it presented a consistency not unlike that of a block of paraffin wax.

Tissue loss and the relative prominence of the skeleton was almost entirely due to a partial loss of voluntary musculature from head to toe. This was most noticeable in the erector spinae group, in the shoulder girdle, in the flattened thenar and hypothenar eminences, and in the calves of the legs. There was a commensurate loss of muscular strength. His grip was weakened bilaterally. In attempting to rise from a supine to an erect position, he found it necessary to roll over on his abdomen, kneel, and climb up his legs in the manner of children with muscular dystrophy.

The remaining significant physical findings may be briefly summarized. The palms and conjunctivae were definitely pale. The eyes showed a suggestive stare, protrusion of the eyeballs, infrequent blinking, and a slow lateral nystagmus on extreme deviation. The left pupil was smaller and the left palpebral fissure

narrower than those on the right. The thyroid gland was not palpable. This important negative finding was confirmed by two other observers. There was a noticeable huskiness to the voice; enunciation, however, was unimpaired. Hyperresonance, distant vesicular breathing, sibilant expiratory rales and a relatively immobile thoracic cage suggested advanced emphysematous changes. The radial and brachial arteries were sclerosed. The external jugular veins were bilaterally distended to a vertical distance of about 10 cm. above the level of the auricles. Sinus tachycardia, a systolic murmur loudest at the apex, and a blood pressure which fluctuated from minute to minute between the limits of 105 and 130 mm. of mercury systolic, and 80 to 100 mm. of mercury diastolic were the only other significant cardiovascular findings. The abdomen was carinated, slightly tender and generally rigid. The neurological findings included a fine tremor of the tongue and outstretched fingers, infrequent fibrillary twitching of the muscles induced by active motion or massage, and absent abdominal reflexes. There was no loss or comparative change in the tendon reflexes, but when attempting to elicit the quadriceps response, it was noticed that individual fiber groups responded irregularly and in sequence, so that a jerkily sustained extension of the lower leg was produced. On a subsequent examination, no response could be obtained even with reinforcement.

The neurologist could find no evidence of fibrillary twitching at the time of his examination. All the tendon reflexes except the ankle jerk were present and evaluated as 1 plus. The patient was unable to squat and resume a standing position without falling. In the opinion of the neurologist the clinical picture did not conform to any of the known muscular dystrophies or atrophies, and he believed that a metabolic origin was the probable cause of the muscular wasting. At the time of his examination, the results of the basal metabolism tests had not been reported.

On the basis of the history and physical examination, the patient was considered to have diabetes mellitus, secondary anemia, arteriosclerosis, chronic pulmonary emphysema, and progressive muscular atrophy. In addition, a provisional diagnosis was made of "endocrinopathy; possibly thyrotoxicosis."

SPECIAL EXAMINATIONS: The results of laboratory studies were as follows: Five complete blood counts revealed a moderate secondary anemia and a relative lymphocytosis. Expressed in milligrams per 100 cc., the levels of blood urea, cholesterol, calcium, and phosphorus were 18 mg., 186 mg., 9.4 mg., and 3.7 mg., respectively. Five days after admission, the fasting blood sugar level was 150 mg. per 100 cc. and sugar was present in the urine, although the patient was on a diabetic diet and had received between 15 and 20 units of insulin daily during the preceding 5 days. Except for the anticipated glycosuria, the urine was not remarkable; at no time during hospitalization did ketonuria occur. No ova or parasites were found in the stools on two occasions. The sedimentation rate of the red blood cells was 6 mm. in 1 hour. Kahn tests on the blood and spinal fluid were negative.

A radiogram of the chest revealed moderate enlargement of the pulmonary conus, calcification of the aortic knob, a general accentuation of the linear trunk markings, and moderate osteoporosis of the bones in the shoulder girdle. Two preoperative electrocardiograms revealed sinus tachycardia (rate 110); P–R intervals of 0.16 second and 0.20 second; a Q–R–S interval of 0.10 second; a tendency toward right axis deviation; and an early convexity of the R–S–T interval in lead IV with a sudden drop below the iso-electric line. The postthyroidectomy electro-cardiogram revealed a normal sinus rhythm and pulse rate of 80; a P–R interval of 0.16 second; a Q–R–S interval of 0.11 second; and a diphasic T-wave in lead IV, manifesting less inversion than on the previous records.

Four determinations of the basal metabolic rate varied from plus 33 percent to plus 45 percent.

OPERATION: On September 9, a thyroidectomy was advised by the surgical consultant. By September 30, the day of operation, the patient's diabetes was stabilized on a 2,200–2,500 calorie diet and 20 units of insulin administered daily. His basal metabolic rate had been reduced to plus 12 percent on iodine medication. He had lost an additional 10 pounds during his 6 weeks of hospitalization. At operation, a subtotal thyroidectomy, the gland was found to be diffusely enlarged with its lateral lobes wrapped closely around the trachea.

On November 11, six weeks following operation and approximately 12 weeks following admission, the patient was discharged from the hospital. Subjective improvement was complete. His weight of 120 pounds on the day of discharge represented a gain of 17 pounds during the postoperative period although, because of continued preoperative weight loss, the actual gain since admission was only 10 pounds. He presented a visible filling-out of his musculature and most of his marked asthenia had disappeared. Basal metabolic rates varied from minus 14 percent to minus 1 percent. His diabetic status showed rapid improvement and he left the hospital practically sugar-free, maintained on a 2,500 calorie diet and the administration of 5 units of protamine insulin daily.

COMMENT

Although the condition presented by this patient was unfamiliar and puzzling, its identification with an extremely rare clinical entity was unfortunately not recognized until shortly before the patient left the hospital. Consequently, the scientific value of this report is lessened by the omission of several special procedures, the necessity for which has only been realized in retrospect. Glucose tolerance tests and values of urinary creatine should have been determined before and after thyroidectomy, as should also the electrical response of the skeletal muscles. A dermatologist should have been consulted with respect to the peculiar condition of the skin of this patient's thorax.[5] A biopsy of skeletal muscle would have been valuable because of the reputed rarity of myopathological reports in severe thyrotoxicosis.[6]

The possibility that these cases of chronic thyrotoxic myopathy are actually instances of true progressive muscular atrophy complicated by an incidental thyrotoxicosis has been given careful consideration. To anyone familiar with the latter more common neurological disorder, the many gross differences between the two conditions are readily apparent. It is true that the thyrotoxic myopathy may be confidently diagnosed as progressive muscular atrophy when the case is first seen, but the explanation for this error lies in the average clinician's lack of familiarity with either or both conditions. The moderate depression of the deep reflexes and the fibrillary twitching

[5] Because of the supposed relation between scleroderma and disfunction of the thyroid gland. See Osler's Principles and Practice of Medicine, edi. 12, D. Appleton-Century Co., New York, 1935, pp. 903 and 1131. In a case of total atrophy of one-half of the entire body, scleroderma was also a feature. Finesilver, B. and Rosow, H. M.: Total hemiatrophy. J. A. M. A. 110:366, Jan. 1938.

[6] Means, J. H.: The Thyroid and its Diseases. J. B. Lippincott Co., 1937, p. 292.

spell progressive muscular atrophy to the uninitiated. The fact that the extent of the atrophy is out of all proportion to the slight amount of central nervous system involvement is overlooked. The ultimate criterion, of course, is the specific curative effect of thyroidectomy. While this test cannot be said to have been applied rigidly to the case reported here, the marked parallelism to the previously reported cases in other particulars leaves little doubt as to the ultimate outcome.

The occurrence of fibrillary twitching in several of these cases of thyrotoxic myopathy is interesting, inasmuch as the phenomenon is so commonly thought to be a sign of degeneration of the anterior horn cells of the spinal cord. Grinker,[7] however, doubts that a degenerative process within a cell can act in such a way as to increase the function of the cell. His suggestion that the fibrillations "are not central in origin * * *, but are evidences of a disordered muscular metabolism secondary to the loss of the trophic influence of the anterior horn cell" is more consistent with the unquestionable finding of these fibrillations in chronic thyrotoxic myopathy.

The coexistence of diabetes and hyperthyroidism in the patient just described has several implications which merit brief mention. This combination of diseases is always reportable, inasmuch as there is as yet no uniformity of opinion regarding their possible etiological relationship.[8 9 10 11] The aggravation of diabetes by complicating thyrotoxicosis is, however, an accepted clinical fact which should not be forgotten when a previously well-controlled diabetic becomes difficult to manage. Suggestive evidence has been brought forward to the effect that malnutrition brought on by diabetes, voluntary weight reduction, and other diseases may precipitate thyrotoxicosis.[12] The pancreas has been assigned a specific role in the pathogenesis of the myopathies by Meldolesi who has been quoted as saying that if a person having the "myopathic diathesis" sustains a pancreatic injury, he will fall prey to progressive muscular atrophy.[13] In the absence of confirmatory studies and in the face of the failure of Scheman [14] and his associates to confirm Meldolesi's thesis with respect to pseudohypertrophic muscular dystrophy, its obvious application to the present case can only be mentioned in passing.

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[7] Grinker, R. R.: Neurology. Chas. C. Thomas, Balto., Md., 1934. p. 805.

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[13] Foreign Letters. J. A. M. A. 109: 1137, Oct. 1937.

[14] Scheman, L., Lewin, P., and Soskin, S.: Psuedohypertrophic muscular dystrophy. J. A. M. A. 111: 2265, Dec. 1938.

No discussion of myopathic symptoms in thyrotoxicosis would be complete without referring to the increasing number of reports concerning an unknown relationship between hyperthyroidism and myasthenia gravis. Allan,[15] reporting a case in which both diseases occurred together, writes, "Hyperplasia of lymphatic tissue, lymphorrhages in muscle tissue, lymphocytosis, creatinuria, and lowered carbohydrate tolerance may be found both in cases of hyperthyroidism and in cases of myasthenia gravis." He cites the high incidence reported by other authors of hyperthyroidism in the rare condition of familial periodic paralysis. Barton and Branch [16] have reported a case of myasthenia gravis with necropsy which clinically imitated hyperthyroidism. Tachycardia, fine tremor, elevated basal metabolic rate, creatinuria, and a positive Goetsch test were the outstanding findings in their patient. In the light of evidence already referred to [12] that weight reduction measures can precipitate thyrotoxicosis, it is interesting that such a history was obtained. At autopsy the thyroid gland was normal, but the muscle tissue was soft, flabby, friable, pale, and showed a marked variability in the size, shape, and straining reaction of the fibers. Zenker's degeneration was present; all the fibers were widely separated by edematous interstitial tissue containing lymphocytic collections.

Creatinuria occurs so constantly in hyperthyroidism that creatine tolerance tests have been devised which yield results closely paralleling the metabolic rate and which have some diagnostic significance.[17] There is undoubtedly a relationship between the muscular weakness of thyrotoxicosis and creatinuria, perhaps representing a "reparable impairment of the phosphocreatine mechanism." [4] Bodansky and Pilcher,[18] in experiments on rats made thyrotoxic by the administration of thyroxine, found significantly large reductions in the creatine content of both skeletal and cardiac muscle, more marked in the latter. The possible relationship between this finding and the cardiac manifestations of hyperthyroidism is obvious. The occurrence of creatinuria in the muscular dystrophies and atrophies can scarcely be said to link all myopathies with thyroid dysfunction. Probably creatinuria indicates nothing more specific than a generalized disturbance of the metabolism of striated muscle which in turn may be produced by a variety of unrelated factors.

[15] Allan, F. N.: Muscular weakness and hyperthyroidism-myasthenia gravis and goitre. Surg. Clin. N. America 14: 1249, Oct. 1934.

[16] Barton, F. E. and Branch, C. F.: Myasthenia gravis. Report of case with necropsy. J. A. M. A. 109: 2044, Dec. 1937.

[17] Means, J. H.: The Thyroid and its Diseases. J. B. Lippincott Co., Phila. pp. 150 and 298.

[18] Bodansky, M. and Pilcher, J. F.: Heart and skeletal muscle during recovery hyperthyroidism. Proc. Soc. Exper. Biol. & Med. 35: 607, Jan. 1937.

SUMMARY

1. A case of generalized muscular atrophy occurring during the course of moderately severe thyrotoxicosis and preceded by diabetes mellitus has been described.

2. This case fulfills the criteria of *chronic thyrotoxic myopathy* as outlined by Brain, and is possibly the eighth reported instance of this condition.

3. Some of the recent literature concerning possible interrelationships among myopathic conditions, the thyroid gland and the pancreas has been reviewed.

MYASTHENIA GRAVIS [1]

WITH CASE REPORT

By Commander R. L. Nattkemper, Medical Corps, United States Navy

The exact cause of this disease is not known. It occurs most frequently during the third and fourth decades. It is more frequent in females and seems associated with pregnancy at times. It has followed acute infections, such as epidemic encephalitis, influenza, or common colds. Considered also is alcoholism, over-exertion, and mental strain. There may be some relation to the suprarenal, thyroid, or thymus glands. There probably is no hereditary basis. A disturbance of muscle metabolism is present, resulting in a creatinuria, but this is variable and is regarded as a result of the disease and not its cause. Recent work on the physiology of the nervous system indicates that in most of the nerves of the parasympathetic or craniosacral system impulses cause the formation of acetylcholine at the nerve ending and the acetylcholine then initiates the activity of the contiguous organ. Acetylcholine is destroyed, after it has served its impulse-transmitting purpose, by esterase, a substance believed to be present normally in the serum or tissue. It would seem that in myasthenia gravis there is an increase in this inhibitory effect of the esterase on the acetylcholine, this then resulting in the symptomatology.

The most constant findings, though not always present, are an enlarged thymus and collections of lymphocytes in the muscles. There are no characteristic findings in the central nervous system.

The condition is of slow onset and usually begins with a paresis of the ocular muscles. Diplopia, strabismus, and nystagmoid movements may be present. The facial muscles are relaxed and give the characteristic appearance of the myasthenic facies, there being a drooping of the corners of the mouth and an inability to whistle. Usually

[1] From the U. S. Naval Hospital, Mare Island, Calif.

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there then follows an implication of the muscles of mastication, deglutition, and those of the tongue and larynx. The weakness of the pharyngeal muscles may cause a regurgitation of liquids through the nose and the difficulty in swallowing may lead to serious choking attacks. The respiratory muscles may, in the later stages, become involved, leading to dyspnoea and death. Weakness and paresis of the muscles of the trunk and extremities occur. The proximal parts of the extremities are more frequently involved than the distal parts. The heart and sphincters are usually spared. There are no trophic disturbances or fibrillary tremors. No sensory changes occur. There is no pain and reflexes are normal. Mental depression, apathy, and at times suicidal tendencies are present. Diagnoses of hysteria, bulbar paralysis, epidemic encephalitis and the muscular dystrophies may have to be differentiated. There are no characteristic complications.

The process is usually a chronic one, although acute, rapidly fatal cases occur. The subacute types run a course of 1 to 2 years, while chronic cases may last for 20 years or more. The condition may entirely disappear or reappear at intervals. There are frequent remissions. During pregnancy symptoms recede but are more marked during the puerperium and at menstrual periods. Aspiration pneumonia frequently terminates the condition.

Formerly treatment was absolute rest and massive doses of strychnine. This gave benefit in the less severe cases. There then followed a period when various endocrine preparations were used, including the gonads and thymus. X-ray of the thymus gland was tried. The results from such treatment were generally not satisfactory.

In 1930, ephedrine was found to be of value in certain cases and, in 1934, physostygmin was used with definite benefit. At about this time glycine was also used with some benefit.

In 1935, prostigmin first became available; first the prostigmin methyl sulphate for parenteral use and later prostigmin bromide tablets for oral use (dosage: 15 mgms. for oral and 0.5 mgms. for hypodermic use). Since prostigmin is an established specific for this disease as regards the symptomatology, the treatment is largely concerned with the administration of that drug. Prostigmin is analagous to physostygmin. It differs in its action in that it affects the bowel more markedly, has less miotic effect and none on the heart. The dosage of the drug is dependent on the particular case, as also is the time of administration. In some cases only one or two oral tablets of 15 mgms. each may be necessary, while 10 or more over the 24 hours may be required in the severe case.

There are at times certain toxic effects such as salivation, colic, peculiar fluttering sensations in abdomen, diarrhoea, giddiness, fainting, and dyspnoea. Also motor restlessness and twitching of the skeletal muscles may be noted. The toxic manifestations can be

controlled by the use of atropine taken as required, or by giving small doses daily prophylactically, which seem not in any way to inhibit the action of the prostigmin. It would appear that a person with myasthenia gravis is to a degree immune from the toxic action of the drug.

Experiments have been carried out showing that quinine causes a marked exacerbation of the symptoms and should be given, if at all, to a myasthenia gravis case with extreme caution.

Observation of a reasonably large number of cases indicates that prostigmin is a specific for the disease insofar as relieving the symptoms is concerned. There is no other condition of muscular weakness which shows any favorable response to the administration of this drug such as is seen in myasthenia gravis. It has been tried on the normal person, in postinfluenzal encephalitis, progressive bulbar palsy, progressive muscular dystrophy, lesions of central and peripheral nervous systems and other related conditions with no improvement as regards muscle power. Also in these conditions it causes fibrillary tremors, noticeable in the tongue. For this reason it supersedes all other procedures in diagnosis. It is not cumulative nor is tolerance established and, when given over long periods of time, there have been no adverse effects. There is no evidence that it may hasten a remission or cure the disease, but it may help by improving the nutrition of the patient.

There are certain drugs which, given with prostigmin in certain cases, greatly enhance its action. Those that have been used are ephedrine, benzedrine, and potassium chloride.

Prostigmin given hypodermically exerts its action in a few minutes and the effect lasts an hour or more. When given orally its action is manifested in about 30 minutes, reaches its maximum in 2 hours, and wears off in 3 to 5 hours. The dose by mouth is 30 times that given hypodermically. The results following oral use are so satisfactory that this is the preferable form of treatment and subcutaneous medication is required only in emergencies.

The following case report illustrates the characteristics of this disease and the satisfactory results from treatment with prostigmin:

Case Report

An officer, 56 years of age, was admitted to this hospital on February 20, 1939, having been transferred as a patient from the Asiatic station. He had considered himself well in all respects until last August. At that time he was driving a car across country and one morning awakened with a ptosis of the right eyelid, which persisted for about 24 hours. A few more days passed when he noted, in eating, a marked fatigue of his masticatory muscles, but this only lasted a short time. There then passed a month or so during which time he was doing heavy, muscular work and felt fine, when again weakness of jaw muscles occurred and, associated with these symptoms, was a slurring of speech, with collection

of mucus in his throat that could be expelled only with difficulty. This condition, persisted and also noted at this time was diplopia and a weakness and flail-like action of his left leg when walking. These symptoms continued and it was in the latter part of December that there occurred the first severe choking attack, in which mucus collected in his throat and impeded respiration to an alarming degree. It is interesting to note that this officer had an explanation for his symptoms. He attributed the ptosis of his eyelid to the long motor trip he was taking and the difficulty in swallowing and chewing of food to faulty dentures.

This officer had just arrived in Manila when the first severe attack occurred and it seemed that the condition progressed much more rapidly from the first minor symptom shortly after commencing the sea voyage.

During the return trip to this hospital aboard the naval transport, as there was available only a limited amount of the specific drug, prostigmin, his condition at times became very critical and intravenous therapy for nutriment was carried out and also definite benefit followed the use of physostigmin and ephedrine.

The familial history shows no significant factors. He was operated for an acute appendix at 30 years of age, and in 1920 passed a small kidney stone with no similar symptoms before or since. He has always rather supervised his diet, complaining at times of flatulence and chronic constipation. As regards an etiological factor in this case, there is nothing shown as of probable cause except for several months prior to the onset of the symptoms he had been working long hours and under mental strain in connection with his duties.

When admitted to this hospital he appeared chronically ill. There was profound generalized muscular weakness and emaciation. There was present the typical myasthenic facies with ptosis of upper right lid and the lower jaw dropped with slight drooling. Speech was slurred and indistinct. There were no mental abnormalities other than an extreme apprehensiveness from the fear of a choking spell.

The general physical examination was not remarkable. His skin was clear and no glandular enlargements were present. His blood pressure was 160/110. Pulse rate was 90, regular and of fair volume. His heart was not enlarged or displaced and no murmurs were present. There was only slight accentuation of the aortic second sound. The abdomen, genitalia, and extremities were negative. A detailed neurological examination indicated no involvement of the central or peripheral nervous systems. Routine and special laboratory work showed no findings of significance. There was a moderate secondary type of anaemia. Blood Kahn and spinal fluid examinations were normal. Blood chemistry, sedimentation rate, and icterus index were normal. The electrocardiogram and x-ray of heart and chest were not significant, nor did the x-ray show the thymus gland to be present. The eye grounds were normal.

TREATMENT: During the first 24 hours of stay in this hospital there were repeated choking attacks and difficulty in breathing. Nourishment was limited to fluids. He was given 9 injections subcutaneously of 1–2,000 prostigmin methyl sulphate ampules of 1 cc. each, over the period of the first 24 hours and these afforded much relief. Such treatment was continued with the addition of ephedrine for the following 3 days, when prostigmin bromide tablets of 15 mgms. each were given orally. They were given as follows: One tablet at 6 a. m., 12 noon, and 6 p. m., and two tablets at 9 a. m., 3 p. m., and 9 p. m. He also received ephedrine hydrochloride ⅜ grain at 11 a. m. and 6 p. m. In a few days there was much improvement throughout the 24 hours, but in addition to the above, an injection of 1 cc. of 1–2,000 solution of prostigmin was necessary just before breakfast, as he still had some difficulty in swallowing at that time. One week later, in addition to the above, glycine 20 gms. daily with 10 grains of potassium chloride TID was given. Later, benzedrine was also used, but with-

out benefit. Following this medication over a period of a few weeks there was a gradual improvement in all respects and at this time he is ambulant, takes auto trips, and appears well in all respects, excepting for very brief periods at irregular times during the day when there may occur a slight ptosis of the lid or fleeting weakness of masticatory muscles. A lessening of the dosage caused an immediate return of all symptoms. Observation did not show any synergistic action of other drugs and he is continued satisfactorily on the prostigmin and ephedrine. There has been no intolerance to the drug nor any ill side effects and it can be reasonably anticipated that a remission will supervene.

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BORDERLINE ADRENAL CORTEX INSUFFICIENCY [1]

WITH CASE REPORT

By Commander Walter J. Pennell, Medical Corps, United States Navy, and Lieutenant (Jr. Gr.) Lynn S. Beals, Jr., Medical Corps, United States Navy

It is the primary purpose of this case presentation to reemphasize the fact that between organic disease and functional disease there often lies a zone, dangerous to the hasty diagnostician, and of infinite possibilities to the medical detective. All too frequently psychoneurosis, neurasthenia, and psychasthenia become merely descriptive by-words embracing symptomatology for which there exists no specific etiologic explanation. Mental difficulties, lassitude, fatiguability, vasomotor instability, hypotension, and gastro-intestinal symptoms often appear as the presenting factors in many organic diseases—fading out with the amelioration of the particular disease. Medical experience has taught us that the painful shoulder or arm associated with cough and weight loss may be a Pancoast tumor; that pain and paresthesias arising in a hip or buttock may be secondary to pyriformis spasm; that hysterical paralysis of the leg may be due to cord tumor, or that an anxiety neurosis may be due to a brain tumor.

In this particular instance, the case study was undertaken because it represented a type of case seen frequently in this hospital and be-

[1] From the U. S. Naval Hospital, Philadelphia, Pa.

cause the usual diagnosis of neurocirculatory asthenia seemed both inadequate and potentially dangerous to a patient with early organic disease in that from thenceforth indefinitely his treatment would be largely psychotherapeutic with a minimum of active medical treatment.

While it would be a conclusion in error to assume that all neurocirculatory asthenics have organic disease, it is hoped that this case report will illustrate the possibility that organic disease may be discovered in the selected case by moderately diligent study.

Case Report

R. N. McN., a 44-year-old married World War veteran, engaged as a farmer, entered the hospital January 3, 1939, with a chief complaint of "lung trouble" since the World War. He dated his present illness back 4 months prior to entry when he had a recurrence of symptoms which first appeared during and after the war. He had influenza in 1918 and pneumonia in 1919. After convalescence he was returned to his company, still with a heavy cough and weakness. At the close of the war, he was hospitalized before discharge as a tuberculous suspect. A diagnosis of tuberculosis was not established although he was offered $9 a month for his disability. He classified his complaints as weakness, lung trouble, and stomach trouble—stating that they dated back to a gradual onset about 19 years ago, shortly after leaving the army. There had been no radical change but they had been slowly growing more severe.

He had had a cough since that time which was occasionally productive of yellowish, nonsmelling sputum, but only the winter previous to admission was it blood-streaked. He attested to night sweats for the previous 19 years but they were not severe enough to warrant change of bed clothes. He also had frequent cutting pains over his left upper chest radiating between his shoulders, aggravated, like his cough, by a cold and over-exertion.

Nineteen years prior to admission, his family doctor told him he had heart trouble, the result of gas on his stomach and the sensation of fullness under his lower ribs. He had no vomiting, although he was nauseated and had cutting pains under his lower ribs, accompanied by sour eructations for the last 19 years. For 10 to 12 years he had the same kind of pain under his right ribs radiating anteriorly to the left flank, occasionally originating around his umbilicus with a similar radiation. The sharp epigastric pain came on about 10 minutes after eating. He had always been constipated and his stool for the previous 4 to 5 years had been white until 3 weeks prior to admission when it became tarry. Under a doctor's care, he had at that time, he believed, not been receiving oral iron.

Subject to colds with malaise and tightness of his chest, he had two to three colds each summer and an almost continuous one during the winter, associated with precordial pain and dull ache across his chest. Throughout the previous winter, he had had a dull pain in his left arm, said by his physician to be due to low blood pressure, which condition he was aware of 4 years previously. Extremely sensitive subjectively to low temperatures and drafts, he had spent 2 to 3 weeks out of every summer and 5 weeks to 4 months (this present illness) out of every winter in bed because he had become so prostrated. Weakness, vertigo, and blurred vision had occurred with sudden posture changes for the previous few years.

Four months before admission he caught a heavy cold which caused an exacerbation of his gastro-intestinal symptoms, as already mentioned, and ran a tem-

perature up to 101.3° for 2 weeks. He remained in bed until a day or two before Christmas, when he got up for a few minutes, only to find his vertigo and weakness so severe, he had to return to bed, where he remained until admission. He had lost 15 pounds in the last 8 months.

PAST HISTORY: Past history revealed in addition to influenza and pneumonia, a diagnosis of arrested tuberculosis made in 1927, for which he received $50 a month compensation, and pleurisy in 1938. He also had an appendectomy in 1929.

FAMILY HISTORY: Family history was irrelevant except for one brother who is in a State institution with a traumatic psychosis.

PHYSICAL EXAMINATION: Physical examination revealed an undernourished, poorly developed, middle-aged man who appeared chronically ill. His hair was quite gray and the color of his skin was a pasty gray with no obvious pigmentation. Over the left lower flank was a 10-cm. square area of desquamation. The thyroid seemed somewhat enlarged. Eyes, ears, nose, and throat were essentially negative, no pigmentation being noted. His chest was asymmetrical with a diminished anteroposterior diameter on the right and limitation of expansion on the left. The left base was low and narrow and the left apex contracted. The lungs appeared negative except for bronchovesicular breath sounds over the bases. His heart seemed small but was otherwise negative. His blood pressure (left arm) was 110/64 while lying down, but fell to 87/70 on standing, the sounds being difficult to obtain. The upper abdomen was moderately tender and no organs could be palpated despite poor muscle tone and poor nutrition, except the liver which was two fingers breadth below the costal margin, smooth and moderately tender. No costovertebral tenderness could be elicited. The extended fingers showed a fine tremor.

He was placed on a high caloric, high vitamin diet in an effort to build his general condition up while being studied. The differential diagnosis seemed to lie between Addison's disease and neurocirculatory asthenia, or somewhere in the zone between them, sharing elements of each.

He remained in *status quo*, both objectively and subjectively, remaining close to his bed most of the time. He continued to complain of the cutting pains over his left chest and abdomen and vertigo when standing, with his blood pressure ranging from 80–98/50–64, averaging 93/59.

SPECIAL EXAMINATIONS revealed the following:

February 4, 1939.—X-ray of chest—essentially negative; no soft shadows suggestive of an incipient parenchymal tuberculous type of lesion noted. Heart measurements revealed the transverse diameters to be 2.8 cm. less than the maximum normal.

February 6.—Electrocardiogram—negative. RBC, 4,660,000; Hgb, 90 percent; WBC, 13,950; segmented, 53; juvenile, 7; lymphs, 33. Blood urea nitrogen—12 mg. percent. Blood sugar (nonfasting)—81 mg. percent.

February 9.—Gall bladder visualization—normal. G.I. series negative. Barium enema negative. Gastric analysis—normal.

February 13.—Kahn negative. Sedimentation rate, 13 mm. in 1 hour,

February 15 to May 9.—Basal metabolic rate varied (on 13 determinations) from +3 percent to −14 percent, averaging −5.3 percent.

May 2.—Urine—routine and microscopic—negative. RBC, 4,560,000; WBC, 9.400; Hgb, 86; segmented, 46; lymphs., 44; eosins, 2; monos. 8.

May 2 to May 4.—Five sputa examinations, negative for acid-fast bacilli.

May 5.—X-ray of both shoulders and upper thoracic spine negative for bone or joint pathology.

May 11.—Intravenous pyelogram was negative for calcifications in the adrenal areas and for renal pathology.

TREATMENT: Inasmuch as the syndrome presented was largely that of hypotension with a narrow pulse pressure and abnormal carbohydrate metabolism, it was decided to institute the recognized treatment for adrenal cortex insufficiency. It was also felt that since the debility arose largely from an extreme degree of vasomotor instability, it would be of value to determine the patient's reaction to specific pressor substances and also to vasodilator agents. As can be seen, the charts (figs. 21–24) reveal that little objective effect on the blood pressure and pulse resulted.

On March 16, 1939, he was placed on active therapy consisting of five periods as follows (see figs. 25–28):

(*a*) March 16 to March 30.—Oral sodium chloride 7.65 grams per day and Wilder's low potassium diet.

(*b*) March 30 to April 10.—As in (*a*) neosynephrine from 30 milligrams up to 90 milligrams a day.

(*c*) April 13 to April 19.—Sodium chloride 10.2 grams per day plus cortical extract.

(*d*) April 19 to April 27.—Diagnostic test of withdrawal of salt and cortical extract.

(*e*) April 27 to May 3.—Return to high salt, low potassium orally and cortical extract intramuscularly.

In order to evaluate the effects of treatment, charts were made of blood pressure, pulse pressure, pulse rate in the three positions—lying, sitting, and standing. The determinations were made as nearly as possible at the same time each day in the same relation to meals. For each change in position 5 to 15 minutes were allowed to elapse for the purpose of vasomotor readjustment.

During the first three periods of treatment, there was a gradual steady improvement subjectively first, apparent under neosynephrine but most marked under cortical extract. When salt and cortical extract were withdrawn for a week, there was a steady fall in blood pressure and pulse pressure with a corresponding tachycardia, particularly in the standing position. At the same time, the patient regressed, feeling fatigued, exhausted with moderate effort and experiencing a return of his vertigo and "cutting pains" from which he had been free during the period of adrenal cortex substitutional therapy and salt. Furthermore, he contracted a heavy cold and cough, found himself "breaking out in a sweat" without provocation, and lost 3 pounds in weight.

At the end of a week he was given 1,000 cc. of 2 percent saline intravenously, 2 cc. of cortical extract intramuscularly, 4.25 grams of sodium chloride orally, and returned to Wilder's high salt, low potassium diet. Within 2 hours after receiving the intravenous saline and the cortical extract, he subjectively felt "150 percent" better than just prior to it and his blood pressure, pulse pressure, and pulse rate had returned in 4 hours almost to the values just prior to the period of withdrawal of medication. Such a response both to the withdrawal test and the substitution of cortical extract and salt is said by Wilder and other workers to be as close an approximation for a biologic test for adrenal cortex insufficiency as we have at the present time.

Following a week of treatment consisting of Wilder's low potassium diet, oral sodium chloride and adrenal cortex extract intramuscularly, during which he continued to improve further, he was regulated on 10.2 grams of salt per day, the special diet, and ½ cc. of cortical extract a week.

When discharged May 12, 1939, he had gained 19 pounds, had a normal pulse pressure, blood pressure, and pulse rate and felt able to return to his work as a farmer. He was given the advice to continue as regulated in the hospital and to receive cortical extract from his local physician in the amount of ½ cc. once a

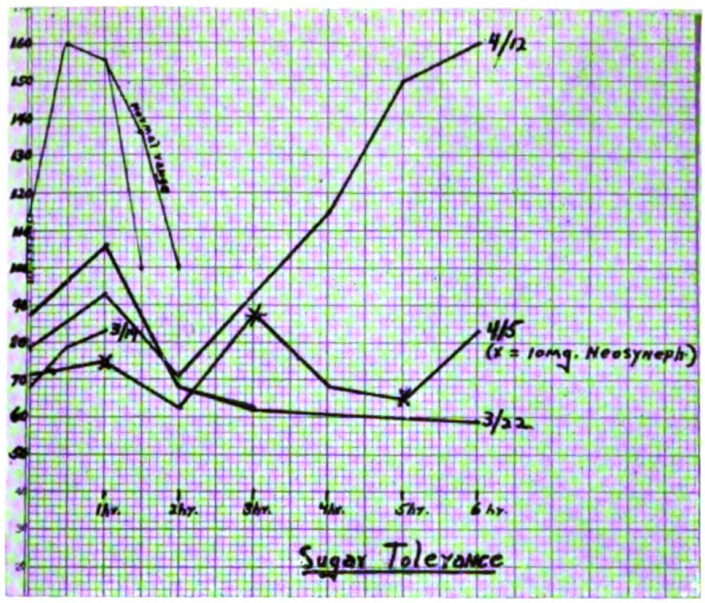

FIGURE 21.—RESPONSE TO SUGAR.

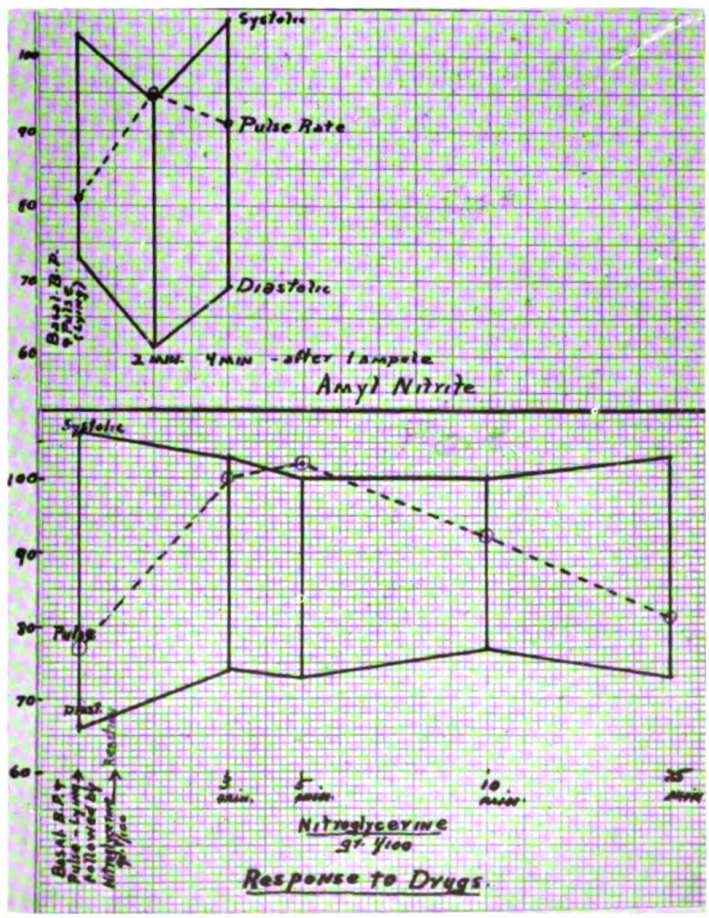

FIGURE 22.—RESPONSE TO AMYL NITRITE AND TO NITROGLYCERINE.

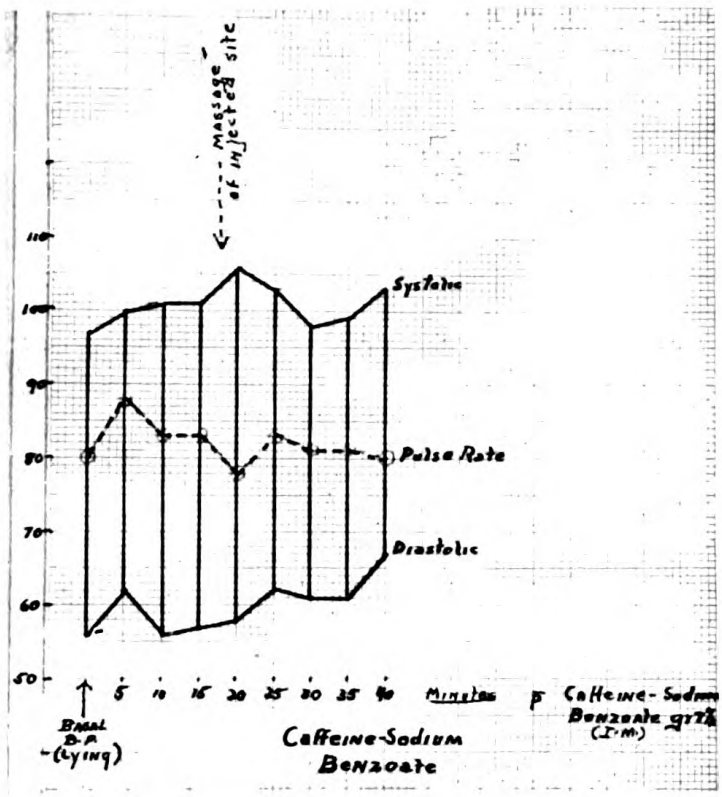

FIGURE 23.—RESPONSE TO CAFFEINE-SODIUM BENZOATE.

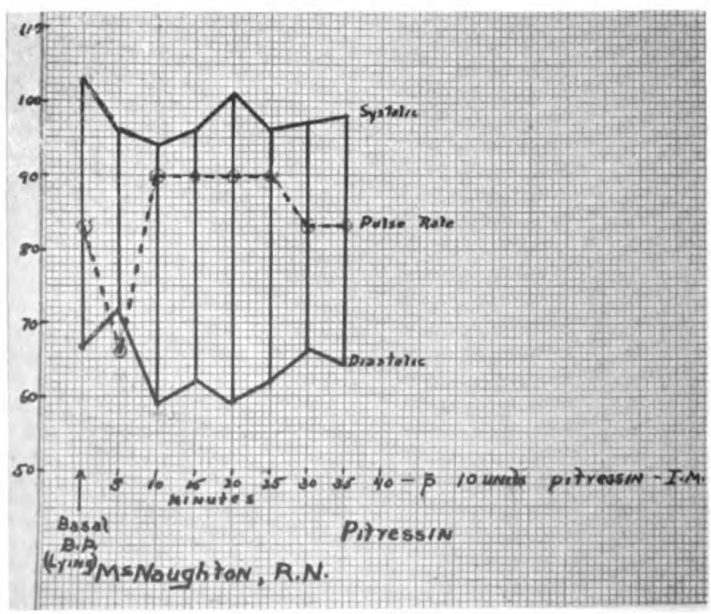

FIGURE 24.—RESPONSE TO PITRESSIN.

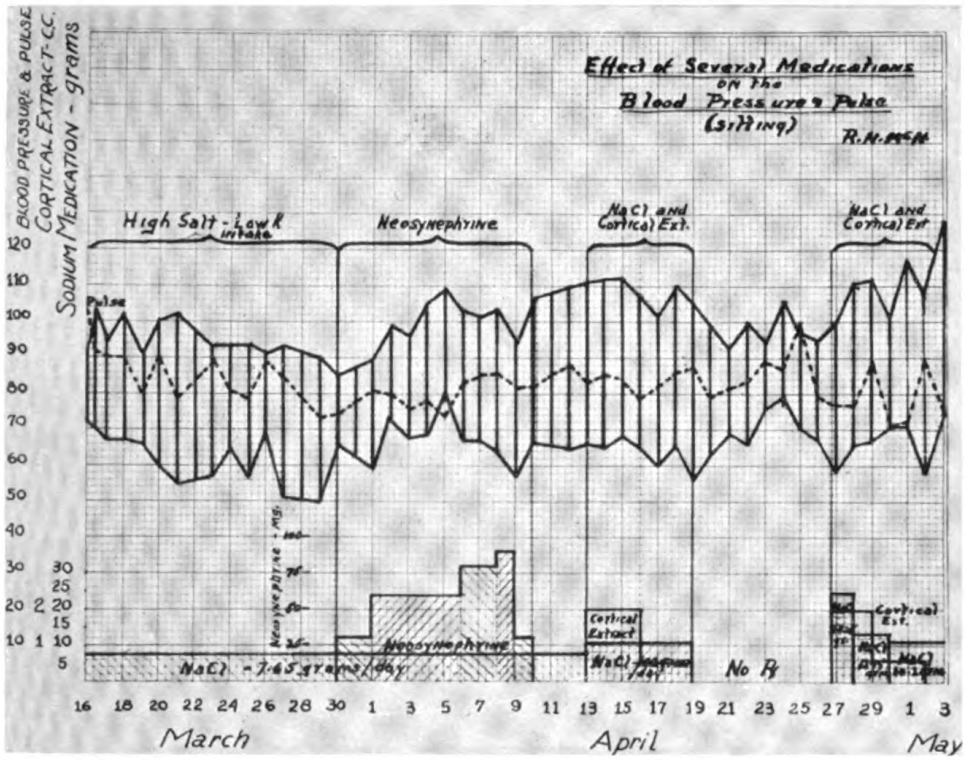

FIGURE 25.—EFFECT OF SEVERAL MEDICATIONS ON THE BLOOD PRESSURE AND PULSE (SITTING).

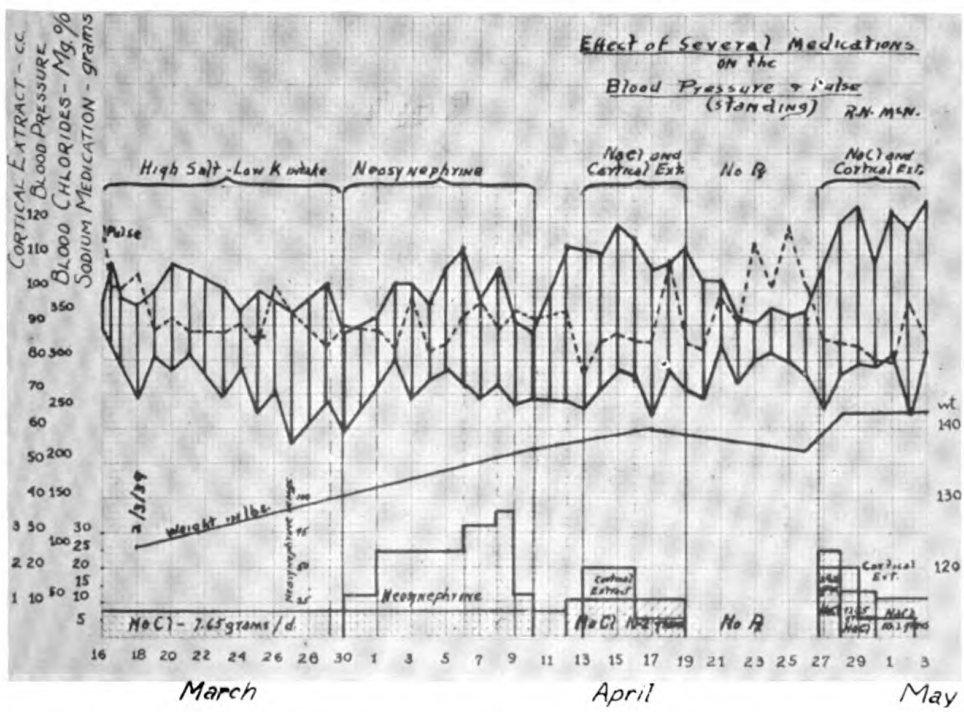

FIGURE 26.—EFFECT OF SEVERAL MEDICATIONS ON THE BLOOD PRESSURE AND PULSE (STANDING).

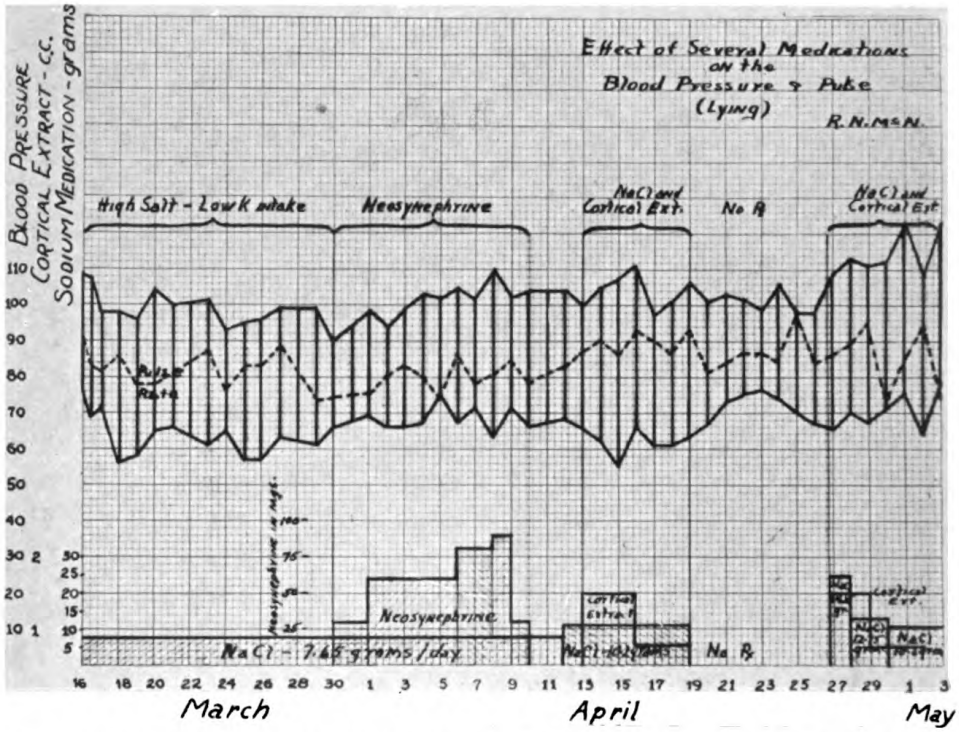

FIGURE 27.—EFFECT OF SEVERAL MEDICATIONS ON THE BLOOD PRESSURE AND PULSE (LYING).

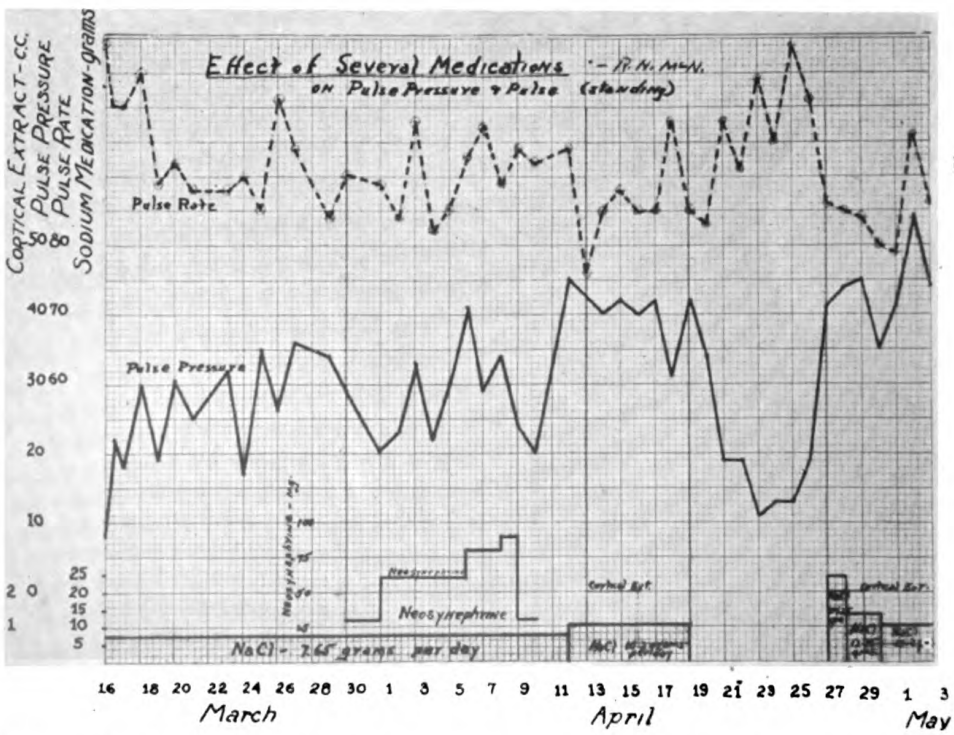

FIGURE 28.—EFFECT OF SEVERAL MEDICATIONS ON PULSE PRESSURE AND PULSE (STANDING).

week. He further was cautioned to avoid hard labor and heavy work in the sun during which he might perspire freely and thus lose some of his chlorides.

CONCLUSIONS

1. In the symptomatology of moderate subclinical levels of adrenal cortex deficiency, psychoneurotic and neurasthenic symptoms may predominate.

2. Pigmentation may be absent (Reifenstein).

3. Duration of symptoms for as long as 16 years has been reported.[11] This case extends, by history, for a period of 20 years.

4. The criteria for establishing this diagnosis are:

(*a*) Hypotension, small pulse pressure and tachycardia.

(*b*) Anorexia and weight loss.

(*c*) Flattened glucose tolerance curve.

(*d*) Response to salt, cortical extract, and low potassium diet, in raising blood pressure, increasing weight and appetite and subjective improvement (Wilder).

(*e*) Loss of improvement, fall in blood pressure, and return of symptoms upon withdrawal of medication and dietary regime.

(*f*) The presence of pigmentation is not essential.

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(*Addendum* on page 407)

LUPUS ERYTHEMATOSUS

WITH CASE REPORT

By Lieutenant Curtis William Schantz, Dental Corps, United States Navy

Contemporary American dental literature contains very little information on lupus erythematosus or Cazenave's disease. The fact that so little has been written about the disease does not classify it with rare maladies in regard to interest for the dental profession. At the onset of the disease, as well as during its course, there is a well marked prev-

alence of oral manifestation; and because subjective symptoms are conspicuous by their absence, except during acute stages, the diagnostic acumen of the observer must be keen.

Clinically there are two types of the disease manifested, namely, the discoid and the migrating or diffuse lesions. The discoid type is more frequently observed, the chronic lesions becoming deep and infiltrated; they progress slowly and are more or less fixed. The disease is usually limited to the face, lips, and scalp, but may be found on any part of the body.

Mouth lesions are present in both types of the disease and involve in most cases the oral mucosa, the rare exception being the tongue. In the mouth, the lesions develop as isolated symmetrical patches on the buccal mucosa, spreading to the adjacent surfaces including the hard palate, the floor of the mouth, the lips, and lastly—in advanced cases—the gums. The oral lesions begin as punctate, bright or pale red, ill-defined spots, sometimes level with the adjacent mucosa and at other instances, elevated. These spots often form an irregularly shaped patch caused by joining with adjacent lesions, and soon present a variable number of superficial erosions partially covered with adherent, moist desquamations. The zone of the lesion is surrounded by engorged blood vessels and the marginal infiltration is very well marked. At a later stage the patch depresses at its center, presenting an indurated erythematous border. Old patches leave firm, whitish scars, irregular in shape.

The lesions on the lips are dark red, sharply defined and irregularly shaped, involving the upper and lower lips. Near the vermilion border, the lips are dry and covered with adherent scales, presenting much the appearance of having recently been covered with cavity lining or collodion. The lesion on the lips is usually painless, but accompanied by a sensation of dryness and stiffness which might easily result from sun or wind burn.

Diagnosis is usually made from the accompanying lesions on the skin of the face. Lupus erythematosus may be differentiated from leukoplakia by the more advanced infiltration in later stages of the disease. Serological and therapeutic tests will eliminate syphilis. Lichen planus is differentiated by the prevailing presence of white-topped papules and the lacelike appearance of the lesion. Tuberculosis and epithelioma must not be overlooked. It is by no means unknown for a lesion of lupus erythematosus to undergo epithelioma-tous degeneration. Tuberculosis of the mouth is usually secondary to pulmonary tuberculosis and may be distinguished by the grayish-white elevations, pinhead in size, consisting of agglomerations of microscopic tubercles.

The cause for lupus erythematosus is unknown and the treatment, therefore, has always been empirical. Gold compounds injected in-

travenously have been used with some success, but not without danger to the patient in the form of dermatitis involving the entire body. Injection of 1 percent solution of gold sodium thiosulphate directly into the lesion has produced good results for Monash and Traub. Radical means of treatment should be limited to the use of electrocautery and then only when esthetics are of no consideration. Deep x-ray therapy is contraindicated in any form of treatment.

Case Report

R. D. H., Pvt., U. S. M. C., age 26, presented himself at the yard dental dispensary, Cavite, P. I., June 24, 1938, for suspected Vincent's infection.

Chief complaint: Sore gums and chapped lips.

Clinical examination: Oral examination disclosed red and highly inflamed gums accompanied by *fetor oris*. The oral mucosa on the buccal surfaces extending downward and including the mucobuccal fold in the lower jaw presented the appearance of recently traumatized tissues. Interspersed were bright red patches slightly raised. The lips presented a dark red scaly surface extending to the vermilion border. The lips were somewhat swollen, painful, and fissured. Temperature 99.1°.

General history: Patient stated that about 1 year previous he had been on duty in Guam and there received a severe sunburn involving principally the lips. From this time on his lips were constantly in sore, chapped condition. Treatment with bland preparations were prescribed which helped materially to ease the discomfort of the dryness of the lips. No other symptoms were noted up to the visit for gum treatment. Previous medical history showed negative serological tests for suspected syphilis. Kahn test at this time was negative, but venereal history of previous gonococcus infection complicated by gonorrheal buboes was recorded. Patient related previous dental history of acute Vincent's infection. All other oral history was negative with no carious teeth present. Diagnosis at this time, Vincent's infection, acute. Patient placed under specific treatment for Vincent's stomatitis.

Treatment: Prophylaxsis and rigid oral hygiene prescribed. Gums treated with salvarsan incorporated with glycerine. Alternately with the above treatment Fowler's solution was used, administered by spray. Following 1 week of this type of treatment no improvement was noted. Temperature slightly above normal. Treatment for Vincent's infection discontinued. Lips kept coated with cocoa butter. Patient was referred to the naval hospital for observation and treatment.

On November 8 he again presented himself at the yard dental dispensary for gum treatment. Oral condition showed little or no improvement, and the lips seemed more thickened and swollen than on the previous visits. In addition to the lip condition a discoid patch had formed on the bridge of the nose. This patch resembled the typical patch described by Ormsby as the discoid type of lupus erythematosus. A biopsy was then taken from the inner border of the upper lip opposite to a corresponding patch on the gum in the area of the upper right central incisor. A v-shaped section was taken under local anesthesia and sent to the naval hospital pathologist for examination.

Microscopical examination: Examination revealed a thinned atrophic mucosa. The submucosa was highly edematous with signs of basophilic degeneration of the collagen fibers. Blood vessels were dilated with a marked lymphocytic infiltration. Numerous circumscribed areas showed a dense collection of lymphocytes resembling giant cells.

Diagnosis: Lupus erythematosus (discoid).

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PAGET'S DISEASE

WITH SPECIAL REFERENCE TO ORAL MANIFESTATIONS[1]

By Lieutenant (Jr. Gr). Herman K. Rendtorff, Dental Corps, United States Navy

In a review of the available dental textbooks and periodicals, there are very few references to be found relative to the oral manifestations of Paget's disease, or osteitis deformans. There are practically no references to be found relative to the importance of oral roentgenograms. The disease has been considered relatively rare but this probably is attributable to the fact that only the more advanced forms of the disease have been recognized. When we consider the structural changes occurring in the maxillary bones and teeth as a result of the attack of the osteitis, we should realize the importance of the oral roentgenograms as an aid in early diagnosis. In the more extensive use of the roentgenogram, we, as a profession, will be more able to diagnose these cases in their incipiency and refer them to the physician for further investigation. By so doing, we may be instrumental in bringing about an early recognition of a disease of bone which is amenable to treatment.

Paget's disease, or osteitis deformans, was first described by Sir James Paget in 1876. There have been about 500 cases reported in the medical literature since that time. Among 20,000 patients examined at the Johns Hopkins Hospital, only 2 cases of osteitis were seen. The disease is one of the skeleton and usually occurs after middle age. Males are more susceptible than females. Since the more general use of the roentgenogram both in medicine and dentistry, the diagnosis has been made in many cases in which the disease otherwise would have remained unrecognized. In many cases, Paget's disease was an incidental finding.

The etiology of Paget's disease is unknown. Gout, rheumatism, syphilis, bacterial infection, trauma, malignant disease, disorders of the endocrine glands and arteriosclerosis have all been advanced as causative factors. There is undoubtedly an hereditary tendency as noted by several observers.[2]

The pathological process of Paget's disease is generally confined to the skull and long bones, but it may attack the whole skeletal framework of the body. The skull is always involved and the calvarium

[1] From the U. S. Naval Dental School, Washington, D. C.
[2] Stafne, E. C., and Austin, L. T., J. A. D. A. 25: 1202-1214, August 1938.

becomes enormously thickened and enlarged. Involvement of the maxillae is probably much more frequent than is commonly supposed. The mandible is rarely affected. Changes wrought in the osseous structures of the jaws during the course of the disease seem to be confined solely to the maxillary bones and teeth, although Seldin [3] recently reported a case of osteitis deformans involving the anterior part of the mandible. The changes are bilateral. Salman [4] reports a case of osteitis deformans involving the maxillary bones in which the chief complaint of the patient was general bilateral enlargement of the maxillary bones. The mandible in this report was normal. The bone involvement in osteitis deformans consists of widespread rarefaction (malacia) and formation of new bone (ossification), the latter essentially of connective tissue origin. Pathologically, the process of the disease seems to be divided into the stages of fibrosis, degeneration, cyst formation, and reossification. In the later stages of the disease, the distinction and differentiation between compact and cancellous bone is lost and the medulla is occupied by calcified fibro-osteoid tissue.

The symptoms of osteitis deformans are usually objective ones. In cases of long duration, the whole calvarium is greatly enlarged and the head is carried forward with the chin nearly resting on the chest. The spine is bent forward and the arms and legs are bowed, particularly the latter because of the body weight. The deformity results in a loss of height, often as much as 6 inches. The subjective symptoms are varied, fatigue, general weakness, cardiovascular symptoms, impairment of sight and hearing, and pain being among those most commonly mentioned. Within the oral cavity, in advanced cases, may be seen an enlargement of the maxillae and an increase in the size of the arch as compared to that of the mandible which is seldom involved. With an increase in the size of the arch, the maxillary teeth will become telescoped over the lower teeth when the jaw is closed. Due to the enlargement of the maxillary bones, the mucous membrane covering them becomes stretched under tension and shows the outlines of numerous fine capillaries. Patients showing marked enlargement of the maxillae experience some discomfort due to accumulation of food in the mucobuccal fold.

A valuable aid in the diagnosis of the osteitis deformans is a study of the concentration of calcium, phosphorus, and phosphotase in the serum. The concentration of calcium and phosphorus in the serum remains normal in Paget's disease, but the values for phosphotase increase from the normal, which are 1 to 5 units per 100 cubic centimeters (Bodansky), to as high as 30 or 40 units per 100 cubic centimeters.

[3] Seldin, Nathaniel A., D. Cosmos, 75: 691-692, July 1933.
[4] Salman, Irving, D. Cosmos, 72: 137-140, February 1930.

The roentgenographic appearance in Paget's disease is probably dependent on the stage at which the disease is seen. The gross picture is that of a combined process of rarefaction and ossification. The normal bony architecture is entirely altered, the bony tissue appearing to be feathery or spongious, with poor organization. Areas of dense bone are often present and small cysts are occasionally seen. The roentgenographic appearance of the skull is generally pathognomonic. The marked asymmetry, general enlargement, and deformity are highly characteristic. The walls of the skull are thickened and spongious in texture, the mass between the inner and outer tables having a sort of frizzly hair appearance.

In a report of a study of Paget's disease based on complete dental roentgenograms that were made in 138 cases in which osteitis deformans had involved one or more bones, Stafne and Austin found that in 23 cases, the dental roentgenograms disclosed evidence of the disease of the maxillae or mandible. In these cases, it was obvious at a glance that an abnormality was present. In other cases, the variations were not so marked as they were in these cases, in which the variations usually consisted of fine, close trabeculations, which were confined to one region or might include the whole maxillae. In these 23 cases in which there was definite evidence of Paget's disease in the dental roentgenograms, the maxillae were involved in 20 cases and the mandible was involved in 3. In the dental roentgenograms which showed evidence of Paget's disease, the findings were so constant and so characteristic that they were considered worthy of report. Of particular interest were the cases in which roentgenographic examinations were made at intervals during the progress of the disease. There was a sufficient number of these cases to suggest that the roentgenologic findings vary with the stage of the disease.

Reports of Paget's disease in dental literature have been very rare, but in a few instances, single cases have been reported in which reference has been made to observations based on the dental roentgenograms. Fox [5] reported that dental roentgenograms made in a case of Paget's disease of the maxillae revealed a cystic involvement of the apices of practically all the maxillary teeth and noted "the presence of knobby growths and irregularity of the roots of all the maxillary teeth showing exostosed and sclerosed conditions of these roots." This report is of interest since the roentgenogram probably showed a marked hyperplasia of cementum and also revealed evidence of the osteoporosis which tends to persist near the root ends of the teeth. In his report of a case of Paget's disease of the mandible, Seldin said that roentgenograms revealed the presence of alternate areas of resorption and calcification and hypercementosis of the teeth. Cahn,[6]

[5] Fox, Lewis, J. A. D. A. 20: 1823–1829, October 1933.
[6] Cahn, Lester R., Arch. Clin. & Oral Path. 1: 141–144, June 1937.

who was able to make microscopic studies of a tooth and its surrounding bone in a case of Paget's disease, found a hyperplasia of the bone and cementum. He suggested that the same condition which causes hyperplasia of the bony trabeculae probably is responsible for hyperplasia of the cementum. Of the 23 cases of Paget's disease studied by Stafne and Austin, dental roentgenograms showed in 18 cases more than from a slight to an excessive amount of hypercementosis of one tooth, and more often several teeth, in the affected region.

Patients with Paget's disease enjoy fair general health and, for the most part, live to old age. Death is rarely a direct result of the osteitis. The thickening of the skull and encroachment on the cranial cavity may cause headache, pressure symptoms or mental change.

At present, no specific or entirely effective treatment for osteitis deformans is known. Treatment for relief of objective symptoms such as analgesia and counterirritation for relief of troublesome pain are indicated. Parathyroid, thyroid, calcium, and suprarenal cortex hormone therapy should be tried. Foci of infection should be looked for and eradicated. Tonics may be useful.

Involvement of the maxillae and mandible in Paget's disease is probably much more common than previously supposed. From the clinical and roentgenographic findings relative to the foregoing study, it seems quite definite that a positive relationship exists between Paget's disease or osteitis deformans and the maxillary structures and teeth in that the normal structures of these tissues are vitally affected by the incidence of the disease.

NOTES AND COMMENTS

CHEMOTHERAPY IN GONORRHEA

During the past year a large amount of data has accumulated on the subject of chemotherapy in gonorrhea. Out of all this evidence, there have emerged two very important matters that strike us as being of particular applicability in the ever-present problem of treatment of gonorrhea in the Navy.

Numerous observers have added their testimony to the truth of these two points in the literature of 1939 and 1940. Conspicuous among these workers are those who presented papers at the 1939 and 1940 annual meetings of the American Neisserian Medical Society and among these deserving special mention are Donald Herrold of the University of Illinois, and Van Slyke, Mahoney and Walcott of the United States Public Health Service who carried on their work at the United States Marine Hospital at Staten Island.

The first of these points has to do with the desirability of deferred treatment with sulfanilamide. It is now well known that if sulfanilamide is withheld for 2 to 3 weeks after the acute onset of gonorrhea, a much larger percentage of early and lasting cures will result than if the sulfanilamide is given in the routine manner at the onset of the disease. It is believed that this phenomenon is to be explained on the theory of interference in the development of immunity by sulfanilamide when this therapy is started before the immunity processes have an opportunity to become well established.

The second point concerns the now well-recognized superiority of sulfapyridine therapy over sulfanilamide therapy in gonorrhea. Van Slyke has recently gone so far as to state without equivocation that "sulfapyridine is so definitely superior to sulfanilamide in the treatment of gonorrhea that sulfanilamide has no longer any place in the treatment of this disease." The Staten Island group has collected elaborate records on some 1,800 cases of acute gonorrhea treated with 3 grams of sulfapyridine on the first day and 2 grams on the succeeding 9 days. Using 10 days as the arbitrary deadline for success or failure of a drug to produce a complete and lasting cure in gonorrhea, the group has concluded that cures may be brought about with sulfapyridine in nearly 70 percent of cases, even though they are ambulatory and working. Gonorrheal complications are practically nil when this

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treatment is used, but many patients will be unable to tolerate the drug because of the nausea and vomiting it often produces. They recommend the taking of several quarts of water daily for a few days at the termination of sulfapyridine treatment in order to dissolve any of the acetyl derivative crystals that may have formed in the kidney pelves.

The Bureau has received a very favorable preliminary report from the medical officer of a carrier on use of this form of therapy in a series of 60 cases. On completion of treatment, these cases were checked on succeeding days by urine examination, prostatic massage, and passage of sounds for effectiveness of treatment. Approximately 92 percent of this series of cases are reported as cured. This report will be published in an early issue of the BULLETIN.

With the current rapid advances being made in chemotherapy, it does not seem unreasonable to hope that we may fairly soon have in our hands a drug even superior to sulfapyridine both as to high therapeutic potency and low toxicity and that gonorrhea may drop from its number one place in medical department problems and worries to a place of minor ranking.

INTERNSHIPS AND POSTGRADUATE INSTRUCTION

Personnel concerned with our naval hospital internships and the postgraduate education of our medical department personnel will be interested in recent suggestions by the Commission on Graduate Medical Education as published in the commission's final report which concludes a 3-year study.

The internship, suggests the commission, should be considered as a basic preparation for the practice of medicine. It should round out and give practical application to the medical school course and, hence, should be closely allied to undergraduate medical education. It should prepare young physicians adequately to begin general practice and should provide them with the essential preparation necessary to undertake further study leading to the practice of a specialty. It should not attempt to train physicians directly for the specialties.

To prepare the intern for general service, he should have experience in internal medicine, surgical diagnosis, minor surgery, treatment of emergencies, pediatrics, obstetrics, and gynecology, and medical department duties as detailed in the bureau's manual. Special attention in these fields should be given to preventive medicine and the care of disease conditions, particularly those causing a high incidence of morbidity and mortality in the service. The whole atmosphere should be educational in character and the intern should learn by example as well as precept.

Postgraduate education the commission defines as study intended to keep a physician abreast of his chosen field of practice but not intended to equip him to enter a new field. Separate and clearly defined types of work are recommended for general practitioners and for specialists. While there has been a marked and rapid increase in interest in the field of postgraduate medical education, there is still need for its further extension and for improvement in the type of opportunities offered. The effect of the work of the specialty boards upon the practice of medicine is discussed in the report, which points out that these boards have provided a well-defined yardstick for measuring an individual physician's competence in his specialty. The entire report stresses the value of adequate training and points out that this will be reflected in improved care of patients.

This bureau's policy relative to postgraduate instruction has closely approximated the stand taken by the commission. Specialization to meet service needs has been encouraged for medical and dental officers who have attained a thorough familiarity with general practice as restricted to service needs. Those who accomplish this are then afforded opportunity to acquire additional professional skill in some particular specialty. Applicants for specialty training are usually assigned to a naval hospital for special training and observation. This assignment approximates a residency as defined by the commission. Officers who complete this assignment and manifest an aptitude for the specialty are then eligible for postgraduate instruction at some civil educational institution. Currently, this latter phase of the bureau's educational program has been temporarily curtailed due to an acute shortage of personnel.

The bureau desires that personnel possessing the required qualifications make application to the appropriate specialty board for certification. This application should be made directly to the board. Those who receive certificates are requested to inform the Chief of the Bureau to that effect to permit incorporation of this information in their official records.

AMERICAN COLLEGE OF PHYSICIANS

The Bureau of Medicine and Surgery has been informed that the following naval medical officers have been elected to membership in the American College of Physicians:

Fellowship

Lieutenant Commander Irwin L. V. Norman (MC), U. S. Navy.

Associateship

Lieutenant Omar J. Brown (MC), U. S. Navy.
Lieutenant Commander William R. Manlove (MC), U. S. Navy.
Lieutenant James J. Sapero (MC), U. S. Navy.
Lieutenant William M. Silliphant (MC), U. S. Navy.
Lieutenant Robert L. Ware (MC), U. S. Navy.
Lieutenant James L. Zundell (MC), U. S. Navy.

Annual Session of the College

The twenty-fifth annual session of the American College of Physicians will be held in Boston, April 21–25, 1941.

Dr. James D. Bruce, president of the College, will have charge of the program of general scientific sessions. Dr. William B. Breed has been appointed general chairman of the session, and will be in charge of the program of clinics and demonstrations in the hospitals and medical schools and of the program of panel and round table discussions to be conducted at the headquarters.

GRADUATE FORTNIGHT

The New York Academy of Medicine will hold its annual graduate fortnight from October 14 to 25, 1940. The purpose of the fortnight is to make a complete study and authoritative presentation of a subject of outstanding importance in the practice of medicine and surgery.

The subject of this year's fortnight is *Infections*. A carefully integrated program will include clinics and clinical demonstrations at many of the hospitals of New York City, evening addresses, and appropriate exhibits. The evening sessions at the Academy will be addressed by recognized authorities in their special fields, drawn from leading medical centers of the United States. The comprehensive exhibit will include books and roentgenographs; pathological and research material; and clinical and laboratory diagnostic and therapeutic methods. It is also planned to provide demonstrations of exhibits.

The subject of the fortnight will include the following:

Experimental basis of chemotherapy in the treatment of bacterial infections.
Clinical bacterial chemotherapy, results obtained and dangers encountered.
A general consideration of bacterial infections.
Recent advances in knowledge of streptococcal infections.
Infections of mouth, pharynx, and upper respiratory tract.
Infections of middle ear and nasal sinuses.
Infections of teeth and surrounding structures.
Influenza.
Pneumococcal infections.
Bacterial meningitis.
Infections of the urinary tract.
Gonococcal infections in the male.
Gonococcal infections in the female.
Osteomyelitis and pyogenic infections of joints.
Wound infections.
Puerperal infections.
Treatment of infections by methods other than chemotherapy.
Virus infections.
Acute poliomyelitis.
Brucellosis—undulant fever.
Rickettsial diseases.
Lymphogranuloma venereum.

Epidemic encephalitis.
Other forms of encephalitis and choriomeningitis.
Exanthematous diseases.

The Academy provides this program for the fundamental purpose of medical education. Consequently, all members of the medical profession are eligible for registration.

ADDENDUM

Since the article on *Borderline Adrenal Cortex Insufficiency* [1] was submitted for publication (1939) by Pennell and Beals, reports have been published regarding the use of a crystalline preparation, desoxycorticosterone acetate, made available synthetically in 1938 as a result of the work of Steiger and Reichstein.

Wilder [2] reports that according to Tooke, Power, and Kepler: [3]

Desoxycorticosterone acetate, given to a patient who is receiving a restricted supply of potassium, causes unusually rapid retention of chloride, sodium, and water, with attendant accumulation of intercellular fluid, increasing volume of blood plasma and disturbing elevation of venous pressure. The increased venous pressure, to judge from work from Visscher's laboratory, may be one of the factors responsible for the sudden cardiac deaths of patients receiving desoxycorticosterone acetate * * *.

Wilder [2] further states that:

The disturbing effects of desoxycorticosterone acetate can readily be controlled by suitable adjustment, at a higher level, of the intake of potassium * * * the intake of sodium is limited to what is represented by 5 gm. of salt, in addition to the salt contained in the food as it comes from the kitchen; also, the food either is made normally rich in potassium or extra potassium is provided in such an amount that the total intake will not be less than 4 gm. This is a right about face for us in the matter of diet for Addison's disease, but the evidence clearly indicates that an allowance of potassium which is dangerous in the extreme for the patient who is unsupported with cortical hormone is what is needed by the same patient receiving desoxycorticosterone acetate * * *.

Desoxycorticosterone acetate, powerful as it is as a regulator of the disturbed salt and water balance of Addison's disease, has been shown to be lacking in that activity of the adrenal cortex which has to do with neoglucogenesis and the maintenance during fasting of a continuous supply of glucose to the tissue. This so-called carbohydrate activity is possessed by some of the crystalline products which Kendall [4] has isolated from adrenal cortical extract. If one of them, with properties like Kendall's compound E_1 were as available as desoxycorticosterone acetate, a suitable mixture of it with desoxycorticosterone acetate would provide * * * just what is wanted. Unfortunately, such a compound is not yet available cheaply and in satisfying amounts, and until it is so available, physicians must guard against hypoglycemia by providing a steady supply of food material from which the organism can obtain the glucose it requires.

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[1] See page 391 of this issue.

[2] Wilder, R. M., Progress in Treatment of Addison's Disease, Proc. Staff Meetings Mayo Clinic, Vol. 15, No. 18, May 1, 1940.

[3] Took, T. B., Power, M. H., and Kepler, E. J., The increased tolerance of patients with Addison's disease to potassium while treated with desoxycortiocosterone acetate, Proc. Staff Meetings Mayo Clinic, Vol. 15, No. 23, June 5, 1940.

[4] Kendall, E. C., A clinical and physiological investigation of the suprarenal cortex. Cold Spring Harbor Symposia on quantitative biology. 5: 299-312, 1937.

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)

TREATMENT IN GENERAL PRACTICE. By *Harry Beckman, M. D., professor of pharmacology at Marquette University School of Medicine, Milwaukee Wis.* Third edition. 780 pages, indexed. W. B. Saunders Co., Philadelphia, 1938. Price $10.

The third edition of this book maintains its reputation as one of the best in the field of therapy. The general plan of the book has not been altered. Certain conditions such as those relating to obstetrics, drug addiction and some tropical diseases have been eliminated and new sections have been added which deal with nutritional disturbances, diseases of the liver and bile passages, disturbances of water, salt and protein balance, and disturbances caused by excessive heat. Many conditions are now considered for the first time such as alkalosis; Menier's disease; colon consciousness; physical allergy; nontropical sprue; myasthenia gravis; prophylaxis of the venereal diseases; and some ear, nose, and throat conditions. Notwithstanding these additions, the book is no larger than previous editions.

Chemotherapy with sulfanilamide is discussed under a chapter headed Sepsis (septicemia). Sulfapyradine and allied chemicals have been developed since this book was published and of course are not included. Both sides of controversies are given with comments by the author and references in the literature. An excellent and comprehensive bibliography, arranged alphabetically, is appended.

This book can be highly recommended as an excellent guide in therapy for daily use.

PRACTICE OF MEDICINE, by *Jonathan Campbell Meakins, M. D., LL. D., professor of medicine and director of the department of medicine, McGill University, physician in chief, Royal Victoria Hospital, Montreal, etc.*, 1365 pages, 521 illustrations, indexed, 26 by 17.5 cm.; C. V. Mosby Co., St. Louis, 1938. Price, $12.50.

The second edition of this most excellent textbook of medicine has been thoroughly revised and brought up to date. The general characteristics of the book have remained the same. Some conditions

have been amplified, others deleted, and the newer discoveries have been included. In the first edition, a distinctive feature was the inclusion of illustrations for the first time in a practice of medicine. This innovation has been continued in the present edition. The numerous illustrations are excellent and representative of the conditions depicted. A comprehensive bibliography is given at the end of each chapter.

Any criticisms which may be made are minor in character. On the whole, this work can be highly recommended to practitioners and students.

SYMPTOMS OF VISCERAL DISEASE, by *Francis Marion Pottenger, A. M., M. D., LL. D., F. A. C. P.; medical director, Pottenger Sanitarium and Clinic for Diseases of the Chest, Monrovia, Calif.; professor of clinical medicine, University of California.* Fifth edition, 442 pages, 97 illustrations. The C. B. Mosby Co., St. Louis, 1938. Price, $5.

The fifth edition of this valuable little book has had to be extensively rewritten in order to keep pace with the many recent advances in our knowledge of physiologic medicine. Visceral pain has been alloted a separate chapter and a chapter has been added describing the vegetative centers in the brain and cord. More emphasis has been given to the endocrines in their relationship to bodily function than in previous editions.

The book is divided into three parts. The first is devoted to a discussion of the physiology of the vegetative nervous system. The close interrelationship between the vegetative nervous system, the glands of internal secretion and the electrolytic balance is stressed. The second part takes up the relationship between the vegetative nervous system and the symptoms of visceral disease, while the third deals with the clinical application of the more important viscerogenic reflexes.

In view of the recent interest in visceral pain, the functional changes in blood pressure and the clinical syndromes which are expressed through the nerves supplying the heart, gastro-intestinal tract and lungs, this stimulating book merits study by both internists and surgeons.

PRACTICE OF ALLERGY by *Warren T. Vaughan, M. D., Richmond, Va.* First edition. 1,034 pages. The C. V. Mosby Co., St. Louis, 1939. Price $11.50.

Dr. Vaughan chose a fitting title for the first edition of his book, written entirely for the physician, his former two editions of Allergy and Applied Immunology from which this one naturally springs having been written for both the physician and the patient.

Specialists in the field of allergy and others already interested in this subject will need no introduction to this very valuable contribution to medicine.

Previously published books devoted to allergy have, in general, been recommended in these reviews because of the growing importance to medical practice of the findings through clinical research in this specialty. On that basis alone this book would be commended for study by the entire profession but it is particularly good and will be found of incalculable worth to anyone doing general or specialized medicine.

The author shows not only a mastery of the subject matter itself but also of the English language in his clear concise and easy diction. Furthermore he evidences a clever artistry in handling a Leica camera as the many original illustrations will show.

A HISTORY OF TROPICAL MEDICINE, by *H. Harold Scott, C. M. G., M. D., F. R. C. P., Lond., D. P. H., D. T. M., and H. Camb., F. R. S. E. director, Bureau of Hygiene and Tropical Diseases; member of the Colonial Advisory Medical Committee; late medical secretary, colonial Medical Research Committee; lecturer in tropical medicine, Westminster Hospital Medical School; Milner Fellow, London School of Tropical Medicine; Colonial Medical Service.* First edition, 2 volumes, 1,165 pages. The Williams & Wilkins Co., Baltimore, Md., 1939. Price $12.50, set.

There has been a great need for a history of tropical medicine because as the author states in the preface: "Here and there scattered in medical works dealing with diseases in the Tropics we find a few notes on the history of these diseases but speaking in any sense other than the narrowest, there is no history of the rise and development of tropical medicine. * * *" Scott has gone far to fill this need. To this reviewer his book is a monumental work before which he feels restrained to unconditional admiration.

The first chapter is of particular interest to naval medical officers. It deals with the development of hygiene and health in the navies and mercantile marine from the early days when commerce was all and the lot of the sailor a merciless fight against scurvy, fevers, and fluxes to the present day of immunized crews banqueting daily on fresh food. The second chapter contains a graphic description of the conditions under which the soldier lived in the warm climates during the eighteenth and nineteenth centuries and the development of measures to keep pestilence out of the armies. In the third chapter the reader is given brief sketches of the conditions in the British colonies and dominions as seen by the early explorers and government officials and in the fourth chapter a similar orientation in India, Australia, and New Zealand.

Then follows the history of the great diseases which have become associated with the Tropics—malaria, yellow fever, trypanosomiasis, leishmaniasis, leprosy, cholera, dysenteries, plague, and some less formidable ailments, dengue, undulant lever, relapsing fever, melioidosis, and ankylostomiasis. There is also a chapter on the history of diseases connected with the food in the Tropics—beriberi, pellagra,

scurvy, and some food poisons. In three short chapters the author adds some very readable historical data on Suez Canal, Panama Canal, and the slave trade and disease. The book is concluded by some brief biographies of a few of the men who have devoted their chief interests to tropical medicine.

The author has a delightful style of writing and it is a pleasure to read his accounts of the many interesting episodes that form the historical background for the present knowledge of tropical medicine.

HEART PATIENTS, Their Study and Care, by *S. Calvin Smith, M. D., Sc. D., formerly special heart examiner for the Surgeon General's office during the World War at home and abroad; author of "Heart Affections: Their Recognition and Treatment"; "Heart Records: Their Interpretation and Preparation"; "How is Your Heart?" (New York and London); "That Heart of Yours."* Lea & Febiger, Philadelphia. 1939. Price $2.

A pocket size compend of 166 pages for the use of the busy practitioner who desires to refresh his memory and keep abreast of the modern trend in heart conditions.

The author's aim has been "to present with clarity and precision all that is useful in older teachings on heart impairment and all that is practical in the maze of modern methods of heart investigation." It is noted that the author is not enthusiastic concerning the use of quinidine sulphate. In fact he states "The writer personally would never use this constricting drug in any heart condition." This the reviewer takes to include such a condition as ventricular tachycardia.

The electrocardiographic comments, particularly under the discussion on coronary occlusion, perhaps due to brevity, are not very informative.

The subject matter in general, however, contains many practical suggestions which the general practitioner will find invaluable in the management of heart patients.

THE ELECTROCARDIOGRAM IN CONGENITAL CARDIAC DISEASE, by *Maurice A. Schnitker, B. Sc., M. D., formerly resident physician, Peter Bent Brigham Hospital and assistant in medicine, Harvard University Medical School, Boston; associate attending physician, Toledo Hospital; junior staff physician, Lucas County Hospital, member active staff, St. Vincent's Hospital, Toledo, Ohio.* 147 pages. Harvard University Press, Cambridge, Mass. 1940. Price $3.

This work consists of a study of 108 cases, in which electrocardiograms were recorded, and of which 106 had autopsy data available and one proved by surgical procedure.

The case reports cover practically the entire range of congenital heart lesions and are divided into the usual three groups, namely, the acyanotic, the late cyanotic or cyanose tardive and the cyanotic with their specific lesion divisions, as coarctation of the aorta, patent ductus arteriosus, tetralogy of Fallot, etc. The cases were selected from the literature and added to by contributions from various sources, including 13 contributed by Dr. Paul D. White.

In his foreword, Dr. Samuel A. Levine, after mentioning Dr. Maude Abbott's part in stimulating interest in our knowledge of congenital heart disease, comments on the need for accurate diagnosis, in view, among other things, of the success of the ligation of patent ductus arteriosus and the possibility of further surgical attacks.

With accuracy in the recognition of congenital abnormalities in mind, Dr. Levine rightly states "we need all the carefully collected data on this subject that we can obtain."

This book will be a valuable addition to the library of those interested in cardiology.

THE ELECTROCARDIOGRAM AND X-RAY CONFIGURATION OF THE HEART, by *Arthur M. Master, B. S., M. D., F. A. C. P., associate in medicine, and chief, cardiographic laboratory, the Mt. Sinai Hospital, New York; associate in medicine, the college of physicians and Surgeons, Columbia University, New York.* 222 pages. 100 illustrations. Lea & Febiger, Philadelphia. 1939. Price $6.50.

This monography presents in atlas form 100 illustrations showing the teleoroentgenogram with corresponding electrocardiogram in various conditions affecting the heart contour and therefore the electrocardiogram, such as age, body position, habitus, obesity, pregnancy, hypertension, valvular disease, pulmonary disease, and deformities of the chest. Clear and concise comments introduce each chapter and illustration.

Those engaged in the interpretation of electrocardiograms should study the material contained in this work giving consideration to the author's remark in the preface: "The reader must appreciate that the pictures are presented to illustrate the premise and are not to be accepted in a statistical sense. The cases cited are merely examples and do not indicate that there are no exceptions."

The book should be included in any library that attempts to include an exhaustive reference on electrocardiography.

HUMAN HELMINTHOLOGY by *Ernest C. Faust, A. B., M. A., Ph. D., professor of parasitology and director of laboratories, Department of Tropical Medicine, Tulane University of Louisiana, New Orleans, La.* Illustrated with 302 engravings. Second edition. 780 pages. Lea and Febiger, Philadelphia, Pa., 1939. Price, $8.50.

This second edition of Faust's *Manual for Physicians, Sanitarians and Medical Zoologists*, will be welcomed by all interested in human helminths. In the intervening 10 years since the first edition appeared much new knowledge has accumulated in this specialized field. The author has incorporated the salient advances by a thorough revision, and the present edition thus offers an up-to-date textbook which will adequately meet the usual needs of clinicians and other workers interested in this subject. The newly added chapter on Anthelmintics and Their Use is of special practical value for physicians.

The book is well illustrated, a point of prime importance to the less experienced in the field.

THE DYSENTERIC DISORDERS, the Diagnosis and Treatment of Dysentery, Sprue, Colitis and other Diarrhoeas in General Practice, by *Philip Manson-Bahr, C. M. G., D. S. O., M. D., F. R. C. P., senior physician to the Hospital for Tropical Diseases, London, director, division of clinical tropical medicine, London School of Hygiene and Tropical Medicine; consulting physician to the Colonial Office and Crown Agents for the Colonies,* Appendix by *W. John Muggleton, M. S. M., technical assistant.* Illustrated with 9 colour, 14 black and white plates, and 106 figures. A William Wood Book published by the William & Wilkins Co., Baltimore, Md. 1939. Pp. 613. Price: $8.50.

The wide experience of the author well qualifies him to write on the subject of dysenteric disorders. The book is designed to be useful to the clinician facing the complex problems presented by patients who exhibit dysentery. From this standpoint much will be found which is of practical value. A further aim of the writer has been to present the unsolved problems pertaining to dysenteric disorders with the hope, as he says, of stimulating research. This has led to the inclusion of much material, which at least, from the standpoint of the practitioner requires the perusal of much that is not essential to his needs and could better have been omitted. There is a great deal of repetition and some parts are verbose.

The appendix is brief to the point of inadequacy. Certainly the colored plate on unstained human intestinal protozoa is misleading to anyone not familiar with their appearance as actually seen under the microscope.

Considering the well-known capabilities of the author, the book on the whole is disappointing.

PRINCIPLES OF PSYCHIATRIC NURSING by *Madelene Elliott Ingram, R. N., formerly on nursing staff of Colorado Psychopathic Hospital, Denver, Colo; head nurse, Butler Hospital, Providence, R. I.; supervisor of female service, Sheppard and Enoch Pratt Hospital; assistant superintendent of nurses and head instructor, Sheppard and Enoch Pratt Hospital, Towson, Md.* With a foreword by *Ross McC. Chapman, M. D., medical superintendent, Sheppard and Enoch Pratt Hospital.* Illustrated. W. B. Saunders Co., Philadelphia and London, 1939. Price $2.75.

An illuminative, practical, straightforward book covering the various features of the nurse's work in caring for the mentally ill. The material on such important subjects as personality development and disorder, classification of psychological conditions, the descriptions of each of the psychoses, general characteristics, miniature case studies, specific treatments, and individual care of psychotic persons is interesting and significant. The chapters on personal and mental hygiene provide the nurse with facts that will be of aid in a better understanding of the child and adult patient and her own mental life. Each chapter closes with general suggestions, summary outline, questions, and suggested references that should be especially helpful to the student and teacher in extending her knowledge of psychiatric nursing.

A TEXTBOOK OF LABORATORY DIAGNOSIS, by *Edwin E. Osgood, M. A., M. D.; associate professor of medicine and head of the division of experimental medicine, University of Oregon Medical School, and member of the staff of Multnomah County Hospital, and the consulting staff of the Doernbecher Memorial Hospital for Children, Portland, Oreg.* Third edition, 676 pages, illustrated with 27 figures and 10 colored plates. The Blakiston Co., Philadelphia, 1940. Price $6.

This book is a new, thoroughly revised and modernized edition of Dr. Osgood's already popular book on laboratory diagnosis. This edition, as in the previous one, is divided into two main parts. The first part deals with various pathologic conditions of the human body and those laboratory procedures which are of value in the diagnosis of each.

The second part gives the technic and the evaluation of these various procedures.

There are three indexes in the book. The first is an index by diseases. Under each disease is listed the laboratory tests that are useful in that condition. The second is an author index, while the third is the usual subject index. All are quite complete, well done, and are great time savers in finding pertinent information.

The book is well printed and bound in a neat, durable washable cover. It is recommended as a text to the medical student as well as a reference book for the internist and the clinical pathologist. The second part of the book is of particular value to the laboratory technician.

GROSS ANATOMY, by *A. Brazier Howell, Associate professor of anatomy, Johns Hopkins University School of Medicine.* First edition, 403 pages. D. Appleton-Century Co., New York and London, 1939. Price $6.

The author's subtitle, "A Brief Systematic Presentation of the Macroscopic Structure of the Human Body," gives, in a few words, the intended purpose of the book. It has been written from the viewpoint of a teacher who has had to contend with the reduction in the number of hours allotted for anatomical instruction. This book should by no means replace the large, detailed, and well illustrated textbooks, even with the additional use of an atlas. It is recognized that the medical curriculum has become more crowded each year; but it is hoped that the instruction time in so fundamental a subject as anatomy will not universally be cut to such a degree that this or some similar book would have to serve as a text. If so, it will soon be that no medical school will really teach anatomy.

The material in the book is orderly and logically arranged and reads very easily. By way of illustrations there are only a few scattered schematic diagrams.

As an adjunct to the study of anatomy, or, as a reading guide in reviewing the more important structures it should serve the student or the practitioner quite excellently.

AN INTRODUCTION TO HUMAN ANATOMY by *Clyde Marshall, M. D., assistant professor of anatomy, School of Medicine, Yale University.* Second edition, revised, with 257 illustrations, 14 in color. W. B. Saunders Co., Philadelphia, 1939. Price $2.50.

This textbook of human anatomy is one which places before the elementary student a simple, accurate, up-to-date and interestingly written account of the nature of this study.

Such subjects as cells and tissues, the digestive system, the nervous system and the topic on developmental anatomy are presented clearly and directly, making it possible for the student to intelligently understand the facts of organic structure and function and the development of the human body. The important points throughout the text have been stressed by the use of finer type and grouped under the heading of Practical Considerations. It is also attractively illustrated with numerous diagrams many in color, which add interest and clarity to the discussion. There is an index to facilitate the use of the book.

A TEXTBOOK OF ANATOMY AND PHYSIOLOGY by *Jesse Feiring Williams, M. D., Sc. D., professor of physical education, Teachers College, Columbia University, New York City.* Sixth edition, revised, with 367 illustrations, 29 in colors. W. B. Saunders Co., Philadelphia, 1939. Price $2.75.

A superior, stable, complete, comprehensive, and accurate depiction of the latest knowledge of anatomy and physiology. The value of this book to teachers and students is in its preparation of material, its stimulative effect, its clarity, and its method of presentation. At the end of each chapter there is a summary of the main points and suggestions on how to study each chapter followed by practical exercises to assist the students to learn thoroughly the subject matter.

References are selected. Glossary includes a detailed explanation of all the chemical and physical terms used. Index is complete. Illustrations throughout the book are splendid.

THE ANATOMY OF THE NERVOUS SYSTEM by *Stephen Walter Ransom, M. D., Ph. D., professor of neurology and director of Neurological Insititute, Northwestern University Medical School, Chicago.* Sixth edition. 507 pages. 382 illustrations, some in color. W. B. Saunders Co., Philadelphia, 1939. Price $6.50.

This well-known book hardly needs an introduction as it is widely used as a text of neuro-anatomy in many medical schools.

It has only been 4 years since the fifth edition appeared, but recent work on the connections of the various thalamic nuclei with the cortex has required a revision which has been ably done.

As in past editions the functional significance of the various structures of the nervous system is stressed, thereby adding value to the book as well as making a dry subject much more interesting.

The book is well done from the publisher's standpoint.

It is invaluable to the student and practitioner of neurology.

A TEXTBOOK OF BACTERIOLOGY (The application of bacteriology and immunology to the etiology, diagnosis, specific therapy, and prevention of infectious diseases for students and practitioners of medicine and public health), by *Hans Zinsser, M. D., consulting bacteriologist to the Peter Bent Brigham Hospital and the Children's Hospital, Boston* and *Stanhope Bayne-Jones, M. D., professor of bacteriology and dean, Yale University Medical School, master of Trumbull College, Yale University, New Haven, Conn.* Eight edition, revised and reset 957 pages. D. Appleton-Century Co., Inc., New York, 1939. Price $8.

This new edition of a well-known and widely recognized textbook of bacteriology may still be considered a requisite to all libraries, teaching institutions, and laboratories having to do with medical bacteriology. It is not looked upon as a textbook suitable for the medical student because it contains far more than the essential tersely stated known facts and most widely accepted concepts. In this respect the title could well be changed to Medical Bacteriology.

The authors have wisely deleted the section on medical protozoology. Whereas only deletions were made and much valuable rewriting was done, it is true with this book, as is so often the case with books of this type whose debut was made so many years ago, that more pruning and rewriting should have been done. Nevertheless it is still a good book and is therefore recommended.

The publishers do an excellent job of printing and binding.

FUNDAMENTALS OF PHARMACY—Theoretical and Practical by *Walter H. Blome, Ph. C., M. S., M. A., Wayne Udiversity College of Pharmacy, Detroit, Mich.*, and *Charles H. Stocking, Ph. C., M. S., University of Michigan College of Pharmacy, Ann Arbor, Mich.*, with a chapter on hospital pharmacy by *Edward C. Watts, B. S. Pharm., assistant chief pharmacist, University of Michigan Hospital., Ann Arbor, Mich.* In one volume, 364 pages including index, with 157 illustrations. First edition. Lea and Febiger, Philadelphia, Pa., 1939. Cloth. Price: $4.50.

There has been a feeling for some time among teachers of pharmacy that a real need exists for a textbook on pharmacy in concise form. The *Fundamentals of Pharmacy* treats of the pharmaceutical art in sufficiently complete style to furnish the student with a working knowledge of the theory and practice of pharmacy.

The book is divided into two parts. Part I is devoted to a discussion of the application of the principles of chemistry, physics, and biology to pharmacy. Part II includes discussions of the manufacture of various types of pharmaceutical preparations.

An important feature of this book is the inclusion of considerable historical material which provides an interesting and informative basis for the study of the different classes of official preparations.

The concluding chapter deals with hospital pharmacy, which is of timely interest because of the effort of leaders in pharmaceutical education to have this important specialty recognized as a necessary phase of public health service. While not very much of the material in this chapter is applicable to pharmacy in naval hospitals, the book

in its entirety is admirably suited as a supplement to the Handbook of the Hospital Corps, United States Navy, for hospital corpsmen preparing for the examination for pharmacist.

DENTAL CARIES: Findings and conclusions on its causes and control, stated in 195 summaries by observers and investigators in 25 countries. *Compiled for the Research Commission of the American Dental Association by the Advisory Committee on Research in Dental Caries: Daniel F. Lynch, chairman, Charles F. Kettering, counselor, William J. Gies, secretary,* New York, 1939. Price: $1.

This publication is a compilation representing answers to a questionnaire sent by the Research Commission of the American Dental Association to observers and investigators in this problem all over the world. It is divided into two parts; the first part dealing with the reports of the contributors, and the second with a general analysis of the findings and conclusions of the contributors.

The volume serves the purpose of exposing the confusion that still exists among the many investigators and observers in this part of the dental caries problem. It analyzes in an excellent outline the sum total of the replies of the contributors stressing the main factors theoretically responsible for dental caries in addition to those facts actually known about the condition.

Unfortunately, some of the replies to the questionnaire have no bearing on the purpose of the publication, reports having been made on results rather than causes of dental caries. In other instances replies contain much pseudo-science rather than results of direct observations or experimentations. Apparently some replies by prominent workers and investigators in the field, because of their extensive work and realization of the many complexities associated with a problem of this nature have hesitated to give any other than the most meagre information. The information imparted, however, can be depended upon to be a result of actual research. This last appears to be more in keeping with a true scientific spirit.

As in all publications there is much theory, some of which cannot be substantiated by direct statistical analysis, observation or experimentation. The contributions especially of the laboratory and field workers to this publication are to be commended.

A compilation such as this undoubtedly required a great expenditure of effort and the Advisory Committee on Research in Dental Caries has done much to answer a long-felt need, namely that of bringing up to date the status as it exists on the problem of the etiology of dental caries. It is a good reference publication and may be recommended as such to all hospitals and large dental activities.

CLINICAL DENTAL ROENTGENOLOGY: Technic and Interpretation, by *John Oppie McCall, D. D. S., F. A. C. D., director, the Murry and Leonie Guggenheim Dental Clinic, former professor of periodontia, New York University College of Dentistry, visiting lecturer in periodontia, New York University College of Dentistry; consultant to the dental service, New York Hospital; associate in public*

health and preventive medicine, Cornell University College of Medicine; lieutenant commander, Dental Corps, United States Naval Reserve; and *Samuel Stanley Wald, D. D. S., F. A. C. D., head of the department of diagnosis and roentgenology, the Murry and Leonie Guggenheim Dental Clinic and School for Dental Hygienists; assistant professor of roentgenology, New York University College of Dentistry; lecturer in dental radiology, New York University College of Medicine; visiting dental surgeon, Nazareth Trade School; consultant to the dental service Community Service Society of New York; lieutenant, Dental Corps, United States Naval Reserve.* First Edition: 319 pages, 1046 illustrations and 355 figures. Published by W. B. Saunders Co., Philadelphia, 1940. Price $5. 50.

The authors, widely recognized as authorities in their field and certainly well qualified to record text-book material, have compiled an excellent edition on dental roentgenology that caters to both the expert and the novice in dentistry. Probably, the best methods of presenting text material involve frequent use of illustrations. From that particular angle, this book deserves much credit. It has some 1,046 illustrations in 319 pages of easy reading type. One hundred and eleven illustrations were devoted to the technic of film placement alone.

Another noteworthy feature is the presence of a concise, yet complete and modern bibliography at the end of each chapter. This alone would distinguish the book from many of the other fine publications along this line that have been released recently. The final outstanding feature is perhaps the best material ever presented on child radiology. The normal and abnormal in teeth and their related structures with excellent illustrations, particularly of the normal dentition from the prenatal period to maturity, are presented in the most modern fashion.

Of special interest to the oral surgeon, is the material on an improved and systematized technic for localization of impacted, supernumerary, and unerupted teeth; fractured roots; teeth and cysts in the maxillary sinus; foreign bodies etc. The reviewer has been able to notice only two items in which it might be considered lacking. The expert in dentistry will miss a chapter on roentgenology in relation to some of the major phases of oral surgery such as Paget's disease (osteitis deformans), fibroma, chondroma, myxoma, and fibro-sarcoma. The novice will miss the diagrammatic sketches that are so helpfully explanatory and used most frequently in current texts.

HANDBOOK OF SKIN DISEASES, by *Leon Hugh Warren, B. A., M. D., M. Sc. (Med.), formerly instructor in dermatology and syphilology at the School of Medicine, Temple University; acting assistant surgeon (dermatology) in the office of dermatoses investigations of the United States Public Health Service; assistant dermatologist, Philadelphia Methodist Hospital; member of the Society for Investigative Dermatology; fellow of the American Academy of Dermatology; fellow of the American Academy of Dermatology and Syphilology,* with a foreword by *Frederick D. Weidman, M. D.* 321 pages. Paul B. Hoeber, Inc., Medical Book Department of Harper & Bros., New York, 1940, price $3.50.

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This is just the kind of text on dermatology that appeals to this reviewer. It is much smaller and briefer than the encyclopedic works in which the general practitioner can lose his way, and even smaller than the usual formal texts on dermatology. Yet it is far above the lowly quiz compend in content, style, dignity, and purpose.

It contains in commendably compressed yet adequate form practically all the information the student or general practitioner might seek in the field of dermatology. The outlook of the author is strictly modern. The reviewer must agree heartily with the writer of the foreword that "the book merits success; the profession needs the book."

MANUAL OF DERMATOLOGY, by *Carroll S. Wright, B. S., M. D., professor of dermatology and syphilology, Temple University School of Medicine; associate professor of dermatology and syphilology, Graduate School of Medicine, University of Pennsylvania; fellow of the College of Physicians of Philadelphia; member of the American Dermatological Association; American Academy of Dermatology, Society for Investigative Dermatology; consulting dermatologist, Temple University Hospital, Philadelphia Municipal Hospital, Shriner's Hospital, Widener Home for Crippled Children, Elwyn Training School, Vineland Training School, Pennsylvania Institution for the Blind.* Published by The Blakiston Co., Philadelphia. 376 pages, 138 illus. Price $4.

This small book concerning diseases of the skin is excellent for the purpose intended, namely, a reference work for students and general practitioners. The essential facts of diagnosis and therepy of the most common skin diseases are briefly and clearly described. Many excellent photographs are included.

In some instances these might have depicted, more profitably, earlier manifestations of the disease illustrated. For instance, most junior medical students can diagnose leprosy when the leonine facies have developed whereas probably few could diagnose an early lesion.

Several eminent dermatologists have recently considered that much of the lore of ponderous tomes may well be discarded.

This is the third miniature dermatological text the writer has seen lately; and, everything considered, this is at once the smallest and best. It fits the pocket but can fill a head.

TUMORS OF THE SKIN, by *Joseph Jordon Eller, M. D.; attending dermatologist, City Hospital, New York City; consulting dermatologist, French Hospital, Broad Street Hospital, New York; Morristown Memorial Hospital, Monmouth Memorial Hospital, Fitkin Memorial Hospital, New Jersey; Norwalk General Hospital, Connecticut; Unity Hospital, Brooklyn; Morelos Hospital, Mexico, etc. Member: American Dermatological Association, New York Dermatological Society, American Board of Dermatology and Syphilolgoy. Fellow: American Academy of Dermatology and Syphilology, New York Academy of Medicine; Panamerican Medical Association, American Association for Cancer Research, Society of Plastic and Reconstructive Surgery, Society for Investigative Dermatology, etc.* First edition, 607 pages, illustrated with 403 engravings. Lea and Febiger, Philadelphia, 1939. Price $10.

In this book most of the benign and malignant tumors of the skin have been brought together under one cover. This saves considerable time in searching numerous books or in skipping around through the larger general pathologies in order to find pertinent information concerning some particular case.

The book is written primarily for the clinical dermatologist and deals mainly with the clinical diagnosis and treatment of skin tumors. Under each condition there is included a sufficient description of the cellular changes to convey a fair idea of the pathology.

There is also a valuable chapter on cutaneous surgery and plastic repair incident to the treatment of skin tumors as well as a rather extensive appendix dealing with radiation physics and biology with dosage tables for the treatment of skin tumors.

The volume is profusely illustrated by black and white engravings of both the gross and microscopic lesions, and is well bound and printed. It is recommended to the practitioner who frequently sees or treats skin tumors.

DIAGNOSIS AND TREATMENT OF DISEASES OF THE HAIR, by *Lee McCarthy, M. D., formerly clinical professor of dermatology, Georgetown University Medical School; attending dermatologist to Garfield Memorial Hospital.* 671 pages with 291 illustrations, 7 in color. C. V. Mosby Co., St. Louis, Mo., 1940. Price $9.50.

In justification of the existence of a medical book, one may offer the reason, the fact, if indeed it is a fact, that the book brings forth something really new, in form if not in substance. There is such a reason and a quite valid one in the case of this book. True, the substance could perhaps be culled by searching through a number of volumes on dermatology and various monographs and texts which include material on the anatomy and histopathology of the hair. But to bring all this information on the subject of hair and nothing but hair together in one volume is something rather unique.

It is doubted if many medical libraries, public or private, contain a comparable volume. Confronted by a case of hair anomaly or malady the practitioner would be grateful to have access to such an authentic and informative volume as this one.

The divisions of the book are so few that they may be enumerated: Anatomy, hygiene, etc.; pigmentation disturbances; atrophies; hypertrophies; inflammatory diseases of follicles; other scalp diseases affecting the hair; endocrine relations; scalp tumors.

There is so little information and so much misinformation extant on this subject, rather particularly concerning cosmetics, that many a layman could read parts of this book with profit and the general practioner could read practically all of it to advantage.

UNTO THE FOURTH GENERATION, Gonorrhea and Syphilis, What the Layman Should Know, by *Irving Simons, B. S., M. D., fellow of the American Urological Association; fellow of the New York Academy of Medicine; passed assistant*

surgeon, U. S. Naval Reserve in charge of urologic surgery at the naval hospital, Norfolk, Va. (1918–19). First edition, 243 pages with 18 illustrations, by M. Emanuel, M. D. E. P. Dutton & Co., Inc., New York, 1940. Price $2.50.

The title is ill chosen because it is misleading. No great grandparent is directly responsible for gonorrhea or syphilis in his great grandchild. Really the author does not infer as much, nor, so far as the reader can learn, does the author believe it, but the book is intended for lay reading and it is difficult enough to avoid lay misinterpretation. With the Biblical quotations that appear, this misinterpretation is still more apt to occur.

In general the book is well and simply written. However, 20 reading hours or 243 pages are probably more than the average layman needs to teach him the early signs and symptoms of venereal disease, which is the prime purpose intended.

Most of the information offered is authentic and ethical. The diagrammatic drawings are clear and useful. However, highly controversial topics are treated with rather dogmatic attitude.

The author inclines to the New World theory as to the origin of syphilis in opposition to the preponderance of authority. He places more reliance upon the complement fixation test for diagnosis of gonorrhea than do most authorities on the subject. If gonorrhea causes as much sterility as the author intimates, it is strange that contraceptives have such vogue. The author is entirely too sanguine concerning prophylaxis nor is this subject sufficiently emphasized.

MODERN UROLOGY FOR NURSES, by *Sheila Maureen Dwyer, R. N., supervisor, J. Bentley Squier Urological Clinic, Presbyterian Hospital, instructor in urological nursing, College of Physicians and Surgeons, Columbia University, New York City* and *George W. Fish, M. D., associate professor of urology, College of Physicians and Surgeons, Columbia University, New York City* with a foreword by *Helen Young, R. N., director of nurses and nursing service, Columbia-Presbyterian Medical Center in the City of New York.* Illustrated with 60 engravings and published by Lea & Febiger, Philadelphia, 1940. Price $3.25.

This little book affords an excellent text, especially for the nurse preparing to specialize in modern urological nursing, though it may be studied with advantage by anyone concerned with a urological service be that person student or graduate nurse, corpsman, intern or doctor.

The text is especially replete with exact lists of supplies essential to proper conduction of various urological maneuvers.

The authors rightly assume that a nurse with proper working knowledge will prepare for various procedures, the nature of which she understands, better than if she acts upon orders merely as an automaton. Special points in urological nursing care are emphasized.

Many valuable suggestions are offered to assist the founder of a new service in laying out floor space to best advantage as well as in

selection of proper instruments and adequate equipment, the parent model being the Squier Urological Clinic at Columbia-Presbyterian Medical Centre.

The text concludes with an outline for a practical teaching program for nurses who intend to specialize in urological nursing.

BLOOD GROUPS AND BLOOD TRANSFUSION, by *Alexander S. Wiener, A. B., M. D., serologist and bacteriologist in the office of the chief medical examiner of New York City.* Second edition. 306 pages, 52 sections. Charles C. Thomas, Publisher, Springfield, Ill., and Baltimore, Md. Price $5.

This second edition has been brought up to date to include all the advances in blood grouping and blood transfusion which have been developed since the first edition appeared in 1935. The four blood groups are presented in a comprehensive manner, including sources of error, heredity, subgroups A and AB, agglutinogens M and N, heredity of agglutinogens M and N, individual differences in human and animal blood, anthropological investigation, and blood groups in relation to disease and tissue transplantation. Blood transfusion is discussed with respect to history, selection of donors, indications, results, technic, and reactions. The advantages and disadvantages of stored blood are included. There are well-written chapters on medico-legal applications of blood tests in disputed parentage and individual identification of stains in forensic cases.

The subject matter is completely and well covered. This book is recommended not only to those interested in blood transfusion but also to those concerned in medico-legal aspects of parentage and identification of blood stains.

FRACTURES by *Paul B. Magnuson, M. D., F. A. C. S., associate professor of surgery, Northwestern University Medical School, attending surgeon, Passavant Memorial Hospital and Wesley Memorial Hospital, Chicago.* Third edition, 511 pages, 317 illustrations. J. B. Lippincott Co., Philadelphia and Montreal. Price $5.

In this third edition, the author has added advances which have been made in the treatment of fractures since the second edition was published in 1936. The section on intracapsular fractures of the neck of the femur has been completely rewritten and illustrated. The first four chapters take up the fundamentals upon which fracture treatment is based, including pathology, repair, anatomical mechanism and the equipment necessary in treatment. The mechanics, symptons, and treatment of fractures are presented in a clear, brief, and concise manner. The methods of treatment are for the greater part those which the author from his vast experience has found satisfactory, many of which are original methods. Some of the mechanical devices which have simplified reduction and maintaining position are not included. The displacements of fragments as result of muscle pull are excellently illustrated. The photographs and

roentgenograms are deserving of comment. The bibliography includes most of the important articles on fracture treatment. This book, although not a voluminous treatise on the subject, should find a place among those concerned in the treatment of fractures.

HANDBOOK OF ORTHOPEDIC SURGERY by *Alfred Rives Shands, Jr., B. A., M. D., medical director, Nemours Foundation, Wilmington, Del., associate professor of surgery in charge of orthopedic surgery, Duke University School of Medicine, Durham, N. C.* (On leave of absence). In collaboration with *Richard Beverly Raney, B. A., M. D., associate of orthopedic surgery, Duke University School of Medicine.* Second edition, 567 pages, 154 illus. The C. V. Mosby Co., St. Louis, 1940. Price $4.25.

In this second edition, the author has brought up to date the new and accepted form of therapy. The purpose of the book is to present the fundamental facts and principles of orthopedic surgery for the medical student and the general practitioner as concisely as possible and still cover the subject in a well-rounded manner. This the author has accomplished.

The subject matter of the text is divided into 24 chapters. This was done because the committee of undergraduate instruction in orthopedic surgery of the American Orthopedic Association in 1934 called attention to the fact that approximately 24 class periods of an hour's duration are profitably employed for undergraduate orthopedic instruction.

The majority of the illustrations are original pen and ink drawings. No photographs or roentgenograms have been reproduced directly. They have all been redrawn to bring out certain characteristic features. The bibliography covers 63 pages and is brought up to date to July 1939.

The book shows great painstaking care in preparing. It should be an excellent book for the student and general practitioner.

TUMORS OF THE HANDS AND FEET, edited by *George T. Pack, B. S., M. D., F. A. C. S., assistant clinical professor of surgery, Yale University School of Medicine, and Cornell University College of Medicine; attending surgeon, Memorial Hospital for Cancer and Allied Diseases.* First edition, 138 pages, illustrated. The C. V. Mosby Co., St. Louis, 1939. Price $3.

The author has segregated from the larger general works on pathology and neoplasms the tumors usually seen in the hands and feet. While these tumors are no different from the same types found in other parts of the body and this monograph adds nothing particularly new to the literature, it nevertheless serves as a guide as to the tumors to be found in these regions and gives the result of treatment that has been used. Subungual melanoma are particularly emphasized. The text is illustrated by numerous rather mediocre pictures in black and white. A comprehensive list of references is given.

THE DIVISION OF PREVENTIVE MEDICINE

Commander C. S. Stephenson, Medical Corps, United States Navy, in charge

INDUSTRIAL INJURIES IN THE UNITED STATES DURING 1938[1]

Industrial accidents during 1938 resulted in the death of 16,400 persons, of whom 15,000 were wage earners and 1,400 self-employed, according to estimates of the United States Bureau of Labor Statistics. Permanent injuries affected a total of 98,900 persons, and temporarily disabling injuries another 1,260,300. The total of all types of injuries was 1,375,600.

These figures indicate marked decreases from those of 1937, for which 19,600 fatalities, 126,700 permanent and 1,691,700 temporary injuries were estimated. These decreases can be explained on the basis of two factors—(*a*) a lowering of the frequency of industrial injuries, and (*b*) a decrease in employment. For the entire group of industries, a 21 percent decrease in disabling injuries per million workers accompanied a 16 percent decrease in employment. In mining and quarrying, for example, the number of disabilities decreased 10 percent per million workers, and employment decreased 13 percent. Similarly, in manufacturing the respective decreases in disabilities per million workers and employment were 29 and 16 percent; in wholesale and retail trade, 4 and 4 percent; in railroads, 15 and 15 percent. On the whole, the rate of disabling injuries per million workers decreased more sharply than did employment, indicating a real improvement in the industrial-injury situation over 1937.

The 19,177 establishments from which reports were received for both 1938 and 1937 experienced a decided improvement in frequency and severity rates. Employment and employee-hours worked decreased by 15 and 22 percent respectively, but the number of fatalities decreased by nearly one-third, permanent injuries by nearly one-half, and temporary injuries by one-third. The total time charge for these injuries decreased by fully 40 percent. Against an average time loss of 4 days for every worker employed during 1937, the 1938 figure was only 2.9 days. For these years the frequency rate, injuries per million hours worked, decreased from 16.77 to 13.98, and the severity rate, average days lost per 1,000 hours worked, decreased from 2.17 to 1.66.

[1] Summarized from the Monthly Labor Review of the Bureau of Labor Statistics, United States Department of Labor, September and October 1939.

Practically all industries surveyed showed decreases in their industrial-injury rates. In only seven industries did total employee-hours worked show an increase over 1937, and in five of these seven, the increases in exposure were accompanied by decreases in frequency rates. With few exceptions, decreases in exposure went along with decreases in frequency and severity rates.

ACCIDENTS IN THE CONSTRUCTION INDUSTRY

A survey covering 412 establishments—58 in heavy and railroad construction, 99 in highway construction, and 255 in building construction, of which about 50 were specialized subcontractors, revealed that during 1938 there were 76 disabling injuries for every million hours worked, and 11 days lost for every thousand hours worked. From this survey it is possible to estimate the total of disabling injuries for the entire construction industry. The safety record for the Government operations was considerably better than that of private construction in 1938, although in the latter also there were a large number of very good safety records in individual companies.

The building-construction group had a frequency rate of 63 disabling injuries per million hours worked, and a severity rate of 7 days of disability per 1,000 hours worked. Heavy and railroad construction (primarily heavy construction), represented by 58 establishments, had a frequency rate of 90, and a severity rate of 15. Highway construction, including 99 establishments, showed a frequency rate of 77 and a severity rate of 11. Heavy and railroad construction, therefore, was the most hazardous, both as to frequency and severity; and building construction was the least hazardous. Nevertheless, these rates are all very much higher than those for manufacturing industries, and indicate the need for greater safety precautions. From the accident reports furnished, it appears that some establishments had very few disabling accidents, while others had a considerable number of such accidents. Further, it is clear that many of them could have been prevented by the observance of proper safety precautions, as is illustrated by the following accidents.

SAFETY PRECAUTIONS

Accidents Due to Their Violation

1. The feet of ladders should be secured against slipping or tilting. The foot of the ladder should not be placed more than one-fourth its length away from the vertical plane of its support and should be secured to prevent all possibility of slipping. Ladders set up for more than occasional use should be secured at top and bottom. Ladders should not be used when arduous and strenuous action on

the part of the workman is required. Ladders equipped with strong self-locking spreaders do not "crawl" easily. Workers should be required to use ladders and not permitted to climb unsafe structures. Often such chance-taking is due to inconveniently placed or unsafe ladders.

A man was working on top of a stepladder putting on window trim. Ladder was resting on terrazzo floor. The ladder started to "crawl" and this caused worker to lose his balance. He fell, striking his head on a miter box and on floor, sustaining serious injuries.

An employee, while descending ladder, caused it to slip accidentally. He fell from a height of 10 feet causing a fracture of the first and second lumbar vertebra when he struck the concrete floor with his back. The injury caused 6 months' disability.

Employee climbed down a wood column form instead of using ladder. When halfway down to landing, he fell on reinforcing-steel dowels and punctured abdomen.

2. Scaffolds should be securely attached to the structure that they are serving. They should be constructed to take the maximum material load which may be placed upon them, as well as the weight of the men. They should be equipped with toeboards to prevent material from falling off and should have guard rails. They should be inspected frequently and kept clear of loose materials and tools.

A painter and his helper fell from a scaffold when a sudden gust of wind blew it down. The painter was bruised, and helper's arm broken.

Carpenter fell from scaffold which he had erected but did not nail securely enough. He was disabled for more than half a year and sustained permanent injury to arm.

In another accident report, a scaffold about 10 feet high, collapsed, and worker fell and broke small bone in foot.

A metal rail fell from scaffold above and struck man on back of head, resulting in fractured skull. The worker was in hospital for about 2 months and was disabled for 90 days.

A carpenter, building forms on a scaffold, missed his footing and fell approximately 26 feet to concrete floor below, sustaining fatal injuries.

A worker stepped on loose steel on scaffold and slipped and fell about 2 feet, breaking his ankle and causing 80 days' lost time.

A wheelbarrow filled with concrete slid off scaffold and fell to floor below, injuring carpenter working beneath scaffold. His hip was fractured.

3. Open spaces, such as skylights and shafts, should be adequately guarded with barricades of sound substantial construction.

One employee while stepping from one part of building to another, fell through skylight into air shaft. He fell four stories down air shaft to the ground, receiving a fractured skull, fractured vertibrae, and shock. Death occurred shortly after the fall.

An employee was working in an elevator shaft when struck by falling barricade made of 2 by 4 inch studding and plywood panels which had been dislodged by wind pressure. After this barricade struck employee, he fell 30 feet into pit. Medical examination showed he had a compound fracture of skull, with injury to hearing. He was released from the hospital with a permanently impaired mentality, a permanent total disability.

4. Men should be instructed in proper and safe methods of lifting and hoisting. Hoisting rigs should be properly designed, installed, and operated. Hoisting cages should be enclosed, usually by heavy wire screens. Hoists should not be operated without proper signals and signals are satisfactory if the engineer can always see the signalman.

A worker strained his arm in attempting to move jammed stone by hand. Crowbars should be used for work of this type.

A worker sprained back while lifting stone to place in wall. He was standing on side slope and slipped.

A worker used a piece of pipe to roll a log. The pipe slipped and the injured fell over the log, suffering severe contusions of the chest. This is an illustration of the use of improper tools. This operation should be performed with timber hooks and crowbars and not with a piece of pipe.

A worker attempted to erect a light pole by chaining the pole to the body of a dump truck and then raising hoist. The chain slipped and pole fell, injuring instep of man guiding pole. A crane was available, if required. Pike hooks should have been used for this operation and probably the available crane as well.

A worker was struck on head by reinforcing rods falling from a wheelbarrow on hoist cage when a high wind tipped barrow. The resulting injury caused a time loss of 11 days.

A man was taking wheelbarrows off hoist at sixth floor. Another workman on second floor wanted the hoist cage and gave signal to engineer to let down cage before man on sixth floor was off cage. The latter worker fell one floor before cage was stopped and he was bruised and shaken up. This accident could have been prevented if the engineer had not moved the hoist until he had received a signal that the man on the sixth floor was off the hoist.

A shaft bucket became entangled with another and raised it 300 feet in shaft. The second bucket fell, killing one man and seriously fracturing the leg of another.

An employee was crushed when struck across his back by wooden piling which fell from leads of pile driver. A fellow employee had failed to fasten securely the piling in the leads.

The gear on a hand-operated iron winch slipped out of mesh, dropping one side of a scaffold. Two men fell 60 feet. One was killed and the other seriously injured. A third worker held on, but was struck in the back by the scaffold. The gear on the iron winch obviously was defective. Equipment of this nature should be thoroughly inspected before using, and should not be used unless in good mechanical condition.

A fireman left his cab without notifying engineer or foreman. The crane revolved and pinned the fireman between cab and sheet piles, fatally crushing him. Before operating crane, the operator should have made sure that no one was in the path of the crane.

A worker was hoisting some 2- by 10- by 10-inch boards from a tunnel shaft. Boards were tied with rope which slipped, releasing the boards which fell to the bottom of shaft. One of the boards fell back into the tunnel after hitting bottom, striking the worker on back, inflicting injury of undetermined severity. Care should be taken before hoisting material that it is securely attached. The load should be lifted off the ground to test fastening, but further lifting should not take place until men below are in the clear.

A rock fell from dipper of shovel and hit a man in the heel as he was rushing away. This caused an injury, with probable incapacity of 6 months. The shovel operator should not have moved the dipper over an area in which a worker was present, and workers should keep clear of working area of dipper.

A worker rode a gravel conveyor to the top of a bin. His clothing caught in the drive chain, pulling him into the driving mechanism and injuring his leg so that it had to be amputated.

5. An unsafe area should be roped off and workers should not be permitted in it.

A worker was walking by overhanging embankment left by a recent flood when a portion of the embankment fell and killed him.

During the placing of sheet piling around cribbing timbers to protect new excavation, the bank caved in suddenly. One man was killed and 3 injured. Such excavations should be shored properly.

An employee working in a quarry had his leg so smashed by rock falling from bank that it had to be amputated. Loose rock should be removed from banks where men are working. Systematic inspection and sealing of banks will prevent such accidents.

A worker was shoveling fine sand into the hopper of a recovery tunnel. He undercut the bank and was suffocated before a rescue could be effected.

While tunneling under a road, a worker was caught in a cave-in, losing one ear and sustaining a jaw fracture and injuries to head, shoulder, and internal organs. The tunnel should have been shored properly.

6. Machinery should be properly guarded and safety devices worn.

An employee, while working around the crusher, had his arm caught in the sprocket chain and pulled through cogwheel between chain and wheel. The arm had to be amputated at the elbow.

While man was cutting wood on power saw, sliver flew up and lodged in his eye, causing a practically total loss of sight in this eye.

An employee was taking apart a water pump so as to move it. In loading some heavy pipe, he caught his finger between the pipe and bed of truck, crushing one finger which had to be amputated.

A worker reached through a drive belt with his foot to open a valve. His foot caught in the belt and several bones were broken.

7. All projecting nails should be carefully removed, hammered in, or bent over in a safe way.

While section of shanty was being lifted from ground to low roof, a section caught on underside of roof and slipped down. A protruding nail lacerated a worker's upper lip, causing serious facial disfigurement.

A worker stepped on protruding nail in scrap lumber, partially covered by snow.

8. Materials should be piled carefully so as to avoid rolling or sliding.

A piece of broken concrete rolled from pile of material being loaded onto truck, striking a worker on shin and causing a bruised shin bone which necessitated an operation.

9. Floors and runways should be kept clean and provide secure footing. When they are wet or slippery, the care required of workers should be more than that ordinarily exercised.

A man slipped off wet plank and fell, hitting head and back on runways, causing an injury to the neck which resulted in paralysis, a permanent total disability.

An employee slipped on wet mortar and landed on handle of a wheelbarrow. Two of his ribs were cracked.

It will be seen from these accidents and the safety suggestions given with them, that the basic reasons for the accidents often were lack of proper supervision and inspection, lack of proper safety instructions, and at times the absence of elementary safety precautions.

REDUCTION IN PERSONNEL INJURIES

REPORT FROM THE U. S. S. "TENNESSEE" FOR 1939

During the calendar year 1939 an effort was made on the U. S. S. *Tennessee* to reduce the incidence of injuries to personnel in order to reduce the loss of working time due to such incidence. The measures taken to effect this objective included: (*a*) Monthly report by all heads of departments of all accidents resulting in injuries, with statement of action taken to prevent recurrence; (*b*) collection and monthly analysis of statistics; (*c*) designation of the medical officer as Safety Officer.

This effort produced gratifying results as a contribution to the sum total of knowledge regarding the cause, frequency, types of injuries incurred, loss in man hours, and prevention of accidents aboard ship. A summary of these results is reported by the medical officer and forwarded officially to the Navy Department with commendatory endorsements. The following comments are abstracted from this report:

The prevention of accidental injuries is of very great importance in the naval service. The hazards incident to large numbers of men working with highly specialized machinery must necessarily produce a constant occurrence of accidents to personnel. These may be classified as those unavoidable and those due to carelessness. In the former group may be placed those due to failure of matériel, and also those occurring in certain emergencies where personnel must think and act primarily in terms of preservation of life and property.

In reviewing the records of injuries received by personnel of this ship sufficiently severe to require medical attention, it is noted that by far the greater number of accidents must be classed as avoidable and due to carelessness on the part of the person injured or of others. It is also to be recalled that a not insignificant number of injuries have been incurred as a result of accidents ashore.

The steps taken in accident prevention have been carried out along various lines. Frequent inspections of working spaces have been made, especially those spaces where extra hazards exist. Lighting, ventilation, and proper use of safety appliances and precautions have been checked frequently. As a result of these surveys, it is the opinion of the medical officer that conditions of work, including safety appliances, are satisfactory. Most of the injuries received may be directly traced to carelessness.

A summary of accidental injuries occurring during 1939 as compiled from detailed monthly reports is reported in table 1.

TABLE 1

Month	Number of injuries	Sick days	Light duty days	Treatment hours	Transferred to hospital	Deaths
January	33	0	(1)	907	4	0
February	44	16	(1)	266	0	0
March	38	25	(1)	105	1	0
April	35	22	(1)	48	1	0
May	22	16	(1)	50	2	0
June	23	0	(1)	46	0	0
July	24	26	29	65.5	0	0
August	36	52	49	75	2	0
September	17	29	0	49	1	0
October	18	3	0	48	0	0
November	18	8	9	28	2	1
December	11	16	3	34.5	3	0
TOTALS	319	211	90	1,717	16	1
Annual rate per 1,000	254.2	168.1	129	1,368	12.7	0.79

[1] No record.

Beginning with the month of June 1939 statistics of all injuries were recorded as to the time of day of occurrence of injury (table 2), type of injury (table 3), cause of injury (table 4), and place of occurrence of injury (table 5).

TABLE 2.—*Time of occurrence*

0000 to 0400	6	0801 to 1200	39	1601 to 2000	18
0401 to 0800	6	1201 to 1600	32	2001 to 2400	5

TABLE 3.—*Type of injury*

Abrasian	5	Intracranial injury	1
Burn, chemical	2	Intraspinal injury	1
Burn, electrical	1	Sprain, joint	7
Burn (steam, hot water, etc.)	5	Strain, muscular	3
Contusion	11	Submersion, non-fatal	4
Dislocation	3	Sunburn	1
Fracture, compound	3	Synovitis, traumatic	1
Fracture, simple	15	Wound, gunshot	1
Friction burns	2	Wound, lacerated	48
Injuries, multiple, extreme	1	Wound, punctured	2

TABLE 4.—*Cause of injury*

Cutting and piercing instruments	14	Handling ammunition, etc	12
Firearms	1	Automobiles, motorcycles	12
Falls of person	14	Falling objects	11
Handling machinery	5	Handling of tools	5
Athletics and recreation	8	Handling of boats	7
Fighting, skylarking	1	Miscellaneous causes	23

TABLE 5.—*Place of occurrence of injury*

INJURIES OCCURRING ON BOARD

Month	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Number of injuries	18	17	12	15	12	8	82
Sick days	19	39	5	0	8	9	80
Light duty days	28	45	0	0	9	0	82
Treatment hours	51.5	56	38	45	25.5	10.5	226.5
Transferred to hospital	0	0	0	0	0	1	1
Died	0	0	0	0	0	0	0

INJURIES OCCURRING ASHORE

Number of injuries	4	5	4	1	4	3	21
Sick days	3	13	20	0	0	7	43
Light duty days	1	0	0	0	0	3	4
Treatment hours	2	10	11	3	8	24	56
Transferred to hospital	0	2	1	0	2	2	7
Died	0	0	0	0	1	0	1

INJURIES OCCURRING IN SMALL BOATS

Number of injuries	2	4	1	1	2	0	10
Sick days	4	0	4	3	0	0	11
Light duty days	0	4	0	0	0	0	4
Treatment hours	12	9	0	0	2.5	0	23.5
Transferred to hospital	0	0	0	0	0	0	0
Died	0	0	0	0	0	0	0

COMMENT

From a study of the statistics of injuries on board this vessel, it appears that the rate of admission for accidental injuries gradually decreased during the year 1939, especially during the period from June to December. It was in June that an intensive accident prevention campaign was started along the lines of inspection and checking of safety devices. Most important of all, perhaps, was the educational campaign to arouse a state of accident consciousness among the crew and also to promote mental alertness during working hours.

The National Safety Council states that 25 percent of all avoidable industrial accidents are due to improper lighting. This may mean insufficient light source or improper placing of lights. Bright lights are in themselves a hazard to eyesight when they produce a glare. Lighting in this vessel was frequently checked with a light meter.

"Flashed eyes," that is, injury to the retina by electric arc welding, actinic rays, etc., may be avoided by strictly enforcing use of safety appliances.

It was found difficult to get proper masks to protect men using spray paint guns. Several cases of eye inflammation resulted from paint spray.

The educational campaign for accident prevention was carried on by division officers. The medical officer suggests that a slide film projector be obtained and suitable picture slides showing accident prevention along naval lines be provided for lectures to the crew. The

ship's photographer could make photographs of shipboard accidents involving both matériel and personnel for instruction purposes.

Fatigue is recognized as a potent factor in the causation of accidental injuries. It is noted from these statistics that a majority of accidents occur during the forenoon working hours. This suggests that there may be some connection between "all night" liberty parties and the incidence of accidents aboard ship. Although no figures have been preserved, it is the opinion of the medical officer that fewer accidents occur during long cruises at sea. The problem of adequate rest is one to be handled by educational measures.

STOOL SURVEY [1]

U. S. S. "PENNSYLVANIA"

A stool survey (225 personnel) for intestinal protozoa and worms has been completed on all officers (2 exceptions), all mess attendants, officers' stewards, officers' cooks, and ship's cooks and bakers. A minimum of one stool was studied of the officers' group. In all others a minimum of 3 stools were studied. In all cases, a fresh preparation was examined in normal saline and several slides were stained by the iron haematoxylin method. Where indicated, water preparations were also examined 24 hours later. Of the 8 cases of *E. histolytica*, there were 3 who gave a history of gastro-intestinal symptoms; all were relieved after a course of carbarsone. Five cases showed large race and in 4 cases the small race *E. histolytica* was found. One case had both large and small present in the same specimen. None of those showing small race gave any history of gastro-intestinal symptoms.

TABLE 1.—*Intestinal protozoa*

PERSONNEL DISTRIBUTION OF INFECTIONS

Number of	Mess attendants			Other personnel		Admissions to sick list	Total cases
	Chamoran	Filipino	Negro	Cooks & bakers	Officers		
Men examined	8	22	23	28	94	50	225
Infections	14	6	22	16	30	35	123
Infections per man	1.75	0.27	0.96	0.57	0.32	0.70	0.55

DISTRIBUTION BY TYPE OF ORGANISM

Type of infection	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
G. lamblia	1	7.1	0	------	3	13.6	3	18.8	4	13.3	9	25.7	20	16.3
E. histolytica	1	7.1	0	------	3	13.6	0	------	3	10.0	1	2.9	8	6.5
E. coli	4	28.6	1	16.7	6	27.3	2	12.5	11	36.7	7	20.0	31	25.2
E. nana	2	14.3	4	66.7	6	27.3	9	56.3	10	33.3	15	42.9	46	37.4
I. butschlii	1	7.1	1	16.6	2	9.1	2	12.4	2	6.7	2	5.7	10	8.1
D. fragilis	0	------	0	------	1	4.5	0	------	0	------	1	2.8	2	1.6
T. trichiura	3	21.4	0	------	0	------	0	------	0	------	0	------	3	2.4
N. americanus	2	14.4	0	------	0	------	0	------	0	------	0	------	2	1.6
N. nana	0	------	0	------	1	4.6	0	------	0	------	0	------	1	0.9

[1] Abstracted from the annual sanitary report for the calendar year 1939.

It will be noted that 9 of the 50 sick bay cases showed *G. lamblia*. Of the 20 *G. lamblia* infections, there were 13 who gave a history of gastro-intestinal symptoms. Of these 13, there were 9 who stated their symptoms had been relieved following 3 days' administration of atabrine.

Results of stool survey are presented in table 1.

FOOD INFECTION

U. S. S. "MEMPHIS"

An attack of food infection occurred among members of the admiral's and warrant officers' messes while alongside dock at the Naval Air Station, San Diego, Calif., on December 15, 1939.

The suspected food was beef broth prepared from meat savings on December 14, kept in the galley overnight, heated and served for luncheon on December 15. The broth was warmed for serving, but definite fact of it being boiled was unlikely. No broth was available for examination.

There were 24 people on the two messes, 7 of whom (1 officer and 6 mess attendants) ate no broth and were not ill. Mild to severe symptoms were noted in 16 of the 17 individuals who ate the broth, the severest symptoms appearing in the person who ate two portions. Two of the cases were admitted to the sick list, 1 for 5 days and 1 for 6 days.

Symptoms developed within 2 to 5 hours after eating. Clinically, the cases were very similar in their course and symptoms. The first indication of illness was nausea, cold perspiration, pallor and collapse, marked diarrhea (bloody in several cases), spasmodic cramps in abdomen, and frontal headache. The symptoms were very sudden in nature and rather severe except in the mild cases. There was no intestinal distention, no ocular symptoms, and no skin eruptions. The symptoms of nausea and vomiting subsided in about 12 hours. Diarrhea and weakness were present for 24 hours.

Excerpts from instructions to galley crew are as follows:

The utmost care should be exercised to insure that food is not contaminated by careless handling after preparation and before serving. The hands must always be washed with soap and hot water before handling food.

Normally the time elapsed between the preparation and serving of food (except bakery products) should not exceed 4 hours. Meats which are to be cooked and then served cold should be cooled and stored in the chill room until just before they are served.

Hash shall not be served when it is necessary to prepare the ingredients the night before or even several hours before the hash is to be cooked and served.

STATISTICS

HEALTH OF THE NAVY

Report for the third quarter, 1939

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of July, August, and September, 1939.

ENTIRE NAVY

Year	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1934	510	69	580	21	120	105
1935	373	53	426	12	92	75
1936	336	67	404	24	88	49
1937	377	61	439	20	101	59
1938	332	57	389	7	72	81
1939	325	52	377	3	59	93

FORCES ASHORE

1934	637	92	730	31	181	64
1935	426	57	484	14	127	43
1936	416	69	486	29	120	38
1937	508	64	571	31	168	37
1938	342	52	394	10	92	39
1939	330	50	380	2	78	48

FORCES AFLOAT

1934	449	59	507	16	91	125
1935	343	51	395	11	72	92
1936	291	66	357	21	70	55
1937	302	60	362	13	63	72
1938	327	60	387	5	60	105
1939	322	54	376	4	49	117

Common infectious diseases of the respiratory type.—There were 2,205 admissions for these diseases reported from the entire Navy for the third quarter of the year 1939—1,207 from forces afloat, 909 from shore stations in the United States, and 89 from foreign shore stations. Catarrhal fever was responsible for 1,541 of the total admissions.

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Ships and shore stations reporting the largest number of cases were as follows:

Ship or station	July	August	September	Total
Naval training station, Norfolk, Va	31	59	68	158
Naval training station, Newport, R. I	43	40	59	142
Naval training station, San Diego, Calif	59	36	18	113
Marine Barracks, Quantico, Va	22	30	31	83
Naval air station, Pensacola, Fla	25	27	21	73
Marine Corps base, San Diego, Calif	15	25	23	63
Fleet air detachment, San Diego, Calif	13	29	12	54
Fourth Marines, Shanghai, China	19	16	19	54
U. S. S. *Enterprise*	21	17	17	55
U. S. S. *Augusta*	11	18	8	37
U. S. S. *Portland*	10	12	14	36
U. S. S. *Ranger*	8	14	10	32

Other infectious diseases.—A fireman, third class, 19 years of age, with 1 year and 7 months' service, was admitted to the sick list on the U. S. S. *Saratoga* with cerebrospinal fever on August 14 and transferred the same day to the naval hospital, San Diego, Calif. He was returned to duty on September 29.

A patient was admitted to the sick list on the U. S. S. *Robin* on June 11, diagnosis undetermined (intracranial injury) and transferred to the naval hospital, San Diego, Calif. Patient complained of headache, nausea, and photophobia, with diminished reflexes on left side, and some paralysis of neck and shoulder muscles was noted. Diagnosis changed to poliomyelitis, anterior, acute. The patient rapidly showed signs of ascending bulbar paralysis, with involvement of muscles of deglutition, and died on July 10.

A case of catarrhal fever, acute, admitted to the naval air station, San Diego, Calif., on August 27, was transferred to the naval hospital the following day where the diagnosis of poliomyelitis, anterior, acute, was established.

The U. S. S. *Enterprise* reported 22 cases of mumps for the quarter; the U. S. S. *Augusta*, 6; the U. S. S. *Texas*, 5; the U. S. S. *Ranger*, 2; and Fourth Marines, Shanghai, China, 4. Four shore stations and 1 ship reported 1 case each for the quarter.

Single cases of chickenpox were reported by the naval air station, Lakehurst, N. J., the United States Navy Company, World's Fair Detachment, New York, N. Y., and the U. S. S. *Brazos*.

A mild case of paratyphoid "A" fever, an aviation cadet, 24 years of age, with 11 months' service, was admitted to the naval hospital, Pensacola, Fla., from the naval air station on July 5 and returned to duty August 18. A course of typhoid vaccine had been completed in September 1938. A report from the naval hospital, Pensacola,

states that this cadet had not been outside of the Pensacola area since January 1, 1939, that there were no other cases of this disease reported in Pensacola at the time, and that no cases of the disease had appeared on the station for at least 2 years.

A private, U. S. M. C., 23 years of age, with 4 years and 4 months' service, was admitted to the sick list on August 9 from the Fourth Marines, Shanghai, China, with paratyphoid "B" fever, and returned to duty October 2. Two courses of straight typhoid vaccine had been completed in May 1935 and June 1938.

The naval training station, Norfolk, Va., reported two mild cases of paratyphoid "B" fever, one on July 31 and one on August 1. Courses of straight typhoid vaccine had been completed in each instance a few weeks prior to admission to the sick list. The questionnaires in these cases state that the probable source of infection was milk purchased in a civilian restaurant. The senior medical officer of the naval training station, Norfolk, Va., reported as follows:

> About the first of August, 1939, an epidemic of paratyphoid "B" fever broke out in Norfolk, Va. It was traced by Public Health authorities to the milk supply. It was discovered that on about July 10, 1939, there was a rain storm which flooded the city storehouse of the Rosedale Dairies. The lower cases of milk were submerged under water, and were later delivered without resterilizing. All cases of paratyphoid "B" were traced to this milk and there were no cases after the incubation period was up.

The sanitary report from the naval training station, Newport, R. I., for the month of July reports 9 cases of rheumatic fever resulting in 802 hospital sick days. The medical officer states that several of the patients gave histories of having become chilled while scrubbing clothes.

A slight epidemic of food poisoning developed aboard the U. S. S. *Simpson* on the night of September 18. Fifteen men were affected and were transferred to the naval dispensary, Guantanamo Bay, Cuba. Investigation of the supper menu and of the articles of food eaten by the men who became ill indicates that cold sliced ham was the food which caused the outbreak. None of the ham, however, was available for bacteriological examination. Other food examined gave negative results on culture for food-poisoning organisms. The ham was cooked and served in part at the noon meal on the previous day. The left-over ham was kept unsliced in the refrigerator until prepared for supper on September 18. It seems evident that the ham was not heated sufficiently in the cooking process to destroy the infection present in the meat and that subsequent storage was not at low enough temperature to prevent growth of the infecting organisms.

There were 91 cases of mild gastro-enteritis, characterized by nausea, vomiting, abdominal cramps, and mild diarrhea, on board the U. S. S. *Mississippi* between August 22 and August 28, 1939. Only one case

was admitted to the sick list. Temperature, pulse, and respiration were normal, and white and differential blood counts were within normal limits. No toxic symptoms were manifested. The cases were evenly distributed through members of the general mess, chief petty officers' mess, warrant officers' mess, junior officers' mess, and wardroom mess. The possibility of food poisoning was considered but no evidence of it could be found. As the cases were evenly distributed throughout the personnel of the ship, suspicion was thrown on the fresh water. Distillation of water in accordance with Bureau of Engineering instructions had been and was being scrupulously carried out. A sample of fresh water submitted for culture was negative for bacteria of the colon group.

Report for the fourth quarter, 1939

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of October, November, and December, 1939.

ENTIRE NAVY

Year	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1934	554	67	622	26	171	114
1935	409	61	470	8	126	73
1936	474	76	550	15	118	96
1937	472	39	511	11	118	93
1938	325	57	382	4	76	81
1939	363	54	417	3	84	98
FORCES ASHORE						
1934	610	77	687	35	192	68
1935	427	64	491	9	151	39
1936	459	98	557	13	121	46
1937	535	49	583	11	169	55
1938	330	55	386	7	104	40
1939	368	54	422	4	117	52
FORCES AFLOAT						
1934	432	61	586	21	160	139
1935	344	59	457	8	111	93
1936	525	63	546	15	115	125
1937	398	34	469	10	89	116
1938	321	59	380	2	59	103
1939	360	54	414	3	64	126

Common infectious diseases of the respiratory type.—During the last quarter of 1939 there were 1,655 cases of these diseases reported from

shore stations in the United States, 68 from shore stations outside the continental limits of the United States, and 1,568 from the forces afloat.

Ships and shore stations reporting the greatest number of admissions for the quarter were as follows:

Ship or station	October	November	December	Total
Naval training station, Norfolk, Va	100	52	280	432
Naval training station, Newport, R. I	83	91	121	295
Marine Barracks, Quantico, Va	46	20	61	127
Marine Corps base, San Diego, Calif	32	30	57	119
U. S. Naval Academy (midshipmen)	24	21	35	80
Naval training station, Great Lakes, Ill	13	6	43	62
Naval training station, San Diego, Calif	9	6	35	50
U. S. S. *Arizona*	4	12	29	45
Receiving station, Philadelphia, Pa	5	8	28	45
U. S. S. *Texas*	4	6	29	39
Fourth Marines, Shanghai, China	13	6	20	39
Naval dispensary, Washington, D. C	14	2	22	38
U. S. S. *Maryland*	13	9	16	38
Naval air station, Norfolk, Va	9	11	15	35
Fleet air detachment, Naval Air Station, San Diego, Calif	14	12	9	35
U. S. S. *Enterprise*	15	4	14	33
U. S. S. *Mississippi*	8	2	21	31
U. S. S. *Portland*	13	7	10	30

Other infectious diseases.—Chickenpox was reported during the quarter as follows: One case from the navy yard, Mare Island, Calif., in October; 1 case each from the U. S. S. *Enterprise* and the Fourth Marines, Shanghai, China, in November; and 1 case each from the U. S. S. *Haraden*, the Naval dispensary, Washington, D. C., and the Naval Training Station, San Diego, Calif., in December.

Two cases of poliomyelitis, anterior, acute, were reported during the quarter, 1 in October from the Receiving Ship, San Diego, Calif., and 1 in November from the U. S. S. *California*.

Three cases of diphtheria were admitted to the sick list on board the U. S. S. *Ellet* and 1 case of scarlet fever on board the U. S. S. *Milwaukee* in December.

The monthly sanitary report for November from the Naval Station, Guam, reported the occurrence of several cases of amebic dysentery among service personnel. Investigation to trace the source of infection was unsuccessful. Most of the patients had eaten in many places during the incubation period of the disease and at one time or another had eaten native vegetables which might have been contaminated. Warnings to the population and to ships against the use of raw vegetables and the usual warnings for the necessity of boiling water were issued. Polluted swimming beaches were closed.

A corporal, United States Marine Corps, was transferred from the Marine Barracks, Quantico, Va., to the Naval Hospital, Washington,

D. C., where the diagnosis of Rocky Mountain spotted fever was established on January 1, 1940. He was returned to duty after 24 days on the sick list.

One case of tularemia was admitted to the sick list on board the U. S. S. *New Mexico* in December and discharged to duty after 19 sick days.

A hospital apprentice, second class, 24 years of age, with 1 year and 1 month's service, on duty at the Naval Hospital, Mare Island, Calif., was admitted to the sick list with cerebrospinal fever on October 24, 1939. He was returned to duty on January 22, 1940.

An apprentice seaman, 18 years of age, with 1 month's service was admitted to the sick list at the Naval Training Station, Great Lakes, with cerebrospinal fever on October 20 and transferred the same day to the naval hospital. He remained on the sick list until December 29.

A moderately severe case of paratyphoid "A" fever (private, U. S. Marine Corps, 21 years of age, with 2 years, 4 months' service) was admitted to hospital from the Fourth Marines, Shanghai, China, on November 13, 1939. Diagnosis was based upon the following clinical evidence: General aches, headache, intermittent fever, splenomegalia, and macular roseola rash. Probable place of infection was a local restaurant in Shanghai, China. Two complete courses of typhoid prophylaxis had been completed. He returned to duty on January 1, 1940.

The regimental surgeon, Fourth Marines, Shanghai, China, reported 2 cases of typhus fever (Brill's type, endemic) and stated that the regimental billets were virtually free of rats and that the reservoir of infection was believed ashore (rickshas and taxicabs).

The commanding officer, naval hospital, Parris Island, S. C., reported 2 cases of typhus fever (endemic). A sergeant, United States Marine Corps, on active duty at and living in Yemassee, S. C., was admitted to the sick list on October 20. The second case, a private, United States Marine Corps, was admitted on November 4. The patient had been living in Beaufort, S. C., and the infection was undoubtedly contracted prior to his entry into the service.

The report states that the disease is endemic in the coastal regions of the South Atlantic States and that, at the request of various doctors in Beaufort, 4 cases had been examined and found positive by the laboratory of the naval hospital, Parris Island, S. C.

Venereal diseases.—The following table of statistical data indicates the frequency of occurrence of venereal diseases during the year 1939 (reported in monthly reports of communicable diseases):

VENEREAL DISEASE

Admission rates per 1,000—Calendar year 1939

BATTLE AND SCOUTING FORCES

Ship	First quarter	Second quarter	Third quarter	Fourth quarter
Arizona	76. 41	56. 29	47. 54	48. 99
Arkansas	115. 38	48. 13	94. 21	127. 78
Astoria	132. 56	281. 21	113. 56	30. 40
Boise	146. 84	87. 52	62. 18	120. 89
Brooklyn	42. 38	109. 67	86. 71	88. 19
California	55. 63	95. 79	75. 14	73. 21
Chester	94. 79	18. 55	43. 61	55. 21
Chicago	35. 53	128. 51	62. 75	63. 24
Cincinnati	90. 53	240. 34	86. 96	238. 66
Colorado	76. 43	158. 39	102. 65	119. 76
Concord	189. 75	62. 07	114. 99	182. 61
Detroit	83. 62	26. 85	49. 56	49. 73
Enterprise	83. 78	74. 27	101. 51	28. 33
Helena	----------	----------	38. 46	88. 08
Honolulu	65. 24	58. 90	54. 19	124. 38
Houston	117. 28	109. 32	158. 23	79. 77
Idaho	28. 62	74. 89	73. 09	89. 53
Indianapolis	80. 38	108. 88	95. 12	81. 76
Langley	38. 10	126. 32	110. 09	612. 24
Lexington	40. 76	70. 02	94. 89	114. 37
Louisville	134. 97	205. 30	180. 06	147. 67
Maryland	42. 46	132. 82	88. 89	56. 11
Memphis	86. 96	115. 16	89. 55	62. 62
Milwaukee	65. 04	122. 45	109. 94	98. 00
Minneapolis	53. 55	123. 49	144. 65	49. 59
Mississippi	51. 61	83. 82	77. 83	70. 37
Nashville	124. 35	305. 38	169. 29	110. 53
Nevada	118. 96	59. 02	41. 99	55. 92
New Mexico	77. 44	113. 37	88. 52	136. 32
New Orleans	81. 16	83. 96	86. 55	30. 40
New York	81. 31	121. 47	140. 06	151. 97
Northampton	54. 87	85. 91	50. 49	51. 87
Oklahoma	76. 29	97. 04	67. 40	66. 60
Omaha	219. 47	159. 42	186. 44	201. 61
Pennsylvania	77. 53	55. 35	39. 01	61. 30
Pensacola	82. 72	123. 78	61. 54	94. 81
Philadelphia	111. 79	87. 74	133. 14	159. 32
Phoenix	126. 15	42. 33	21. 62	84. 51
Portland	64. 52	148. 15	91. 05	75. 35
Quincy	123. 53	171. 60	118. 48	123. 58
Raleigh	121. 00	71. 30	64. 52	71. 11
Ranger	53. 66	155. 93	196. 67	144. 39
Richmond	22. 10	73. 53	54. 37	82. 86
Salt Lake City	48. 19	134. 97	71. 86	71. 75
San Francisco	85. 91	193. 64	170. 21	220. 73
Saratoga	66. 14	70. 13	86. 35	114. 96
Savannah	69. 24	162. 16	123. 66	104. 35
St. Louis	----------	68. 45	110. 28	134. 89
Tennessee	110. 20	120. 93	98. 28	95. 97
Texas	191. 84	115. 07	76. 38	153. 85
Trenton	79. 84	237. 92	328. 86	537. 01
Tuscaloosa	97. 56	94. 12	190. 20	173. 65
Utah	63. 35	110. 24	118. 39	136. 25
Vincennes	95. 24	107. 62	78. 55	160. 26
West Virginia	51. 54	57. 32	118. 36	81. 44
Wichita	132. 94	198. 89	183. 33	185. 24
Wright	209. 40	146. 04	72. 73	53. 48
Wyoming	143. 82	205. 84	120. 48	158. 04
Yorktown	80. 33	88. 59	98. 44	126. 65

ASIATIC FLEET

Ship	First quarter	Second quarter	Third quarter	Fourth quarter
Alden	366. 41	61. 54	276. 92	480. 00
Asheville	510. 64	568. 53	652. 17	828. 40
Augusta	369. 43	596. 82	456. 43	510. 55
Barker	430. 77	366. 41	372. 09	566. 93
Bittern	0. 00	400. 00	66. 67	131. 15
Black Hawk	560. 12	487. 22	588. 59	460. 80
Bulmer	94. 49	283. 46	327. 87	300. 00
Canopus	197. 22	331. 18	345. 86	318. 77
Edsall	212. 12	427. 48	388. 06	338. 46
J. D. Edwards	274. 81	465. 12	180. 45	483. 87
Finch	135. 59	344. 83	562. 50	312. 50
J. D. Ford	539. 68	384. 00	558. 14	645. 16
Guam	482. 76	590. 16	271. 19	771. 93
Heron	271. 19	375. 00	342. 86	193. 55
Isabel	254. 55	525. 25	449. 44	311. 69
Paul Jones	147. 06	120. 30	180. 45	346. 46
Luzon	260. 87	416. 67	360. 00	344. 09
Marblehead	316. 34	338. 86	640. 93	543. 12
Mindanao	326. 53	734. 69	658. 82	523. 81
Napa			0. 00	230. 77
Oahu	369. 23	184. 62	615. 38	61. 54
Parrott	366. 41	369. 23	220. 47	527. 13
Peary	360. 90	300. 75	651. 16	349. 21
Pecos	432. 43	580. 64	455. 45	620. 32
Pigeon	405. 80	347. 83	597. 01	400. 00
Pillsbury	149. 25	151. 52	666. 67	393. 44
Pope	330. 58	450. 12	549. 62	566. 93
Sacramento	471. 79	260. 87	71. 01	687. 38
Stewart	338. 46	421. 05	519. 08	625. 00
Tulsa	439. 56	625. 00	786. 89	1, 000. 00
Tutuila	312. 50	131. 15	344. 83	758. 62
Whipple	231. 88	417. 91	606. 06	229. 51

MORBIDITY

Summary for the quarter ending December 31, 1939

Average strength	Forces afloat 96,285		Forces ashore 58,933		Entire Navy 155,218	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	9, 964	413. 94	6, 210	421. 50	16, 174	416. 81
Diseases only	8, 671	360. 22	5, 421	367. 94	14, 092	363. 15
Injuries and poisonings	1, 293	53. 72	789	53. 55	2, 082	53. 65
Communicable diseases transmissible by oral and nasal discharges (Class VIII):						
(A)	63	2. 62	52	3. 53	115	2. 96
(B)	1, 544	64. 14	1, 726	117. 15	3, 270	84. 27
Venereal diseases	3, 025	125. 67	769	52. 19	3, 794	97. 77

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for the quarter ending December 31, 1939

The following statistics were taken from sanitary reports submitted by naval training stations:

October, November, and December, 1939	Naval training station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period	3, 788	2, 843	2, 623	2, 750
Recruits appearing before Board of Medical Survey	53	0	29	(1)
Recruits recommended for discharge from the service	43	0	29	(1)
Recruits discharged by reason of medical survey	29	0	25	(1)
Recruits held over pending further observation	31	0	(1)	(1)
Recruits transferred to the hospital for treatment, operation or further observation for conditions existing prior to enlistment	0	69	(1)	22

[1] Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Absence, acquired, teeth	13	Constitutional psychopathic state, sexual psychopathy	1
Absence, congenital, nasal septum	3	Curvature, spine	1
Acne, vulgaris	1	Deafness, bilateral	6
Adhesions, abdominal	1	Deafness, unilateral	2
Adhesions, inferior turbinates, bilateral	1	Defective physical development	2
Amnesia	1	Deformity, acquired	9
Amputation, traumatic, right index finger	1	Deformity, congenital	1
Ankylosis, right elbow	1	Dementia praecox	4
Arthritis, chronic	2	Deviation, nasal septum	3
Astigmatism, compound, myopic	1	Dislocation, articular cartilage, left knee	1
Calculus, kidney, bilateral	1	Dislocation, chronic, recurrent, left shoulder	1
Cardiac arrhythmia (pulsus trigeminus)	1	Dyspituitarism	1
Caries, teeth	9	Eczema	1
Cicatrix, skin	2	Effort syndrome	2
Color blindness	4	Encephalitis, chronic	1
Constitutional psychopathic inferiority, without psychosis	6	Enuresis	16
Constitutional psychopathic state, inadequate personality	4	Epilepsy	9
		Epiphysitis, left anterior	1
		Exuberant callus, left ulna	1
		Flat foot	14

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Gonococcus infection, prostate	1	Psychoneurosis, hysteria	4
Hammertoe	1	Psychoneurosis, neurasthenia	1
Headache	1	Psychoneurosis, psychasthenia	1
Hernia, inguinal, direct	2	Pterygium	1
Hernia, inguinal, indirect	8	Pyelitis, chronic	1
Hypertension, arterial	8	Sinusitis, maxillary	1
Hypochondriasis	1	Somnambulism	1
Loss of substance of bone	1	Spur, nasal septum	1
Malocclusion, teeth	8	Strabismus	1
Metatarsalgia	1	Syncope	2
Migraine	1	Syphilis	1
Myopia	15	Tachycardia	1
Nephritis, chronic	11	Tenosynovitis, chronic	1
Neuritis, optic	1	Trachoma	1
Nystagmus, marked	1	Tuberculosis, pulmonary, chronic, active (far advanced with cavitation)	1
Osteomyelitis	1	Ulcer, duodenum	1
Otitis interna	1	Ulcer, stomach	1
Otitis, media, chronic	15	Union of fracture, faulty	1
Paralysis, external rectus, left eye	1	Valvular heart disease, aortic and mitral	1
Perforated nasal septum	1	Valvular heart disease, mitral insufficiency	4
Pes cavus	2	Varicose veins	1
Polypus, rectal	1		
Psychoneurosis, anxiety neurosis	1		

DEATHS

During the fourth quarter ending December 31, 1939

Cause		Navy			Marine Corps		Nurse Corps	Total
Principal	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		10,484	2,301	119,532	1,422	21,041	438	155,218
Diseases								
Abscess, brain	None			1				1
Abscess, liver	None			1				1
Angina, Vincent's	Septicemia			1				1
Appendicitis, acute	Obstruction, intestinal, from paralytic cause.	1						1
Appendicitis, acute	Peritonitis, general, acute.			1		1		2
Calculus, kidney	Septicemia (staphylococcus).			1				1
Carcinoma, gall bladder, squamous cell.	None				1			1
Carcinoma, lung, bronchogenic, undifferentiated.	None	1						1
Carcinoma, lung, squamous cell.	Pleurisy, suppurative	1						1
Cellulitis, foot	Septicemia, (staphylococcus aureus).				1			1
Coronary heart disease, arteriosclerotic.	None	1		1				2
Embolism, pulmonary	Appendicitis, acute			1				1
Endocarditis, acute	Infarction, lungs and kidneys.					1		1
Epilepsy	None					1		1
Granulocytopenia, malignant.	None			1				1
Lymphosarcoma, retroperitoneal.	None	1						1
Melanoma, generalized	None			1				1
Myocarditis, chronic	Arteriosclerosis, general			1				1
Pneumonia, broncho	Cholelithiasis and atelectasis.			1				1
Pneumonia, broncho	Nephritis, acute			3				3
Syphilis, cardiovascular	Thrombosis, coronary artery.			1				1
Thrombosis, coronary artery.	None	2						2
Thrombosis, coronary artery.	Appendicitis, acute			1				1
Thrombosis, coronary artery.	Arterial hypertension	1						1
Tuberculosis, meningeal	None			1				1
Tuberculosis, pulmonary, chronic, active.	None			2		1		3
Ulcer, stomach, perforated	Hemorrhage, stomach			1				1
Total for diseases		8		20	2	4		34
Injuries and poisonings								
Crush, chest	Embolism, pulmonary	1						1
Drowning	None			6				6
Drowning	Psychosis, unclassified			1				1
Fracture, compound, skull	None			2		1		3
Fracture, simple, skull	Intracranial injury			1				1
Fracture, simple, cervical vertebrae.	Compression, cervical cord.			1				1
Intracranial injury	Alcoholism, acute			1				1
Intracranial injury	Hemorrhage, traumatic, subdural.			1				1
Injuries, multiple, extreme	None	4		15		1		20
Wound, gunshot, chest	None					1		1
Wound, gunshot, head	None	1		3				4
Wound, incised, neck	Psychosis, unclassified			1				1
Poisoning, acute, methyl alcohol.	None			1				1
Total for injuries and poisonings.		6		33		3		42
Grand total		14		53	2	7		76
Annual death rate per 1,000:								
All causes		5.34		1.77	5.63	1.33		1.96
Disease only		3.05		.67	5.63	.76		.87
Drowning				.23				.18
Poisonings				.03				.03
Other injuries		2.29		.84		.57		.88

VOLUME XXXVIII OCTOBER 1940 NUMBER 4

United States Naval Medical Bulletin

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

Issued Quarterly by the Bureau of Medicine and Surgery

Navy Department

Washington, D. C.

VOL. XXXVIII OCTOBER 1940 NO. 4

UNITED STATES NAVAL MEDICAL BULLETIN

FOR THE INFORMATION OF
THE MEDICAL DEPARTMENT OF THE NAVY

DIVISION OF PUBLICATIONS
THE BUREAU OF MEDICINE AND SURGERY

THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY

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

Compiled and published under the authority of Naval Appropriation Act for fiscal year 1941, approved June 11, 1940

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1940

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

NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary.

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Volume XII, 1918, Nos. 1 and 3.
Volume XXIV, 1926, Nos. 1 and 4.
Volume XXV, 1927, No. 1.
Volume XXVII, 1929, Nos. 3 and 4.
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SUBSCRIPTION PRICE OF THE BULLETIN

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

Yearly subscription, beginning July 1, $1; for foreign subscriptions add 35 cents for postage.

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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 Medical Department personnel, and reports from various sources, notes, and comments on topics of professional 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 professional interest.

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

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

ROSS T. MCINTIRE,
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.

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EBEN E. SMITH, *Editor*,
Commander, Medical Corps, United States Navy.

U. S. NAVAL MEDICAL BULLETIN

VOL. XXXVIII OCTOBER 1940 No. 4

SPECIAL ARTICLES

THE DIAGNOSIS AND TREATMENT OF HEAD INJURIES

By Lieutenant Commander Gerald W. Smith, Medical Corps, United States Navy *

The extent of functional impairment and final pathologic change of the brain caused by craniocerebral injury depends not only on the nature and extent of the lesion but also upon the kind of treatment instituted.

During the past 15 years the conservative treatment of head trauma has rapidly gained in use and with this change from the more radical method of treatment, the mortality rate has decreased even though the incidence has greatly increased. A generation ago radical subtemporal decompression was usually resorted to for relief of increased intracranial pressure. It was the accepted method of treatment for basal fractures of the skull.[1] This procedure only superimposed surgical shock on traumatic shock and resulted in an inexcusable high mortality rate.

In most clinics now, operative intervention is not found indicated in more than 5 to 10 percent of head injury cases and then only when complicated by subdural or epidural hemorrhage or when depressed skull fractures are encountered. Surgical decompression, which provides for greater space in the cranial cavity for the tightly compressed brain, is rarely resorted to now that the results of physiological decompression are so satisfactory.

The purpose of this paper is to discuss the various types of craniocerebral injury and the method of treatment used in the neurosurgical clinic of Dr. Temple Fay at the Temple University Hospital, Philadelphia. The type of treatment for head injuries here discussed has been used with good results in this clinic since 1929 in over 1,000 cases. I have had the opportunity to examine many of these patients in the neurosurgical out-patient clinic and to aid in the management and treatment of 165 acute head trauma cases admitted to the neurosurgical service during the past year.

It is difficult to compare head injury mortality statistics in various clinics because the same index or measuring rod is not used. One

* On assignment from the U. S. Navy to the Department of Neurology and Neurosurgery, Temple University School of Medicine, Clinic of Dr. Temple Fay, Temple University Hospital, Philadelphia, Pa.

[1] Da Costa, J. C.: Modern Surgery, W. B. Saunders Co., Philadelphia, 1923.

clinic includes in its statistics patients with head injury who expire within a few minutes to 3 hours after admission, while another clinic will not include this group because death of the patient probably would have ensued without treatment or with any method of treatment. The kind of case that may be admitted to the clinic also varies. If patients are included who have been only momentarily stunned from head trauma, the mortality rate will be low.

In this clinic patients receiving a slight cerebral concussion are seldom admitted to the neurosurgical service unless definite focal neurological signs or changes in their spinal fluid are manifested. They remain in the accident dispensary 6 to 8 hours and are observed closely for any development of neurological signs of significance. If they are free from any residual effects of the injury, they are sent home and told to return immediately if they become drowsy or vomit and to report to the neurosurgical outpatient clinic within 3 days for reexamination.

There are numerous classifications for craniocerebral injury but none is completely satisfactory. However, for purposes of prognosis the following simple classification, compiled by Dr. Michael Scott, with the mortality rate for each type of case experienced in the neurosurgical clinic, Temple University Hospital, is found useful.

TABLE 1.—*Classification of craniocerebral injuries with mortality rates*

Classification	Number of cases	Died before 3 hours		Died after 3 hours		Total deaths	
		Number	Percent	Number	Percent	Number	Percent
Concussion	342	3	0. 8	16	4. 6	19	5. 4
Skull fracture with clear spinal fluid	53	4	7. 5	3	5. 7	7	13. 2
Blood in spinal fluid, concussion but no fracture	115	6	5. 2	24	20. 8	30	26
Skull fracture, blood in spinal fluid, concussion	145	14	9. 6	47	32. 4	61	42
Total	655	27	4. 1	90	13. 7	117	17. 8

Of this group of 655 cases only 50 cases or 7.6 percent required operation as a means of relief from intracranial pressure following trauma. These operations consisted of explorations for complicating epidural or subdural hemorrhage and were accomplished only when definite neurological or x-ray evidence was present that an expanding intracranial lesion existed.

The presence or absence of skull fracture complicating head trauma is not usually of much significance in regards to management and treatment unless there is a massive depressed fracture. It is important, however, from a medicolegal point of view. Unfortunately, x-ray evidence of a small linear skull fracture is often of more significance

to the members of the jury than a history of cerebral contusion, laceration with severe shock and bloody spinal fluid, but without fracture of the skull. The location and extent of the skull fracture is important in regard to prognosis. The highest mortality follows fractures of the base of the skull, the posterior fossa being the most vulnerable of the 3 cranial fossa because of the adjacency of the vital centers. Basal fractures are more apt to be compound; therefore there is more danger of meningitis. The continuity of the pharynx, roof of the nares, orbit or ears with the base of the skull permits access of the air to the seat of the fracture and allows blood and cerebrospinal fluid to flow externally. Sixty percent of the fractures of the vault involve the base. The danger of fracture of the skull lies in the possibility of brain injury, hemorrhage or secondary infection.

CEREBRAL CONCUSSION

Gross [2] states, and it has been verified many times, that "There is no lesion of the head so trifling, on the one hand, as not to endanger life, or so severe on the other, that it may not be followed by recovery." What may appear at first to be just a slight concussion may prove to be complicated by a laceration of the middle meningeal artery with slow development of an epidural hemorrhage. Unless every patient who has received a head injury, resulting in even a transient period of unconsciousness, is kept under observation from 6 to 12 hours, such complications will be unobserved and death of the patient may occur from cerebral compression.

Concussion may be produced by a blow to the head or indirectly by a fall on the buttock which momentarily displaces the brain causing unconsciousness either profound or momentary.[2] What happens during a concussion is not clearly understood. There is a momentary interruption of the function of the nerve cells; beyond this the explanation is only theoretical. It has been suggested that concussion is produced by a jarring of the neurons, by separation of the synapses, by temporary increase in coagulation of the cell plasma, by the advance and recession of waves of cerebral fluid or by vasomotor changes. Strauss and Savitsky [3] define concussion "As a series of events resulting from a blow to the head severe enough to cause disruption of intracranial equilibrium." Numerous cases of death from concussion have been reported and autopsy failed to reveal any organic lesion and the spinal fluid was free from red blood cells. It is improbable that laceration of the brain or rupture of the vessels occurs in slight concussion for recovery of the patient is too rapid and too complete.

Following a blow to the head producing concussion the patient may not necessarily fall. He may "see stars" and become momentarily

[2] Gross, S. D.: System of Surgery, Vol. 2. Henry C. Lea, Philadelphia, 1872.

[3] Strauss, I. and Savitsky, N.: Head injury; neurologic and psychiatric aspects. Arch. Neurol. and Psychiat. 31: 853 May 1934.

dazed and confused. I have seen football players continue to play for several minutes after such an experience. Subsequently the patient with concussion may become weak, dizzy, pale, nauseated, and may vomit. In several hours he may show no residual effects from his experience. In a more severe case the patient will fall in a state of complete relaxation. Frequently the fall caused by the blow produces extensive craniocerebral damage. This has been observed in the prize-fight ring. A light blow received to the head but forceful enough to cause the fighter to be momentarily dazed, relaxed, and off balance may cause him to fall backward and strike his head with sufficient force to produce skull fracture and subarachnoid bleeding.

CEREBRAL CONTUSION

Contusion or bruising of the brain occurs from trauma to the head more forceful than that found producing cerebral concussion. With contusion there is rupturing of some of the small vessels of the brain with more severe damage to the cerebral structures. The period of unconsciousness is more prolonged and recovery of the patient is not so rapid. Symptoms indicating cortical irritation are manifested such as irritability, restlessness, fretfulness, and the patient may remain semistuporous for hours. Shock is more likely to be associated with cerebral contusion than with uncomplicated concussion. Skull fracture may or may not occur. Hemorrhage into the subarachnoid space is frequently found when spinal tap is performed. When hemorrhage into the subarachnoid space occurs meningeal symptoms develop. Headache is severe and usually localized in the occipital region and back of neck. Nuchal rigidity may be pronounced and the slightest turning of the head produces excruciating pain. The temperature is usually elevated from 100° to 101° F. due to the meningeal irritation and a leukocytosis occurs. Unilateral focal neurological signs may not appear if no laceration of the cortex or localized edema complicates the contusion. A generalized increase in deep reflexes, bilateral positive Babinski and Hoffmann signs may appear. A retrograde amnesia is not uncommonly found.

Weed [4] has shown that blood in the subarachnoid space produces violent reaction of the meninges and causes a disturbance in the elimination of the spinal fluid. Hence it is imperative that the spinal fluid be withdrawn at appropriate intervals by lumbar tap. Withdrawal of the bloody spinal fluid favors clotting of the vessels which may be responsible for the bleeding in the subarachnoid system.

The following case report illustrates a typical case of cerebral contusion complicated by skull fracture and subarachnoid hemorrhage:

[4] Weed, L. H.: Forces concerned in absorption of cerebrospinal fluid. Am. J. Physiol. 114: 40, Dec. 1935.

Case Report

M. R. a lady aged 33 years stepped from a street car, February 4, 1940, and was struck and hurled 30 feet by a passing automobile. She was brought immediately to Temple University accident dispensary unconscious and in state of shock. A 2-inch scalp laceration was over her right temporal region. She was treated for shock and the scalp hemorrhage was controlled by packing. Subsequent to the shock reaction spinal tap revealed initial pressure of 14 mm. hg. and bloody spinal fluid. She complained of severe occipital headache and pain in her neck.

Physical examination showed numerous contusions and abrasions of the arms and legs, laceration of the scalp, but no evidence of internal injuries. Neurological examination elicited extreme nuchal rigidity, unequal and irregular pupils from a previous bilateral iridectomy. The pupils reacted normally to light and accommodation. Left extremities showed some weakness and increase in deep reflexes. An equivocal left Babinski sign was present. The patient was transferred to the neurosurgical service.

Clinical Course.—She continued to be drowsy and semistuporous. Temperature became elevated to 101° F., pulse rate ranged between 68 and 90 per minute. Daily spinal drainages were done; 20 to 40 cc. were removed at each drainage. Following drainages she became mentally clearer and more alert, pulse increased in rate, and the pulse pressure fell from 60 to 40. Repeated neurological examinations were made to determine any increase or decrease in focal signs. After the third day no focal signs were present and the headache and nuchal rigidity diminished in severity. A vertical fracture 6 cm. in length was visualized in the left occipital bone by x-ray examination. During the period of spinal drainages she received 32 ounces of fluid per day and a dry diet. On the fifth day after admission the spinal fluid was wine colored, on the eighth day it was xanthochromic, and on the eleventh day it was clear. Spinal drainage was discontinued on the eighth day after admission and the fluid intake was reduced to 20 ounces per day and gradually elevated to 32 ounces by the fifteenth day when she was discharged with instructions to continue at that fluid level, remain on a dry diet, and report to the neurosurgical outpatient clinic in 3 weeks. At the end of the third month no fluid limitation was considered necessary. She had no complaints or residual symptoms from her experience.

Comment.—This case illustrates the value of daily spinal drainage when subarachnoid hemorrhage is present. Ample fluid (32 ounces in 24 hours) must be given during the period blood is in the spinal fluid for sufficient spinal fluid formation to enable satisfactory and frequent drainage. Prior to spinal drainage the pulse became slower, the pulse pressure increased, and the patient became drowsier, indicating that intracranial pressure had increased with resultant impairment of cerebral circulation.

On the fourth day after admission x-ray examination was made. It is important that the patient's general condition be sufficiently good before transfer to the x-ray department where further moving must be made. I have seen patients in other clinics moved from the accident dispensary to a stretcher and taken to the x-ray department in another part of the hospital for skull x-ray almost before they had recovered from the initial period of shock. Nothing is gained by an early x-ray examination and much can be lost. The fracture line in the occipital bone demonstrated in this case 4 days after the patient's admission to the hospital was interesting information but it did not alter the subsequent treatment or prognosis. Had this patient been subjected to the changes of position usually associated with x-ray examination early after her admission she might not have recovered.

It is my opinion that when head injuries occur on board ship it is important to keep the patient in the sick bay under constant observation and care until the medical officer is certain that no harmful effects can occur by having the patient moved from the ship to a hospital ship or hospital ashore. There is no emergency here that a medical officer aboard ship cannot satisfactorily handle. The treatment of shock can be done in one place as well as in another. Subsequent to the period of shock, treatment for head injury can be followed, if need be, for several days aboard ship where the accident occurred until the patient's condition warrants transfer. Should the patient expire during the first 3 hours even though all possible treatment had been received, it is probable that death would have occurred even sooner had attempts been made to effect his transfer. The patient should receive constant attention during the critical stage. The question of transfer should be in the background and should be considered only when the physical condition of the patient is such that transfer cannot jeopardize his chance for recovery. It is realized that often the commanding officer feels as most laymen, that after any serious accident the sooner the injured person is removed to a hospital the better. This is not true in cases of head injury where rest, quiet, and constant attention are imperative, for at times only slight superimposed trauma as produced by moving may bring about the death of the patient. I have seen patients with head injury kept in bed and treated in a hospital accident dispensary 8 to 10 hours after injury because moving the patient to a stretcher, taking him upstairs, and again lifting him into bed were considered dangerous. If moving a head injury case from an accident ward to another part of the same building is sometimes considered contraindicated, it is easy to visualize what may happen by transferring prematurely a severe head trauma case from one ship to another.

EPIDURAL HEMORRHAGE

Epidural hemorrhage may occur from even trivial blows over the squamous portion of the temporal bone. This portion of the skull is thin and brittle and overlies the middle meningeal artery and its branches which lie as if embossed on the outer surface of the dura. A fracture of this portion of the skull will frequently lacerate the artery causing bleeding to occur between the dura and skull. As the bleeding progresses and overcomes the intracranial tension, the dura is stripped from the skull over its lateral convexity and may produce additional tearing of the middle meningeal artery. In the very young and the old, stripping of the dura from the skull is more difficult and hemorrhage is therefore not usually as large as found in individuals of middle age. The roughened surface of the exposed skull favors clotting and therefore the accumulation of blood is slow.

Most clinicians are familiar with the typical clinical picture of epidural hemorrhage which is characterized by an initial unconscious period, followed by a lucid interval of several minutes to hours, only to be superseded by gradual progressing mental dullness, stupor, and finally unconsciousness. The hemorrhage enlarges and causes compression of the brain with resultant anoxemia of the cerebral cells. Unfortunately there is often such an overlapping of these three clinical phases that the lucid interval may be unobserved. Too often a history is not obtainable and by the time the posttraumatic patient has arrived at the accident dispensary the patient may be in deep coma. Unless the clinician is experienced in recognizing the clinical signs of acute cerebral compression due to an expanding hemorrhagic lesion, diagnosis of the condition is not made and death of the patient ensues. Careful observation of the focal and general neurological signs manifested should elicit evidence of expanding intracranial lesion. Whether the lesion be an epidural lesion or a subdural lesion is not especially important if the increase in intracranial pressure cannot be controlled by dehydration methods, since operative interference should be undertaken for either condition. Operative intervention for epidural hemorrhage should be done as soon as the diagnosis has been made after the period of shock is over. On the other hand it is found better to delay operation in cases of subdural hemorrhage for 5 to 10 days if it is found possible to control the intracranial pressure by conservative measures.

Kennedy and Wortis [5] endeavor to differentiate epidural and subdural lesions clinically. They find in their series of 72 cases of acute subdural hematoma and 17 cases of epidural hemorrhage that subdural hemorrhage is more likely to occur in all age groups, while epidural hemorrhage is more likely to occur in the young and middle aged. Frequently a fracture line is seen crossing the middle meningeal artery or its branches above the site of an epidural hemorrhage while in acute subdural hemorrhage the lesion is more often contralateral to the skull fracture or site of the trauma. The lucid interval is shorter in epidural hemorrhage than in the acute subdural lesions. If paresis occurs in epidural hemorrhage it is more often contralateral to the trauma while in acute subdural hemorrhage the paresis is just as apt to be ipsilateral to the side of the trauma. They noted that epidural hemorrhage is usually unilateral while the acute subdural hemorrhage is more apt to be bilateral. A 43.7 percent mortality after operation was noted in this series of cases of acute subdural hemorrhage as compared to 54.5 percent mortality after operation in the cases with epidural hemorrhage.

[5] Kennedy, F. and Wortis, H.: Epidural hemorrhage; study of 72 cases of hematoma and 17 cases of hemorrhage. Surg. Gynec. & Obst. 63: 732-742, Dec. 1936.

Usually with trauma severe enough to produce an epidural hemorrhage there is some underlying cortical damage. The spinal fluid may be slightly bloody or xanthochromic though more frequently it is found to be clear.

The localization of epidural hemorrhage is not usually difficult. The pupils are often unequal in size and the dilated pupil is commonly found on the side of the lesion. The presence of other focal neurological signs as hemiparesis, facial weakness, ocular palsies, inequality of deep and superficial reflexes and positive Babinski and Hoffmann signs aid in the localization of the lesion. However, when there is considerable increase in intracranial pressure focal neurological signs become less dependable. Evidence on x-ray examination of a calcified pineal gland which is displaced from its normal midline position is conclusive of an intracranial space-taking lesion; and if the x-ray examination is repeated several hours later and measurement of the position of the pineal gland shows a further shift from the midline position, then it is obvious that the intracranial lesion is a rapidly expanding one.

Surgical intervention for epidural hemorrhage consists in making a trephine opening over the site of the suspected hemorrhage, removing the epidural clot and ligating the proximal and distal ends of the lacerated meningeal artery to prevent further bleeding into the epidural space. If difficulty is encountered in ligating the middle meningeal artery proximal to its laceration it may be necessary to rongeur down to the base of the middle fossa, retract the temporal lobe and dura and occlude the foramen spinosum with a small cotton plug at the point of entrance of the artery into the cranial cavity.

The following case report of a patient with epidural hemorrhage is typical:

Case Report

P. S., laborer, age 65, was struck in a fight February 16, 1940, by a fellow workman and fell to the floor striking his head. He was taken to Temple University accident dispensary several hours after the injury in a semistuporous condition.

Physical Examination.—His temperature was 96° F., respiration rate 16 per minute; pulse was weak, 118 per minute. Blood pressure was 100/50. A contused area was present over his right temporoparietal area of scalp. His pupils were equal and reacted to light and accommodation. Tendon reflexes were hyperactive, slightly increased on the left side. After the period of shock had passed he was able to move all his extremities. A spinal tap revealed initial pressure of 14 mm. hg. and 8 cc. of clear spinal fluid were removed. The patient was admitted to the neurosurgical service.

Clinical Course.—Within several hours the semistupor progressed into stupor. His head was turned to the right. His eyes were directed to the right and upward. The right pupil was 5 mm. and the left, 3 mm. in diameter. Both pupils responded to light stimuli. There was weakness of the extremities on the left which involved the left side of the face below the eye. The tendon reflexes were increased throughout but they were more active on the left side. Hoffmann and Babinski

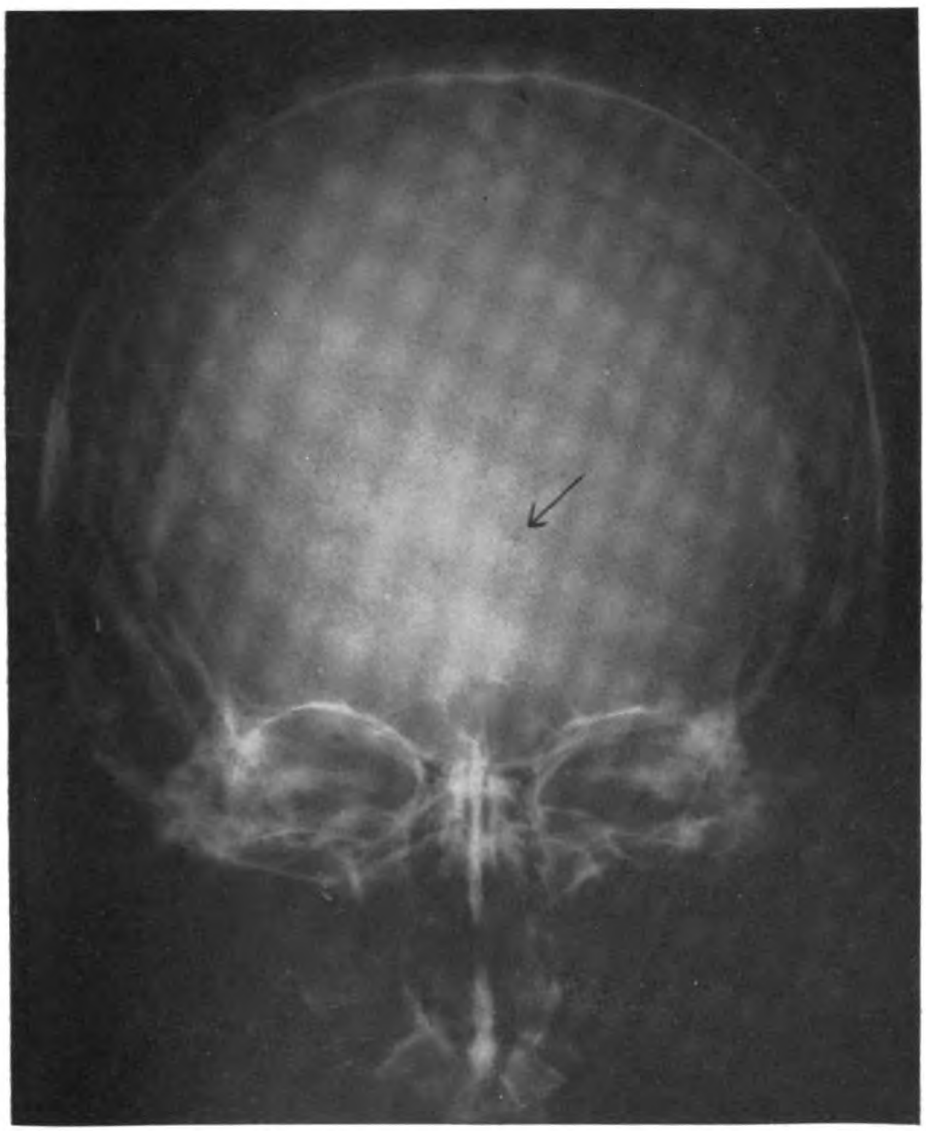

FIGURE 1.—ANTEROPOSTERIOR VIEW SHOWING CALCIFIED PINEAL GLAND DISPLACED.

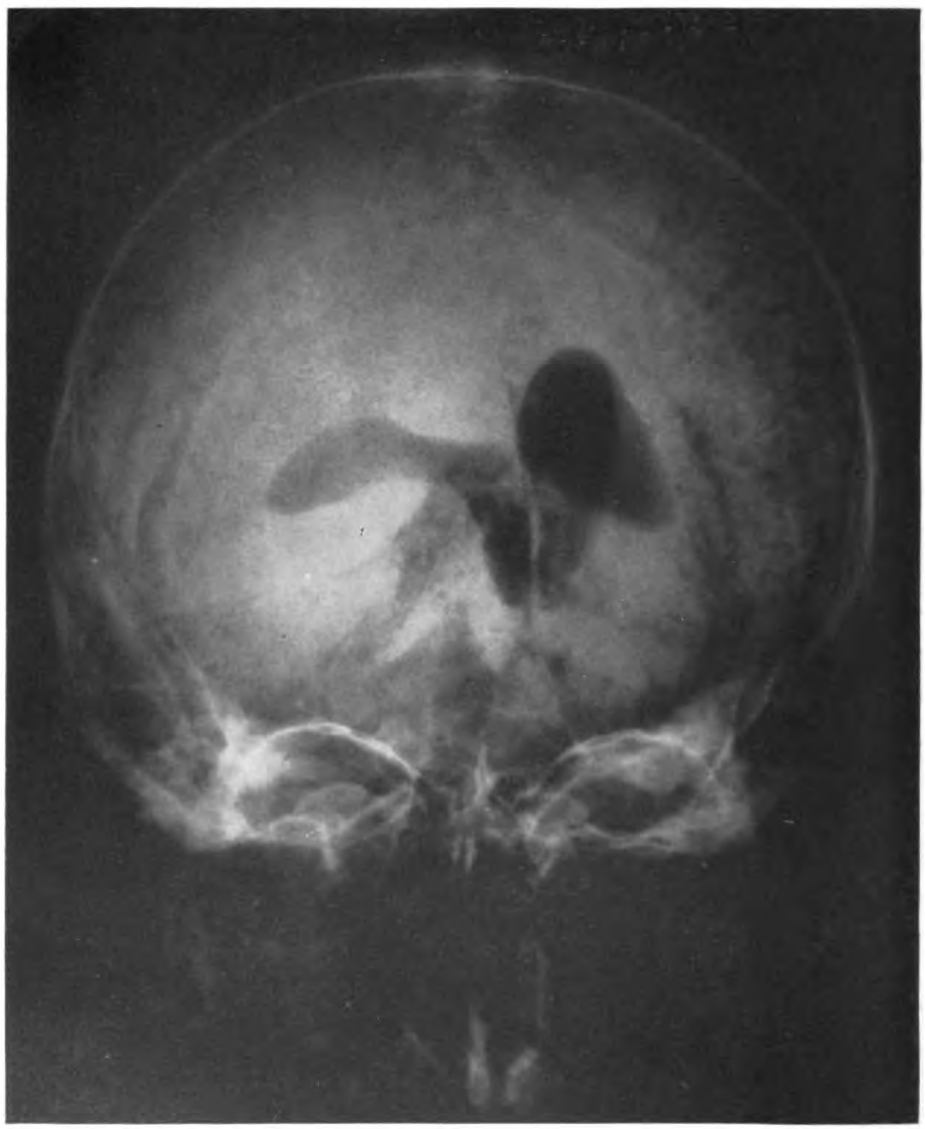

FIGURE 2.--ANTEROPOSTERIOR VIEW SHOWING REPLACEMENT OF CEREBROSPINAL FLUID BY 150 CC. OF AIR. (SEE PAGE 459)

signs were positive on the left. The superficial abdominal and cremasteric reflexes were abolished on the left, present on the right. Corneal reflex was active bilaterally. The patient responded to painful stimulation but he could not be aroused by calling his name. The stupor deepened. The scalp over the right parietal bone became more edematous and pitted on pressure. Both pupils became fixed to light; the right pupil remained dilated and larger than the left. The right eye ball deviated laterally while left remained fixed in the midline. He moved his right extremities but not the left. A generalized increase in muscular tone developed being greater on the left. Temperature rose to 99.2° F., respiration rate decreased to 16 per minute and became stertorous in character. Pulse rate was 65 per minute. The blood pressure was 150/75, with a pulse pressure of 75.

The laboratory examinations were essentially normal except for an elevated blood sugar content of 214 mgm. per 100 cc. of blood, which is not an unusual occurrence in cases of severe head trauma.

X-ray of skull (fig. 1) with mobile unit was difficult due to patient's restlessness. No fracture line was detected. The pineal gland was well calcified and in the anteroposterior plane it was displaced from the right toward the left a distance of 7 mm. from the normal midline position, indicating a mass lesion on the right side.

Operation.—The patient was taken to the operating room and Dr. Michael Scott performed a right exploratory trephine under local anesthesia. A 2-inch linear incision was made in the right midposterior parietal region which uncovered a transverse linear fracture. A trephine opening the size of a silver dollar was made. Upon removal of the button of bone, a huge epidural clot about 2 inches in thickness was encountered. This clot extended in all directions and after being aspirated, the branches of the posterior meningeal artery were found to be lacerated. These were ligated. The bleeding also extended from the anterior portion of the middle meningeal artery. Since this could not be controlled from the posterior opening, another trephine opening was made in the anterotemporal region. The middle meningeal artery was exposed and ligated. Through and through irrigation was done in the epidural space and all bleeding controlled by electrocoagulation. The dura was greatly depressed and at least 6 to 8 ounces of clot were removed. The dura was not opened because there was no evidence of any subdural bleeding. The anterior trephine opening was then closed. A drain was placed in the posterior opening and the incision closed in the usual manner with interrupted silk sutures.

Subsequent to operation the patient became more alert, answered questions, and responded to simple commands. The pupils reacted to light and accommodation and remained unequal; the right being larger than the left. The left arm continued to be paretic but the patient could move his left leg. On the second postoperative day a pneumococcic type-four pneumonia developed. The patient failed to respond to repeated blood transfusions and intravenous injections of type 4 rabbit sera and expired 7 days after admission.

Comment: In cases where epidural hemorrhage is suspected, early x-ray examination may reveal a fracture over the middle meningeal artery groove or a shift in position of the pineal gland. When this is the case it adds confirmatory evidence for establishing the diagnosis of epidural hemorrhage. This is one instance where early x-ray examination is of value but if such an examination necessitates moving the patient, the advantage of learning that a fracture is present or that the pineal gland is displaced does not offset the possible harm that may be done to the patient by having him moved about. In this case a mobile x-ray unit was used and no fracture line was visible. However, the shift in the position of the pineal gland toward the left coincided with the neurological findings. The progressively slow pulse, slow stertorous respiration, ele-

vated pulse pressure, and deepening of the stupor indicated increased intracranial pressure. The pupils were unequal and the right was larger than the left. The left hemiplegia, exaggeration of deep tendon reflexes on the left, left hemiplegia and positive Babinski and Hoffmann signs on the left pointed to a lesion in the contralateral cerebral hemisphere. These findings with a history of a lucid interval, though brief, preceding stupor left no doubt as to the correct diagnosis.

This case emphasizes the importance of frequent and careful neurological examinations and careful recording every 15 minutes of the pulse, respiration, temperature, and blood-pressure readings. Prior to operation as the intracranial pressure gradually rose, the pulse became slowed and the pulse pressure approached and crossed the pulse rate line on the clinical chart, indicating that surgical decompression had to be accomplished immediately.

SUBDURAL HEMATOMA

Acute subdural hemorrhage occurs more frequently than is commonly believed. Kennedy and Wortis [5] reported 89 cases of acute intracranial hemorrhage admitted at the Bellevue Hospital between 1930 and 1936. They found 72 cases of acute subdural hematoma as compared to only 17 cases of epidural hemorrhage. Virchow [6] described the pathology of subdural hematoma in 1857 and pointed out that in chronic alcoholics, luetics, and in the insane, a subdural hemorrhagic cystic process which he termed pachymeningitis hemorrhagica interna frequently occurred following craniocerebral injury. A severe head trauma is usually the cause, but undoubtedly alcohol, syphilis, brain tumor, and various abnormal conditions of the blood or blood-forming organs (avitaminosis, leukemia, scurvy, anemia) are predisposing factors to such an accident.

Trauma severe enough to cause subdural hemorrhage is usually severe enough to produce some underlying brain injury. Hemorrhage into the subdural space may occur from laceration of the dural sinuses, rupturing of the cortical veins as they bridge the subdural space, or by leakage of blood from a ruptured meningeal artery or vein through a lacerated dura. Hemorrhage occurring independently of skull fracture in the adult usually is due to venous origin. Due to the anatomical characteristics of the subdural space the burden of developing a clot is thrown on a relatively avascular dura, for the arachnoid plays little part in aiding clot formation. Leary and Edwards [7] have pointed out that the lack of adequate provision of venous drainage of the granulation tissue and the necessity of organization of the clot from only one surface, are responsible for the poorest efforts at repair found anywhere in the body.

If the subdural hematoma is not removed surgically, and it cannot be removed from the subdural space by any method of dehydration, it acts as an expanding mass and death of the patient may ensue.

[6] Virchow, R.: Haematoma durae matris, Verhandl. d. phys-med. Gesellsch. zu Wurzburg. 7: 134–142, 1857.

[7] Leary, T., and Edwards, E. A.: Subdural space and its lining. Arch. Neurol & Psychiat. 29: 691, Apr., 1933.

Several hours after the collection of blood in the subdural space a fibrovascular layer attempts to envelop the blood. The layer is complete in 2 to 3 weeks. Merritt and Munro [8] have noted that this admixture of blood may become a solid clot or a collection of cerebrospinal fluid with a high protein content and they point out that those intermediate clots possessing any especial degree of fluidity are probably expanding lesions. It is evidenced that they do increase in size from their clinical course. Putnam [9] has shown that red blood cells in the subdural space gradually disintegrate with liberation of hemoglobin from the cell stroma with its reduction to small protein aggregates which produce an increase in osmotic pressure. He believes that added volume of the hematoma, at least in part, is derived from the plasma of circulating blood. The dilated capillaries in the outer wall of the hematoma usually lie close to the lumen of the cyst. An exchange of fluid between the two is therefore probable. Frequently the dilated capillaries in the wall of the hematoma rupture or the hematoma may enlarge by intermittent bleeding at the original source.

Months or even years after head trauma, complicated by subdural hematoma, symptoms of headache, restlessness, irritability, insomnia, convulsions, mental impairment, and personality changes may occur due to increase in size of the capsulated subdural mass. Neurological focal signs are often present such as hemiparesis, ocular palsies, transient or permanent hemiplegia; choked disk and sensory changes may be manifested. The diagnosis is often obscure, confused with brain tumor, epilepsy, and traumatic neurosis. Neurological focal signs may not be present, however, as in the case cited by Elvidge[10] of a patient who was unconscious for 3 days following head injury and recovered, though symptoms of headache and insomnia persisted. Three years later the patient became confused, disturbed, and hallucinated. Petit mal convulsive seizures developed which later became grand mal in type. Six years afterward the patient received a small depressed skull fracture from a bullet wound and on exploration a large, heavily encapsulated subdural organized collection of blood was found which had complicated the cerebral concussion 6 years before and undoubtedly caused the personality change, psychosis, and the convulsive attacks which subsequently occurred.

The diagnosis of acute subdural hematoma is often difficult. The development or progression of focal symptoms as hemiparesis, hemiplegia, aphasia, facial weakness, pupillary changes, is significant. There is a tendency in these cases to an "in and out" state of con-

[8] Merritt, H. H., and Munro, D.: Surgical pathology of subdural hematoma based on study of 105 cases. Arch. Neurol & Psychiat. 35: 64–78, Jan. 1936.

[9] Putnam, T. J.: Chronic subdural hematoma; its pathology, its relation to pachymeningitis hemorrhagica, and its surgical treatment. Arch. Surg. 11: 329, Sept. 1925.

[10] Elvidge, A. R.: Head injuries. Canadian M. A. Jour. 38: 26–33, Jan. 1938.

sciousness though frequently stupor occurs and becomes progressively deeper. A slow pulse rate of 65 or under, shallow and irregular respiration, an elevated pulse pressure over 60, nose itching, and frequent yawning are signs indicating increased intracranial pressure.

The subdural space is an enclosed cavity and does not communicate normally with the underlying subarachnoid space. Hence bleeding into this space does not necessarily cause bleeding into the subarachnoid space. The spinal fluid early after injury will sometimes be pink or slightly bloody due to associated underlying brain damage with rupture of the cortical vessels; or if the arachnoid is lacerated the subdural blood will extravasate into the subarachnoid space and a bloody spinal fluid will result.

Subdural fluid or blood collection cannot be removed by any means other than by operation. However, it is frequently possible to control the state of the cerebral circulation by repeated lumbar taps, magnesium sulphate enemas, intravenous hypertonic solutions, and restriction of fluid until the patient's general condition is improved sufficiently to withstand operation. If satisfactory cerebral circulation can be maintained by these methods, better results are obtained by delaying operation for 7 to 10 days. A safe delay will also offer better opportunity for localization of the lesion due to the opportunity for focal neurological signs to develop.

Temple Fay [11] has pointed out that whenever one encounters severe head injury cases with signs of greatly increased intracranial pressure and rapidly expanding lesion, immediate operation is associated with a 90 percent mortality. Edema and swelling accompany the severely lacerated brain and when a small trephine opening is made in the skull and the dura opened, the expanding brain will herniate through the dural orifice as soon as the subdural clot has been evacuated, producing further trauma to the already softened brain structure. Therefore, if early operation must be undertaken in these cases of combined subarachnoid and subdural hemorrhage, the operator should employ a large cranial flap so as to permit the opening of the dura over half the hemisphere with removal of the bone for decompressive purposes. However, it is important that every possible effort at control of the greatly increased intracranial pressure by physiological decompression be done in order to preserve the patient long enough so that removal of the subdural clot may be safely accomplished on the seventh to tenth day after the injury.

Mention here must be made of a subdural hydroma or hygroma which is a localized collection of fluid in the subdural space which occurs subsequent to head trauma. This condition can only be differentiated from hematoma at operation, for fluid is found in place

[11] Fay, Temple: Treatment of cerebral trauma based upon laws of cerebral hydrodynamics. Surg. Clinics of N. A., 17: 1661–1682, Dec. 1937.

of the usual hematoma. The signs and symptoms and treatment of this condition differ in no way from those of subdural hematoma.

The following case report of a patient with subdural hematoma is typical.

Case Report

J. T., a rug spinner, aged 56, was struck on the right side of his head with a bottle September 3, 1939. He received a slight scalp laceration but he was not unconscious. A history of chronic alcoholism was obtained, otherwise the past history was irrelevant. A week after his head injury he developed a left frontal headache which became progressively more annoying though he continued his work. His wife noticed that he slept more than normally and that he seemed drowsy most of the time. He staggered occasionally when he walked and on bending over became dizzy and fell. Headache persisted and increased in severity. Personality changes were noticed by his friends. He was normally a quiet, reserved individual but he changed to a talkative, blustering type, behaved in a silly manner, took no interest in his work or family, neglected his clothes and became careless in his habits. He had to give up his work October 12, 1939, due to weakness of his left arm and leg and attacks of vertigo. Frequent vomiting spells occurred. He was admitted to the neurosurgical service, Temple University Hospital, November 2, 1939.

Physical Examination.—Neurological examination showed some clouding of consciousness, lack of insight and euphoria. Pupils were 3 mm. in diameter and active to light and accommodation. There was no choking of the disk. The perimetric fields were normal. Weakness of the lower two-thirds of the face on the left side existed. Considerable weakness and impairment in volitional movements were noted in left upper and lower extremities. All deep reflexes were exaggerated but they were more increased on the left. Hoffmann and Babinski signs were negative. Sensation was intact for all modalities. Gait was hemiparetic in type. Percussion dullness was elicited over right frontoparietal area. Temperature was normal, pulse ranged from 65 to 72 per minute and respiration rate was 20 per minute. Blood pressure was 130/60, with pulse pressure of 60.

X-ray examination of skull showed the pineal gland displaced 7 mm. toward the left and 1.5 mm. beyond the dorsal limit of its normal position.

Encephalogram was done with displacement of 150 cc. of cerebrospinal fluid with air. The initial spinal fluid pressure was 7 mm. of hg. The first portion of the spinal fluid removed was clear but the last portion was xanthochromic. The cerebrospinal fluid contained 49 white cells per cu. mm. and numerous red blood cells; differential cell count showed polymorphonuclears 17, lymphocytes 68, and mononuclears 15 percent.

The encephalogram (fig. 2) revealed the presence of a mass lesion on the right. All the midline structures were displaced toward the left and the right lateral ventricle was displaced downward. More displacement was noted on the anterior than posterior horn of the right ventricle indicating that a mass lesion was well forward and in the right frontoparietal region.

Operation.—An exploratory right craniotomy was performed. The posterior limb of a temporoparietal flap was made and a burr opening revealed a chronic subdural hematoma. An osteoplastic flap was reflected and the clot, which was composed of a light, porous, friable, old hematoma of pale milk chocolate color, extended over the temporoparietal area. There were two pillars which bridged between the thick membrane on the surface of the dura and arachnoid which were fully organized and contained blood vessels. The firm walls of the cystic hematoma were removed by curettage and by irrigation. Hemostasis was secured and

the dura sutured. A small drain was left in the subdural space. Closure was made in the usual manner with interrupted silk.

Convalescence was uneventful. The patient was discharged from the hospital December 15, 1939, with no residual neurological symptoms other than slight weakness of left arm and leg. When examined 6 months later, full recovery had been made and he had returned to his former occupation.

COMMENT.—This case is typical of the clinical course of chronic subdural hematoma. The initial head injury was apparently very trivial and was superimposed on a chronic alcoholic individual. The onset of such symptoms as personality change, headache, weakness of extremities and vertigo was gradual though marked with intermittency. The differential diagnosis in this case was not difficult though brain tumor and brain abscess had to be excluded. The history was suggestive of chronic subdural hematoma though it is not uncommon to find a patient with brain tumor who gives a history of previous head injury. It is more important to exclude brain abscess than tumor because the operative procedure for abscess and subdural hematoma differs considerably. Any space taking cerebral lesion must be considered potentially as abscess and frequently its exclusion is met with difficulty.[12] The absence of low-grade fever, leukocytosis, increase in polymorphonuclear cells in the spinal fluid and increase in blood sedimentation rate aided in ruling out abscess.

GENERAL CONSIDERATIONS IN THE TREATMENT OF HEAD INJURY

There is no routine treatment applicable in every craniocerebral injury. The management depends absolutely on the symptoms and signs manifested in each individual case. These signs and symptoms may vary considerably in the more serious cases within a short period, indicating increase in cerebral volume, threatening medullary compression, impaired cerebral circulation and increase in intracranial pressure. Especially is this true within the first 24 hours following the injury.

To properly evaluate the clinical signs and determine the degree of intracranial pressure and its underlying pathologic cause, at least some conception of cerebral hydrodynamics and volume relationships is necessary in order to institute competent treatment.[12 13 14 15]

The Monro Kellie doctrine, formulated in the early nineteenth century, introduced certain principles of physics relative to the flow of fluids through the closed nonexpanding cranial walls.

There are three volume components which fill the intracranial cavity:

1. Cellular content, which largely deals with the function of the nervous system, forms one component, though many of the cells enter into the formation of the walls of the blood vessels, the meninges and dura. The volume of this component may vary considerably depending on the presence of swelling or edema.

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[12] Smith, Gerald: Brain abscess, a review of 20 proven cases. U. S. Nav. Med. Bull. **38**: 358, July 1940.

[13] Swift, G. W.: Conservative treatment of cerebrocranial injuries. Am. J. Surg. **42**: 510, Dec. 1938.

[14] Fay, Temple: Treatment of acute and chronic cases of cerebral trauma by methods of dehydration. Ann. Surg. **101**: 76, Jan. 1935.

[15] Craig, W. M.: Physiology pathology and treatment of craniocerebral injuries. N. E. J. Med. **212**: 77, Apr. 1935.

2. The blood within the arteriovenous vessels constitutes one component and its volume fluctuates depending upon whether ischemia or hyperemia of the brain is present.

3. The third component and the least essential of the three, consists of the free cerebrospinal fluid in the intracranial subarachnoid space and ventricles together with the interstitial fluid lying outside the blood vessels and brain cells. This extracellular or interstitial fluid comprises a considerable portion of the brain cavity.

It has long been recognized that following head trauma, increase in cerebrospinal fluid and swelling of the brain substance by edema occur,[15] [16] and that the increase in the 2 components within the skull would mean a decrease in the third, or blood volume, which would lead to serious cerebral anemia and anoxemia of the brain cells.[17] [18]

Rational treatment of head injury aims to maintain sufficient blood volume in the cranial cavity to insure normal function of the brain. Head trauma may impair the function of the brain by direct laceration, by accumulation of extravasated blood or fluid in the subdural or epidural spaces, increasing the volume of cerebrospinal fluid or by causing swelling and edema of the brain substance. All these effects of head trauma cause increase in intracranial pressure and cerebral compression. To overcome this cerebral compression and provide more space in the cranial cavity for better circulation only one component may be reduced in volume. It is obvious that one cannot decrease the size of the brain volume if the patient is to recover his mental faculties. Surgical decompression will increase the size of the cranial cavity allowing more blood to enter, but when accomplished it is met frequently with disaster because the swollen brain herniates through the dura opening and causes damage to the cortex. The blood volume component cannot be reduced because cerebral circulation must be maintained to insure the patient's recovery. Therefore the only component in the cranial cavity which is relatively unimportant and which can be reduced with safety is the interstitial and cerebrospinal fluid.

Reduction of the cerebrospinal fluid volume can be accomplished by spinal drainage or ventricular tap. By introduction of hypertonic solutions intravenously or into the alimentary canal the interstitial fluid content in the brain is reduced allowing added space for an increased blood volume in the cranial cavity with resultant improvement in the brain cells in terms of repair, nourishment and function.[18]

Howe [19] first noted in experimental work on animals that glucose

[16] Lehman, E. and Parker, W.: Unsolved problems of brain injury. Internat. Clin. **3**: 181, Sept. 1935.

[17] Werder, J. D.: Drainage of cerebrospinal fluid in treatment of acute head injury. Arch. Surg. **34**: 424, 1937.

[18] Weed, L. H. and Hughson, W.: Systemic effects of the intravenous injection of solutions of various concentrations with special reference to cerebrospinal fluid. Am. J. Physio. **58**: 53, Nov. 1921.

[19] Howe, H. S.: Reduction of normal cerebrospinal fluid pressure by intravenous administration of hypertonic solutions; experimental studies on cats. Arch. Neurol and Psychiat. **14**: 315–326, Sept. 1925.

solution administered intravenously favored absorption of interstitial fluid from the brain. Fay and Peet [11] were the first to apply this observation to cases with edema of the brain with the result that its use became widespread and a necessary adjunct to the treatment of severe cerebral trauma cases associated with shock.

The cerebrospinal fluid is derived from the blood. When the blood volume becomes reduced the cerebrospinal fluid in the ventricular reservoir is called upon to help replace the fluid loss. Hence the blood volume and cerebrospinal fluid are complementary and changes in one component affects the other reciprocally. So by reducing blood volume by magnesium sulphate by mouth or rectum, bleeding or sweating, spinal fluid volume is diminished. This reduction in spinal fluid volume provides additional space in the cranial cavity for improved circulation. However depletion of the cerebral interstitial and ventricular fluid will not suffice unless steps are taken by sufficient limitation of fluid intake to maintain a state of dehydration. The beneficial effect of spinal drainage and hypertonic solutions by vein and bowel obtained by allowing for better cerebral circulation would soon be lost if the patient were allowed to immediately replace the fluid by being given fluid by mouth or if enemas or large infusions of saline were administered. It has been found that an adult post-traumatic patient receiving 20 ounces of fluid per 24 hours and a dry diet will not produce sufficient spinal fluid to increase intracranial pressure.

As successful treatment of head injuries depends upon supplying sufficient oxygen to the traumatized brain to insure return of normal cerebral function, certain necessary requirements must be fulfilled before recovery of the patient is possible:

1. In order to furnish the lungs with the required amount of oxygen it is imperative that the air passageways be open and kept open. A trachea nearly occluded with mucus or a retracted tongue can be easily overlooked. It has been shown experimentally that brain cells become swollen and chromolysis occurs after 90 seconds of complete anoxemia and that they degenerate completely after 4 minutes in such a state. A patient in coma lying for several hours with insufficient oxygen supply to the lung bed will be seriously hindered in making a satisfactory recovery even though every other form of therapy is provided. A vicious cycle is also produced in that the anoxemic brain cells tend to become edematous due to an increased permeability of their cellular walls.[20] This in turn reduces the cranial space for necessary blood volume in the cranial cavity and thereby increases the cellular anoxemia.

2. The lung bed must be adequate to utilize the oxygen delivered. If pneumonia, atelectasis, pleural effusion, or increase in interstitial

[20] Landis, E. M.: Micro-injection studies of capillary permeability; effect of lack of oxygen on permeability of capillary wall to fluid and to plasma proteins. Am. Jour. Physiol. 83: 528–543, Jan. 1928.

fluid interferes with proper oxygenation of the blood, steps must be immediately taken to remedy it.

3. Ample blood volume must be maintained for transporting oxygen from the lung bed to the brain. In shock, fluid leaves the vessels to pass out into the subcutaneous tissues,visceral and cranial cavities, and considerable fluid is lost from the blood volume onto the skin surfaces as evidenced by clammy and cold extremities. Without adequate blood volume a cerebral anoxemia ensues.

4. It is necessary in order to supply the brain with sufficient oxygen that the blood going to the brain contain an adequate number of red blood cells and sufficient hemoglobin content. After extensive hemorrhage or in feeble undernourished individuals who have received head injury, blood transfusions are necessary to restore these constituents to the blood volume.

5. The heart action must be satisfactorily maintained in order to force blood to the cerebral structures. Cardiac stimulants may be required to increase the heart function.

6. Space in the cranial cavity must be provided to allow enough blood to enter in order to provide nourishment to the brain cells. All other requirements to successful treatment may be met, but if there is present high intracranial pressure from a space taking lesion, massive depressed fracture, cerebral edema, or increase in spinal fluid in the ventricles, sufficient cerebral circulation cannot be maintained because cranial space for adequate blood volume is lacking, hence recovery of the patient is impossible. To supply the required space for a cerebral blood volume compatible with life, dehydration methods of treatment are valuable. The method of treatment discussed here is based upon the principle that ample oxygen supply to the brain must be maintained. When this is kept in mind, the rationale of the management of the patient with head injury is more easily understood.

EVALUATION OF CLINICAL SIGNS

The proper interpretation of temperature, pulse, respiration, and blood-pressure readings is of most importance in the management of head trauma cases. These clinical signs afford us a means of determining the true state of the patient in terms of intracranial pressure and cerebral function.

Posttraumatic patients in shock have a subnormal temperature which is largely due to evaporation of moisture from the skin. The pulse is weak and rapid because the heart must increase its rate to keep the arterial tree filled in the face of a low-blood volume. The respiration rate is fast and shallow due to the premature loss of oxygen before it reaches the periphery with resultant cellular anoxemia.[21]

[21] Macleod, J. J. R.: Physiology and Biochemistry in Modern Medicine, C. V. Mosby Company, St. Louis, publishers, 1922.

There is a fall in blood pressure, especially the diastolic, which goes below 60 mm. of hg.

After the patient recovers from shock the careful observation and recording of the temperature, pulse, respiration rate, and blood-pressure readings are just as important as before. As the intracranial pressure rises the temperature becomes elevated due to disturbance in the vasomotor or heat-regulating centers in the brain. Temperatures rapidly rising to 106° and 107° F. indicate edema or hemorrhage within the brain substance, usually in the region between the medulla and the basal ganglion. Rapidly rising temperature above 105° F. almost invariably means a terminal condition and strongly suggests an intramedullary hemorrhage. This type of hyperpyrexia does not respond to the usual methods of body-heat reduction forms of therapy.

With increased intracranial pressure the pulse rate becomes slower due to irritation of the vagal center in the medulla. A pulse rate below 68 per minute is significant and the attending physician should search for the cause of the vagal irritation which may be due to bloody spinal fluid, or expanding subdural or epidural hemorrhage. Highly elevated intracranial pressure produces a paralysis of the vagus center with resultant loss of the center's regulatory action on the heart and a rate of 120 per minute will probably ensue.

A respiratory rate of 16 per minute or under is usually associated with increased intracranial pressure. Shallow respirations of the Cheyne-Stokes type develop with edema of the respiratory center, hemorrhage into the floor of the fourth ventricle or with herniation of the brain stem into the foramen magnum.

Careful observation of the systolic and diastolic blood pressure is important in the management of head trauma cases for these readings probably give the most accurate criteria as to the true state of the intracranial pathology. As the intracranial pressure increases there is a corresponding rise in pulse pressure. When the pulse pressure approaches or crosses the pulse rate level on the chart, valuable information is obtained indicating that there is a failure in the cerebral compensatory mechanism and that efforts must be made to reduce immediately the intracranial pressure. However, by watching and recording the pulse pressure every 15 to 30 minutes in the early case, the physician is warned of increasing intracranial pressure by a gradual increasing pulse pressure. Proper acute methods of dehydration can then be instituted instead of waiting until the cerebral compensatory mechanisms fail.

A pulse rate of 120 or more per minute in the absence of elevated temperature usually means that the patient is in need of more fluid to supplant a low-blood volume. A fast pulse, low-blood pressure and a low-pulse pressure indicate a failing peripheral circulation. Steps must be taken to increase the fluid in order to combat circulatory

failure. A slow pulse with a high pulse pressure is interpreted as a failing cerebral circulation due to increased intracranial pressure. In this case all possible methods must be taken to reduce the elevated pressure in order to allow for an improved cerebral circulation. Spinal drainage, magnesium sulphate enema, hypertonic glucose and salt solutions given intravenously will usually bring about the beneficial effects desired.

TREATMENT DURING PERIOD OF SHOCK

Shock is invariably present subsequent to severe craniocerebral injury and consideration for its treatment is of paramount importance and takes precedence over treatment of the cerebral condition. Immediately upon admission of the patient, general inspection of the patient and careful recording of the temperature, pulse, respiration and blood pressure readings will give information if shock is present.

Shock is recognized by the presence of the following clinical signs: (*a*) Fast and thready pulse, usually above 120 per minute. (*b*) Subnormal temperature. (*c*) Cold clammy skin. (*d*) Low blood pressure; diastolic pressure is usually below 60 mm. of hg.

If the patient is in shock, warm dry clothing and heat should be applied to the body surfaces. Atropine sulphate gr. 1/100 (0.65 mg.) is given hypodermically to prevent further loss of fluid from the skin surfaces to limit further reduction in blood volume. Pituitrin (surgical) 15 minums (1 cc.) given hypodermically acts as a vasoconstrictor of the peripheral circulatory bed which reduces the blood volume container, improves diastolic pressure and provides for better oxygenation. Strychnine sulphate, $\frac{1}{30}$ gr. (0.002 gm.) given hypodermically is useful for its stimulating effect. Cardiac stimulants such as caffeine, sodium benzoate, and adrenalin are seldom indicated ordinarily because the heart is usually sufficiently active. However, with signs of failing circulation these cardiac stimulants may be given. Fifty to seventy-five cc. of 50 percent glucose are given intravenously depending on whether the patient is obese, thin, emaciated, or alcoholic. Larger doses are given to the fat patients for they are in need of greater dehydration. Hypertonic glucose solution given intravenously draws from the tissues fluid necessary for replenishment of blood volume, thus improving blood pressure and the general circulation. This withdrawal of fluid from the tissues aids in reducing cerebral edema. However, the repeated and indiscriminate use of intravenous glucose solution is not recommended. Gregerson and Wright [22] have shown that glucose has only a brief salutary effect and then migrates to the tissues. This migration, as pointed out by

[22] Gregerson, M., and Wright, L.: Effect of intravenous injection of sucrose and glucose upon reducing power of cerebrospinal fluid, before and after hydrolysis. Am. J. Physiol. 112: 97, May 1935.

Milles and Hurwitz,[23] produces a secondary rise in intracranial pressure and also may cause secondary shock.

Experimental work with the use of sucrose intravenously has shown that this sugar is not hydrolyzed or stored in the body tissues or blood when given intravenously. It is suggested that 50 percent sucrose may be given in 100 to 200 cc. amounts intravenously instead of 50 percent glucose for dehydration. It is pointed out that 92 percent of the sucrose given is eliminated in the urine in 24 hours and that the marked diuretic effect produces a rapid drop in cerebrospinal pressure within 2 hours after injection.[24] [25]

In this clinic sucrose is seldom used. Its value has not been proven as yet in a large series of trauma cases. It has no nourishing value. Also a few reports on its possible injurious effect to the kidneys have been published.[26] If used instead of glucose during the period of shock it is possible that the period of shock may be prolonged due to its marked and rapid dehydration effect.

If sufficient blood volume cannot be maintained by intravenous use of 50 to 70 cc. of 50 percent glucose solution, 100 to 150 cc. of normal saline solution may be given where shock is severe. Large quantities of saline solution given intravenously are contra-indicated, for, if the patient survives the shock, he may succumb to respiratory paralysis from cerebral edema produced by the excessive amount of fluid injected. In cases where shock persists it is better to give repeated small amounts (50 to 100 cc.) of normal saline intravenously than an initial large quantity. The period of shock may be considered present as long as the temperature remains subnormal and the pulse rate above 120 per minute.

Control of bleeding points of the scalp should be accomplished and the presence or absence of simple, depressed, or comminuted fracture can be ascertained by gloved finger inspection of the wound. If there is persistent bleeding from the nose, vasoline gauze strips may be packed into the nasal cavity on the side of the bleeding. If cerebrospinal leak, or bleeding from the ear is present the external ear is carefully cleansed and a tampon soaked in dichloramine-T or 2 percent mercurochrome solution is inserted into the auditory canal. Cleansing of the scalp wound with simple antiseptics is done and dressings are applied, but no attempt is made at surgical intervention as elevation of a depressed or suturing of a large wound for these procedures should be done in the operating room and are not indicated until all evidence of shock has disappeared. X-ray examina-

[23] Milles, G., and Hurwitz, P.: Effects of hypertonic solutions on cerebrospinal fluid pressure with special reference to secondary rise and toxicity. Arch. Surg. 24: 591, Apr. 1932.

[24] Bullock, L. T., Gregerson, M. T., and Kinney, R.: Use of hypertonic sucrose solution intravenously to reduce cerebrospinal fluid pressure without a secondary rise. Am. J. Physiol. 112: 82, May 1935.

[25] Masserman, J. H.: Effects of intravenous administration of hypertonic solutions of sucrose. The Johns Hopkins Hospital Bull. 57: 12, July 1935.

[26] Barker, H.: Dangers of rapid diuresis. Ill. M. J. 72: 313, 1937.

tion, clinical procedures and all unnecessary movement of the patient likewise should be avoided at this stage. All effort should be centered on getting the patient over the state of shock and on the subsequent treatment for increased intracranial pressure which will usually develop. Temperature, pulse, respiration, and blood pressure readings should be recorded every 15 minutes. During the period of shock the necessary stimulants are repeated as often as may be required until the period of shock is over.

TREATMENT DURING THE ACUTE CEREBRAL PHASE

A careful physical and neurological examination of the patient is made. Dislocation of the joints, fracture, and internal injuries must be looked for and if present the necessary treatment should be instituted. Morphine is **never** indicated in head trauma cases as it masks the clinical signs and is dangerous because of its depressing effect on the respiratory center which is already probably affected by the increased intracranial pressure. Neurological examination will elicit any focal signs if they are present. The patient's state of consciousness should be accurately recorded for it is important to know if any subsequent change occurs since progressive increase in mental dullness, confusion, stupor or coma indicates a rising intracranial pressure.

Symptoms of restlessness, irritability and hyperactivity indicate cortical irritation and sedatives may be necessary. Chloral hydrate and sodium bromide, *a.a.* 10 gr. have been found useful in these cases. If the patient is unable to take these by mouth, double the dose may be given by rectum.

The size, shape, and reaction of the pupils are noted. Unequal pupils are of localizing significance. If the patient is unable to speak it must be learned whether this inability is due to aphasia, dysarthria, or stupor. Facial weakness can be demonstrated by noting angle of the mouth and the degree of relaxation of the cheeks during expiration, the flabby cheek being on the paretic side. Supraorbital pressure will cause a facial grimace and by comparing the two sides it can be ascertained which side of the face is the weaker. This is an important localizing procedure if the patient is not in deep coma and unresponsive. Nuchal rigidity usually indicates subarachnoid bleeding or an injury to the cervical spine. The patient's movements in bed are observed. The extremities on one side may move about more than the other, indicating a hemiparesis. A paretic lower extremity will usually show as an eversion of the foot and an outward rotation of the thigh. A flattening of the muscular contour of the thigh may signify loss of tone and the presence of a flaccid paralysis of that extremity. Elevating the arms or legs and letting them fall by their own weight to the bed will demonstrate any paralysis. The affected extremity will fall flail-like. The normal extremity's fall will be more

delayed. The sensory examination may be limited to the effect of painful stimulation, depending on the state of consciousness of the patient. The degree of stupor can be fairly well determined by applying supraorbital pressure or by pinching the skin along the medial surface of the thigh. Failure of response indicates deep stupor or coma.

The presence, degree of activity, and evidence of any asymmetry of the deep and superficial reflexes are noted. The presence of the Babinski phenomenon is of significance though its absence does not rule out pyramidal tract irritation. The presence of a positive Hoffman sign often accompanies hyperreflexia and spasticity as found in hemiplegic states but its presence does not necessarily indicate pyramidal tract involvement since this sign has been noted by the writer frequently in emotional and hypothermic states.[27]

After the period of shock has passed, spinal puncture is performed. This procedure is contraindicated in cases with compound fracture of the skull with cerebrospinal leakage from the ear or nose because of the reverse in flow of the fluid which may occur with resultant infection. Spinal puncture requires a careful pressure reading by manometer. Normal spinal fluid pressure ranges from 6 to 10 mm. of hg. If the spinal fluid pressure is elevated to 14 mm. of hg. or over and is clear in color, the clinician must seriously consider the presence of an expanding lesion and watch carefully for the development of focal neurological signs. Hemorrhage usually occurs in the epidural or subdural space and as these spaces do not normally communicate with the subarachnoid system, the spinal fluid will not be bloody. If the spinal fluid is found to be bloody, the pressure is taken and all the spinal fluid which can be removed is slowly drained from the spinal canal, with the patient in the recumbent position. The purpose of this drainage is not only to reduce the intracranial pressure and prevent subsequent rise in pressure but also to remove the blood from the subarachnoid space and facilitate normal absorption of the spinal fluid by the pacchionian system. It is important to do daily spinal drainage until the spinal fluid becomes clear. This usually takes from 6 to 8 days. If, however, spinal drainage is not done until several days after the patient's admission to the hospital, the blood in the spinal fluid may cause a noncommunicating type of hydrocephalus, due to the blockage in the ventricular system which prevents the ventricles from freely communicating with the basal cistern. Withdrawal of fluid by spinal tap after this complication has occurred is dangerous because the sudden release of fluid from about the base favors movement of the brain structures down into the incisura of the tentorium or the foramen magnum with resultant immediate respiratory paraly-

[27] Fay, Temple and Smith, Gerald: Neurological changes observed in humans in state of hypothermia. In press.

sis. Therefore, emphasis is placed on doing a spinal tap early after the case is seen and not to delay until one is forced to relieve the high-intracranial pressure when such a procedure is wrought with danger.

If the above procedure is followed, valuable data will be obtained regarding temperature, pulse, respiration, blood pressure, pulse pressure, the presence of focal and general neurological signs, spinal fluid pressure and the nature of the spinal fluid. Sufficient information is now available to determine the subsequent treatment. The patient is now transferred to the proper hospital service and the following routine requested:

1. Temperature, pulse, and respiration recorded every 15 minutes.
2. Complete blood count.
3. Urinalysis.
4. Typing of patient's blood (when severe blood loss).
5. Blood Wasserman.
6. Position flat in bed.
7. Blood pressure every half hour.
8. Ice bag to head.
9. Pulse pressure (the difference between the systolic and diastolic readings) is charted separately with the pulse every half hour.
10. A dry diet.
11. The fluid intake and output are carefully recorded on the chart. The amount of fluids allowed depends on several factors. If the patient is a fat hydrated type of individual, less fluids are required because his fluid reservoirs are large and filled. On the other hand, if the patient is thin and emaciated more fluid is necessary, for the patient has little stored from which to draw. The amount of fluid per 24 hours allowed the patient also depends on the nature of the spinal fluid.

(a) If the spinal fluid is free from gross blood, further spinal fluid drainage will probably not be indicated. Total fluids, 600 cc. per 24 hours are allowed.

(b) If the spinal fluid contains blood, daily or more frequent spinal drainage becomes necessary for removal of the blood. Total fluids, 900 cc. are allowed daily.

A patient receiving 600 cc. of fluid per day, including milk, broth, fruit juices, water, and coffee, will not have an increase in the amount of spinal fluid after the second day. This will control the spinal fluid pressure and volume and allow for adequate cerebral circulation.

When one encounters bloody spinal fluid it is necessary to do daily spinal drainage and in order to slowly drain off at least 45 to 60 cc., which is desirable, ample fluid, 900 to 960 cc., must be given to provide for the formation of this amount of spinal fluid.

Solid foods are usually taken and retained better than soft foods. However, if soft foods or food with high-water content are given to

the patient, they must be included in the total daily fluid allowance and estimated as an equivalent amount of water.

Thirst is a common and early complaint. It is found that a listerine gargle, moistening the lips with mineral oil or by giving small amounts of fluid at regular intervals will allay this discomfort.

The following medications may be prescribed if found indicated. Urotropin is given 3 times a day in 5-grain doses. This drug tends to prevent infection in the central nervous system and is ordered routinely in this clinic before and after every spinal puncture. Its actual beneficial effect has, however, never been proven. Crowe [28] first reported the use of urotropin in 1909 in treatment of head trauma with cerebrospinal leak through the ear and nose and its use in head trauma cases has continued.

If cortical irritation has occurred with symptoms of irritability, restlessness, and insomnia, chloral hydrate and sodium bromide, *a. a.*, 10 gr. (0.65 gm.) are useful sedatives. If the pulse rate is over 120 per minute, digalin 20 minims (1.25 cc.) alternated every 4 hours with pituitrin, 15 minims (1 cc.) may be given if found indicated. Sulfanilamide and sulfapyridine are useful as a preventive for meningitis from streptococcic, meningococcic or pneumonococcic origin if the circumstances warrant their use.

If the signs and symptoms point to an increase in intracranial pressure it may be necessary to repeat the spinal drainage or repeat 50 cc. of 50 percent glucose solution intravenously and give a magnesium sulphate enema. The proportion of the ingredients used for the enema are as follows: magnesium sulphate crystals, 90 gm.; glycerin, 30 cc.; water, 180 cc. Or, if the patient is able to swallow, 45 gm. of magnesium sulphate crystals in 180 cc. of water are given by mouth. When rapid and immediate dehydration is desired, 20 cc. of 20 percent sodium chloride solution may be given intravenously.

TREATMENT DURING TEN-DAY PRECONVALESCENT PERIOD

Careful recording of the temperature, pulse, respiration, and blood-pressure readings at 4-hour intervals are made. Frequent neurological examinations for the presence of any focal signs should be continued. Careful observations of the patient's state of consciousness are noted. If the spinal fluid is bloody, daily spinal drainage (patient in recumbent position) is accomplished, the cells counted and the fluid saved for daily comparison. Careful recording of the fluid intake and output is continued. If the patient had clear spinal fluid the fluid intake is raised to 960 cc. per 24 hours and maintained at that level. After the seventh to tenth day, if the bloody spinal fluid becomes xanthochromic or clear, daily spinal drainages are discontinued and the fluid intake is reduced to 600 cc. per 24 hours for

[28] Crowe, S. J.: Use of urotropin. The Johns Hopkins Hospital Bull. April 1909.

several days to prevent the necessity for any further drainages for relief of pressure which usually occurs when regular spinal drainages are discontinued. The fluid level is gradually raised to 960 cc. per day and the patient is discharged at that level.

TREATMENT DURING THE CONVALESCENT PERIOD

For the following 3 months after discharge the patient is allowed 960 cc. of fluid per 24 hours. Sweets and salts are restricted to reduce thirst. The use of alcohol in any form is discouraged. The patient is given instructions regarding the diet he is to follow which consists largely of solid foods. It should be limited to moderate amounts of red and white meats, eggs, non-salty fish, dry cereals, toast and butter. The following vegetables are recommended because of their comparatively low-water content: Potato (baked, fried, French); peas; beans (navy and lima); carrots; beets; parsnips; rice; and corn.

Vegetables which should be avoided because of their high-water content are tomatoes, spinach, asparagus, turnips, cabbage, all greens, string beans, cauliflower, sweet potatoes, onions, and squash. All soggy dishes as apple sauce, puddings, gravy, stews, and macaroni should be eliminated from the diet. If these foods are taken they should be exchanged for a proportionate amount of allotted liquids.

Symptoms of headache, irritability, mental dullness, loss of attention and easy fatigability, which are commonly associated with post-traumatic syndrome have been largely avoided. It is believed that the infrequent occurrence of these symptoms following head injury is due to the careful detailed control of water balance through the acute phase and during the 3 months following the patient's discharge. Formerly 6 to 9 months were considered required for patients to readjust themselves socially and industrially. Quite frequently partial or complete disability occurred. Eighty percent of the patients treated with this method have returned to work and normal activity within 3 months following their traumatic episode.[29]

SUMMARY

With modern fast transportation methods the incidence of head injury becomes greater and its treatment is a greater problem to the medical profession. A method of treatment covering the period of shock, acute cerebral phase, preconvalescent, and convalescent stages is given which can be followed on board ship, in a dispensary or well-equipped hospital. The rationale of the treatment is based on the maintainance of adequate cerebral circulation, which must be maintained before complete recovery can be accomplished. The usual cause for impairment of cerebral circulation following head

[29]Scott, Michael: End result in series of 655 head trauma cases. Arch. Neurol. & Psychiat. 40: 605, 1938.

trauma is primarily physical in that the required space is not available in the cranial cavity for necessary blood volume due to increase in the interstitial and cerebrospinal fluid. Adequate space for increased blood volume in the cranial cavity can be provided in most instances by the conservative form of therapy outlined. Surgical decompression, which formerly was resorted to in a high percentage of patients with serious head injury, is now found necessary in only rare instances due to the beneficial effect obtained from physiological decompression.

The importance of recording the temperature, pulse, and respiratory rate and blood pressure at frequent intervals during the acute cerebral phase is pointed out. Accurate evaluation of these clinical signs, which affords an index to the degree of intracranial pathology, is necessary before intelligent treatment can be instituted. The pulse pressure is considered one of the most important of the clinical signs, for changes in this reading indicate fluctuation in intracranial pressure which must be constantly known and controlled throughout management of the case.

Various types of head injury (cerebral concussion and contusion; skull fracture; subarachnoid, epidural and subdural hemorrhage) are discussed from a diagnostic and prognostic point of view and certain classical case histories are included.

The vital necessity for avoidance of unnecessary clinical procedures, examinations or movement of the patient during the period of shock and acute injury is stressed.

EXTRADURAL HEMORRHAGE [1]

WITH CASE REPORT

By Lieutenant Commander Robert E. Baker, Medical Corps, United States Navy

In the treatment of head injuries there are a great many controversial features. Subtemporal decompressions, formerly widely practiced for the relief of increased intracranial pressure, are now seldom resorted to except in certain clinics, notably Dandy's, and then in less than 10 percent of the cases of severe head injury. Dehydration measures, including the restriction of fluid intake, the intravenous injection of hypertonic solutions of saline, glucose, and more recently sucrose, and purgation, have been widely adopted in the treatment of these cases during the past few years, largely due to the favorable statistics presented by Temple Fay and others using these measures.

The question of spinal puncture and lumbar drainage is perhaps the most controversial one of all. One can find excellent authority for any aspect of this subject. Dandy is strongly opposed to spinal

[1] Read at the 371st meeting of the Medical Association of the Isthmian Canal Zone at Colon, R. P.

punctures, even for the determination of the pressure of the spinal fluid, and says that in the presence of intracranial pressure from whatever source, lumbar punctures are always dangerous. He states that when lumbar puncture is performed soon after an accident, the additional room that is immediately available allows renewed bleeding to take place. His chief objection, however, is that he feels that a traumatic cerebral edema results from the readjustment of the brain to equalize the changed intracranial pressure. On the other hand, Fay advocates lumbar puncture as soon as the period of shock is over and if the fluid is found to be bloody, the complete drainage of all fluid obtainable in the horizontal position, 30 to 70 cc. if necessary. He then continues the lumbar drainage as often as a rise in pulse pressure and respiratory changes may indicate, every 4 to 6 hours if necessary. He believes that reestablishment of the cerebrospinal fluid circulating function is usually possible by the eighth to the tenth day following injury and then he discontinues drainage. Frazier and Grant take a middle course regarding spinal drainage. They advocate the recording of the spinal fluid pressure and if it is above the normal, removing sufficient fluid to permit the excess pressure to be lowered by one-half. Fay admits the possibility of foramenal hernia but states that it will not result from spinal puncture if the ventricles are freely communicating with the basal cistern, and that occlusion by clot in the third or fourth ventricle or Aqueduct of Sylvius is extremely rare, so that if spinal puncture is done early, foramenal hernia will not be likely. If delayed until cerebral edema occurs and the fluid spaces are obliterated, then this danger becomes very real.

With such excellent authorities for each of the methods given above, it is easy to see why there is such confusion in the mind of the average physician as to the correct procedure to follow in any given case. There is a small group of cases, however, regarding which there is little controversy. This group includes extradural hemorrhage, depressed fracture of the skull, and compound comminuted fractures of the skull. It is generally agreed that these cases require operative intervention and the sooner the better. Subdural hemorrhage is also recognized as a surgical lesion but is ordinarily not so urgent in character as the group first mentioned. It usually causes later symptoms.

Early diagnosis and treatment of extradural hemorrhage is admittedly preferred and in a case with typical symptoms would probably not be difficult. However, when the hemorrhage is slow and no localizing symptoms, such as paralysis or convulsions, are present, it is not always easy to determine the diagnosis without carefully observing the patient over a reasonable length of time. Frequent blood pressure, temperature, pulse, and respiration readings should

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be taken and charted, watching carefully for any indication of a break in compensation. The most commonly observed symptom of extradural hemorrhage is that of an interval of consciousness after a head injury, followed by the onset of coma which gradually deepens in extent. A decrease in the pulse rate and an increase in the pulse pressure are indications of increasing pressure, likewise a sudden rise in the temperature curve. A progressive motor loss over the contralateral side of the body and focal or Jacksonian convulsions are diagnostic, although not pathognomonic, if present. When motor weakness develops, the order and character of the paralysis are most significant. It always begins in the face, extends to the arm, and the leg, because the hemorrhage developing in the temporal region from branches of the middle meningeal artery extends mesially and reaches the face center first, the arm center next, and lastly, the leg center. The presence of a slight facial weakness should therefore make one very suspicious of an extradural hemorrhage. Convulsions have the same significance as motor weakness and paralysis and may precede the onset of paralysis. They, likewise, in extradural hemorrhage, begin first in the face, then the arm, and lastly, the leg. If there is any doubt as to which side is involved Dandy recommends making a perforator opening on the right side, and if this reveals no hemorrhage, making another on the opposite side. There is only one treatment for extradural hemorrhage, once it has been successfully diagnosed, and that is evacuation of the clot and ligation of the bleeding vessel.

The following brief case report of a case of extradural hemorrhage which was successfully diagnosed and treated is interesting because of the fact that no paralysis or convulsions were present to assist in localization, although the lucid interval of consciousness was present with a later deepening coma.

Case Report

P. C. J., a 23-year-old, single, white seaman, first class, was admitted on a stretcher on October 14, 1937. He had attempted to change seats while riding in the rear of a moving truck and lost his balance, falling to the pavement which he struck with the back of his head. An x-ray taken by the admitting physician showed a simple linear fracture of the skull about 12 cm. in length extending from the posterior inferior aspect of the right parietal bone through the right temporal bone, apparently ending in the sphenoidal sinus. He was conscious at the time of admission but appeared mentally dull and his reaction time was slow. He complained of severe right-sided headache. There were no neurological abnormalities noted at the time of admission. He vomited several times soon after admission.

Physical examination was negative except for a slight contusion of the scalp in the postparietal region on the right side. The pulse was 38, and there was a marked variability in the force of the individual beats. B. P. 124/90; R. 20; T. 97.4° F. (axillary). The skin felt cold to the hand of the examiner. Lumbar puncture showed bloody spinal fluid under a pressure of 36 mm. hg. and 27 cc.

of this was aspirated, reducing the pressure to 7 mm. hg. The red cell count of the spinal fluid was 43,000 per cmm. He was placed under the usual routine of dehydration with a dry diet and restricted fluid intake and given treatment for shock. He was carefully observed during the night with frequent readings of the B. P., T., P., and R. At 8:00 the following morning it was noted that, although his general condition seemed slightly improved as shown by the above curves, there was an increase in the mental stupor. At this time his B. P. was 112/68; P. 52; and R. 26; ax. T. 99° F. (axillary). Lumbar puncture revealed bloody fluid under pressure of 26 mm. hg. 27 cc. spinal fluid were aspirated, reducing the pressure to 6 mm. hg. The red cell count of the fluid was 36,400 per cmm.

At 10:30 a. m. T. 101.8° F., P. 70, R. 32. Coma was deepening, respiration became stertorous, and he was very restless. At 11:00 a. m. the B. P. was 164/80; axillary T. 102.8° F.; P. 80; and R. 28. The respiration became increasingly stertorous and he developed Cheyne-Stokes type of respiration. The coma increased in depth and his general condition was very poor. The Babinski was positive on both sides, the cremasteric and abdominal reflexes were absent on both sides, the right pupil was dilated, and the knee jerks were hyperactive. The patient appeared to be rapidly going bad and surgical intervention was deemed urgent.

Operation: A subtemporal decompression was decided upon on the right side for the following reasons: (*a*) The right pupil was dilated, (*b*) the X-ray showed the fracture on the right side of the skull, and (*c*) when conscious the patient had complained of a right-sided headache. There were no localizing signs such as paralysis or convulsions present to assist in determining the side of the lesion. At 1:25 p. m. a subtemporal decompression was made under local anesthesia on the right side in the usual manner. An oblique incision of the skin, galea, and aponeurosis of the temporal muscle was made and the fibers of the temporal muscle separated by blunt dissection. The periosteal tissues were stripped back and held by mastoid retractors and a perforator opening made in the skull. On reaching the extradural space a dark clot was seen. The opening was then enlarged by means of Hudson burrs and rongeurs and a very extensive clot was found spreading over the temporal and parietal regions. This was carefully removed and two bleeding points, branches of the middle meningeal artery on the dura, were ligated with silk suture ligatures. The dura showed no tendency to bulge and it was decided that there was no indication for opening the dura and it was not incised. A moderate sized gauze pack was inserted for postoperative hemostasis and the wound was closed. The change in the patient's general condition upon removal of the clot was quite dramatic. He became conscious and asked for a glass of water, his respiration improved, the restlessness diminished, and all symptoms improved. The packing was removed the following morning and there was no evidence of further bleeding. On the 17th (2 days after operation) lumbar puncture revealed clear fluid under 10 mm. hg. pressure. However, a red cell count showed 2,400 per cmm. He was continued on dehydration therapy for 2 weeks and then his fluid intake was gradually increased. He was allowed to sit up 3 weeks after operation and the next day was allowed to get up a few minutes each day. He has made an uneventful convalescence and has had no further symptoms.

Four months after operation (2–16–38) patient was seen and examined. He had just returned from convalescent leave and was symptom free except for a mild headache about 1 week ago. While on leave he traveled from Coco Solo to Iowa and back without difficulty. He considered himself able to do regular duties. A letter from the patient, August 1940, reports that he is in good health

and that he has not been sick since his recovery from the injury. In the fall of 1939 he was examined when discharged and reenlisted and was found to be physically fit for duty.

PACHYMENINGITIS HEMORRHAGICA INTERNA

WITH CASE REPORT

By Lieutenant Earl F. Evans, Medical Corps, United States Navy

Pachymeningitis hemorrhagica interna is defined as a chronic congestion in the internal layers of the dura, associated with repeated small hemorrhages and the organization of a new membrane which tends to extend and thicken. This view as expressed by Jelliffe and White is in contradistinction to that expressed by Cecil, who refers to the condition under the heading of "Subdural hemorrhage," stating that following subdural hemorrhages of traumatic origin, "If this blood is not evacuated it becomes organized, and pachymeningitis interna hemorrhagica with its chronic headache results." This view as expressed by Cecil is in conformity with the writings of earlier investigators such as Morgagni, who described the condition during the eighteenth century. Baillarger in 1854 described the condition as a primary hemorrhage, which following a chronic course was followed by new tissue membrane formation. However, in 1856 Hesche and Virchow first presented the view of an inflammatory nature of the productive membrane, and the consequent hemorrhage due to rich formation of new blood vessels. The majority of the more recent investigators of the condition support the view, that there is a chronic inflammatory process involving the inner surface of the dura with new blood vessel formation prior to the occurrence of the subdural hemorrhages.

As to the frequency of this condition, most authors state that it occurs infrequently. However, Jelliffe and White, quoting from hospital autopsy reports, state that it is by no means infrequent. This difference of opinion may be due to the fact that pachymeningitis hemorrhagica interna, like so many other conditions in medicine, parades under a variety of titles, some of which are: Cerebral and cervical hypertrophic meningitis, hyperplastic fibrinous meningitis, sclerogummatous meningitis, pachymeningitis interna hemorrhagica, hematoma of the dura, chronic subdural hematoma, hemorrhagic pachymeningitis. This condition has been diagnosed in the Pensacola Naval Hospital in four cases since 1919 which would classify the condition certainly as being relatively infrequent in the class of patients treated in the Navy.

Pachymeningitis hemorrhagica interna is usually described as being of two main divisions, the syphilitic and the nonsyphilitic. Of the four cases reviewed one had active syphilis, one had a history of previous luetic infection but with a negative blood and spinal fluid

seriology, and two had no history or seriological evidence of lues. In two of these cases there was no history of head trauma; in the other two a traumatic etiology could not be definitely eliminated.

The nonsyphilitic variety may follow fracture of the skull or even concussion, it may be present in chronic alcoholism, in certain of the dementias especially dementia paralytica and senile dementia; and also in certain of the blood dyscrasias associated with hemorrhages, scurvy, purpura, hemophila, and the leukemias. It should be noted in this connection that alcohol and the dementias afford frequent opportunity for head injuries. Although the condition is usually a disorder of advanced years it may occur at any age and may be found in children.

The clinical course may be asymptomatic or characterized by vague general cerebral symptoms or obscured by the concurrent disease. The onset however, may be sudden and dramatic as was the case in two of our patients. However, this statement is not altogether accurate as in one of these two cases we were later, on close questioning, able to obtain a history of headache, vague malaise, and weakness for some months prior to the dramatic episode of unconsciousness and in another a history of prior complaint was not obtained as the patient died without regaining consciousness. In traumatic cases the onset may be rapid but as a rule it is gradual, following in several hours to several months after the injury. Apoplectic forms may simulate cerebral thrombosis or hemorrhage. However, it can usually be differentiated by the absence of localizing signs such as motor tract disturbance or cranial nerve involvement and the prominent evidence of meningeal irritation. Until prolonged observation and time-consuming laboratory procedures have been obtained it may be difficult to differentiate from tuberculous meningitis, brain tumor, and brain abscess.

The early signs may be those of cerebral irritation, such as headache, vomiting, delirium, unilateral convulsions, or even apoplectiform attacks.

There may be a complete change of personality. Later the condition may progress to stupor and coma. The headache is usually of gradually increasing discomfort until it becomes a severe cephalgia. There may be persisting stereotype muscular movements, chewing, automatic arm, hand or leg movements indicative of a local irritative lesion of the motor area. The constant putting of the hands to the head by the comatous or stuporous patient has been frequently mentioned as an important sign. It is a frequent sign in paresis. There may be rigidity of the neck with retraction of the head and a positive Kernig's sign. A transient positive Babinski reaction is an important sign and one which occurred in our last case where the Babinski reaction would be positive one day and negative a day or two later

only to become positive again in a day or two. As in any meningeal hemorrhage local percussion tenderness may be especially marked. Involvement of the eye muscles or basal cranial nerves is rare. Evidence of increased intracranial pressure may be seen in the fundi, but it is of a mild or moderate degree. Pupillary irregularities may be present, but except in those cases which are a complication of a cerebrospinal luetic infection the criteria of a true Argyll-Robertson pupil are not found. Temperature changes are inconstant but some irregular temperature elevation usually occurs.

There are no characteristic blood changes. However, the spinal fluid gives important data. There may be increased spinal fluid pressure.

The fluid is frankly bloody, yellow, or of a true xanthrochromic character. The Kahn reaction is of no value as lues may be a frequent concomitant condition.

Most recent writers describe the pathology as being that of a productive inflammation causing the formation of a thin yellowish-brown membrane on the inner surface of the dura. This process advances with the formation of new blood vessels, then their walls give way with resulting hemorrhage formation. Proliferation of connective tissue with perivascular lymphocytic infiltration progresses until the enormous hyperplasia of the dura may extend to involve the arachnoid, pia, and invade or displace the underlying cerebral tissue. Lesions of a gummatous character may be produced. The base of the brain is rarely affected, the pathological changes being usually located over the parietal, temporal, and frontal lobes. The process may extend to involve the spinal cord. In the variety due to syphilis the process may be confined to the cervical region of the spinal cord. In the brain the surface of the hematoma toward the brain is thin and covered with a mesothelial membrane. The dural side is thick and the organized granulation tissue contains large spaces lined with mesothelium containing large blood spaces and fibrin. These spaces anastomose freely with each other and with the capillaries.

The prognosis in these cases must be guarded as the condition may become chronic with remissions and exacerbations. Some patients may recover completely. Usually however, the disorder is progressive, death occurring early or after a long psychotic period of irregular excitement or depression.

The therapy of pachymeningitis hemorrhagica interna may be either surgical or palliative with frequent spinal taps. Brain puncture with removal of the clots has been of service. Stimulation of elimination is useful. Analgesics and hypnotics may be necessary to control the pain or the excitement. Appropriate treatment of any concomitant condition should, of course, be instituted.

Case Report

A. C. K. (VAP), aged 38 years, was admitted to the Naval Hospital, Pensacola, Fla., on September 11, 1937, in a semiconscious condition, complaining of severe headache and soreness of the muscles of the neck. Two days previous to admission patient was found lying unconscious on the floor of an outhouse by his wife who states the patient regained consciousness of a few minutes after she found him, but soon lapsed back into his present stuporous condition. Patient gave a history of having struck his head on an overhanging beam about 3 weeks before onset. There was no unconsciousness immediately following injury; merely headache for a few hours. He had a history of headaches for the past several months. There was no history of lues.

Physical examination.—Temperature 98.8° F. on admission; later ranging from 101°–102.6° F. Pulse 40 on admission, later ranging from 110–120. Patient was mentally confused, stuporous, and answered questions incoherently. Pupils were equal and contracted. Fundi were normal except for slight edema of the medial side of each disk. There was no facial or cranial nerve involvement or evident paresis or paralysis of extremities. Patient had stiffness of neck, retraction of head and Kernig's, Brudzinski, and Babinski signs were positive bilaterally. Tendon reflexes were hyperactive, but equal bilaterally. Abdominal and cremasteric reflexes were present. Spinal puncture revealed a grossly bloody fluid, under slightly increased pressure, smears and cultures negative for organisms, globulin increased, Kahn negative, L. C. G. 0000000000. Daily spinal fluids revealed similar findings. Blood: W. B. C. 19,750, R. B. C. 4,700,000, polys 85, lymphs 14, monos 1, Kahn negative.

Clinical course.—Patient was extremely restless except for a few hours following each spinal tap and required morphine for relief of headache and restlessness. Repeated blood and spinal fluid cultures were negative for organisms. During the following 10 days patient exhibited a transient Babinski reflex which would be positive on one day and on the following 1 or 2 days would be negative, only to return in a day or two. Automatic hand and arm movements persisted especially constantly putting the hands to his head. Temperature gradually rose to 103°–103.6° F. Blood in the spinal fluid diminished after 5 to 6 days until a xanthochromic fluid with only a small number of undissolved red blood cells was obtained during the last few days. On admission the urine was normal but for 2 days before death albumin and numerous hyalin and granular casts appeared. Patient became progressively worse, lapsing into coma, with increasing nitrogen retention in the blood. Death intervened on the twelfth day after admission. Autopsy was performed and the following pathological changes were found:

Autopsy report.—The dura shows evidence of thickening, inflammation, and the presence of several subdural hemorrhages over the parietal, temporal, and frontal lobes on both sides. There is marked congestion of the superficial cerebral vessels. On incision of the dura there is evident roughening and thickening of the inner surface due to an acute inflammation, hemorrhage, and new blood vessel formation. This process of inflammation and hemorrhage extends inwardly to involve the arachnoid and pia, being especially marked over the right temporal and frontal lobes. The brain stem was divided and the brain removed. The basal regions reveal no evident pathology, except for the presence of bloody yellow fluid in the subarachnoid space. On incision of the right frontal lobe through an overlying area of subdural hemorrhage, a large blood clot in an early stage of organization is found occupying approximately three-fourths of the right frontal lobe; being continuous with the overlying subdural hemorrhage and extending into the anterior portion of the lateral ventricle where a small amount of bloody

yellow fluid is found. On removal of this large clot, the remaining cavity in the brain shows practically no evidence of a limiting wall formation.

SUMMARY

A brief résumé of the history, clinical course, physical and laboratory findings, pathology, and treatment of pachymeningitis hemorrhagica interna has been recorded. A review of the records of the Pensacola Naval Hospital revealed that the diagnosis of pachymeningitis hemorrhagica interna has been made in four cases since 1919. A recent case of this disease with autopsy findings is presented.

In this small series of cases the mortality was 50 percent; two cases recovering without residuals incapacitating them for duty, and two cases terminating in death. The author realizes that nothing new or original has been herein presented and that no conclusions can be drawn from such a small series of cases. However, this presentation is made for the interest it might stimulate in regard to the relative difficulty in diagnosis of this condition and the necessity for a guarded prognosis in these cases.

SPONTANEOUS SUBARACHNOID HEMORRHAGE

WITH CASE REPORTS

By Commander H. D. Hubbard, Medical Corps, United States Navy

Believing that patients presenting this type of cerebral accident are not frequently seen or, rather, not readily recognized as such, two cases admitted to the U. S. Naval Hospital, San Diego, Calif., are submitted.

The clinical picture of the sudden rupture of a small cerebral aneurysm, with subsequent hemorrhage within the subarachnoid space, is one that when once seen and recognized can thereafter readily be diagnosed.

The various etiological factors that at one time or another have been thought responsible for this condition are syphilis, chronic arterial hypertension, and congenital aneurysms in or near the circle of Willis. Most observers believe that congenital defect is the most important predisposing cause. The aneurysm may be present at birth or may develop after birth as the result of a congenital defect in the media of the vessel involved.

Whatever the causative agent, the symptoms are so characteristic that a diagnosis can almost certainly be made from them alone. There may be prodromal symptoms of apprehension, irritability, dizziness, fainting attacks, blurring of vision, muscular twitchings, and headaches, but these are not constant.

The patient generally gives a history of some sudden exertion, followed immediately by what he describes as a "snap in the head" and by nausea and vomiting. A severe headache develops, more

pronounced in the occipital region, and pain occurs in the back of the neck. Rigidity of the muscles of the neck develops with the accompanying signs of meningeal irritation such as a positive Kernig and Brudzinski. The temperature, pulse, and respirations are increased and leukocytosis develops. Spinal puncture reveals gross blood thoroughly mixed with the spinal fluid and when this fluid is allowed to stand and the cells to settle, xanthochromia is noted. The number of white blood cells in the spinal fluid is considerably increased, the increase depending upon the amount of hemorrhage and meningeal irritation. More serious symptoms may develop depending upon the amount of hemorrhage. These may vary from a state of mental confusion to that of a prolonged coma or sudden death. If the hemorrhage is so extensive as to cause compression of the medulla there will be a disturbance of pulse, blood pressure, respiration, etc. There may be various signs of paralysis indicating definite cerebral damage. Papiolledema may or may not develop. Tendon reflexes will be diminished and sometimes lost.

After the diagnosis has once been made the prognosis should be guarded. If there is recovery from one attack there may be a recurrence with death in a subsequent attack. Opinion differs as to the value of repeated lumbar punctures in the treatment of this condition. In these cases the treatment consisted only of absolute rest in bed, under careful observation. for a period of 8 weeks.

Case Reports

Case 1. N. D.—White male, age 32, of the U. S. Coast Guard. Admitted September 28, 1937, with diagnosis undetermined (myositis acute, neck muscles).

Chief complaints.—Severe headache, pain in the occipital region, pain in the posterior cervical region radiating to the mid dorsal spine, and inability to move the head in any direction without severe pain.

Family history.—Has no bearing. He has lost track of his father. Mother, wife, and two children are living and well.

Habits.—Normal. He has been 10 years in the U. S. Coast Guard service. He uses tobacco moderately, alcohol sparingly, and drugs not at all. He drinks one cup of coffee daily.

Past history.—Usual childhood diseases. Pneumonia. Dislocation of right shoulder. Specific urethritis. Lues is denied. No operations.

History of present complaint.—Six hours prior to admission the patient was firing a service rifle in the prone position on the rifle range, when a sudden severe "snaplike" pain occurred in his head. He retired from the firing position, became nauseated, and vomited. Pain then developed in the back of the neck radiating down the dorsal spine but was more severe in the nape of the neck. Marked generalized headache developed. These pains occurred almost immediately following what he described as "something snapped in my head."

Physical examination.—Examination revealed a patient wearing an expression of severe pain. His face was flushed and he held his head rigid to prevent motion of the neck. The eyes reacted normally and there were no abnormal findings in the eye grounds. There was no ringing in the ears. There was marked tenderness over the occipital protuberance.

The pulse was full and regular, 120 beats per minute. The blood pressure was 130/80. The temperature was 100.2° and the respirations were 24. The extremities were normal except for an old deformity of the right shoulder. The deep reflexes were diminished but equal. There were dissociated areas of sensation about the shoulders and upper part of the chest, the left side being more sensitive than the right. There was some hyperesthesia of the soles of the feet but there were no pathological reflexes.

The stereoscopic x-ray of the skull revealed no evidence of bone injury or pathology. The calvarium showed a normal thickness with the sutures closed. The convolutions and vessel markings were normal. The sella turcica and clinoids were normal. All sinuses were clear. The cervical and thoracic spines showed no pathology.

The blood Kahn was negative and the urine showed no albumen or sugar. Lumbar puncture revealed a spinal fluid so thoroughly mixed with blood that it had lost all resemblance to spinal fluid and appeared as if coming from a large vein. It seemed to flow under no increase in pressure although no manometric reading was taken. The fluid was reported to contain many red blood cells but only an occasional white blood cell. No organisms were found and the culture was negative.

The blood picture on the first day showed 4,790,000 red blood cells and 12,700 white blood cells with 77 percent neutrophiles. The hemoglobin was 90 percent.

Twenty-four hours following the first lumbar puncture another spinal tap was done but there appeared to be no change in the appearance of the spinal fluid. The WBC. count was 8,800 with 75 percent neutrophiles. The headache was not as severe and the temperature, pulse, and respiration were only slightly elevated. The blood pressure was 130/90.

Eleven days after admission the temperature, pulse, and respiration became normal. The patient made an uneventful recovery with no signs of residuals.

Case 2. G. B.—White male, age 43. Pensioner. Admitted October 5, 1937, with diagnosis undetermined (intracranial hemorrhage).

CHIEF COMPLAINTS.—Pain in the head, more marked in the occipital region, with nausea and occasional vomiting.

FAMILY HISTORY.—Has no bearing; father died of nephritis at 38 years. Mother died of carcinoma of the stomach at 48 years. Three sisters are living and well.

HABITS.—Normal. Tobacco, alcohol, and other stimulants used moderately. Drugs not at all.

PAST HISTORY.—He was a pugilist for 6 years. There were the usual childhood diseases. Influenza in 1918. Appendectomy in 1920. The nose had been broken several times. The right eye was enucleated in 1927 because of glaucoma. Venereal disease denied.

HISTORY OF PRESENT COMPLAINT.—For the past 2 years he had noticed a rhythmical contraction of the leg muscles while at rest (Parkinsonian syndrome). During the past year there developed a blurring of vision, dizziness, and headache with an apprehension of impending illness.

Three days prior to admission, while shopping, there suddenly developed a severe pain in the back of the neck which radiated to the head. Following this pain he lost consciousness and did not recover for a period of 24 hours. While unconscious he is said to have vomited frequently.

PHYSICAL EXAMINATION.—Upon admission he did not appear ill. The essential findings were as follows:

Pain was elicited on anterior flexion of the neck.

The left eye showed no abnormality.

The left nostril was obstructed due to an old injury to the nose. The uvula was deviated to the right. There was a decreased patellar reflex on the left side

and an atypical bilateral Kernig and Brudzinski. The other reflexes were normal.

The spinal fluid was under mild pressure and so mixed with blood that it had lost the appearance of spinal fluid. It contained no organisms and showed no growth in culture media.

The blood and spinal fluid Kahn were negative. The urine was negative. The blood pressure was 110/90. The temperature, pulse and respirations were normal.

The red blood count was 4,640,000 with 90 percent hemoglobin and the white blood count was 13,000.

The stereoscopic examination of the skull showed the calvarium to be of average thickness with the sutures closed. The vessel and convolution markings were normal. The anterior clinoids were somewhat depressed and there was calcification in the region of the pineal body. All sinuses and the mastoid areas were clear. There was no evidence of bone injury or pathology.

While these cases differ somewhat in their history and onset the diagnosis was established upon the appearance of the spinal fluid and the lack of evidence pointing to either an accident causing a skull or brain injury, arterial degenerative disease, or a bleeding brain tumor.

It is felt that, if the signs and symptons of spontaneous subarachnoid hemorrhage are kept in mind in differentiating other allied conditions, these cases will not be overlooked.

A HEAD INJURY SURVEY

I. ETIOLOGY AND PREVENTION

By Lieutenant Commander Hans v. Briesen, Medical Corps, United States Naval Reserve [1]

The case records of patients admitted to the Los Angeles County Hospital with a head-injury problem during the 6 years from July 1928 to July 1934 have been reviewed. There were 5,912 such patients. Information was collected about age, sex, manner of injury, objective and subjective evidence, roentgen ray evidence, clinical evidence, diagnostic procedures, diagnosis, treatment, complications, and results. The following paper contains the thoughts regarding cause and prevention suggested by this work, and is especially concerned with the problem of automobile accidents.

ETIOLOGY

To work out adequately the etiology of an injury a number of interlacing and coordinating factors must be considered. Without attempting to evaluate their importance they may be stated as follows:

1. Personality of the injured, including age, sex, occupation, activity, and mental state at time of accident.

[1] Neurosurgical Staff, Los Angeles Orthopaedic Hospital; Neurosurgical Staff, Good Hope Hospital Association Clinic, Los Angeles, Calif.

2. The place at which the accident occurred, including its approaches, visibility, and topography; and the time of day.

3. The manner of injury.

It is unfortunate that in this survey age and manner of injury are the only factors that can be used. Figure 3 illustrates their relationship.

EXPLANATION OF FIGURE 3.—The height of the column in each instance represents the number of cases in that age. Each coordinate in the horizontal line represents 1 year. The coordinates that are left unfilled and which show themselves as indentations from the base line are the number of each age which were recorded in the charts as known acute alcoholics. Where there is only one case of an age and that case is an alcoholic, the coordinate square has been left hollow. The hollow coordinate square or rectangle on the top of the columns in sections E, G, and O, of the chart represent cases into which other factors have entered; for example, the majority of the cases so designated in E were due to epilepsy. However, one of them, the top of age 19 column, was a fall caused by hunger; and another, the top of the age 30 column, was a fall caused by being stabbed. In G, the two hollow squares at the top of the column represent known suicide attempts. The hollow square at the top of O column of airplane accidents represents a glider injury. The small letters throughout the "hit by" and "miscellaneous" injuries are keys to type of injuries listed below.

For the purpose of general discussion as to etiology only a few of the miscellaneous injuries will be considered. The large masses of cases, however, will be discussed more thoroughly. An attempt was made to discover occupation and to find out if there was any connection between the type of occupation and injury. However, this was rather hopeless and as regards occupation only one inconclusive remark can be made, and it must be regarded as a personal opinion. It was noted that one of the auto-pedestrian victims in the middle twenties was an artist, and the question arose whether or not preoccupation of this type of mind might not have been an etiological factor.

A. ALL AUTOMOBILE ACCIDENTS.—3,604 cases—61 percent.

A–1. *Automobile collisions.*—1,966 cases—54.5 percent of A; 33.3 percent of all. It is noted that the great mass of cases are from the age of 14 to the age of 40. In this great mass of cases we have our highest percentage of alcoholics.

A–2. *Auto-pedestrian accidents.*—1,581 cases—43.9 percent of A; 26.79 percent of all. It is noted in auto-pedestrian that at the age of 6 we find our highest number of cases, that is, 64 cases, and then there are not so many until one comes again to a high column at the age of 40 and 50. It is also noticed that the great mass of cases in this chart are below the age of 14 and above the age of 43. It is also noticed that the alcoholic columns rise higher in the middle age group, the youngest alcoholic being a single case in the 18-year-old group. The highest percentage in the group is at the age of 41.

A–3. *Auto-bicycle accidents.*—It is noted that the mass comes between the age of 11 and the age of 15.

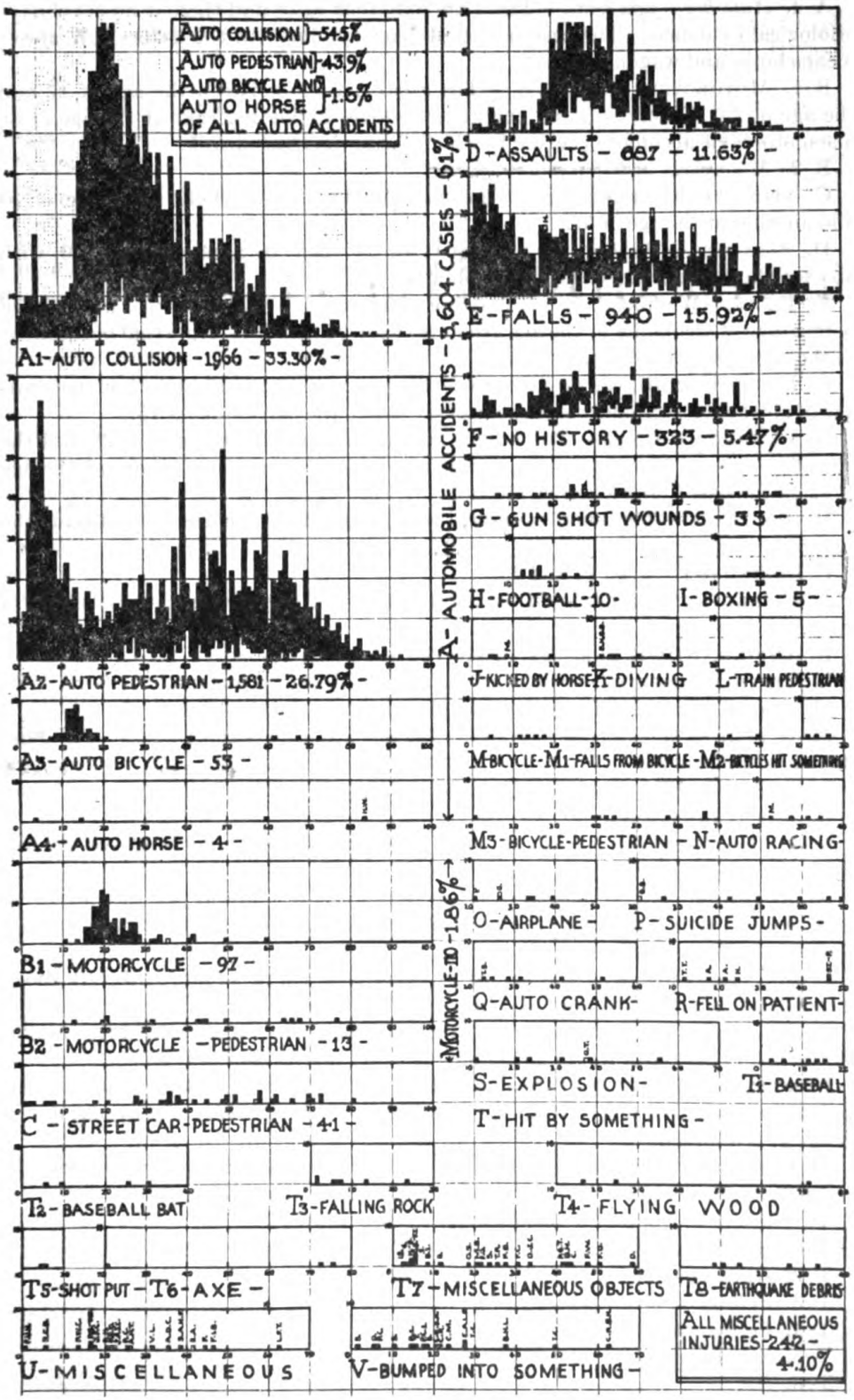

FIGURE 3.—Classified charts illustrating head injury survey.

A–4. *Auto-horse accidents.*—There are only four cases and there is no conclusive etiological evidence. The case in the 84-year column with the letters HW above means horse and wagon.

B–1. MOTORCYCLE ACCIDENTS.—The great mass is between the age of 17 and the age of 23. There is noted one hollow square indicating that at the age of 36 one motorcycle accident occurred in which the patient was drunk.

B–2. *Motorcycle-pedestrian accidents.*

C. STREET CAR-PEDESTRIAN ACCIDENTS.—We have an alcoholic at the age of 40 and another at the age of 57.

D. ASSAULTS.—We find the mass of cases between the ages of 20 and 40, with an exceedingly high percentage of alcoholics.

E. FALLS.—We find the great massing of cases below the age of 16, with a high percentage of alcoholics in the age group between 20 and 70. Note that one patient at the age of 81 was drunk.

F. PATIENTS WHO GAVE NO HISTORY.—There is noted again a rather high percentage of alcoholics, especially at the age of 40, where the percentage is 75.

G. GUNSHOT WOUNDS.—The hollow square labeled S at the age of 28 and the one at the age of 50 were known suicide attempts. The hollow square at the age of 72 on the base line is a known alcoholic.

H. FOOTBALL.—The small number of injuries mass themselves between the ages of 10 and 20.

I. BOXING.—Between 17 and 25.

J. KICKED BY A HORSE.—M at the age of 9 represents one case kicked by a mule.

K. DIVING. SHOB, at age 12, means "struck head on board."

L. TRAIN-PEDESTRIAN.—Shows no particular etiological factors.

M. BICYCLE INJURIES is a group excluding auto-bicycle injuries.

M–1. *Falls from bicycle.*—This shows the age group between 1 and 20, although there is one at age 70.

M–2. *Bicycle hit something.*—These have not been labeled but they are: a bump in the street, a tree, a fence.

M–3. *Bicycle-pedestrian.*—Again show the massing of accidents above the age of 40.

N. AUTOMOBILE RACING.—M over age 23 represents a motorcycle race accident.

O. AIRPLANE ACCIDENTS.—P over age 21 represents one case hit by a propeller. G over age 27 represents a glider injury.

P. SUICIDE JUMPS.—At the age of 21 there is a hollow square with OB above it representing a drunken man who jumped off a bridge.

Q. AUTOMOBILE CRANK INJURIES.—TB at age 23 means "thrown by auto crank." In the rest of the cases we do not know the exact mechanism of injury.

R. FELL ON PATIENT.—At the age of 12 TT represents truck tire; A at 18 years and 22 years, automobiles; H at age 25 a horse; ST at age 47, a steel tank; and P at age 47 is a piano.

S. EXPLOSION.—At the age of 38 there is a hollow square indicating an alcoholic, and GT, which should be over 39, represents a gas tank explosion.

T. The entire group of T means HIT BY SOMETHING.

T–1. *Baseball.*

T–2. *Baseball bat.*

T–3. *Falling rocks.*

T–4. *Flying wood.*

T–5. *Shot put.*

T–6. *Ax.*

T–7. *Miscellaneous objects.*—In T–7 symbols mean as follows: IB, Iron bedstead; G, glassware; H, hammer; B, board; GB, golf ball; FT, falling tree; S, swing;

B–1, bucket; OS, object from shelf; MB, milk bottle; FO, falling object; S–1, swing; TR, tire rim; FB, falling board; FC, falling case; DJC, door of jail cell; PGT, power grinder tool; BH, bale of hay; C, chisel; FW, falling wrench; D, door.

T–8: *Earthquake debris.*

U. MISCELLANEOUS INJURIES.—In miscellaneous injuries symbols are as follows: FUH, found unconscious in front of home; RCB, riding car bumper; PWC, playing with catapult; PB, playing basketball; WF, wrestling—5 foot fall; JFC, jumped from car; RH, roughhousing; PBO, park bench overturned; JFJC, jumped from jail car; RC, roller-coaster accident; AHT, attempt to hop truck; VL, under landslide; AOC, auto over cliff; SAHF, suicide attempt by hanging—fell; EA, elevator accident; F, flood; FIB, fell in bathtub; LFT, leaped in front of train.

V. BUMPED INTO SOMETHING.

There are some errors in the symbols on this chart. B at the age of 2 bumped into a bedstead; D at the age of 6, into a door; S, 11, into a shelf; B, 14, a beam; T, 15, a tree. At the age of 18 and 19 there is a personal collision playing baseball. CRBH at the age of 21, bumped head on car rail, is an erorr and this is simply a bumped head, nothing else being known about the case etiologically, and the same applies to age 63. However, the CR at the age of 22 is a bumped head on a car rail. CM at age 24 is a cement mixer, and EALP at age 28 means "evading an auto, bumped head on lamp post." BLH at the age of 38, is a simple bump. TC, at age 28, is top of car, as is also the other TC at age 50. All miscellaneous injuries sum up to 242 cases, which are 4.10 percent of all.

The etiological factors involved in the injuries are almost self-evident. With the possible exception of "being hit by earthquake debris," shown on chart T–8, it seems hardly questionable that most of these accidents might have been avoided. It is granted that they are the natural risk that people take who ride bicycles, move pianos, work under automobiles, and play vigorous games. However, to go back to the larger masses of cases it is seen in automobile accidents that the general trend follows very closely the development of the personality and its disintegration. It is at the age of 6 that a child first knows what he himself wishes to do, and feels confident to disregard the advice of his guardians. It is again at the age of 40 that one begins to be slightly inattentive, and between the ages of 14 and 40, as shown in chart A–1, one is vigorous and active. At this point the similarity between automobile collisions, chart A–1, and assaults, chart D; and the similarity between auto-pedestrian, chart A–2, and falls, chart E, should be recognized.

Although these charts prove nothing they do show a very definite trend. The masses of cases in auto-pedestrians and in falls show the inattention of immaturity and the inattention of old age, and the masses of cases of automobile collisions and assaults show the inattention of vitality. It is granted that in chart A–1 the 93-year-old patient who suffered a head injury in an automobile collision may have been victimized. However, in the case of the 93-year-old patient in A–2, auto-pedestrian, personality was a partial factor. It is also granted that a number of patients in the entire group have been victimized. However, a thoughtful consideration of these facts

indicates that irrespective of locality of the accident or the time, there is always an element of carelessness in any accident that occurs. Other factors being equal, if we can eliminate the carelessness of immaturity, the carelessness of old age, the carelessness of vitality, the carelessness of alcoholism, it is conceivable that we can reduce the number of accidents by a tremendous percentage.

It seems necessary to make a complicated chart such as this to convince people that automobile accidents are due to carelessness. The purpose of such a chart is to attempt to drive this fact home as a logical conclusion to people who look upon accidents as the result of several uncontrollable, coincidental factors. There have not been many accidents in which the carelessness of some individual has not played a part and without which carelessness the accident might have been avoided. A collision or auto-pedestrian injury usually results from the carelessness of more than one person involved, but the elimination of such carelessness on the part of one individual is enough to prevent the accident.

PREVENTION

Starting with the chart and taking into consideration the injuries, it is evident that in chart E, the boy of 19 who fainted from hunger and hit his head is a case which illustrates that sociological factors must be marshalled to prevent such a type of injury. In chart G, gunshot wounds, sociological factors are probably responsible for the majority. In the miscellaneous injuries the case of the 1½-year-old child who was found unconscious in front of his home brings up the question of prevention that must be handled by sociologists and politicians. This is essentially so for falls, chart E, in the age group from 1 to 7. These latter cases suggest the establishment of children's care clinics in neighborhoods where mothers are so busy that they cannot keep their eyes upon their infants constantly. These problems are complex and will probably not be thoroughly worked out for many years. However, the problem of the automobile can definitely be worked upon.

One hundred years ago a death by shooting was accepted more readily than it is today. Justice, so-called, was administered without much regard to possible contributing factors. Today when a man is shot the entire situation is studied from the standpoint of personality, cause, punishment, and prevention by a staff of trained investigators. We treat our automobile accidents today as we treated shooting then. Very shortly, accidental deaths, let us call them *murders by fate*, will be investigated as thoroughly as murders by man are now, for from a cosmic viewpoint a death of a member of the human species is just as important when accidentally caused as when planned and carried out by man himself. The reason we thoroughly investigate murder by man is so that we can trace down the culprit and prevent its

recurrence. For this same reason a thorough investigation of all the above-mentioned etiological factors must be done in all accidents. A step is already being taken by our police department. Only a short time ago when an automobile collision took place and no personal injury occurred the case was one for the civil-law courts. At the present time the police department is investigating all such accidents and if by tire markings on the street or any other clue, they discover that the accident has been caused by a law violation, the case is brought into the criminal courts.

Traffic laws, devices, and propaganda are constantly before the eyes of the public. Legislators, sociologists, and safety societies are closing in on the problem and doing excellent work. The number of injuries from accidents is definitely, if slowly, decreasing. Excellent roads and efficient signal systems also facilitate traffic, prevent delay, and discourage haste, and each single item in the system has been and is valuable. There are, however, in addition two psychological factors that this work wishes to foster. They have to do with the raising of the driving-public morale to the extent that the etiological factor of carelessness will be reduced to a minimum.

The question as to whether or not public morale can be raised can be answered by the fact that we no longer solve our individual differences of opinion with firearms as we did a very few decades ago. Individual morale sometimes suffers, but public morale advances with civilization and its requirements, and this advance in morale will be definitely seen in the driving public within a very few years.

Probably the best example of excellent group-driving morale is shown by the picnics of one of the major oil companies. In 1937 this company had a picnic at Santa Cruz. The roads through the mountains to this community are definitely hazardous. Some time before the picnic the company carried out a campaign among its employees with posters emphasizing the fact that upon this picnic those attending should be careful not to have an accident. There were not less than five thousand automobiles at this picnic. There were no accidents! The following year this same company had a picnic at the Santa Anita race track on a Saturday afternoon when interurban traffic is always heavy. Large signs were erected warning employees to be careful. Again there were no accidents!

It is granted that the general morale of this company's employees is higher than that of the average automobile driver. With these people special safety campaigns are hardly necessary. They are drilled from the moment of their employment to become safety conscious and for 10 years at least this company has had a safety department, with a safety engineer in charge, whose duty it is to foresee hazards and eliminate them. As an example, in their buildings there are signs in front of the elevators with instructions as to

how to get in and out of an elevator. In short, here is a group which has done more or less what must be done for society in general.

The methods here advocated can be classified under personal fear and personal honor. These suggestions are in no way offered as a displacement of existing laws and rules, but are offered as an additional requirement of a psychological nature which should call to the attention of a great number of people who never considered driving as a particular hazard that the danger from driving is as great as that of illness, not only to the person driving but also to other individuals in the community; and because of this, driving is a responsibility as well as a privilege.

The accident problem has been approached in every conceivable manner by all interested groups. My experience has convinced me that it is a national one. It has attained the magnitude of warfare, and the majority of accidents are unnecessary and preventable.

In attempting to determine of what people are afraid the following questions have been asked many drivers. The first is: Do you admit that you might have an automobile accident? Most drivers will admit that this is within the realm of possibility but is exceedingly remote. Then: Do you think you might have appendicitis, typhoid fever, tuberculosis, stomach trouble? It has been definitely found that the common illnesses are much more closely felt as a menace by the general public than the possibility of an accident. Almost anyone will admit that if a gun is pointed directly at him he will be alarmed. Our propaganda must closely compare automobile accidents with illnesses to which people do admit they may be subject, and automobiles must be compared with lethal weapons of which people are universally afraid, and of which they are exceedingly careful. People also fear disgrace and the time must come when an automobile accident is not only unfortunate to the individual, but also disgraceful.

TABLE 1.—*Death rate table* [1]

Cause of death	Number	Percentage liability	Cause of death	Number	Percentage liability
Tuberculosis	70,080	200.6	Mastoiditis	4,112	12.1
Lobar pneumonia	57,658	169.25	Anemia	3,481	10.025
Bronchopneumonia	42,621	125.25	Typhoid fever	3,442	10.01
AUTOMOBILE ACCIDENTS	34,183	100	Alcoholism	3,349	9.82
Influenza	28,230	83	Meningitis	2,763	8.12
Appendicitis	16,142	47.45	Scarlet fever	2,718	7.96
Ulcer of stomach and duodenum	8,430	24.78	Acute poliomyelitis and polioencephalitis	1,040	3.6

[1] From Mortality Statistics for 1935, published by Bureau of the Census, U. S. Dept. of Commerce.

Table 1, taken from the mortality statistics of 1935, published by the United States Department of Commerce, Bureau of Census, illustrates the death rates for a few of the more common illnesses with which people are familiar. It will be seen that tuberculosis, broncho-

pneumonia, and lobar pneumonia are more dangerous from the standpoint of death than automobiles. However, it is felt that if all of the age groups above that of the active automobile drivers were eliminated, the chance of death from an automobile accident would probably be equal to the chance of death from any of these other causes. With all the other illnesses mentioned the chances of death are less than the chances of death from automobile accidents. Where there is a 100-percent chance of death from an automobile accident, there is only an 83-percent chance from influenza; a 47-percent chance from appendicitis; a 24-percent chance from ulcer of the stomach, etc. In other words, the danger from automobiles to the public in general is higher than from influenza, is twice as great as that of appendicitis, 4 times as great as that of stomach trouble, 10 times as great as that of mastoiditis, anemia, typhoid fever, alcoholism, meningitis, and scarlet fever, and is 30 times as great as that of infantile paralysis. It would be extremely interesting to work out the disability statistics on these same groups, the probability being that partial and permanent disabilities from automobile accidents would outweigh all of the others in a startling manner.

There occur approximately 35,000 automobile deaths yearly in the United States. This, with the great number of disabilities in those who do not die, constitutes a tremendous expense to the individual, to the community, and to the Government.

The educational side of accident-prevention propaganda has been left as a matter of volition to the licensees. If they wish to pay no attention to it there is no requirement that they do so, yet anyone who will read the pamphlets, *A Trio of Warnings* and *Sudden Death and How to Avoid It* will realize instantly the psychological change that takes place in regard to the driving of an automobile. These pamphlets deal with explanations of speeds and braking efficiency, with hazards of driving, and contain some gruesome stories of automobile accidents which are very effective. If every driver would read both of these pamphlets it alone would decrease the number of automobile accidents.

From earliest understanding people must be taught that automobiles are lethal machines. They must be treated like loaded guns. Among people accustomed to handling guns the pointing of an empty weapon at a person is truly a moral sin and infinitely more dangerous than pointing a gun that is known to be loaded, for the simple reason that it is the gun that is considered harmless that is handled with carelessness and causes a death. Automobiles are comparable to loaded guns. To one who will not take this fact seriously the privilege of driving should be denied.

One of the most difficult things to get anyone to do is to make a promise. The reason is that a promise, even to very young children,

means that at least an attempt must be made to keep it. There is considerable difference of opinion as to what percentage of people are honorable. A large percentage will take a personal promise seriously, regardless of how dishonorable they may otherwise be. Therefore it is recommended that all drivers' honor be reached by the taking of an oath upon their application for, or renewal of, a license. Such an oath should be so worded and of proper length to impress upon every driver who takes it both the privilege and the responsibility that driving entails.

OATH

I do hereby solemnly swear that in receiving a license to operate a motor vehicle I am conscious of the obligations involved; that careless or reckless driving on my part may result in damage and destruction of property, but more important, in disability or loss of life to myself or others. In consideration of the granting of this license I promise to drive in accordance with the laws of the States over whose roads I may travel; to avoid recklessness or carelessness that may in any way make accidents more likely; to use good sense and judgment in the use of intoxicants when I am the responsible person in the transference of a motor vehicle from one place to another. In short, inasmuch as my life, and the life of others, may be in jeopardy should I not do so, I swear that in accepting a license to drive I do so recognizing that it CONSTITUTES A SOLEMN AND SACRED TRUST GRANTED BY THE PEOPLE, to uphold which I will do all in my power.

Good citizens and comparatively safe drivers do not want to take an oath any more than they want to take a pledge to stop drinking. They do not feel it is necessary for them.

It is here proposed that after passing the required test and proving the necessary skill and knowledge, every driver be required to read the pamphlets *A Trio of Warnings* and *Sudden Death and How to Avoid It*, and, after he has read and understood these, required to stand erect and sincere, and make an oath—not sign it—stand up and say it seriously. If he will not read and digest these pamphlets, if he will not take an oath, if he has a twinkle in his eye or a smirk on his face while taking it, he should be sent away to return when he is more serious. If people object, wondering why they should be forced to go through such a fuss to receive a driver's license, they can be shown photographs of accidents or escorted through the accident ward of any hospital. Morgues are also educational!

After these routines have been completed the applicant is given a license upon which the oath is printed and which he must sign. Arrest and punishment under our present system is a matter of expense and inconvenience. Under a system in which these psychological factors are taken into consideration it will also become a matter of shame.

The psychological approach, with the impressing upon automobile drivers that the privilege of driving is also a responsibility, must be included in addition to the traffic rules and regulations now in effect. Until all drivers understand the dangers and responsibilities by com-

pulsion and not by volition, accidents will continue in spite of laws and regulations. The component of lack of moral responsibility has a large part to play with the number of accidents. For instance, a man who is in a hurry for the time being loses some moral responsibility. Likewise, vitality without well-developed moral responsibility becomes a factor in accidents. It is the meeting of a careless, vigorous personality with an incompetent one that causes the accident and if such facts are forced into the understanding of automobile drivers there should be fewer accidents.

The cost of accidents and their consequent disability is infinitely greater than the cost of administration of such additional requirements as these, or of propaganda to establish such requirements. Safety propaganda alone is unsatisfactory because at present it has a "take it or leave it" quality. The automobile accident problem has become so vast that the Nation can no longer depend upon the willingness of individuals to accept propaganda. In a situation that takes a yearly toll of 35,000 lives it is necessary to force people to realize the danger.

Opposition will come from people who object to being personally disturbed by additional regulations and who may be essentially safe drivers themselves, and people who do not believe that public morale can be changed.

The period can be looked for when we, with a large driving public, will not have a single injury. Although this is an optimistic viewpoint and its realization may be doubtful, there is no doubt that if we attempt by every means to eliminate all injuries we will at least succeed in eliminating a great many.

GENERAL SUMMARY

1. The etiology of every automobile accident must be studied by a group of men trained to do this from every possible etiological angle, including personality of the people involved, their activities and mental state at the time, the location of the accident and surroundings, and the manner of injury. The blame must be definitely placed and a recurrence prevented.

2. Propaganda must be organized indicating that automobiles are lethal machines, that care must be taken in their use, and by required reading of this propaganda, caution instilled into the driving public.

3. This propaganda must show people that automobile accidents are as likely to happen to them as any of the more common illnesses.

4. A definite personal and solemn promise must be printed upon the driver's license and the driver must seriously and solemnly repeat this promise before he signs it on his license.

COMPOUND FRACTURES OF THE EXTREMITIES *

RESULTS OF TREATMENT

By Robert V. Funsten, M. D., University of Virginia, and Lieutenant (jr. gr.) Charles J. Frankel, Medical Corps, United States Naval Reserve

The lack of uniformity in the treatment of compound fractures has been subjected to criticism in several recent papers.[1] One clinic may present a series of cases with excellent results while another may present results equally as satisfactory despite the use of a different technic. Examination, however, into the various technics reveals that they differ only in a few details; that fundamentally they are the same.

Since 1932 we have treated here at the University Hospital over 250 cases of compound fractures of the extremities which were severe enough to require hospitalization of the patient. From 1932 to 1935 we followed a technic which we thought embodied the lessons that had been learned during the late war.[2] In 1935, however, we modified our process of treatment in a few details.

Of the 250 cases seen during the 7-year period, 194 were followed sufficiently to collect adequate data.

We were especially interested in studying the development of gas gangrene in our series, inasmuch as this section of the country had been notorious for the high incidence of gas bacillus infection. Stone and Holsinger [3] in 1934 reported a series of cases seen in this hospital. Sixty-seven cases of gas gangrene were seen in a 12-year period. Of the compound fractures 12.3 percent developed gas gangrene. The mortality of the entire group was 25 percent.

In 1932 our method of treatment closely followed that of Bohler.[4] All patients in shock were first treated for it before any attempt was made to treat the fracture or the compound wound. When the patient's condition permitted, he was removed to the operating room where under general anesthesia (in this respect we differed from Bohler) the leg was shaved and cleaned with soap and water, benzene, ether, iodine, and alcohol, and then draped. Care was taken to avoid washing dirt into the wound. The wound was swabbed with iodine and alcohol and then a debridement was undertaken. Necrosed tissue was removed as was bone devoid of any attachment. If the wound was seen earlier than 8 hours after injury, an attempt was made to close the wound and reduce the fracture by one form of traction or another, and apply a circular padded cast. Those wounds seen after 8 hours were treated as potentially infected cases. They

* From the Department of Orthopedic Surgery, University of Virginia.

[1] Caldwell, E. H.: Treatment of compound fractures, Am. J. Surg. 43: 554-559, February 1939.

[2] Bohler, L.: Treatment of Fractures. Wilhelm Maudrich, Spitalgasse 1, Vienna, 1932.

[3] Stone, C. S., Jr. and Holsinger, H. B.: Diagnosis and treatment of gas bacillus infection, Virginia M. Monthly, 61: 200-204, July 1934.

[4] Bohler, L.: Treatment of Fractures. 4th ed. Wood, 1935.

were debrided and then treated by the Orr method or by the application of wet dressings.

In 1935 we changed our procedure slightly.[5] In the preparation of the extremity we covered the wound with sterile gauze. The extremity was then shaved, cleaned, washed, and prepared in the former method. We were careful not to pour any iodine or alcohol into the wound. Debridement was done and then from 2 to 4 quarts of normal saline was run into the wound. Care was taken to place the irrigation bottle at or about the level of the operating table, to avoid forcing the fluid into the wound under great pressure. At this stage all instruments were discarded, clean drapes were applied, gloves and gowns changed, and the last stage of repair begun. Fractures were then reduced and primary closure attempted in those cases seen within 8 hours from the time of injury. Circular padded casts were applied. All cases were given tetanus antitoxin. If the indication demanded it, gas serum was given prophylactically.

Sumner Koch [6] expresses the fundamentals of treatment of compound fractures as follows: (*a*) In the preparation of the field do not add furthur contamination nor add additional injury. (*b*) In the treatment of the wound, an open wound should be converted to a closed one and a contaminated one to a clean one. He added that chemical antiseptics do not safeguard the patient and in his cases he has discarded their use entirely. Iodine, he states, causes injury to sensitive exposed tissues.

In the treatment of wounds in which gas infection is present the Army Manual states: [7]

1. Operate as early as the diagnosis is made.
2. In cases in the extremities avoid the use of tourniquets.
3. Make incisions longitudinally, and half again as long as thought necessary in both skin and fascia.
4. Go between muscles rather than through or across them.
5. Excise all discolored, noncontractile muscle, and all other infected tissue. By the removal of an entire infected muscle or group of muscles the spread of infection may be stopped and amputation may at times be avoided.
6. Leave the wound wide open. Dress the wound with gauze, laid in, not packed in.
7. Carrell tubes should be placed deep in the wound for purposes of irrigation only if it is sure that they will be properly cared for.
8. Parts that have inadequate blood supply, or those in which gangrene is already present, should be amputated at once.
9. When amputation is necessary, disarticulation should be done when feasible. If this is not feasible, the guillotine amputation is the operation of choice.

There are some who feel that no compound fracture should be closed; that immediate closure of the wound causes potential risks

[5] Key, J. A. and Conwell, H. E.: Management of fractures, dislocations, and sprains. C. V. Mosby Co. publishers, 1937.

[6] Koch, S.: Immediate treatment of compound injuries, Bull. Am. Coll. Surgs. 18: 25-29, Sept. 1934.

[7] Army Medical Manual, 1934.

that overshadow the convenience of closure. McBride [8] advocates the Orr treatment. His methods consist of a thorough debridement followed by packing the wound loosely with vaseline and where possible the application of a cast.

Sherman [9] after a thorough debridement uses internal fixation, a method which in his hands has given excellent results, but which is not widely used in this Country or abroad.

Conwell [5] advocates the use of Dakin's solution instead of saline in the irrigation of the wound. It is questionable whether the Dakin's has more than a mechanical flushing action at the moment.

Abroad, as here, the clinics vary only in detail. Mannheimer [10] in a résumé of the European literature concluded that most surgeons regard debridement and primary suture as important and that results were better with the use of a consistent technic.

Imhert [11] presented a series of cases treated by the Bohler method and his cases also show good results. He reported that internal fixation was not regarded favorably in his clinic.

There is universal condemnation of closure without debridement and of failure to provide adequate immobilization.[1]

Daland [12] in 1934 reported 236 cases treated at the Massachusetts General Hospital. The method used then was practically the same as is used in our clinic today. Daland reported 18 percent of the cases became infected. He found fewer indications for the use of gas serum due to the thorough debridement. He reported no cases of gas bacillus infection.

Jensen [13] recently suggested the local implantation of sulfanilamide crystals in compound wounds, and showed in his series that the drug markedly decreased postoperative infections. This view is likewise held by Key [14] and many others. Speed [15] feels that internal fixation can be used with impunity in cases treated by the drug. It is still too early to conclusively evaluate the new addition to the armamentarium. Many men, including the authors, are experimentally checking the use of the drug and its associated compounds, in vitro and in vivo.

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[8] McBride, E. D.: Treatment of compound fractures; analysis of 100 cases, South, M. J. **32**: 243–247, Mar. 1939.

[9] Foster, G. V.: Compound fractures of long bones; review of 304 cases treated by debridement, Carrel-Dakin technic, open reduction and plating when indicated, Surg. Gynec. & Obs. **56**: 529–538, Feb. 1933.

[10] Mannheimer, E.: Treatment of open fractures at surgical clinic of Serafim Hospital in Stockholm during years 1924 to 1933. Nord. Med. tidskr. **10**: 1297–1305, Aug. 1935.

[11] Imhert. Rev. de Chir. 1936.

[12] Daland, E. M.: Study of 236 compound fractures treated at Mass. General Hospital, New Eng. J. Med. **210**: 983, May 1934.

[13] Jensen, N. K., Johnsrud, L. W., and Nelson, M. C.: Local implantation of sulfanilamide in compound fractures; preliminary report, Surgery **6**: 1–12, July 1939.

[14] Key, J. A. and Burford.: Paper read before Orthopedic Section, S. M. A., Nov. 1939.

[15] Speed, J. S.: Paper read before Orthopedic Section of S. M. A., Nov. 1939.

We have found that children respond to the same treatment as adults. Shock is always controlled before treatment is instituted. We do not agree that children respond better to a more conservative type of treatment.[16]

We have divided our 194 cases into (*a*) 96 treated by debridement and the use of iodine in the wound, and (*b*) 83 treated with debridement and extensive irrigation with saline. Fifteen cases received miscellaneous treatment. (See table 1.) Those cases seen within 8 hours after injury were considered potentially clean, those seen after 8 hours were considered potentially infected.

TABLE 1

Group	Tetanus infection	Gas bacillus infection	Nonunion	Delayed union	Primary healing	Hospitalization days
a	12. 5	4. 1	2	3	39	12. 9
b	4	3. 6	1. 8	3	59	9. 8

The total mortality for the series of cases was 5.7 percent. The causes of death were: Pulmonary embolism, 4; hemorrhage and shock, 2; tetanus, 2; and anesthesia, pneumonia, and septicemia, 1 each.

Of the seven cases of gas bacillus infection, all showed clinical symptoms. Four were amputated. There was no mortality.

Of the infected cases (osteomyelitis) nine cases were seen within the first 8-hour period for an average of 3½ hours after the injury. Ten cases were seen after the 8-hour period varying from 8 hours to 16 days, or an average of 4½ days after the accident.

All of the gas infections were in cases with extensive wounds. They had all received the therapeutic as well as prophylactic gas serum. All except one case entered the hospital within the 8-hour period.

In analysis of our results, a marked drop in mortality and incidence of gas bacillus infection at this hospital is difficult to explain fully. Certainly in the period before 1932, cases were repaired carefully, serum was used in a large number of cases and its efficacy was questioned then as it sometimes is now. We feel that if any single factor is responsible, it is the thorough debridement that is done today. We believe also that it is extremely important to distinguish clinical gas infection from bacteriological gas infection.[4] It was noticed during the war that many cases with extensive wounds gave positive cultures, but showed no clinical symptoms of the disease. It is

[16] Beekman, F.: Compound fracture in childhood, Am. J. Surg. 39: 312-318, Feb. 1938.

obvious folly to amputate merely on laboratory evidence of the disease.

Our treatment of gas infections has been to amputate only where necessary. The gas serum is given prophylactically and therapeutically; wounds are opened widely; and the Dakin's tubes are placed throughout the muscle planes and the tubes irrigated with either Dakin's or peroxide. Lately we have used oral sulfanilamide along with the other treatment. We have had no experience with the use of deep x-ray therapy.[17]

Nineteen cases developed osteomyelitis; 14 of these healed quickly under incision and drainage; 5, however, went on to formation of chronic, disabling osteomyelitis. We believe that compound fracture with extensive damage will always carry with it the possibility of osteomyelitis. That the percentage is no higher can be attributed to the advances surgery has learned from the last war. It is interesting to note that though the last war forcibly brought to light these principles, the same fundamentals were voiced by Sir Ashley Cooper [1] over 100 years ago.

We were fortunate in having few cases of delayed and nonunion. Those long bones which required from 3 to 6 months to heal were classified as delayed union. Those which failed to show any union in 6 months or more were classified as nonunion. The treatment in both groups was first conservative; the application of skin casts and walking irons, the use of diathermy,[18] and finally open operation when no response to the early treatment was shown. Open operation consisted of the use of various types of autogenous bone grafts.

Caldwell [19] has listed among the causes of nonunion: (*a*) Extensive damage to periosteum; (*b*) washing away of blood clot; (*c*) loss of fragments; (*d*) poor immobilization; (*e*) use of strong chemical agents, such as Dakin's, which may have an inhibitory action.

In our cases we can be certain only that the periosteum was badly damaged. Beyond that it is difficult to evaluate the actual causes of nonunion.

Two of our cases died from shock before any repair could be attempted. On entry into the hospital, both cases—shotgun wounds—were in a marked state of collapse, and gave histories of the loss of large amounts of blood. Transfusions were given to no avail. Blalock's [20] recent work on shock due to hemorrhage well explains why these patients failed to respond to treatments. Two cases developed

[17] Kelly, J. F., Dowell, D. A., Russum, B. C., and Collen, F. E.: Practical and experimental aspects of roentgen treatment of Bacillus welchii (gas gangrene) and other gas-forming infections, Radiology 31: 608–619, Nov. 1938.

[18] Voshell, A. F.: Delayed union of fractures; analysis of cases, Arch. Phys. Therapy, 18: 561–564, Sept. 1937.

[19] Caldwell, G. A.: Surgical measures for prevention of gas gangrene, South. Surgeon, 5: 141–152, April 1936.

[20] Blalock, A.: Lecture, University of Virginia Clinics, 1939.

tetanus 19 and 38 days after entering, respectively. Prophylactic serum had been given and despite the use of large amounts of therapeutic serum the course of the disease when once started was never arrested. It will be interesting to follow the recent work begun at Johns Hopkins suggesting the necessity of a different serum for the late manifestations of the disease. Four cases developed pulmonary embolism in 14, 7, 5, and 4 days, respectively. Two were in old individuals and two in young adults. Homan [21] in a recent lecture here suggested that patients who have had serious trauma to the extremities should be forced to keep the extremities elevated; that any other position was conducive to the formation of thrombi. One case developed pneumonia and died shortly afterwards. It may be added here that all cases were done under general anesthesia.

We claim no originality in our treatment of compound fractures. We arrived at our present method by combining what we believe to be the best principles of the various methods in vogue. Our results compare favorably with other clinics and we do not insist that our treatment is the only one and should be used to the exclusion of all others. In our hands it has been satisfactory and we believe that any method which adheres to good surgical principles and which may vary in many ways from ours will give equally as satisfactory results.

SUMMARY

1. An examination was made of the various types of procedures used in treating compound fractures. It is found that fundamentally most of them differ only in a few details.

2. One hundred and ninety-four cases are presented. A comparison is made between the results obtained through using iodine in the wound and a later method avoiding the use of antiseptics in the wound.

3. We believe that careful adherence to the surgical principles laid down over 100 years ago will provide good results.

4. The treatment of compound fractures is time consuming, often tedious, and requires patience and judgment.

5. The marked drop in the incidence of gas bacillus infection here can be explained only partially by the change in the method of treatment.

6. The end results as to infection and mortality compare favorably with other clinics.

SPRAINED ANKLES [1]

TREATMENT BY NOVOCAINE AND ADRENALIN INJECTIONS

By Lieutenant Commander C. R. Ball, Medical Corps, United States Navy

The treatment of sprained joints is a daily procedure among medical officers on duty with men actively engaged in athletics. We are all

[21] Homan, J.: Lecture, University of Virginia Clinics, 1939.

[1] From United States Naval Academy.

familiar with the changes that have gradually taken place in the treatment of sprains. It was once a common procedure to apply a cast for several weeks. The cast has been replaced in most clinics by an elastic bandage and earlier, use of physiotherapy.

In the late fall of 1938 we began to treat the athletes in organized athletics in the Naval Academy by the injection of novocaine and adrenalin. This form of treatment was started in an effort to keep the player out of the hospital and to hasten his return to the various sports. Since that time 25 cases of sprained ankles have been treated and in each case the result has been excellent.

The procedure that we follow here in Misery Hall will be briefly described. An elastic bandage is applied to the injured ankle just as soon after the injury occurs as is possible. This bandage is applied on the field, by the hospital corpsman, a coach, manager, or by the injured athlete himself. He is carried or walks with aid to Misery Hall. The elastic bandage is left in place and the leg is placed in a bucket of ice water. We have recently been using a rubber apparatus containing small compartments of water similar to the tray of an ice box. This rubber apparatus is placed in the ice box instead of the tray to freeze. This rubber apparatus is flexible and is placed around the elastic bandage where it is held in place by a towel or a second elastic bandage. An hour later the bandages are removed and the ankle carefully dried. The skin over and around the injured ligament is painted twice with Scott's solution or iodine. A small sterile syringe with a 1-inch needle and containing 1 cc. of a 2-percent novocaine and adrenalin is used for the injection.[2] A small amount of novocaine is injected ahead of the needle as we proceed to the area of the injured ligament where the bulk of the solution is injected. The plunger is pulled back frequently to be sure that the needle has not entered a blood vessel.

The needle is withdrawn and a piece of gauze saturated with alochol is placed over the injured area. An elastic bandage is applied to the ankle and over the alcohol sponge.

The injured player walks to sick quarters or to his room with the aid of crutches and is turned in bed for the night. It has been necessary in several cases to give a small dose of codein at bed time. The following day he walks to his classes with the aid of crutches. A balm cotton dressing is substituted for the alcohol sponge and the elastic bandage again applied. The third day he discards the crutches and is given a whirlpool bath for 45 minutes at a temperature of 105° to

[2] The procaine-epinephrine solution used for this series of cases was manufactured by the Abbott Laboratories. Each 1 cc. sterile ampule contained: Procaine hydrochloride (USP), 0.02 gm.; epinephrine (Abbott), 0.00004 gm.; Sodium bisulphite, 0.001 gm., in isotonic solution.

110° F. The balm dressing, elastic bandage, and whirlpool bath are repeated daily until all subjective and objective symptoms have disappeared.

In several cases the pain has entirely disappeared at the end of 3 days and the player has resumed his place on the team. Two weeks is the maximum time that is usually required for complete recovery.

The ankle is always strapped with adhesive before returning to duty on the field and removed after the game. In case the individual is sensitive to adhesive, the tape is placed over a gauze wrap. It is important to insist upon daily treatment until the injured ankle is completely restored to it's normal function. When the injury is severe it is well to use the whirlpool bath for ½ hour in the morning as well as in the afternoon.

The ligaments of the ankle in the frequency of injury were as follows: (*a*) anterior tibio-fibular; (*b*) lateral collateral; (*c*) medial collateral; (*d*) tibio-astragaloid. Not infrequently several ligaments were injured and it was necessary to inject all of them.

The pathology of sprained ankles has not been definitely settled. For a long time it was thought that the severity of a sprain depended on the injury to the ligaments involved. The few cases in which it has been possible to study the injured joint have thrown some doubt that the injured ligament is responsible for the major symptoms. It was found in some cases that the ligaments were not torn although the pain and disability were rather marked. We do know that the capsular ligament which is thickened in places to form the ligaments which I have mentioned is richly supplied with nerves.

The disability then is probably due to a combination of the pathological findings, namely: Torn fibers in the ligament and capsule, tendon sheath, blood vessels, capillaries, nerves, subcutaneous tissue and skin. The pain is caused by a lack of oxygen. The decrease in oxygen is caused by the hemorrhage and edema. In order to prevent hemorrhage and edema which cut off the oxygen supply and interfere with normal function, an elastic bandage should be applied to the site of injury as soon after the trauma as is possible.

The period of disability is greatly reduced when hemorrhage in the subcutaneous tissue is prevented. The most important treatment in all closed athletic injuries is to apply an elastic bandage at once, no matter how small the force causing the injury may be.

The sprained ankle is always x-rayed and if a fracture of the fibula is found in addition to the sprain, it is necessary to keep the patient on crutches for a longer period of time.

When the injured ankle is not seen until the next day or even later, the injection is then given because the period of disability can still be shortened.

We compared the results obtained in the 25 cases injected with our previous treatment. The average case under the injection treatment was returned to duty at least 2 weeks sooner. The players soon recognized the advantage of the injection treatment and would request it. We had several players of a visiting team request an injection after having talked with one of our players.

We have not used the injection treatment in a sufficient number of other joint injuries and other types of injuries to make a report at this time.

We may summarize and state that our sprained ankle cases are returned to duty earlier with our present treatment. The treatment consists of an elastic bandage, ice water, an injection, alcohol dressing, balm dressing, and whirlpool bath.

INTRAVENOUS ANESTHESIA

By Lieutenant Commander J. R. Fulton, Medical Corps, United States Navy

The intravenous method of anesthesia is theoretically more simple than the use of an anesthetic gas by inhalation. However, the use of this method requires that the surgeon and the anesthetist understand its application and the limits of its usefulness. Intravenous anesthetics should be administered absolutely on the same principle as inhalation anesthetics and not according to the patient's weight, age, or size. They should be given only by experienced physicians or anesthetists and according to the fractional method introduced by Lundy.[1 2 3]

The first record of intravenous anesthesia was by Orr of France in 1872 with chloral hydrate the agent. Others were introduced, such as, hedanol, paraldehyde, soninifene, magnesium sulfate, ipral, and avertin. Each had brief but extensive use. The use of sodium amytal to produce surgical anesthesia was reported [4] in 1929. Nembutal was introduced in 1930.[5] These latter longer acting barbiturates were soon seen to have many disadvantages as anesthetic agents and are now reserved for intravenous use for the control of convulsions associated with epilepsy, eclampsia, tetanus, and strychnine poisoning. Sodium amytal and nembutal as surgical anesthetics have the disadvantages of prolonged recovery and, too frequently, restlessness and excitation of a sufficient degree to require restraint.

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[1] Lundy, J. S.: Intravenous anesthesia, Am. J. Surg., **34**: 559–570, Dec. 1936.

[2] Lundy, J. S.: Intravenous and regional anesthesia, Ann. Surg. **110**: 878, Nov. 1939.

[3] Lundy, J. S.: A technical description of the intravenous administration of pentothal sodium based on experience in more than 12,500 administrations. Paper read before meeting of Ohio Soc. of Anesth., Toledo, Ohio, May 1939.

[4] Zerfas, L. G., and others: Induction of anesthesia in man by intravenous injection of sodium iso-amyl-ethyl barbiturate, Proc. Soc. Exper. Biol. & Med. **26**: 399–403, Feb. 1929.

[5] Lundy, J. S.: Intravenous anesthesia; particularly hypnotic anesthesia and toxic effects of certain new derivatives of barbituric acid, Anesth. & Analg. **9**: 210–217, Sept. 1930.

The introduction of the so-called ultrashort acting barbiturates opened up the field of intravenous anesthesia and provided us with a method which has proved comparatively safe, satisfactory, and essentially free from complication when the agents are administered by experienced persons for suitable types of operations. Evipal soluble was introduced in 1932, and pentothal sodium in 1934. Evipal is comparable to pentothal in action, but it is said to be from 30 to 50 percent less potent.[6 7] Pentothal is closely related to sodium amytal and nembutal. Sodium amytal and nembutal have the same empirical formula but differ only in the position of one of the methyl groups. Pentothal differs from nembutal by the replacement of one atom of oxygen by a sulfur atom on the urea side of the molecule.

Captains Seeley [8] and Kendrick [9] of the United States Army Medical Corps and their co-workers have recently shown that sodium amytal and nembutal, when given to animals intravenously, will delay the onset of surgical and traumatic shock from 6 to 8 hours and, if administered when the animal is already in shock, will reverse this process. Because of its chemical similarity, it is reasonable to assume that pentothal sodium will produce the same effect. The introduction of pentothal sodium for surgical anesthesia has given us a drug and a method of anesthesia for wartime conditions that, in my opinion, will be of great value. The fact that pentothal sodium produces rapid induction and recovery, is nonexplosive, probably allays shock, does not require elaborate equipment to administer, all would seem to make it a valuable addition to our armamentarium in evacuation hospitals, battle-dressing stations, and in the sick bays of combatant ships when a great number of wounded men must be taken care of by a limited medical personnel.

APPLICATION

Although the trend in the late literature is continually towards the displacement of other anesthetic agents by pentothal sodium, it is still the consensus [2 6 7 10 11] of opinion that this method of anesthesia is most applicable for short and minor surgical procedures ranging from 15 to 30 minutes, especially in cases in which the operative procedure does not involve the respiratory passages or when extensive muscular

[6] Adams, R. C.: Present status of intravenous administration of pentothal sodium in institution and private practice. Can. M. A. J., 38: 330, Apr. 1938.

[7] Garofola, M.: Present status of pentothal sodium as an anesthetic agent. J. Conn. State Med. Soc., 2: 550–557, Nov. 1938.

[8] Seeley, S. F., Essex, H. E., and Mann, F. C.: Comparative studies on traumatic shock under ether and under sodium amytal anesthesia. Ann. Surg., 104: 332–338, Sept. 1936.

[9] Kendrick, D. B.: Results of intravenous and intra-arterial administration of fluids in traumatic shock produced experimentally. Surgery, 6: 520, Oct. 1939.

[10] Adams, R. C.: Intravenous anesthesia. Surg. Gynec. & Obst., 68: 719–721, Mar. 1939.

[11] Tovell, R. M. and Garofola, M.: Evaluation of intravenous anesthesia, N. Y. State J. of Med., 39: 2026–20–32, Nov. 1939.

relaxation, particularly abdominal, is not required. The more ardent enthusiasts recommend the method as being practically ideal for almost every type of operation.[12] [13] [14] However, it must be remembered that the method and the drug are entirely safe only in the hands of those experienced in its use. Until such time as the medical officers of the Service have become more familiar with the drug and the method of administration, I believe its use should be on a conservative basis.

In certain cases its use may be preferable for intraabdominal operations, but in many of these instances a safer and more satisfactory anesthesia can be obtained if an abdominal wall block is used as a supplement to intravenous anesthesia. [6] [10] It may be used to supplement local or spinal anesthesia when the effect of the latter is wearing off. It appears to be an ideal anesthesia for the reduction of simple fractures, dislocations, and other emergency procedures. [15] It is an excellent anesthesia for almost any orthopedic procedure. It may be used for a quick and pleasant induction preliminary to the administration of an inhalation anesthetic. Tovell [11] has emphasized its usefulness as an aid in evaluating the prognosis of patients suffering from Raynaud's disease or essential hypertension. The lowest point to which the blood pressure falls, with a dose sufficient to raise the temperature of the extremities to a maximal, corresponds fairly well in most cases with the level of blood pressure that will obtain following neurosurgical intervention. The drug is of particular value for gynecological operations, for operations about the thorax, thoracic wall, breasts, and axilla. All surgical procedures on the breast from biopsy to radical amputation may be done under intravenous anesthesia. Pentothal produces sufficient relaxation of the anal sphincter to permit any operation about the anus or rectum. Intravenous anesthesia has displaced low spinal and sacral anesthesia to a great extent in transurethral and cystoscopic operations and manipulations.[6] [7] [13] It is particularly well suited for operations about the eye, and operations about the head and neck, and most plastic operations, even though of long duration, can be done under pentothal sodium. In operations about the head it removes the anesthetist and the equipment from the operative field and lessens the danger of contamination. This method is particularly suited to certain operations on the brain, spinal cord, and nerves, such as craniotomy for abscess of the brain, elevation of depressed fractures, encephalography, avulsion and suture of nerves,

[12] Carraway, B. M. and Carraway, C. N.: Intravenous anesthesia, Amer. J. Surg. 39: 576–580, Mar. 1938.
[13] Carraway, B. M.: Pentothal sodium with nasal oxygen, Anesth. & Analg., 18: 259–269, Sept.–Oct. 1939.
[14] Porter, A. R.: Intravenous anesthesia, Memphis, M. J., 15: 3–6, Jan. 1940.
[15] Marcus, P. S.: Pentothal: anesthetic agent of choice for the reduction of simple fractures, New Eng. J. Med. 222: 137–140, 1940.

and repair of lacerations of the scalp. It is, of course, preferable to an explosive inhalation agent in operations when the electrocautery is being used. Pentothal sodium anesthesia will be found of particular value in a number of minor and emergency procedures such as opening of abscesses, removing of packs, painful examinations and dressings, in fact, in any case in which a prompt but transient effect is desired.

It should be remembered that pentothal sodium is a sulfur containing compound and most observers [4 6 10] agree that it should not be administered to a patient who has been receiving sulfanilamide, unless the latter drug has been withdrawn for at least 24 to 48 hours before operation. Lundy [3] has called attention to the fact that on operating upon inflammatory lesions of the side of the neck the incision should not be made until the patient is deeply anesthetized. Under light anesthesia an incision of an inflammatory lesion in this area may affect the carotid sinus reflex and cause a sudden cessation of the heart beat. According to Weese,[16] this does not occur under deep anesthesia.

PRELIMINARY MEDICATION

Proper premedication is of greatest importance. The necessity of atropine cannot be too strongly urged.[11 13 17] Attention has been called to the occurrence of temporary closure of the glottis and a hyperactive state of the laryngeal reflex, further complicated by frequent coughing. In addition, hiccoughs or sneezing may occur. It has been suggested that these phenomenon are due to parasympathetic overactivity. For this, the preanesthetic administration of atropine is shown to be an adequate prophylactic measure.[17] In addition, atropine minimizes the likelihood of production of excessive secretions. Sufficient nembutal to render the patient sleepy before being brought to the operating room gives a much smoother anesthetic, minimizes apprehension, and reduces the amount of pentothal required. For the average adult a grain and a half of nembutal is given 1 hour before operation. Atropine, grain 1/150, is usually combined, except in the aged or markedly debilitated patient, with morphine, grain ¼, one-half hour before operation. For short operative procedures, all preliminary medication, except the atropine, may be omitted. It is generally conceded that for long operative procedures the administration of adequate preliminary medication will reduce the amount of

[16] Weese, H.: Concerning mechanism of anesthesia accidents in sublingual phlegmons, Anesth. & Analg. 18: 15-21, Jan. 1939.

[17] Ruth, H. S., Tovell, R. M., Milligan, A. D., Charleroy, D. K.: Pentothal sodium, J. A. M. A., 113: 1864-1868, Nov. 1939.

pentothal that otherwise would be required and will assist in the production of a smooth and rapid induction.

CONTRAINDICATIONS

Most observers [6 7 10 11] agree the drug should not be used in children under the age of 10 to 12 years because of their susceptibility to respiratory depression, the smallness of the air passages, and the difficulty of maintaining a patent airway. Others do not agree with this observation and use it for short operations in children as young as 3 years. It is contraindicated in patients with pulmonary disease that reduces pulmonary ventilation, such as bronchiectasis, advanced pulmonary tuberculosis, asthma, or emphysema. Any marked degree of cardiac insufficiency with dyspnoea contraindicates its use. Tovell [11] and others have stated that in operating on tumors of the neck that encroach on the lumen of the glottis or trachea, pentothal is contraindicated. The consensus of opinion is that the method should not be used for operations in the vicinity of the pharynx, operations in which it will be difficult to maintain a free airway, and in cases in which blood and mucus may act as a potential obstruction in the airway. Moderate liver or renal involvement does not constitute a contraindication. Evidence of gross hepatic disease is thought by many to be a definite contraindication, yet others have reported its use in cases where the patient has been deeply jaundiced. In either case, pentothal should be given with extreme care in patients who show renal or hepatic damage and large doses should be avoided. Porter [18] states that any case that is a bad surgical risk under any other anesthesia remains a bad risk under pentothal. Old and debilitated patients tolerate the drug well although much smaller doses are necessary to produce adequate surgical anesthesia. Except for the most brief and minor procedures, pentothal sodium should not be given intravenously unless the facilities for administering oxygen and carbon dioxide are available.

ADMINISTRATION

To properly administer intravenous anesthesia, 2 anesthetists are required, a competent nurse at the head to elevate the chin, maintain an efficient airway, and to check the blood pressure, and a physician anesthetist to administer the drug and watch the patient's condition. The drug is now being used in a 2½ percent solution in a great many hospitals and clinics. It is felt that the 2½ percent solution has many advantages over the 5 percent. With the more dilute solution, induction is slower, it is felt that the anesthetist has better control of

[18] Porter, A. R.: Intravenous anesthesia, Memphis M. J., **15:** 3-6, Jan. 1940.

the patient, probably less of the drug is used and the danger of phlebitis is minimized. The solution is prepared by diluting the contents of the 1-gram ampule in 40 cc. of distilled water. The solution is best injected with a 20 cc. syringe with an eccentric outlet or through a two-way stopcock attached to the needle and continuous intravenous drip between injections as described recently by Ruth and Tovell.[17]

The patient should be brought to the operating room with an empty bladder, rectum, and preferably an empty stomach. If the patient has eaten within the past 4 hours, he should be kept in Trendelenburg position during induction and the anesthetist should be prepared to use suction if necessary.[7] The patient should be secured on the operating table, the operative site prepared, and the patient draped before inserting the needle into the vein. A great amount of time under anesthesia will be saved by this procedure. When the surgeon is ready, 4 to 5 cc. of the 2½ percent solution are injected and then the patient is told to count slowly. If no signs of drowsiness appear in 30 seconds, i. e., yawning or slurred speech, an additional 2 cc. are injected every 10 to 15 seconds until the patient ceases to count. The surgeon should wait 30 to 45 seconds before beginning the operation to allow the maximal effect of the drug to take place. The needle is maintained within the vein and, after induction, injections of small amounts are made as necessary from time to time to maintain anesthesia and the degree of relaxation desired.

Pentothal, like all barbiturates, is a respiratory depressant and the amount of depression depends on the amount of drug in the circulation at any one time. At times respirations become so shallow they are almost imperceptible. Lundy [1 2] has advised the use of a cotton or paper butterfly over the nose and mouth, in the current of the flow of air, as an aid to the determination of the depth of respiration. Respiration is the best single guide to the depth of anesthesia. When the respirations are shallow, the patient is deeply anesthetized and, conversely, when the respirations deepen, the patient is light. No single dose should be so large as to markedly depress respiration.

Most of the other signs of anesthesia are subject to swift change and none, except respiration, is very reliable. The lid reflex is lost, the blood pressure may show an initial drop of 10 to 15 mm. of mercury early but later returns to normal. The pulse may be slightly elevated or normal. The degree of relaxation of the jaw is a good guide to the depth of anesthesia, but this cannot always be depended upon. The signs of overdosage are an arrest of, or imperceptible, respiration accompanied by cyanosis and later by marked relaxation of the muscles and a feeble pulse. In these circumstances administration should be discontinued at once, an airway should be established, by

endo-tracheal intubation if necessary, and artificial respiration by forced gentle administration of 5 percent carbon dioxide and 95-percent oxygen by rhythmic compression of the bag of the gas machine should be employed. The intravenous use of respiratory stimulants [3 7 13] has been recommended. These include metrazol, coramine, and picrotoxin.

Since May 1938, Carraway [12 13] has been using continuous nasal oxygen with pentothal intravenously and it has been found that by using this method that respirations are more nearly normal and cyanosis is less frequent, and that a greater amount of the barbiturate can be administered when employing this method. In November 1938, Organe and Broad [19] advocated the use of nitrous oxide and oxygen with pentothal anesthesia and at the present writing several clinics [19 20] and hospitals are using nitrous oxide 50 percent, and oxygen 50 percent along with the pentothal intravenously. Respirations are more vigorous and can be noted by watching the exchange in the bag. When this technic is used, much more profound anesthesia and relaxation can be secured.

Recovery from anesthesia is rapid and quiet and the majority of patients recover without nausea or vomiting. The length of the recovery period, of course, depends upon the amount of the drug administered, and following brief procedures the patient is awake in a few minutes. The swallowing reflex returns early and though the patient may continue to sleep for an hour or so after a prolonged pentothal anesthesia, he usually can be aroused easily and his color remains good. This method is a very satisfactory anesthesia from the point of view of the patient. The rapid induction, the awakening as though from a refreshing sleep without nausea, vomiting, or headache, is greatly appreciated, and several patients in my experience who have taken pentothal, who were unfortunate enough to have to undergo a second operation, have insisted that they be given pentothal for the latter operation.

SUMMARY

Intravenous pentothal sodium is the anesthesia of choice for many types of short and minor procedures. It has been proven to be a safe and satisfactory anesthetic when administered by competent anesthetists. Special care should be taken to maintain an efficient airway. Facilities for administering carbon dioxide and oxygen should always be available when this drug is being used. The importance of preliminary medication, particularly atropine, cannot be

[19] Organe, G., and Broad, R. J. B.: Pentothal with nitrous oxide and oxygen, Lancet. 2: 1170, Nov. 1938.

[20] Lundy, J. S., Tuohy, E. B., Adams, R. C., and Mousel, L. H.: Annual Report for 1939 of the Section on Anesthesia: Proc. Staff Meet., Mayo Clinic, 15: 241-254, Apr. 1940.

too strongly stressed. For prolonged and major surgical procedures, continuous nasal oxygen or 50-percent nitrous oxide and 50-percent oxygen should be given throughout the period of the intravenous administration of the drug.

SUCTION-SIPHONAGE MACHINE

By Commander M. D. Willcutts, Medical Corps, United States Navy, and Lieutenant Commander E. P. Kunkel, Medical Corps, United States Navy

Suction siphonage has become established as an important and often life saving measure in both medical and surgical therapeusis. It is not intended in this communication to enumerate the various conditions in which its use has been found to be effective.

We wish, however, to present a simple suction-siphonage apparatus which we have found to work satisfactorily. We do not claim any originality for the apparatus. One of us (M. W.) while visiting at Johns Hopkins Hospital, Baltimore, Md., brought back the idea which led us to build our present machine.

Before our present suction-siphonage apparatus came into being we used the old method of pouring the water from one bottle to another. This was a cumbersome procedure and required the services of two corpsmen to make the water changes. Frequently the funnel or the rubber cork which closed the suction chamber of the upper bottle, became misplaced and much time was lost in getting the apparatus in working order, and, at its best, we do not believe that it ever worked as well as the present machine.

The suction-siphonage machine now in use affords a mild suction, removing accumulations by siphonage rather than by suction. The top of the apparatus is about as high as the bed. Due to the mildness of suction there is little danger in traumatizing the stomach or duodenal mucosa. The suction is not so strong as to cause the mucous membrane to plug off the openings in the Levine tube. This affords a continuous siphonage which is much more effective than in the usually employed suction-siphonage machine placed at a higher level.

Very little description is necessary as figures 4 and 5 are self-explanatory. Figure 4 illustrates the apparatus as assembled and ready for use. Figure 5 is a schematic drawing of the apparatus. The two bottles *B* and *C* including the rubber tubing connections are encased in a wooden case. This case is supported on a trunnion which permits the bottles to be tilted through an arc of 180°. The water in the bottles is colored with a distinctive dye in order that the amount of water remaining in the bottles can be readily ascertained. On the top and the bottom of the case is a notice to the effect that

the screw clamps *A* and *A'* must be closed when down and open when up. The reason for this is obvious—for if screw clamp *A* were up and closed no air would be allowed to escape to bottle *C* and hence there would be no flow of water from bottle *B* to *C*. And if *A* were open when down all the water would run out on the deck and no vacuum would be obtained in bottle *C*.

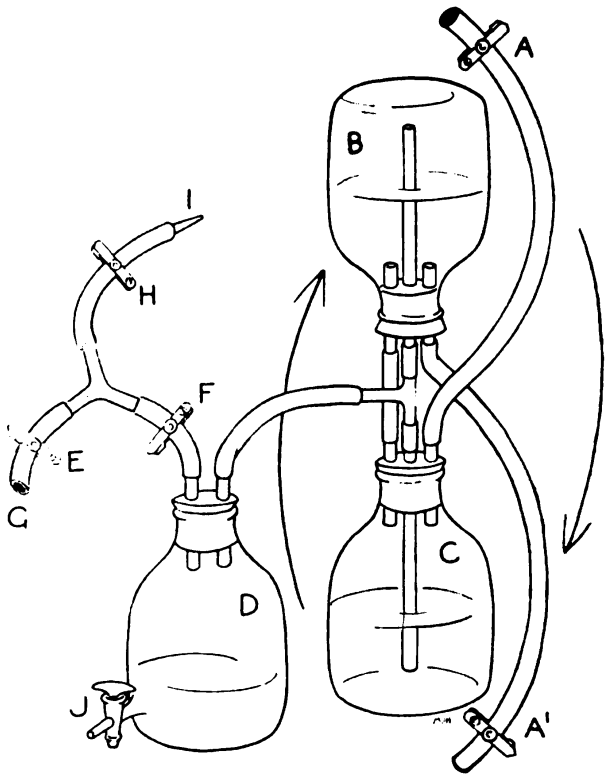

FIGURE 5.—Schematic drawing showing hook-up of suction-siphonage apparatus.

The bottles *B* and *C* should be turned back and forth in the same 180° arc and not through 360° for this would result in kinking the rubber tube leading from the *T* tube to the waste bottle *D*.

It is not necessary for the waste bottle *D* to have a spigot—but with a spigot the refuse material can be removed from the bottle without losing the vacuum in the bottle.

A Levine tube, after it has been put in place, is connected to *I*. When working the suction-siphonage apparatus, screw clamp *H* and *F* are open and *E* closed. When it is desired to give fluids or clean out the Levine tube, screw clamp *F* is closed and *E* and *H* open, the fluid is administered through opening *G* with a 20 or 50 cc. Luer syringe.

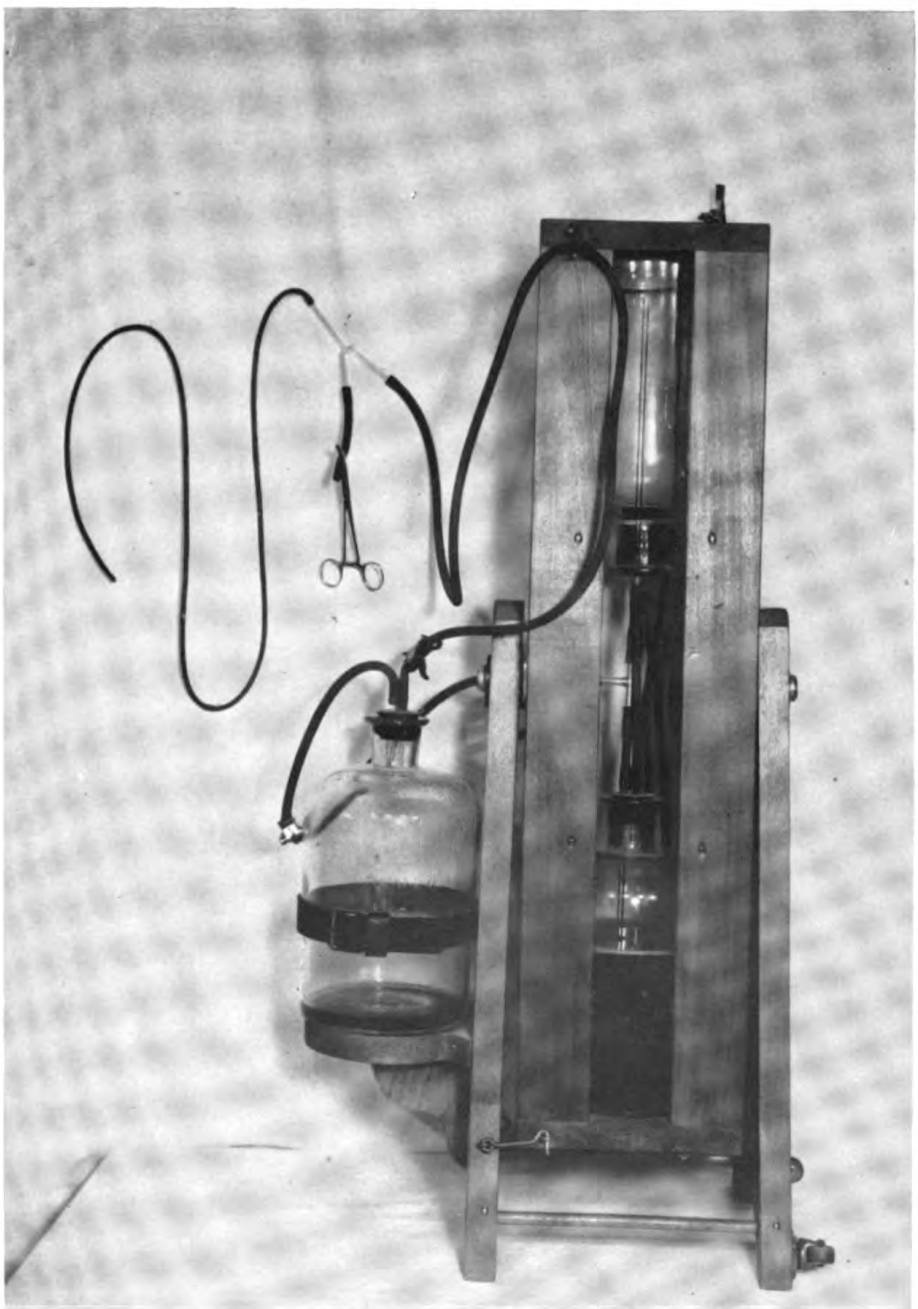

FIGURE 4.—SUCTION-SIPHONAGE APPARATUS ASSEMBLED AND READY FOR USE.

The bottles are of 5 liter capacity but an ordinary gallon jug may be used. The dimensions of the case are 8½ by 8½ by 33 inches. The case clears the deck by 4 inches. The height is about that of the standard Navy hospital bed.

This apparatus can be easily assembled and the wooden case and trunnion made by any carpenter.

CONCLUSION

1. This simple suction-siphonage apparatus can be easily assembled at shore establishments or aboard ship.
2. The suction is mild, thus there is little danger in traumatizing the mucous lining of the stomach or duodenum.
3. It has proved to be most satisfactory in any case where suction siphonage is indicated.

COTTON SUTURES

USE IN GENERAL SURGERY

By Commander J. B. Logue, Medical Corps, United States Navy, and Lieutenant Commander W. E. Walsh, Medical Corps, United States Navy

Suture material has always been one of the most important problems facing the surgeon, for, on its ultimate fate to a large degree rests the result of the operative procedure.

From earliest times surgeons have searched without success for an ideal material for sutures. Catgut, though now universally used, is far from the ideal sought after, due to its relatively high cost, bulkiness, difficulty in sterilization, difficulty in transportation, its uncertainty of time of absorption, and the reaction of the tissues to its use.

Halsted[1] was never a convert to the use of catgut and continued the use of silk as a suture material long after it had been abandoned by the majority of surgeons.

Halsted's excellent and beautiful surgical procedures were no doubt due to the meticulous care and delicate handling of tissues which he taught should be used in all operative procedures. He believed in using interrupted sutures as fine as was compatible with the structures to be approximated.

Moorehead at his clinic at the Post Graduate Hospital, New York City, used to remark to his students when demonstrating traumatic wounds of the hand, that the best suture material for these cases was number 50, turkey red, cotton thread.

There has been a definite trend in recent years by surgeons to use nonabsorbable suture material, such as silk, linen, wire, and now cotton.

[1] Halsted: Surgical Papers, 1: 29.

Wolff and Priestly,[2] Mayo Clinic, and Bower[3] in their observations on the absorption time of catgut found that small sizes of chromic catgut are absorbed more slowly than larger sizes due to a more thorough penetration of the chromicing agent of the smaller sizes and that labels indicating absorption time are fallacious due to various individual factors.

Meade and Ochsner[4] show graphically the comparative tissue reactions to catgut, silk, linen, and cotton suture material. "The last produced less cellular exudation and earlier healing than any of the others. Whereas dry cotton has less tensile strength, size for size, than catgut, silk, or linen, after being placed in tissues it showed much less decrease in tensile (break) strength than the others."

Their investigation further showed a 10-percent increase in tensile strength of cotton when boiled 20 minutes. When placed in tissues, cotton lost 10 percent of its tensile strength in 14 days, whereas catgut loses from 50 to 75 percent and silk 35 percent. Due to a high coefficient of friction a square knot in cotton held better than catgut or silk and the suture could be cut closer to the knot. They further found that there was practically no ingrowth of granulation tissue in cotton sutures due to the compact structure of the thread and, because of this lack of ingrowth of granulation tissue, they are of the opinion that there is less likelihood of sinus formation.

Catgut is a foreign protein and is absorbed by an exudative reaction which, when excessive, delays wound healing or as Gage[4] describes it "wet healing" in contradistinction to the absence of tissue reaction to cotton or "dry healing."

Following a study of Meade and Ochsner's[4] article it was decided to use spool cotton on the surgical service of the United States Naval Hospital, Pearl Harbor, T. H. Their instructions as to size and preparation of suture material was followed. Number 50, white, was used for ligatures and numbers 30 and 20 for structures requiring greater strength. Number 20, black was used for skin closure. All sutures were either autoclaved for 15 minutes at 15 pounds pressure or boiled for 20 minutes.

In a personal communication Meade[5] states that "quilting" cotton is superior to number 30 and "heavy duty" cotton in that there is less tendency to adhere to the rubber glove. We have had no experience with "quilting cotton."

Since January 1940 we have used spool cotton on 172 cases. The

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[2] Wolff, L. H., and Priestley, J. T.: Absorption of catgut in human beings; preliminary report, Proc. Staff. Meet., Mayo Clin. 14: 149–153, March 1939.

[3] Bower, J. O., Burns, J. C., and Mengle, H. A.: Superiority of very fine catgut in gastrointestinal surgery. Am. J. Surg. 47: 20–32, Jan. 1940.

[4] Meade, W. H., and Ochsner, A.: Spool cotton as suture material, J. A. M. A. 113: 2230–2231, Dec. 1939.

[5] Personal communication.

first cases were bilateral inguinal hernias. We decided to use spool cotton on one side and catgut on the other side.

The staff and patients on whom the operations were performed were impressed by the marked difference in the tissue reaction. The side on which cotton had been used presented a flat, pale, flexible scar free from the underlying structures while the side on which catgut had been used generally showed considerable tissue reaction and frequently a collection of sterile serum with consequent delay in healing. The use of cotton was gradually extended until one of us (J. B. L.) used it practically on all cases. In this series there were eight arthrotomies of the knee and the wounds healed with very little tissue reaction, a thin, flexible scar and no serum.

In addition to arthrotomies on the knee and other joints the following types of operations were performed: craniotomies, modified, Nicola operations, tenorrhaphies, cholecystectomies, appendectomies, herniorrhaphies, excisions of bursae, repair of ruptured biceps, amputations, partial gastrectomies, intestinal obstruction, laparotomies, open reduction of fractures, repair of early traumatic cases and compound fractures. There was no wound infection in the series that could be attributed to the suture material.

In two cases of appendiceal abscess and one with a post appendectomy abscess of the abdominal wall suture sinuses developed and closed promptly after excision of the scar and granulation tissue, the secondary closure being accomplished with cotton sutures.

In two cases of cholecystectomy superficial suture sinuses developed and healed rapidly on removal of the offending sutures. A few cases of appendectomy in our early series developed a small amount of serum as a result of using a continuous suture in the fascia of the external oblique in contravention of Halsted's tenets and was in no wise the fault of the suture material.

The scars in our series have shown less tissue reaction than we have experienced with any of the usual materials used for closure including Michel clips. They are pale and flexible with little tendency to adhere to the underlying structures and there is a minimum amount of serum. In the material removed following excision of scars we have found very little ingrowth of tissue into the suture material.

CONCLUSIONS

1. Spool cotton was used in 172 general surgical cases.
2. Results have been highly satisfactory due to minimal irritation of the tissue resulting in "dry healing."
3. Scars have shown slight reaction and are pale, flexible and freely movable over underlying structures.
4. Cotton thread is a cheap, readily available, transportable suture material.

5. It is easily sterilized by autoclaving or by boiling, care being exercised not to fracture the fibrils.

6. It is an invaluable suture material in peacetime and of the utmost importance in wartime, military surgery.

7. Cotton suture material is definitely more difficult to handle than catgut and prolongs the operative period but we feel that the more rapid and satisfactory healing of wounds with a consequent early return to duty status of the patient more than offsets this disadvantage.

8. If the sound principles laid down by Halsted for the use of nonabsorbable suture material are strictly adhered to we feel that cotton suture material will be more generally used.

SURGICAL STERILIZATION

By Weeden B. Underwood, Research Engineer, American Sterilizer Company

INTRODUCTION

The introduction within the past very few years of what can properly be described as "precision" sterilizers has brought about the imperative need for entire revision of practices relating to sterilization.

Our reason for describing the modern sterilizer as "precisionlike" is that it is controlled by temperature. With each performance of the machine, we know precisely what sterilizing effect is being applied to the load. We can and do reproduce exactly the same kind of steam in every cycle, whereas under the old system of pressure control, one never knew what temperature of steam was being applied. It might be, for example, at 20 pounds pressure, a maximum of 259° F. attained in a brief interval of time, or with no air evacuation it might not be higher than 228 degrees, after perhaps an hour or more exposure.

This statement is in no sense overdrawn. The hazards commonly encountered in attempts to sterilize in pressure steam without measuring the temperature of the steam are enormous.

Now bear in mind, please, that the accepted standards for surgical sterilization, as commonly in vogue today, were set up prior to 1933, around the performance of machines of this type, controlled only by gauging the pressure. Obviously those standards, to produce some reasonable margin of safety, had to provide exposure periods far in excess of what would be required if the sterilizing quality of the steam were definitely known. The generally accepted standard for routine surgical supply sterilization under this system came to be 20 pounds pressure for 1 hour.

Sterilizer manufacturers suddenly found in 1933 a way to eliminate this inefficiency of performance, but in far too many cases the technic in the surgery has failed to recognize this change, and is still adhering to the old standards at rather terrific expense. The net cost to

hospitals from this practice is far too great to pass over lightly. Properly prepared surgical loads need never be sterilized for more than 30 minutes, and the modern sterilizer will give a uniform performance showing a greater margin of safety with this exposure than the pressure-controlled sterilizer will average in more than double the exposure.

This saving of time is by no means the only factor of economy involved. With the modern temperature-controlled sterilizer the method of use always insures the development of temperature indicated, which at 15 pounds pressure means 250° F. This temperature, or in fact any temperature within the recognized sterilizing range, maintained for materially more than 30 minutes, will definitely bring about premature disintegration of even the coarser fabrics, such as muslin. When those fabrics begin to turn brown after repeated sterilization, that means the beginning of a rotting-out process brought about by too much heat, or by prolonged exposure to the proper temperature.

On the other hand, supplies which have been sterilized safely, but not oversterilized, will stand up much longer. Unfortunately there appear to be no exact figures relating to the amount of destruction that occurs annually in any hospital from oversterilization, but from the writer's personal observation the figures would in many cases be rather startling.

REGULATION OF STERILIZERS

To set up standards of sterilization which can be followed safely, necessitates other standards with respect to the use of the sterilizer. For all surgical processes, regardless of what the load may be—dry goods, fluids, instruments or utensils—there are many excellent reasons for maintenance of the same maximum operating temperature. There is no practical reason for changing the sterilizer regulation with respect to pressure and temperature, once it has been properly adjusted. Much investigative work indicates that this range should be 250° to 254° F., corresponding to pure steam at 15 to 17 pounds' pressure. It is usually not practicable to attempt closer automatic regulation, nor is it greatly desirable.

DEFINITION OF PERIOD OF EXPOSURE

With pressure and temperature regulation established, it is necessary to define exactly what is meant by "period of exposure." When steam is turned to the chamber for sterilizing, temperature as shown by the thermometer will advance with the initial rapid discharge of air at a rapid and uniform rate to about 240° F. Then the rate of advance will slow down materially at an increasingly slower rate until the maximum temperature is obtained.

When the thermometer indicates 240° F., the temperature of the steam surrounding the load will be several degrees higher—close to 250° F. This is a natural and to be expected lag accounted for in the action of the thermostatic valve which controls the chamber discharge. But knowing that this lag does exist, it must be taken into consideration, particularly in sterilizing heat-sensitive fluids for example, for any degree of overexposure may be critical. It means that when the thermometer indicates 240° F., the load is being subjected to nearly the full sterilizing influence of the steam, and in this respect it is well to bear in mind that measurable sterilizing effect begins at temperatures far below 250° F. Anson Hoyt, Albert L. Chaney, and Korine Cavell outline this fact clearly in the December 1938 Journal of Bacteriology. It is indicated that the resistant pathogenic organisms, such as spores of *C. oedematiens*, *C. welchii* and *C. tetani* are destroyed in direct contact with steam in 1 minute at 250° F., in 4 minutes at 240° F., and in 10 minutes at 230° F.

This will explain why we define period of exposure as beginning when the thermometer indicates 240° F., and continuing for the specified length of time during which the thermometer will slowly advance to the maximum of 250 to 254° F.

The idea seems to be prevalent among surgical supervisors that the larger the sterilizer the more time needed for sterilization. This is by no means true, assuming of course, that the load has been arranged properly for sterilization. The factor which normally fixes the period of exposure for the load, is not the size of the load, but the size and arrangement of the individual packages which make up the load. The arrangement of the packs in the sterilizer can be made to accommodate proper sterilization, but the individual packs must be analyzed to see what period of time must be allowed for penetration of steam through them. This is an important detail which needs study and thought, a detail which should be largely standardized so that all hospitals may follow the same general plan.

The old-fashioned lap set which included everything needed for a major operation from gowns to table covers, has no legitimate place in modern surgery. It may have been a convenient way to handle supplies, but it did impose handicaps which we are now recognizing. Such packs are too large for safe sterilization in any reasonable period of time. When so much material is crowded together in one pack, the tendency is to wrap the pack so tightly, in order to get it to hold together, that steam penetration is gravely retarded. We can still wrap up a lot of material in one pack when the occasion demands that, and sterilize with an ample margin of safety in 30 minutes but we must recognize the limitations and the various features which relate to the business of facilitating steam penetration.

As a rough outline of what the largest size pack should be, the sugges-

tion is made that the dimensions should not greatly exceed 20 inches in length, 16 inches in width, 12 inches in depth (fig. 6). Articles such as table covers, towels, gowns should be done up in flat packs—not rolled, and in the make-up of the pack, such flat articles should all lie flat relative to each other, to facilitate steam penetration as indicated by figure 7.

The basic reason for this detail is very interesting and full understanding of it will guide the nurse in proper arrangement of all kinds

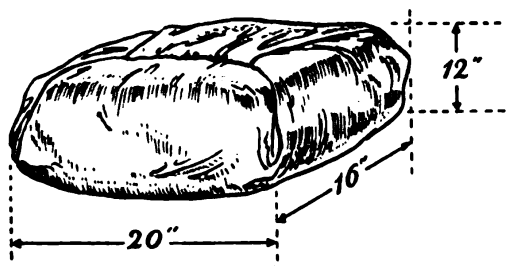

FIGURE 6.—Illustrates maximum permissible size of pack.

of loads. The movement of air and steam in the sterilizer is always directed from the top of the chamber toward the bottom. There is no appreciable sidewise motion of the gaseous content of the chamber.

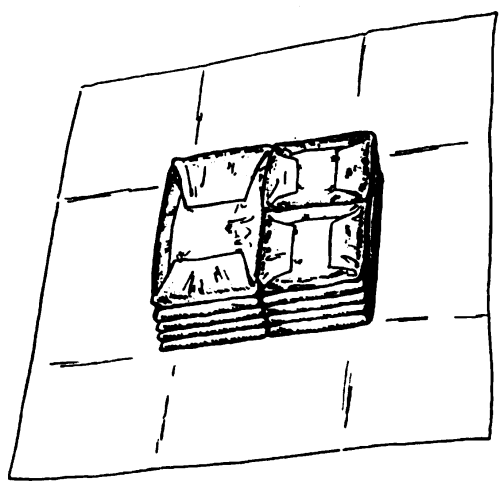

FIGURE 7.—Illustrates correct arrangement of articles in pack.

The top to bottom movement is governed by the difference in weight between air and steam, air being much heavier. When steam is admitted to the sterilizing chamber, it floats immediately to the top areas and compresses the air in the bottom. The air finds exit through the chamber drain piping at the bottom front end of the chamber, and it continues to flow freely as steam enters until hot steam finally follows through to the thermostatic valve, which then closes off and retains the steam, opening thereafter only from time to time as condensate or air pockets gravitate through. In this

manner, air evacuation is normally complete to a remarkable degree because air and steam are very reluctant to mix.

But we do not depend at all upon the accuracy and reliability of the thermostatic valve. Sometimes those valves close off from fatigue or the line becomes clogged with sediment so that air discharge is retarded or shut off altogether. In that case the pressure would build up, but the temperature would not advance. Instead, we measure the temperature of the discharge as it leaves the chamber, using a reliable thermometer. In this way, the operator is able to gauge her performance with absolute certainty that the steam temperature in the chamber is within the prescribed limits. If the sterilizer performs normally, the temperature will advance to the prescribed

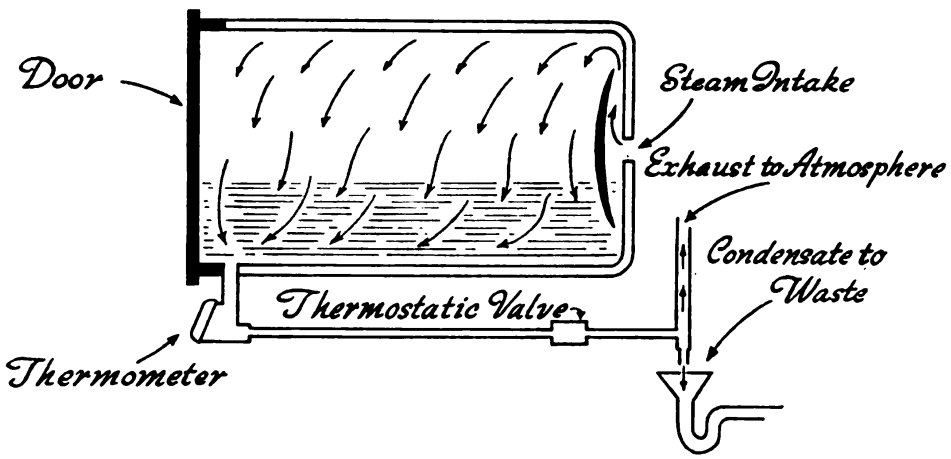

FIGURE 8.—Illustrates the flow of air and steam through the modern sterilizer.

range promptly and that range is automatically controlled. If the thermostatic valve has become clogged or for any reason the evacuation of air is interrupted, the difficulty will be apparent at once by failure of the thermometer to indicate temperature. In any event, the performance is safeguarded. Figure 8 illustrates how the modern sterilizer makes use of steam.

The known direction of the air and steam flow within the chamber leads directly to the proper way to arrange the load for most advantageous steam permeation. It must be remembered that every molecule of air in the chamber in contact with steam will gravitate below that steam. This holds true for the air trapped in the pores of the packages. As steam enters the pack from above, there must be a corresponding movement of air downward out of the pack. If this movement is retarded, the penetration of steam will be retarded and furthermore the pores in all fabrics tend to close up as the fabric is moistened, as in the presence of steam, thus slowing down the penetration. It must be obvious then that the more layers of fabrics imposed one on top of another in any given pack,

the more resistance to the downward flow of steam through the pack. Take for example, a pack made up of four gowns wrapped flat, stacked four deep, as illustrated in figure 9. If placed in the chamber flat side down, steam will have to penetrate all the many layers of fabric from top to bottom, and there will be a definite lag accounted for in this way. If, however, the pack is placed on edge, even though the material is rather tightly wrapped, there will be interstices between layers through which air can escape downward, so that steam can enter. In this case, to be specific, penetration will occur in just about half the time, if the pack is placed in the sterilizer on edge. This law holds true for all sorts of fabric materials.

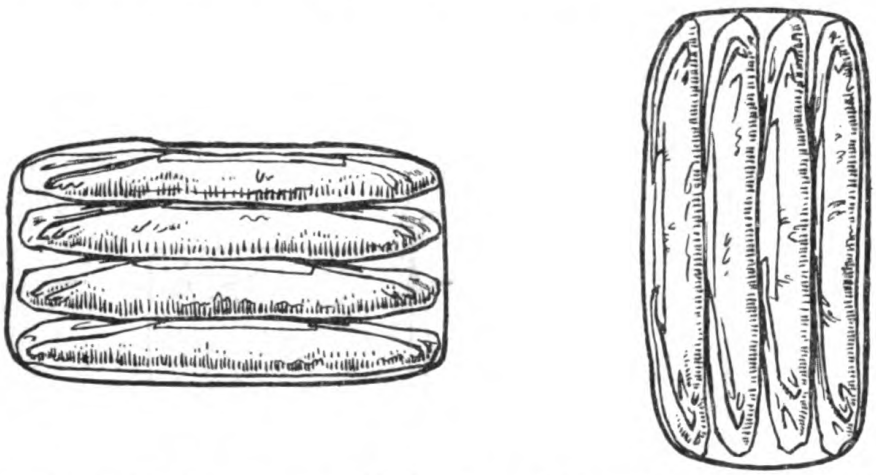

FIGURE 9.—Illustrates arrangement of packs in sterilizer; left is incorrect, right is correct.

Again, we have a similar application of the law in the sterilization of dressings, cotton pledgets, any porous supplies contained in enamel-ware jars, with loose-fitting covers. Placed upright in the sterilizer, even with the cover off, all air is trapped securely in the jar. It cannot get out, and in sterilizing in a perfectly performing machine, the effect will be equivalent to a completely air-trapped sterilizer. Some steam will enter the jar, but the ultimate temperature will be very low, relatively.

On the other hand, if the jar is placed on its side with the cover held on loosely with a piece of cotton tape, air will flow out and steam will take its place almost instantly. Sterilization will occur in a brief interval. Figure 10 illustrates this detail.

This illustrates a principle which the operator can constantly use as a guide. Imagine the article, such as the jar of dressings, filled with water. Right side up, the water is retained in the jar. Held on its side, the water promptly flows out. The same law holds for air in the presence of steam as in the sterilizer. The heavier air will flow out, and steam will take its place, if the jar is placed on its side.

Another similar application of the law is found in the sterilization of needles contained in bottles or test tubes. The stopper can never be anything less porous than a tight cotton stopper and the tube or bottle must be placed in the sterilizer on its side for the same reasons outlined above. Otherwise, sterilization will be decidedly questionable. With any tight stopper such as cork or rubber, regardless of how the tube or bottle be placed in the sterilizer, no steam can enter at all, and there will be no appreciable sterilizing effect even if sterilization is continued for hours.

Figure 11 illustrates the proper method of arranging surgical packs in a large sterilizer. In this case the machine is 32 inches wide, 48 inches high, and 84 inches deep, so in order to be sure of getting steam in contact with lower tiers, there are shelves arranged conveniently to separate the goods. In this way steam attacks the tiers in

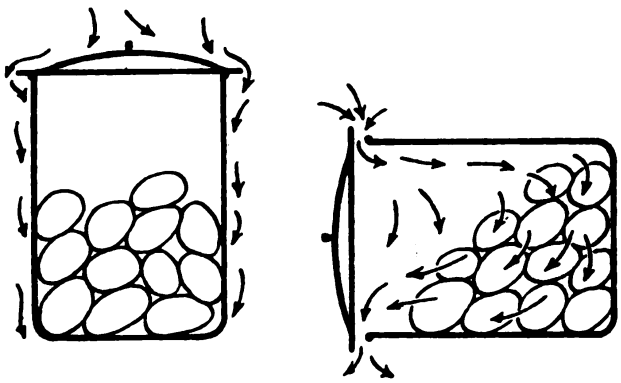

FIGURE 10.—Illustrates arrangement of jars in sterilizer; left is incorrect, right is correct.

the lower areas almost as quickly as those in the top, even though the complete load is very heavy, sterilization occurs with a nice margin of safety in 30 minutes. The heaviest pack is no larger than that illustrated in figure 6.

If the sterilizer is fairly deep and there is no provision for shelving, then the load must be arranged to accommodate the passage of steam to the lower packs. This can be done very satisfactorily by crossing the packs, stacking them like cordwood, leaving sufficient space between the packs throughout so that open spaces are left for the circulation of steam around the load from top to bottom as shown by figure 12. But under no condition should any heavy mass of material be located directly in contact and above another similar mass. This leaves no steam space at all above the lower pack and steam to enter it must first pass through the upper pack. Remember that steam is a gas. It will fill all the open spaces in the sterilizer very quickly and those open spaces largely govern steam circulation and penetration. The purpose must be to see that each pack is placed in the sterilizer on edge and that the packs are not crowded so close together

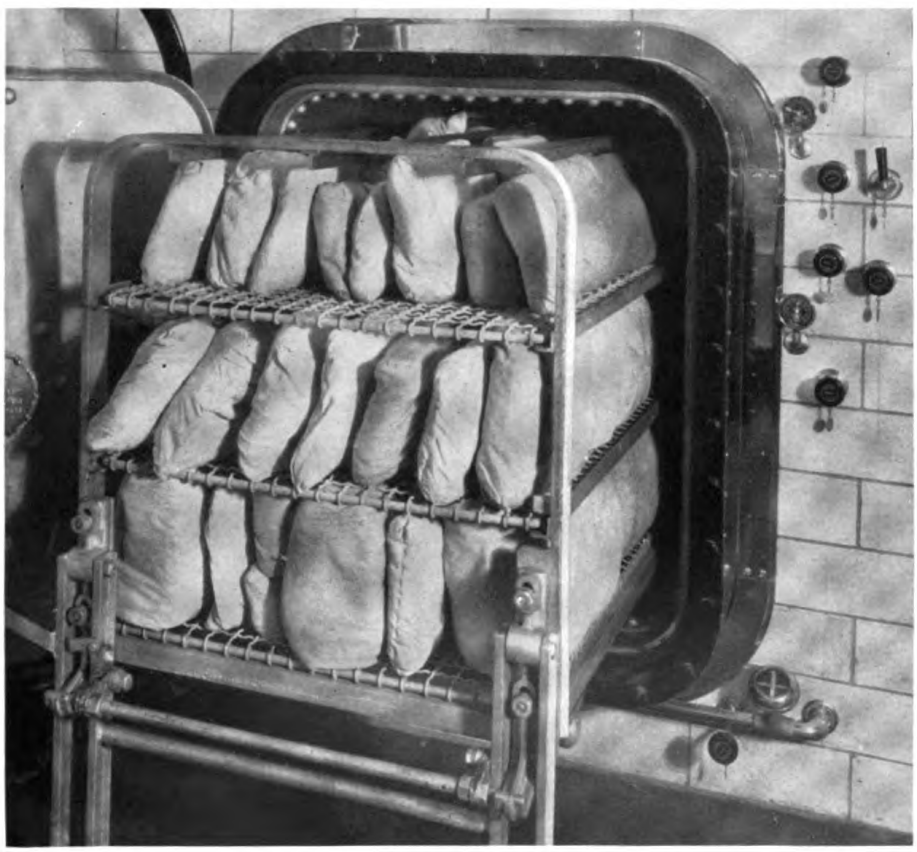

FIGURE 11.—ILLUSTRATES CORRECT ARRANGEMENT OF PACKS IN A LARGE STERILIZER WITH SHELVING.

FIGURE 13.—ILLUSTRATES PROPER WAY TO LOAD A DRUM.

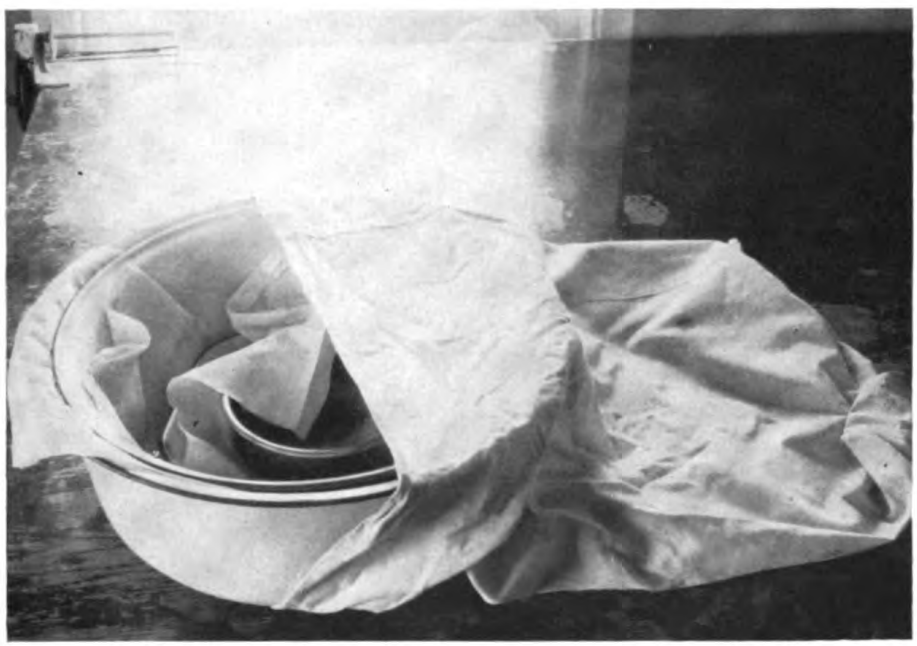

FIGURE 14.—ILLUSTRATES PACKING OF UTENSILS FOR STERILIZATION.

from any angle as to shut off the free contact of steam with individual packs.

Drums always present something of a problem in sterilizing, under the best of conditions. Enclosure in metal restricts the passage of steam to the contents and if the drum is improperly packed, the lag is extreme, necessitates abnormal periods of exposure. The older forms of drums were assumed to be dustproof, a totally erroneous impression. They were equipped with sliding bands around the sides with which portholes could be opened for sterilization, and closed when the drums were taken out. The covers were never approxi-

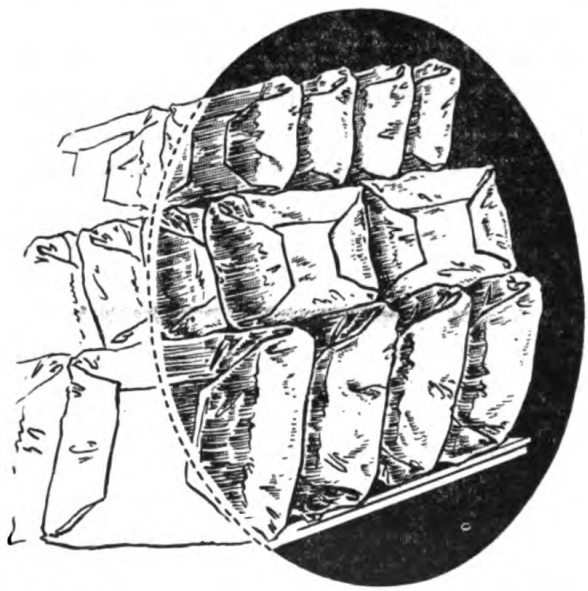

FIGURE 12.—Illustrates correct arrangement of packs in a large sterilizer not equipped with shelving.

mately dustproof, nor were the sliding bands surely tight, and the use of the sliding bands does restrict the size of the port openings through which steam may enter.

The newer drums have no sliding bands and have more and better distributed port openings which aid materially in getting the steam through the load. With either type of drum, however, safety from contamination through air contacts necessitates lining the drum throughout with two thicknesses of muslin, the same sort of covering as is used for exposed packs and used for the same purpose, to filter out dust particles which are surely drawn into the drum as it cools down following sterilization. Never use covers for drum lining made from canvas because canvas seriously retards the flow of steam.

Figure 13 illustrates the proper way to load the drum. The flat towels or sheets, etc., are placed loosely in the drum as shown. Then when the drum is placed on edge in the sterilizer, free passage for

steam is provided. Under no condition should the drum be packed tightly. Under ordinary conditions of loading, it is usually safer to sterilize drums by themselves and to expose for 45 minutes, whereas the same materials packed as described herein, but not in drums, are safely sterilized with a large margin, in 30 minutes.

STERILIZATION OF UTENSILS

Except for floor service where it would be thoroughly impractical to provide the more expensive pressure apparatus for the few utensils involved, it is no longer considered good practice to boil utensils used in surgery. Boiling under the best of conditions, while it does render the utensils sterile, leaves them with a coating of lime or scale which can never be scrubbed or scoured off in the usual process of cleaning. The result is most unsatisfactory, dirty appearing utensils. Pressure steam sterilization on the contrary, leaves no deposits whatever, and complete sterilization is accomplished with a large margin of safety in 15 minutes. The utensils used in surgery can be wrapped in muslin and stored dry, just the same as dressings, ready for use. Figure 14 illustrates how they can be nested and put up in suitable bags which are more convenient for packing. Of course the utensils are placed in the sterilizer on edge, to avoid pocketing air in them and to facilitate drainage of condensate from them.

STERILIZATION OF INSTRUMENTS

Routine sterilization of instruments by boiling requires 20 minutes exposure. In emergency, exposure is often reduced to 10 minutes and far too often, exposure is limited to 3 to 5 minutes. Boiling water never exceeds a temperature of 212° F., and the sterilizing effect is a product of this moderate temperature plus superabundance of moisture. The shorter periods will perhaps kill vegetative organisms such as streptococcus, but are not adequate for spores in the resistant stages.

Pressure steam on the contrary quickly heats instruments to a saturated-with-moisture steam temperature of 250° F., and it has been authoritatively shown that the resistant pathogenic spores are destroyed in 1 minute at 250° F. in direct contact with steam.

In sterilizing instruments we have the great advantage of this immediate and direct contact with steam, plus the second advantage of an abundance of moisture, the film of condensate which immediately deposits on the cool instruments as steam contacts them. The moisture factor is exceedingly important, essential to the quick break-down of crusts of blood or pus or tissue possibly concealed in joints or crevices.

For routine sterilization it is excellent practice to establish an exposure period of 10 minutes with the sterilizer regulated to maintain

254° maximum temperature. For emergency, the period of exposure can safely be reduced to 5 minutes, still retaining a considerable margin of safety. We are critical of exposure periods, regardless of the temperature used, of less than 5 minutes because of that possible factor of concealed crusts of dried matter in the joints and crevices. For their break-down, time is required, not added temperature, time in which

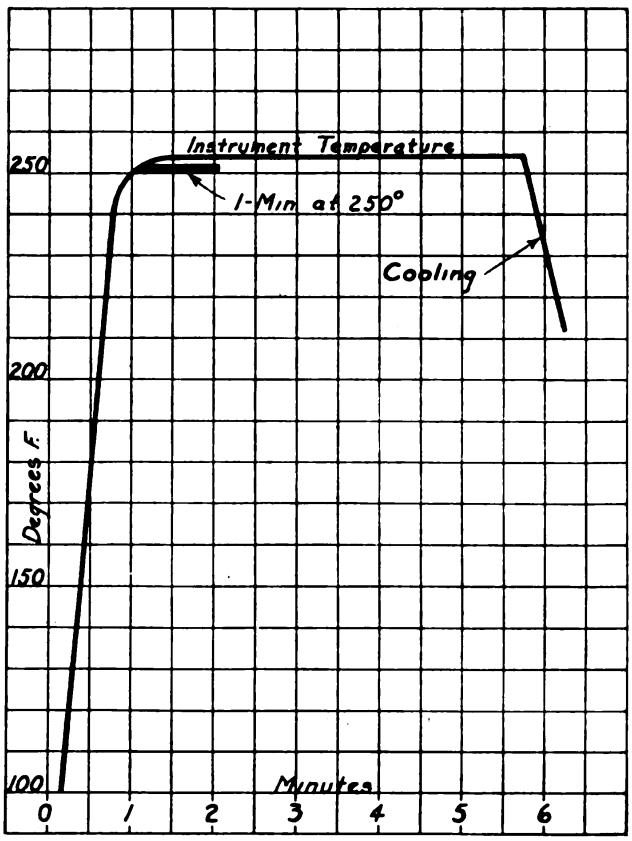

FIGURE 15.—Chart illustrates sterilizer temperature curve for 5-minute emergency sterilization of instruments.

the moisture factor can do its work—soften the crusts for permeation with steam. In extreme emergency, the period of exposure is often reduced to 3 minutes and it is true that this exposure undoubtedly carries a greater margin of safety than any of the so-called emergency processes used in boiling instruments. Figure 15 illustrates the actual development of instrument temperature in a modern sterilizer of the pressure type. This temperature chart shows the extreme speed with which instruments are directly contacted with steam in the pressure instrument sterilizer. Temperature has advanced to 250° F. in exactly 1 minute. Proper valving also permits exhaust of steam following sterilization in less than 1 minute. The com-

plete chart illustrates the recommended period of 5-minute exposure for emergency.

The loading equipment which has done much to popularize pressure steam sterilization of instruments is illustrated in figures 16 and 17. This type of tray carrier for pressure instrument sterilizers greatly simplifies the nurses' work. The sterilizer has two trays, each handled independently. The tray carrier permits the tray to be withdrawn half its length so that the operator can reach the rear handle without burning her arms or contaminating the load.

These machines are now coming into wide use for another very important purpose—washing and sterilizing septic instruments. The special trays adapted for this purpose are illustrated in figure 17. Instead of soaking infected instruments in some strong chemical sterilizing fluid for an hour or more, then laboriously washing them by hand—with some element of danger to the operator, the instruments are now taken direct from the operating table, jointed instruments are opened, and all are placed in an unperforated tray, covered with hot water to which is added in solution, one tablespoonful of trisodium phosphate. The tray of instruments is then sterilized for 15 to 20 minutes, routine process, which renders the instruments perfectly sterile and essentially clean, except for some tiny shreds of tissue which may adhere to joints. All that is needed now is to wipe off the instruments while still wet to get rid of the shreds of tissue, then resterilize them routinely. Practically all work of cleaning is eliminated and all danger in handling is avoided.

SOLUTION STERILIZATION

Based upon inaccurate assumptions, the idea is pretty general that certain heat sensitive fluids, such as procaine, cannot be sterilized successfully in a pressure sterilizer, but must be sterilized by the fractional method in nonpressure steam on three successive days, and some hospitals follow this practice which is expensive and tedious. Or, if they do use the pressure method, they find some fluids reacting badly to sterilization.

It is quite true that careless handling of the sterilizer, use of too much heat or unduly prolonged exposure to sterilization will destroy or seriously impair the usefulness of many fluids. When difficulty is encountered, the cause is, almost invariably, oversterilization. Calculating the period of exposure to accommodate the load does permit perfectly satisfactory sterilization of nearly every fluid encountered in the laboratory, as well as those commonly used in the surgery, in any modern temperature-controlled pressure sterilizer.

Calculating the exposure period to accommodate the load, however, refers specifically to the individual flask or bottle, not to the number of units in the sterilizer. For example, 1,000 cc. flasks of saline or dex-

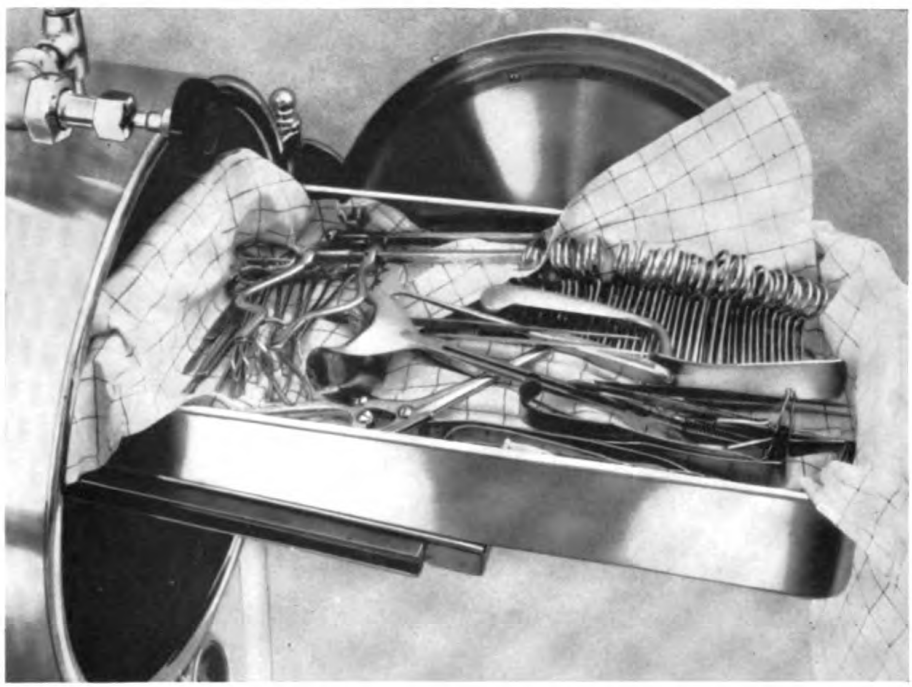

FIGURE 16.—ILLUSTRATES TRAY CARRIER FOR PRESSURE STERILIZATION OF INSTRUMENTS.

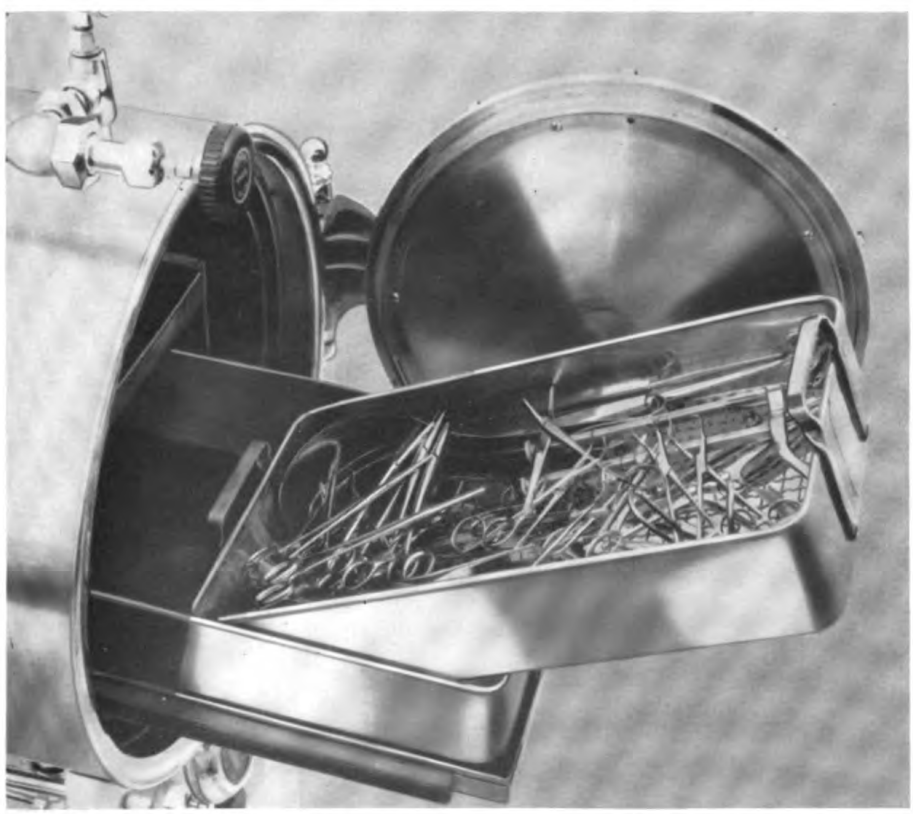

FIGURE 17.—ILLUSTRATES TRAYS FOR PRESSURE STERILIZATION OF INSTRUMENTS.

trose are normally and properly sterilized in 15 minutes exposure, with the sterilizer operating at a maximum controlled temperature of 254° F., while the average small bottle or flask of procaine, 1 to 2 ounces of fluid, will receive the same sterilizing effect by exposure for 8 minutes. Obviously, the two containers cannot be sterilized in the same load. Sterilization for 15 minutes would badly discolor the procaine.

The period of exposure is definitely not in direct ratio to the quantity of fluids nor to the capacity of the flasks. It cannot be calculated in this way. The relation is established proportional to the area of the container exposed to the heating effect of the surrounding steam, per unit of volume of the fluid.

Each solution load must be made up of containers of uniform size. The period of exposure, assuming that the load is made up of units of the same size, will be exactly the same whether the sterilizer contains one flask or some larger number. If the load is heavy, the time required to build up to this sterilizing range, as indicated by the thermometer, will be greater, but the actual exposure after this range has been attained will be just the same.

The following data have been prepared by the use of a potentiometer with thermocouples to establish proper exposure periods for various sizes of Florence pattern flasks. In each case the container was filled to about three-quarters of capacity.

	Exposure period
2,000 cc. flasks	20 mins.
1,500 cc. flasks	15 mins.
1,000 cc. flasks	15 mins.
500 cc. flasks	12 mins.
250 cc. flasks	8 mins.
125 cc. flasks	8 mins.
50 cc. flasks	6 mins.
2 ounce ordinary straight-sided thick glass bottles commonly used for procaine	8 mins.

Rapid exhaust of pressure from the sterilizer, following sterilization will surely cause violent ebullition of the fluid which will result in blown stoppers and an undue loss of fluid. At the best, under most careful regulation of heat, there will be a loss of about 3 percent which cannot be avoided. The average loss is about 5 percent and in some instances, an extra 5 percent is provided, of distilled water, in making up solutions to cover this expected loss by evaporation, leaving the fluid finally very close to the concentration intended.

The practice of permitting the entire sterilizer to cool down slowly after solution sterilization, literally prolongs the period of exposure to sterilizing influence and in the case of heat-sensitive fluids, such as procaine, the additional exposure may cause discoloration.

The most satisfactory method for cooling down solutions, after sterilization, calls for some skill and care in the regulation of the exhaust, as follows: At the close of the period of exposure, turn off all

heat, close the valve admitting steam to the chamber, then adjust the exhaust valve from the chamber, very slightly open to a degree that will permit exhaust of pressure to zero in not less than 7 to 10 minutes. Under this method the fluid can exhaust its heat without too violent ebullition at the same rate at which the pressure is reduced. This method has been followed successfully for many years by large institutions and it is recommended.

STERILIZATION OF RUBBER GOODS

Sterilization of rubber goods is always something of a problem because the period of exposure must be limited to 15 to 20 minutes to avoid serious destruction. This factor necessitates unusual care in preparation so that steam in the limited period of exposure can do a maximum of work.

It must be remembered that rubber is not porous. Steam will not pass through the walls though heat is readily transmitted. Again we have the problem of contacting the surfaces which must be sterile with the actual steam so that both moisture and heat are made use of. For the sake of emphasis, compare these data, relating to destructive effect of dry heat versus steam in sterilizing:

Dry heat requires at least 300° F. for at least 1 hour.
Direct contact with steam at 250° F. for 1 minute sterilizes.

It is obvious then, that heat conducted through rubber walls, without the moisture of the steam, would have very little sterilizing effect. In sterilizing rubber gloves the essential detail is of course, to render the entire outer surface of the glove which contacts the wound, absolutely sterile. It is less important to sterilize the inner walls because at best the surgeon's hands are not sterile. However, the need for great care in preparation for sterilization is clear.

If any two surfaces of rubber are held closely in contact with each other in the sterilizer, heat will conduct through the rubber but there will be no moisture contacting the surfaces that are held together. If the hand of the glove is collapsed in the sterilizer, steam cannot permeate to the fingers. If the wrist of the glove is folded back on itself as is usual in sterilizing, those surfaces contacting each other will exclude the moisture of the steam. How can we guard against these obvious faults?

The nearest practical approach to perfection with respect to the inside walls of the fingers and hand sections of rubber glove, is to place in the hand of the glove a crinkled mass of tough paper, perhaps quarter of an inch thick, extending in as far as the fingers, holding the hand surfaces of the glove well apart for the intake of steam. If the fingers are not collapsed by pressure in wrapping, they will hold open quite satisfactorily for the intake of steam. It seems impractical

to attempt to force paper or any other porous material into the fingers themselves. Paper does not restrict the passage of steam noticeably.

For the wrist section, it is highly desirable to place two or three thicknesses of crinkled tough paper or a pad of gauze within the fold to hold the surfaces apart. This will insure steam contact with those surfaces.

Then, in wrapping the glove, great care must be taken not to fold the glove at all since that will bring outer surfaces of the glove in contact with each other. Instead, let the glove assume its natural spread-out position with the wrist folded back, and cover it loosely with two thicknesses of muslin in the least restrictive manner possible. Place glove packs in the sterilizer preferably on edge, very loosely arranged so there is no crowding whatever, and sterilize gloves always by themselves. The period of exposure should never exceed 15 to 20 minutes.

Rubber tubing presents still more difficult problems since the obvious necessity is to sterilize the inside of the tubing. It is the writer's firm belief after much observation and thought that a large percentage of the reactions usually charged to something else, is directly due to improperly cleansed tubing. That is not a part of this story except incidentally, but it should be clear that if rubber tubing is used in which blood or any other foreign matter is permitted to harden, cleansing will necessitate some heroic procedure such as unusual mechanical cleansing with wicking drawn through the bore, or a very long continued flow of some cleansing fluid, followed of course by thorough rinsing with distillate.

However, the point is that perfect sterilization will be found altogether inadequate to render the tubing fit for use unless it has previously been properly cleansed.

In sterilizing, the difficulty involved hinges on the detail of getting steam to circulate through the tubing. It is not safe to depend upon the heat conducted through the walls. There must be moisture also, and if the tubing is coiled up in a tray, in accordance with the usual method, all air within the tubing will be perfectly trapped there and steam cannot enter. To arrive at the correct answer, we refer again to that rule of evacuating water from the container to be sterilized.

Figure 18 illustrates one very practical method of sterilizing the inside and the outside of tubing. The cylinder around which the tubing has been spiraled is plain cardboard, about 5 inches in diameter and 10 inches high, rolled up into the cylinder form and covered with muslin. The outfit undergoing sterilization is a standard Abbott transfusion set. Note that it is spiraled around the cylinder in such a manner that if the tubing were filled with water, it would all drain out. Similarly, in the presence of steam in the sterilizer the air will all drain out and steam will take its place almost instantly. Of course

the outfit is to be covered with two thicknesses of muslin and it must be sterilized in upright position.

With this preparation, perfect sterilization of the tubing will occur in 15 minutes' exposure and exposure should not exceed 20 minutes.

It is easily possible to improvise a similar cylinder from hand towels, around which the tubing can be spiraled and secured with safety pins, care being taken not to close off the bore of the tubing by pinching.

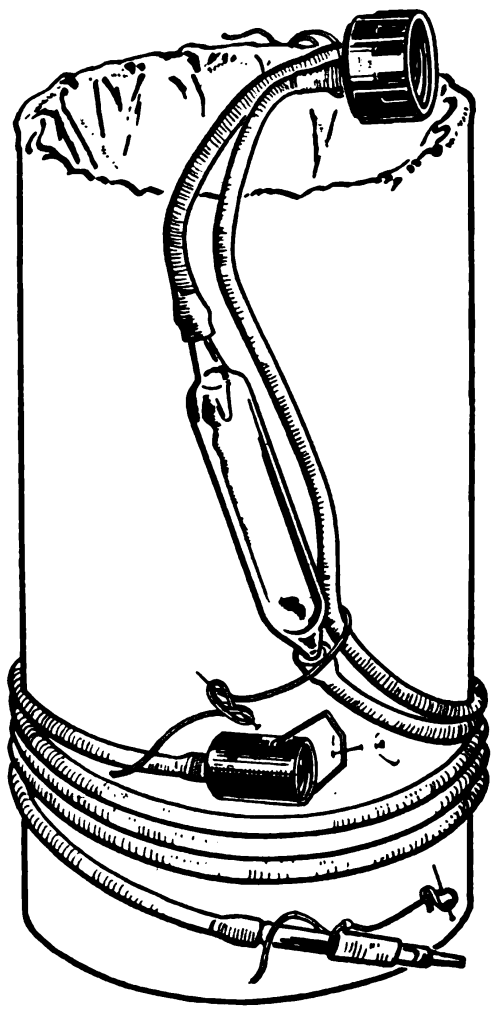

FIGURE 18.—Illustrates method for pressure sterilization of rubber tubing.

CARRELL-DAKIN SOLUTION [1]

METHOD OF PREPARATION FROM A CONVENIENT SOURCE

By Chief Pharmacist P. S. Gault, United States Navy, and Pharmacist's Mate, Second Class, E. E. Ozburn, United States Navy

In 1915 Dr. Alexis Carrell and H. D. Dakin introduced to the medical and surgical professions a solution of sodium hypochlorite (NaOCl) which is employed in the treatment of infected and post-surgical wounds.

[1] From U. S. Naval Medical School, U. S. Naval Medical Center, Washington, D. C.

The efficacy of this solution for infected wounds will not be touched upon here for it is well known. However, it is desired to call attention to some other conditions which may occur in the naval service for which its use is more or less specific, namely, mustard-gas burns,[2] poison-ivy poisoning and the so-called athlete's foot. With these conditions in mind a simplified method is suggested whereby this solution can be readily prepared aboard ship or in the field with the minimum of equipment and technic.

Modified Dakin's solution, as defined by the U. S. P. XI, contains not less than 0.45 gm. and not more than 0.50 gm. of sodium hypochlorite (NaOCl) in each 100 cc. of solution. The pH is controlled by buffering with sodium bicarbonate, so that a quick flash of red color is produced with alcoholic phenolphthalein (minimum alkalinity), but no pink color is produced when powdered phenolphthalein is agitated with another portion of the solution (maximum alkalinity).

The major problem on small ships and stations is to have a readily available source of the stronger sodium hypochlorite from which Dakin's solution can be made by dilution. Naturally, the best source would be sodium hypochlorite of 100 percent purity, but as the salt exists only in solution under ordinary conditions, this would be impossible.

There are four principal methods of obtaining this chemical in solution that might well be reviewed:

METHOD 1. *From calcium hypochlorite, $Ca(OCl)_2$.*—In this method the calcium salt is treated with sodium carbonate, and an exchange reaction takes place with the precipitation of calcium carbonate and the solution of the sodium hypochlorite according to the following equation:

$$Ca(OCl)_2 + Na_2CO_3 \longrightarrow 2NaOCl + CaCO_3$$

The calcium carbonate is then filtered off and the filtrate used for the preparation of Dakin's solution. This method is perhaps the most universally used and has the advantage that a high-test hypochlorite (Mathieson Alkali Works) containing a high percentage of available chlorine and almost completely water soluble, from which one can make a large quantity of Dakin's solution from a small amount of calcium hypochlorite, is on the market. Use of a high test hypochlorite is open to the following criticisms: (*a*) Even though great strides have been made in the manufacture of $Ca(OCl)_2$ in the past few years, it is still affected by heat, light, and moisture, and one can never accept with certainty the stated percentage on the container, especially if the container has once been opened; (*b*) an excess of sodium carbonate must be used to precipitate any calcium chloride left in solution (even then, some $Ca(OH)_2$ is frequently left in solution which may precipitate out later); and (*c*) one must filter the solution, dilute, buffer, assay and then adjust to desired percentage strength and re-assay, which requires time, standard solutions, apparatus, and some knowledge of analytical chemistry.

[2] Manual of the Medical Department, U. S. Navy, par. 868 (e).

METHOD 2. *From liquid chlorine and sodium carbonate.*—In this method a measured amount of chlorine is passed into a solution of sodium carbonate of known strength, resulting in the reactions shown in the following equations:

(1) $Na_2CO_3 + 2H_2O \longrightarrow 2NaOH + H_2CO_3$ (hydrolysis).
(2) $2NaOH + Cl_2 \longrightarrow NaOCl + NaCl + H_2O$

This method also requires special apparatus and technic and is the procedure employed at the United States Naval Medical School.

METHOD 3. *By the electrolysis of sodium chloride.*—In this method an exact amount of sodium chloride is placed in a measured amount of water, two electrodes (usually of some noble metal) inserted and a current applied, resulting in the reactions shown in the following equations:

(1) The NaCl ionizes: $NaCl \longrightarrow Na^+ + Cl^-$
(2) The sodium ion migrates to the negative electrode and gains one electron to become metallic sodium:

$$Na^+ + 1 \text{ electron} \longrightarrow Na^\circ$$

(3) The metallic sodium immediately reacts with the water to form sodium hydroxide and hydrogen gas:

$$2Na^\circ + 2H_2O \longrightarrow 2NaOH + H_2$$

(4) The chloride ion meanwhile loses one electron to the positive electrode becoming chlorine gas:

$$2Cl^- - 2 \text{ electrons} \longrightarrow Cl_2$$

(5) The chlorine gas immediately reacts with the sodium hydroxide simultaneously formed (reaction 3) to give NaOCl in solution:

$$2NaOH + Cl_2 \rightarrow NaOCl + NaCl + H_2O$$

By adjusting the original amount of NaCl and water as well as the current, the percentage of NaOCl can readily be controlled. The electrolysis appliances on the market are said to give an excellent product but are bulky and expensive.

METHOD 4. *By dilution of a strong hypochlorite solution.*—This method could be classed under any of the others, depending on how the strong solution was prepared. Generally these strong solutions are prepared by the electrolysis of sodium chloride solution (method 3). Method 4 is the one proposed as the most convenient for use in the naval service, particularly on small stations and ships or in the field. This method utilizes the various sodium hypochlorite solutions used as bleaching agents in households, laundries, etc., which are readily available in stores ashore and are carried on board ship by many ship's service stores. They are sold under various trade names and consist usually of 5 to 6 percent solutions of NaOCl made electrolytically from sodium chloride solutions. One quart of such a preparation is sufficient to make 10 quarts of Dakin's solution.

SUGGESTED METHOD OF PREPARATION

One volume of the commercial solution (5 to 6 percent strength of NaOCl) is diluted with 9 volumes of 1 percent sodium bicarbonate solution. Mix well and test as follows: To 20 cc. of this solution add about 20 milligrams of powdered phenolphthalein and shake gently for about 1 minute; if no red color is produced in the mixture the test indicates that the solution is not too alkaline in reaction. To 5 cc. of the solution add 0.5 cc. of 1 percent alcoholic solution of phenol-

phthalein; if a flash of red color is produced the test indicates that the solution is not acid in reaction. If these tests react as indicated the solution has the correct reaction and is ready for use. Should a red color be produced in the test with powdered phenolphthalein, this indicates that the solution is too alkaline in reaction. If no red color is produced in the test with alcoholic phenolphthalein the solution is acid in reaction. In either case the solution may be adjusted to the correct reaction by the addition of powdered sodium bicarbonate in the proportion of 1 gram to 1 liter of solution.

As the solution prepared by the proposed process contains approximately the same amount of sodium hypochlorite as that prepared by Carrell-Dakin and is relatively free of caustic alkali, it should have the same therapeutic effect. In fact, no difference in its action was noted with cases treated at the Naval Hospital, Washington, D. C.

The manufacturer of the commercial solution which was employed in connection with this work states that the solution loses approximately 1 percent of NaOCl per year under ordinary storage conditions. This was verified in the laboratory at this school. If ship's service stores carried a product containing not less than 5 percent of NaOCl for crew's use, there would be assured a turnover in the product and at the same time it would be available for emergency use.

Findings at the naval medical school indicate that the solution is best preserved in a cool dark place, as heat and light accelerate its decomposition. Most of the commercial products are furnished in amber colored glass bottles with rubber stoppers. The Dakin's solutions prepared from these commercial products are quite stable if kept in an ice box after their preparation.

CONCLUSIONS

The various methods of preparing Dakin's solution are briefly reviewed.

A cheap convenient source of sodium hypochlorite is suggested.

A method is suggested whereby anyone without special equipment or chemical training can prepare Dakin's solution from this source by diluting 1 volume of commercial sodium hypochlorite solution with 9 volumes of 1 percent sodium bicarbonate solution.

POSTOPERATIVE ATELECTASIS *

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

INTRODUCTION

Postoperative atelectasis is defined as the collapse of a lung or portion of a lung in such a manner that the affected portion contains little or no air and is greatly decreased in size.[1] It may be termed

* From the United States Naval Hospital, Brooklyn, N. Y.

[1] Iglauer, S.: Pulmonary collapse following tonsillectomy under local anesthesia; report of case, Arch. Otolaryng. 25: 382-388, April 1937.

massive when more than one lobe is involved, lobar when a single lobe is involved, and lobular when only the lobule of the lung is affected.[2] It has been suggested that the term *delectasis* be susbtituted as a single word to replace the terms postoperative atelectasis or postoperative massive collapse.[3]

In the opinion of Frederick Lord [1] "the clinical recognition of atelectasis as a common and important complication of various broncho-pulmonary conditions is the most important single advance in physical diagnosis since the time of Laennec."

If not quickly terminated by restoration of ventilation and drainage, the atelectasis is slowly succeeded by suppuration, first in the form of drowned lung, purulent bronchitis, or bronchopneumonia, with later abscess formation or possibly bronchiectasis. It is in diagnosing early cases as pneumonia, rather than as atelectasis, that danger lies.[4]

HISTORY

Massive collapse was first recognized and differentiated from lobar and lobular pneumonia at autopsy by Schenk [4] in 1811. His findings were confirmed repeatedly in the next 20 years and in 1832 the condition was given the name atelectasis by Joerg.[4] By the middle of the century various investigators, notably Traube [4] in 1836 and Mendellsohn [5] in 1844, had produced atelectasis experimentally by artificial occlusion of a bronchus. It was then that W. T. Gardner [4] of the University of Edinburgh in 1850, formulated the theory that atelectasis was caused by a mucous plug which obstructed the bronchi and acted as a ball valve, allowing air to escape during expiration but preventing the access of air on inspiration. Credence was lent to this theory in 1860 by Bartels [4] who demonstrated mucous secretion in the bronchi corresponding to the atelectatic part of the lung at autopsy.

This condition was first recognized clinically in 1886 by William Pasteur [4] of Middlesex Hospital, London, who collected 14 cases in the next 3 years.

The invention of the bronchoscope in 1897 and its improvement by Jackson in 1904 offered a new method of clinical investigation and treatment. However, it was not until 1925 that Tucker [2] of Philadelphia demonstrated by bronchoscopic examination the cause of postoperative massive collapse in a case to be complete obstruction

[2] Tucker, G.: Bronchoscopic observations on obstructive pulmonary atelectasis, Arch. Otolaryng. 13: 315-325, March 1931.

[3] Watterson, W. H.: Detelectasis, M. Bull. Vet. Admin. 12: 149-153, Oct. 1935.

[4] Carson, W. J.: Postoperative pulmonary atelectasis, West. J. Surg. 44: 683-687, Dec. 1936.

[5] Maier, R. J.: Massive collapse (postoperative massive atelectasis), Ill. M. J. 68: 498-505, Dec. 1935.

of the main bronchus and its subdivisions tributary to the area of atelectatic lung by thick tenacious secretion.

INCIDENCE

As so aptly stated by Snyder [6] the incidence of postoperative atelectasis varies with the diligence with which it is sought and the diligence with which prophylactic measures are enforced. It is variously reported as accounting for 10 to 70 percent of all postoperative pulmonary complications. Brunn and Bill [4] reporting the postoperative complications in 456 major operations in the clinic of thoracic surgery at the University of California, 1930, had 33 pulmonary complications, 67 percent of which were atelectasis. Eliason and McLaughlin [7] at the University of Pennsylvania Hospital, 1934, found a total of 129 cases of postoperative pulmonary complications following 8,864 operations in 10 years, about 25 percent of which were atelectasis. In 1935 Christopher and Schaffer [7] at Evanston Hospital reported 12,494 operations over a 5-year period, with 77 postoperative pulmonary complications, about 10 percent of which were atelectasis. It is noteworthy that 80 percent of these occurred following lower abdominal surgery in which muscles playing an important part as accessory muscles of respiration, *i. e.* the obliques, were cut, whereas most observers have found that postoperative atelectasis occurs most frequently following operations on the upper abdomen.

PATHOLOGY [4 5]

On opening the thorax at autopsy the atelectatic lung is found smaller than normal, completely airless, solid, and dark in color. If placed in water it sinks. The decrease in the size of the lung is compensated for by traction displacement of surrounding structures. The heart and trachea are drawn to the affected side and may lie completely within it. The diaphragm on the affected side is elevated and the ribs depressed. The contralateral lung and the unaffected portion of the diseased lung are emphysematous. On section considerable thin serum can be expressed. The bronchus and its divisions tributory to the involved area are occluded by tenacious mucous secretion.

Microscopic sections show the alveoli collapsed or partially collapsed. Collapsed alveoli have their lining epithelial cells closely packed together; and those partially collapsed are filled with thin fluid containing few phagocytic cells. The walls of the alveoli appear

[6] Snyder, H. E.: Postoperative pulmonary atelectasis; report of 11 cases, Ann. Surg. 102: 5-15, July 1935.
[7] Christopher, F. and Schaffer, J. M.: Postoperative atelectasis, Am. J. Surg. 32: 197-203, May 1936.

moderately edematous with little or no inflammatory reaction. Some of the bronchioles are collapsed; others contain thin fluid and their walls are edematous. The capillaries, arterioles, and venules are dilated and filled with red blood cells.

CLINICAL PHYSIOLOGY [8]

Henderson draws attention to the part played by postoperative loss of tonus and intratissue pressure in connection with the occurrence of atelectasis. Due to this postoperative loss of tonus, especially in the diaphragm and accessory muscles of respiration, there is a marked loss of vital capacity. The diaphragm may be relaxed to such an extent that in x-ray pictures its shadow is as much as 7 to 8 cm. headward from the normal position. All this results in such a diminution in the size of the thoracic cavity and such deflation of the lungs that many minute deaerated areas are produced. This may be termed "collapse without symptoms," and has been found in more than 80 percent of all laparotomies. With the lungs partially deflated, it sometimes happens that mucus accumulates in one or another of the major airways until it is completely closed. In that part of the lung which is thus occluded the gases contained in the occluded lobe or lung are absorbed into the blood and the lobe or lung becomes completely collapsed. As one lung collapses it draws the mediastinum after it and atmospheric pressure inflates the other lung. Hence, massive collapse is almost necessarily confined to one lung, for the collapse of one lung tends to expand the other.

ETIOLOGY

It appears to be the general belief that the direct cause of atelectasis is the obstruction of a bronchus and associated bronchioles with mucous secretions and the subsequent absorption of air from the lung or portion of lung tributary thereto. There have been many suggestions relative to predisposing causes which act as the forerunner of this occurrence. Among these may be cited the following:

(1) Reduction of vital capacity and high diaphragm due to loss of tonus,[8] splinting of muscles from pain and meteorism, tight abdominal bandages, and air under the diaphragm.[1] Overholt states that after upper abdominal operations vital capacity is reduced 64 percent and after lower abdominal operations, 40 percent. He found that morphine increased the vital capacity by reducing pain.

(2) Spasmodic reflex of the bronchioles causing them to become

[8] Henderson, Y.: Wesley M. Carpenter lecture: atelectasis, massive collapse and related postoperative conditions, Bull. New York Acad. Med. 11: 639–656, Nov. 1935.

occluded, thereby permitting future absorption of air in the segment of lung involved.[9] Sante [9] believes this reflex to be due to infection or insult to the region of the vagus supply.

(3) Active compression on lower lobes of lungs by elevated diaphragm.[1]

(4) Bronchospasm and edema, possibly due to allergic effect of the injection of procaine hydrochloride.[1] In this connection Brown [5] cites a case without excessive mucus thought to be due to acute edema of the mucous membrane of the bronchi similar to angioneurotic edema and relieved by the administration of epinephrin and cocaine.

(5) Respiratory disturbance due to inflammation affecting the muscles of the crus [9] situated behind the peritoneum and placing out of action one-half of the diaphragm and its synergistic and antagonistic muscles.

(6) Brown [7] believes that spinal anesthesia predisposes to atelectasis due to its marked depression of the respiratory center, not only during the operation but for a considerable time thereafter.

(7) Local infection in the upper respiratory tract, local or general infections elsewhere, and debilitated states.[6]

(8) Preoperative and postoperative medication. Atropine, by tending to thicken secretions, would seem to be favorable toward the production of atelectasis. Unguarded use of morphine, sufficient to depress the cough reflex, would obviously predispose to its development.

SYMPTOMATOLOGY

Symptoms vary with the suddenness of the onset of bronchial plugging, size of plugged bronchus, alterations in intrapleural pressure, and disturbance in position of mediastinal structures.[7] The symptoms may be so slight that the condition is discovered incidentally during physical examination.

Usually the onset is sudden, within the first three postoperative days, and is ushered in with a sharp rise in temperature, 102° to 104°, rapid pulse, and respiratory rate of 30 to 40 per minute. There is dyspnea, orthopnea, cyanosis, and pain in the chest. Pain is influenced by the location and extent of adhesions. Sputum is scant at first, but becomes profuse, mucopurulent, and may contain blood. Snyder [6] states the sputum is never rusty or bloody. The white blood count ranges between 15 and 20 thousand.

PHYSICAL FINDINGS

The patient appears cyanotic, anxious, and dyspneic. Physical signs in the chest vary with the amount of lung tissue involved. In a typical case the usual findings are as follows: On inspection the

[9] Wilson, H. B.: Massive collapse of lung complicating parturition, Am. J. Obst. & Gynec.: **31**: 667-670 April 1936.

affected side appears more or less fixed and the intercostal spaces are narrowed and depressed. On percussion the affected side is found to be dull or flat, the diaphragm is high and immobile, and the area of cardiac dullness is shifted toward the affected side. On auscultation breath sounds and voice sounds are absent or suppressed with a bronchial quality. Later there are rales. The unaffected side is hyperresonant and the breath sounds are increased. In children scoliosis is often noted.

X-RAY FINDINGS [7]

The half of the diaphragm on the affected side is elevated, the rib interspaces are narrowed, the mediastinal structures deviated to the affected side, and the lung tissue affected gives a dense homogeneous shadow. The space lost by atelectasis is taken up by a compensatory emphysema of the uninvolved lung.

The fluoroscope may play an important part in the diagnosis in children, in whom the mediastinum is seen to move toward the affected side on inspiration.

DIFFERENTIAL DIAGNOSIS

Traction displacement of the heart with unilateral elevation of the diaphragm serve to distinguish atelectasis from pneumonia, pneumothorax, hydrothorax, dilatation of the stomach, ileus, peritonitis, pleurisy, pulmonary embolism with infarction, cardiac failure, coronary thrombosis, dextrocardia, massive tuberculosis, and diaphragmatic hernia, with which conditions it has been confused.

PROPHYLAXIS

Preventive measures are aimed at the maintenance of normal respiratory excursions, normal blood volume, normal body temperature, and the avoidance of dehydration. Effective active measures are the administration of mixtures of carbon dioxide and oxygen, the administration of intravenous fluids, enforced deep breathing exercises every hour, frequent change in position, and the use of expectorants.

The time to think of atelectasis is before, during, and after operation, and there are many things to avoid in order to decrease the frequency of its occurrence.

An attempt should be made to avoid the following: Operating in the presence of respiratory infections; the use of atropine, since it thickens secretions; the aspiration of a foreign body or secretions during vomiting and with inhalation anesthesia; pressure on patient's chest by assistants or instruments; prolonged operating time; postoperative distension of the bowel and stomach; and the administration of frequent large doses of morphine.

TREATMENT

An attempt should be made to dislodge the offending mucous plug by placing the patient on the unaffected side with the head low, rolling him back and forth and slapping him over the affected area. Deep breathing is enforced by the inhalation of carbon dioxide and oxygen and coughing is encouraged. Morphine in sufficient doses to control the pain, but not to depress the respiratory system and cough reflex are advocated. If the treatment proves successful, it should be followed up with repeated postural drainage and the other active measures already cited as of value in prevention. For intractable cases bronchoscopy is necessary.

According to Tucker [2] it is not possible to aspirate the obstructive secretion from every small bronchus, but when the air gets into a lobe of the lung and can find its way into the obstructed air vesicles, the patient can cough out the secretion that could not be removed at bronchoscopic aspiration. The essential thing is that the larger bronchi be opened by aspirating the obstructing secretion, allowing the air to get in. Following bronchoscopy it is necessary to maintain drainage by continuance of previously mentioned therapeutic measures.

PROGNOSIS AND MORTALITY

Under proper treatment complete relief of symptoms can usually be expected within a few days, although in some unresolved cases a productive cough persists for weeks. Relief of the initial distress is usually abrupt, with fall in temperature, pulse and respiration in a matter of a few minutes or at most only a few hours.

The mortality of atelectasis *per se* is low, but, as Faulkner [7] points out, postoperative atelectasis is of more than academic interest because it may be complicated either by a bronchopneumonia or a lobar pneumonia with fatal results.

SUMMARY OF CASES

Over a period of approximately 3 years, 14 cases of postoperative atelectasis occurred in the United States Naval Hospital, Brooklyn, N. Y.

In 1935, 73 major operations were performed, in which there were 2 cases of postoperative atelectasis, an incidence of 2.6 percent. In 1936 among 262 major operations, there were also 2 cases of postoperative collapse, an incidence of 0.76 percent. In 1937 the incidence increased to 3.14 percent, with the occurrence of 10 cases in 318 operations.

Analysis of the cases of postoperative atelectasis occurring in 1937 reveals the following facts: Seven occurred following inguinal hernior-

rhaphy; and one each following suture of perforated duodenal ulcer, gastrostomy, and varicocelectomy. Varicocelectomy is the only minor operation included, and that because it was followed by atelectasis. Local anesthesia was used in eight cases and spinal in two cases.

Three cases occurred within 24 hours after operation, 5 cases in 48 hours; 1 in 72 hours, and 1 in 9 days.

In 5 of the cases the collapse was massive and required bronchoscopy and 5 were partial, resolving by crisis with postural drainage and the repeated administration of oxygen-carbon dioxide mixture. Two cases eventuated in death, 1 from bronchopneumonia and peritonitis, the other from carcinoma.

INSULIN TREATMENT OF BURNS

By Lieutenant Stephen E. Flynn, Medical Corps, United States Navy

The treatment of burns with insulin has been used locally in a number of cases, but only recently have I used insulin internally and externally in the treatment of severe burns and discontinued the use of tannic acid sprays and the forcing of large amounts of fluids internally.

I heartily agree with Trusler, Egbert, and Williams[1] that the tannic-acid theory of the treatment of burns is fallacious. This form of treatment is not only fallacious but it adds insult to injury by sealing in the dead cells and their toxins, and any infection that may form under the tannic acid crust has to be eliminated by the circulatory system, increasing the chances of penumonia and pyelitis, two major complications following severe burns.

Much of toxemia caused by cell destruction from the burn can be eliminated from the burned area by doing a debridement, procuring a good drainage, and setting up a reaction.

The administering of insulin in dextrose intravenously in burn cases is a new treatment and the results have been striking in cases that are in shock and where life and death hang in the balance. After setting up a reaction, 50 cc. of 50 percent dextrose is given intravenously along with 15 units of U–20 insulin. The insulin steps up the metabolism by helping the shocked system in the catabolism of the dextrose. After every severe burn there is a great loss of blood sugar and the intravenous injections of 50 cc. of 50 percent dextrose with 15 units of insulin should be given daily, until the patient's blood sugar is within normal limits, or at any time when the patient appears to be losing ground. Care should be taken that no more than 15 units of insulin are given at any one time.

The use of 500 cc. of 10 percent dextrose has been discontinued in

[1] Trusler, H. M., Egbert, H. L., and Williams, H. S.: Burn Shock: Water intoxication as a complication, J. A. M. A. 113: 2207, Dec. 1939.

severe burn cases for it only makes the tissues "water logged" and causes excessive work upon the circulatory system. The patient becomes water intoxicated when large amounts of fluids are given.

Shock from severe burns is not due to the loss of body fluids alone but mostly due to the loss of blood plasma that due to the increased permeability of the capillaries passes into the body tissues, acts as an irritant to the tissues, and causes shock. A big factor in secondary shock is the loss of the hormones in the plasma that have escaped into the tissues and the failure of the glands of internal secretion to restore this loss due to the initial shock. At this critical time the addition of insulin in dextrose intravenously plays a big factor in restoration of life. The sooner dextrose and insulin are given in shock and followed with whole blood, or blood plasma transfusion, the more rapid will be the results in combatting shock.

Blood plasma has many advantages over whole blood in time of emergency because pool blood plasma can be given without any regard for grouping. This allows plasma to be given without any delay. Although whole blood is the best restorative agent it requires time-consuming laboratory tests, leading to delay in treating severe shock.

A blood chemical analysis should be done frequently in order to keep as close as possible within normal limits, as the success in recovery depends greatly upon this factor. Five hundred cubic centimeters of whole blood are given to restore this normal relationship unless the hematocrit reading becomes too high, and then the same amount of blood plasma is given intravenously whenever the fluid balance falls below normal.

Before debridement one quarter grain of morphine sulphate should be given. If the patient is a child the dose should be regulated according to Young's law. After debridement the patient is kept in an electric-heat cradle with the temperature around 90° or higher if necessary. At this stage of the treatment in combatting burn shock the administration of oxygen inhalations is helpful and very necessary.

Oxygen therapy to be useful must be continuous as long as shock or cyanosis is present. The oxygen tent is the most satisfactory method in the administration of oxygen but is also the most troublesome and difficult to operate. The nasal catheter method is the simplest and costs comparatively little but a large cylinder with flow meter are needed as for the tent. The success of the nasal catheter method depends on care in placing the catheter and keeping it there. A good rate of oxygen flow is around 6 liters per minute. This is an adequate rate of flow and the cost at this rate is not great. After oxygen therapy has been started, the burned area is cleansed with hydrogen peroxide, following which insulin is sprayed on the burned surface with an atomizer twice a day. The local application of

insulin to the burned area has given very encouraging results. It increases the metabolism of new cell growth and forms healthy granulation tissue which decreases the length of disability.

I have not attempted to discuss the treatment of cases where shock is not a problem, for they will usually recover without any treatment. But I have found, that where repair has been sluggish, 10 to 15 drops of insulin dropped directly on the raw burned area, and then covered with a dressing, has stimulated the growth of healthy granulation tissue and hastened recovery.

In cases where skin graft is necessary I have found the Thiersch graft most satisfactory, but often the grafts become sluggish in growth and the application of hydrogen peroxide followed by insulin locally around the graft border will stimulate their activity. These grafts must be watched daily and at any sign of inactivity more insulin should be applied to be area.

Insulin ointment has been used by some writers with success but I have had better results by not using vaseline for it forms an oily film over the raw surface that will not be removed with hydrogen peroxide and it keeps the insulin from having its full effect in stimulating new growth.

SUMMARY

1. Tannic acid sprayed over burned area seals in toxins or any infection that may develop under the crust.

2. In the treatment of burn shock the following procedure is recommended:

(*a*) Administer oxygen and give blood plasma without delay. After typing, Wassermann, and chemical analysis of the blood are completed, give blood transfusion if indicated.

(*b*) Dextrose 50 cc. of 50 percent with insulin should be given early.

(*c*) Debridement and setting up a reaction.

(*d*) Keep blood chemical analysis within normal limits by proper administration of fluids. Indiscriminate forcing of large amount of fluids may cause a fatal water intoxication.

3. Insulin applied locally on burned area stimulates healthy granulation tissue.

4. Thiersch grafts most satisfactory for repair, followed by application of insulin daily to stimulate growth of grafts.

BRONCHOGENIC CARCINOMA

A REVIEW OF THIRTY VERIFIED CASES

By Commander P. P. Maher, Medical Corps, United States Navy, and Lieutenant A. H. Staderman, Medical Corps, United States Navy

The purpose of this paper is to present an analysis of proven cases of primary lung carcinoma which were observed in the United States Naval Hospital, Philadelphia, Pennsylvania, during the period from January 1, 1937, to August 1, 1939.

The population of this hospital consisted predominantly of veterans of the World War. The average daily census was about 550. Active service patients numbered about 200 per day. The average age of the veterans was approximately 44 years. All were males.

INCIDENCE

There were 17,503 admissions (see table 1) during the period of 31 months. Of these, 1,978 had laboratory tests only, in conjunction with out-patient examinations. The remainder, or 15,525, constituted the body of in-patients.

TABLE 1.—*Hospital admissions*

	1937	1938	1939 (to Aug.1)	Total
In-patients	5, 441	6, 047	4, 037	15, 525
For tests only	768	1, 048	162	1, 978
Total	6, 209	7, 095	4, 199	17, 503

DEATHS.—Deaths numbered 866 during the period. The coroner's cases, which totaled 77, seldom were autopsied in the hospital, and hence were of no practical importance in this series. The remainder or 789 cases, form the basis of this study.

TABLE 2.—*Deaths*[a]

	1937	1938	1939 (to Aug. 1)	Total
In-patients	268	319	202	789
Autopsies	114	156	99	369

[a] Percentage of autopsies, 46.9.

Death was due to cancer in 128 cases. The majority of these were confirmed on autopsy, while the remainder had the diagnosis established prior to death by microscopic examination of biopsied material. Cancer of the colon led with 28 cases or 21.8 percent.

Bronchogenic cancer was a close second with 24 cases or 18.7 percent. Cancer of the stomach followed with 18 cases, or 14.1 percent.

TABLE 3.—*Cancers verified by microscopic study*

	1937	1938	1939 (to Aug. 1)	Total	
				Number	Percent
All cancers	31	61	36	128	
By organs:					
Colon	4	11	13	28	21.8
Bronchus	6	10	8	24	18.7
Stomach	3	10	5	18	14.1
Testicle	3	3	1	7	5.5
Pancreas	1	5		6	4.7
Bladder	3		2	5	3.9
Esophagus	1	1	2	4	3.1

A bronchogenic carcinoma was found in each group of 646.6 patients.

RACE.—Race apparently is of no significance in this disease.

TABLE 4.—*Race incidence in 30 cases*

White	27	Colored	3

The incidence of the disease in the colored population is believed to be practically the same as in the white population.

AGE.—The youngest patient was 37.5 years of age. The oldest patient was 67 years of age. The average age was 50.15 years.

TABLE 5.—*Age incidence in 30 cases*

Decades:	*Incidence*	Decades—Continued	*Incidence*
30–39	2	50–59	8
40–49	14	60–67	6

ETIOLOGY

The cause is unknown. Irritation is believed by many authorities to be a factor in the causation of this disease, but proof is lacking. Only 3 patients gave a history suggestive of an occupational hazard. The majority, however, stated that they had smoked tobacco, especially cigarettes, to a moderate degree. None of the patients were agricultural workers.

TABLE 6.—*Statistical summary of cases reviewed*

Case	Initials	Age	Race	Occupation	Onset	Admission	Death	Duration in months
1	E. G.	48	W	Automotive engineer	(?)	Oct. 25, 1936	Jan. 2, 1937	
2	L. R.	48	W	Meat cutter	Feb. 16, 1937	Feb. 23, 1937	Mar. 20, 1937	2
3	C. D.	50	C	Porter	Feb. 1, 1937	Feb. 27, 1937	May 11, 1937	3.4
4	C. K.	57	W	Unknown	Jan. 31, 1937	Mar. 31, 1937	Apr. 23, 1937	2.8
5	W. L.	67	W	Glass blower	Nov. 18, 1936	Apr. 18, 1937	June 17, 1937	8
6	W. W.	45	W	Captain, U. S. M. C.	Dec. 11, 1936	May 11, 1937	Oct. 24, 1937	11.5
7	A. R.	37.5	W	Driller	Feb. 14, 1937	Aug. 14, 1937	Sept. 24, 1937	7.3
8	E. D.	50.5	W	Architect	Aug. 1, 1937	Sept. 29, 1937	June 26, 1938	11
9	T. D.	51.5	W	Cook	Oct. 14, 1937	Nov. 19, 1937	May 24, 1938	8.3
10	F. F.	44	W	Captain U. S. A.				
11	C. R.	50	W	Electrician	Jan. 1, 1938	Feb. 18, 1938	Apr. 9, 1938	4.3
12	G. L.	42	W	Clerk	Sept. 5, 1937	Mar. 5, 1938	Mar. 27, 1938	7.6
13	H. W.	47	C	Laborer	Dec. 31, 1937	Mar. 31, 1938	Apr. 9, 1938	4.3
14	E. W.	40	W	Salesman	Nov. 18, 1937	Apr. 18, 1938	Aug. 21, 1938	10
15	B. E.	60	W	Cable splicer	Dec. 1, 1937	May 11, 1938	June 25, 1938	8
16	J. McM.	49.5	W	Gas station worker	(?)	May 24, 1938	Oct. 15, 1938	
17	M. G.	62.5	W	Showman	Apr. 24, 1938	June 24, 1938	July 20, 1938	3
18	M. B.	45	W	Credit man	Feb. 1, 1938	July 20, 1938	(1)	
19	S. G.	45	W	Miner	July 20, 1938	Sept. 20, 1938	Oct. 17, 1939	15
20	J. McG.	50	W	Teamster	Aug. 4, 1938	Oct. 4, 1938	Dec. 15, 1938	4.3
21	A. C.	47.5	W	Merchant	Nov. 1, 1938	Dec. 16, 1938	July 23, 1939	10
22	W. W.	47	C	Laborer	Aug. 11, 1938	Aug. 11, 1939	(2)	
23	W. G.	63	W	do	Dec. 20, 1938	Jan. 10, 1939	Apr. 11, 1939	4.6
24	M. S.	44	W	Unknown	Jan. 10, 1939	Jan. 31, 1939	July 10, 1939	7
25	C. S.	67	W	Baker	Jan. 1, 1939	Mar. 17, 1939	Mar. 27, 1939	4
26	F. M.	38.5	W	Iron worker	Feb. 1, 1939	Mar. 20, 1939	May 14, 1939	3.5
27	T. S.	58	W	Chief water tender, U. S. N.	Feb. 1, 1939	Mar. 24, 1939	Apr. 21, 1939	2.6
28	C. H.	43	W	Truck driver	Mar. 23, 1939	June 9, 1939	July 23, 1939	4
29	E. C.	61.5	W	Cable worker	(?)	June 13, 1939	June 30, 1939	
30	C. S.	51	W	Bartender	May 17, 1939	June 28, 1939	July 26, 1939	2.4

[1] Living June 1940.
[2] Living August 1940.

The past medical histories were not remarkable.

The family histories also yielded information of no significance. No familial predisposition to cancer could be definitely established.

MORBID ANATOMY

The cancer rises from the basement membrane of the bronchus during the process of metaplasia. The pavement cells are undifferentiated. The most malignant, and, incidentally, the most radiosensitive, tumors persist as undifferentiated types—but the intermediate forms are prone to develop into the glandular, or the adenocarcinomatous types. The squamous cell types of primary carcinoma of the bronchus are probably the least virulent. The so-called "oat-cell" and the medullary forms are considered to be variants of the undifferentiated type of new growth.

Bronchogenic carcinoma manifests a predilection for the hilar areas. The majority of the tumors in this series were located in the main bronchi. A few parenchymatous tumors arose in bronchi of the second order, while the peripheral growths had their origin in bronchi of the third order.

The tumor usually grows into the lumen of the bronchus. Rarely it may encircle it. In either event the lumen is at first partially constricted due to invasion and it is eventually occluded. Infection

distal to the narrowed lumen, first, in the form of bronchitis, and, later, bronchiectasis, atelectasis, abscess or empyema soon develops.

Following a bronchostenosis there is a collapse of the pulmonary salient. The collapse may be partial, lobar, or massive. The negative pressure is increased in the atelectatic area. The rib cage contracts, the dome of the diaphragm rises and the medistinal contents shift toward the involved side because of the increased negative pressure.

The duration of life following bronchostenosis and infection distal to the obstruction is short.

THE LYMPHATIC SYSTEM OF THE LUNGS

Knowledge of the intrapulmonary lymphatic system has been contributed by Miller.[1] This system consists of two sets of vessels, the superficial and the deep. The superficial or the pleural set communicates with the deep or the intrapulmonary set by short connecting vessels equipped by valves which open toward the pleura. Except for the lower surface of the lower lobes, the flow of lymph in the superficial vessels immediately beneath the pleural surface is in the direction of the hilum and eventually into the lymph nodes of the bilum. The flow of lymph in the deep system is through lymphatics along the pulmonary blood vessels and bronchi toward the hilum and eventually into the lymph nodes of the hilum. It is seen that the flow of both the deep and the superficial lymphatics is toward the hilum and eventually through the mediastinal nodes to the thoracic duct or the bronchomediastinal trunk.

TABLE 7.—*Lobe of lung involved in 30 cases*

Right lung	Number	Left lung	Number
Upper lobe	10	Upper lobe	8
Middle lobe	1	Lower lobe	4
Lower lobe	6		
Total	17	Total	12
Percent	56. 7	Percent	40

Site unknown, 1, percent 3.3.

[1] Miller, W. S.: The Lung, published by Charles C. Thomas, Springfield, Ill. 1937.

METASTASES

The cases with extensive metastatic lesions in the skeleton and the brain had small primary adenocarcinomas of the bronchi (cases 1, 7, 16, and 24). Two of these, cases 1 and 7, developed from secondary bronchi.

Case 7 had a negative roentgenogram of the chest. X-ray examination of the ischium, however, revealed an extensive, destructive, osteolytic process in the left ischial ramus.

The two patients, cases 16 and 24, who had craniotomies performed at another hospital for primary tumors of the brain showed postoperatively small lesions in the lung fields on the roentgenograms. The brain lesions were secondary deposits of carcinoma primary in the bronchi. These findings were confirmed at postmortem examinations.

Two patients who harbored undifferentiated bronchogenic carcinoma had widespread metastatic lesions of the skull, brain, brain stem, and pelvis.

The cases with squamous carcinoma were least virulent. The carcinomatous spread was most often restricted to the hilar areas, the mediastinum, the liver, the adrenals, and the kidneys.

It is apparent that small bronchial carcinomas, especially those of the adenocarcinomatous or the undifferentiated type, arising from the secondary and tertiary bronchi, may have a widespread metastatic dissemination before signs referable to the lungs are present. The symptomatology centers around these secondary manifestations, thus confusing the true nature of the lesion.

TABLE 8.—*Metastases in 17 autopsied cases*

Tissue	Number	Percentage
Hilar glands	9	53
Mediastinal glands	9	53
Kidneys	7	41
Liver	6	35
Brain	5	29. 5
Adrenals	4	23. 6
Ipsolateral pleura	3	18
Abdominal lymph nodes	3	18
Pancreas	3	18
Pelvis	2	12
Pericardium	2	12
Contralateral pleura	1	6
Supraclavicular glands, bilateral	1	6
Heart	1	6
Rib	1	6
Skull	1	6

SYMPTOMS

TABLE 9.—*Symptoms in 27 cases*

Symptom	Number	Percentage
Cough	18	66. 6
Loss of weight	18	66. 6
Dyspnea	11	40. 7
Hemoptysis	10	37. 0
Weakness	9	33. 3
Pain—thoracic	9	33. 3
Expectoration	9	33. 3
Pain—extrathoracic	6	22. 2
Headache	4	14. 8
Hoarseness	4	14. 8
Mental confusion	4	14. 8
Night sweats	4	14. 8
Anorexia	4	14. 8
Fever	2	6. 4
Wheezing	2	6. 4
Hiccough	1	3. 7

COUGH.—Cough was the most frequent and the earliest of the symptoms. This at first was nonproductive but later in the disease it was accompanied by mucopurulent or purulent sputum. The expectoration was usually less than that in bronchiectasis or lung abscess.

WEIGHT LOSS.—This symptom was present as frequently as cough. However, it appeared much later in the course of the disease and it was not mentioned as a first symptom in a single instance.

PAIN.—Pain of the thoracic type was not an early symptom. It was frequently described as being dull, aching, and continuous. It was worse at night. The extrathoracic type of pain was the presenting symptom in five cases, or 18.5 percent. Headache and sciatica predominated.

HEMOPTYSIS.—This symptom was complained of in only one instance but it was present at some time during the course of the disease in 37 percent of the cases. It is believed this symptom was frequently minimized and often not mentioned. However, the amount of blood streaking of the sputum early in the disease may be so slight that the patient fails to recognize it. Hemoptysis was of the massive type in only one case in this series.

DYSPNEA.—This was present in 40.7 percent of the cases. It was usually a late symptom. It appeared to be proportional to the number of lobes of the lung involved in the process and the toxic effects of the disease.

WEAKNESS.—This was also a frequent complaint. It was a late symptom.

TABLE 10.—*First symptoms in 27 cases*

Symptom	Number	Percentage
Cough	7	25. 9
Pain—thoracic	6	22. 2
Pain—extrathoracic	5	18. 5
Headache	4	14. 8
Hemoptysis	1	3. 7
Weakness	1	3. 7
Hoarseness	1	3. 7
Wheezing	1	3. 7
Mental confusion	1	3. 7
	27	100. 0

PHYSICAL FINDINGS

Many of these patients when first seen appeared to be in a surprisingly good state of health. Their color was good and the majority were ambulatory.

TABLE 11.—*Physical findings in 27 cases*

Physical finding	Number	Percentage
Weight loss—more than 10 pounds	20	74. 0
Impaired resonance	19	70. 3
Diminished breath sounds	13	48. 1
Moist rales	8	29. 6
Bronchial breathing	7	25. 9
Respiratory lag	7	25. 9
Apices contracted	6	22. 2
Vocal resonance increased	5	18. 5
Liver enlarged	5	18. 5
Whispered voice increased	4	14. 8
Vocal resonance diminished	4	14. 8
Whispered voice diminished	4	14. 8
Venous engorgement of head and arm	4	14. 8
Recurrent laryngeal nerve paralysis	3	11. 1
Trachea deviated to affected side	3	11. 1
Supraclavicular adenopathy	2	6. 4
Sciatica	2	6. 4

ROENTGEN FINDINGS

The roentgen findings showed atelectasis in 25 of the 27 cases. The phenomenon of atelectasis, or collapse of the lung, was due in each case to bronchostenosis caused by malignant invasion. The air in the pulmonary salient distal to the occluded bronchus was absorbed, thereby producing a collapse of the affected portion of the lung, with secondary shift of the mediastinal contents to the affected side, and narrowing of the intercostal spaces on the same side. Elevation of the dome of the diaphragm was noted in 6 cases. The above physical factors produced a moderately dense, homogenous shadow with con-

cave margins, most characteristic when found in the upper lobes. Frequently evidences of secondary infection were manifest.

A shadow of the tumor was apparent on the roentgenogram in only 20.6 percent of the patients.

TABLE 12.—*X-ray findings in 29 cases*

X-ray finding		Number	Percent
Atelectasis		27	93. 0
Massive	2		
Lobar	13		
Partial	12		
Narrowing of the rib interspaces on the side of the neoplasm		20	68. 8
Shift of the mediastinal contents to the side of the neoplasm		14	48. 0
Elevation of the dome of the diaphragm on the side of the neoplasm		6	20. 6
Primary tumor nodule		6	20. 6
With no atelectasis	2		
With secondary atelectasis	4		
Osteolytic lesions of skeleton		4	13. 8
Skull	2		
Pelvis	2		
Negative chest x-ray with peripheral metastases		1	3. 45
No roentgenogram of chest		1	3. 45
Obstructive emphysema		1	3. 45

Of these 30 cases, 1 case had no roentgenogram and the diagnosis was established at autopsy. In 2 cases craniotomy was performed before the diagnosis of primary cancer of the lung was suspected. Of the remaining 27 cases, the roentgen diagnosis was correct in 20, or 74 percent. In 7 cases the initial roentgenograms suggested the following erroneous diagnoses:

Case 1.—Lymphoblastoma.

Case 2.—Pleurisy with effusion, massive, left.

Case 5.—Fibrosis and pleurisy left upper lobe.

Case 7.—Roentgenogram negative. At autopsy a small primary carcinoma was found in a secondary bronchus to the left lower lobe.

Case 9.—Fibrosis right upper lobe.

Case 11.—Lung abscess.

Case 23.—Increased trunk markings.

The shift of the mediastinum to the affected side was present in 14 cases or 48 percent. This sign was thought to be of considerable prognostic importance when a marked shift was present for it represented a mediastinum apparently free from malignant invasion. Cases with little or no mediastinal shift and a large atelectalic involvement of the lobar type at operation or autopsy revealed a "frozen" mediastinum.

ILLUSTRATIVE ROENTGENOGRAMS

Case 7.—A. R. Roentgenogram (fig. 19) revealed an extensive, destructive, osteolytic process in the left ischial ramus. Because of persistent intense pain in

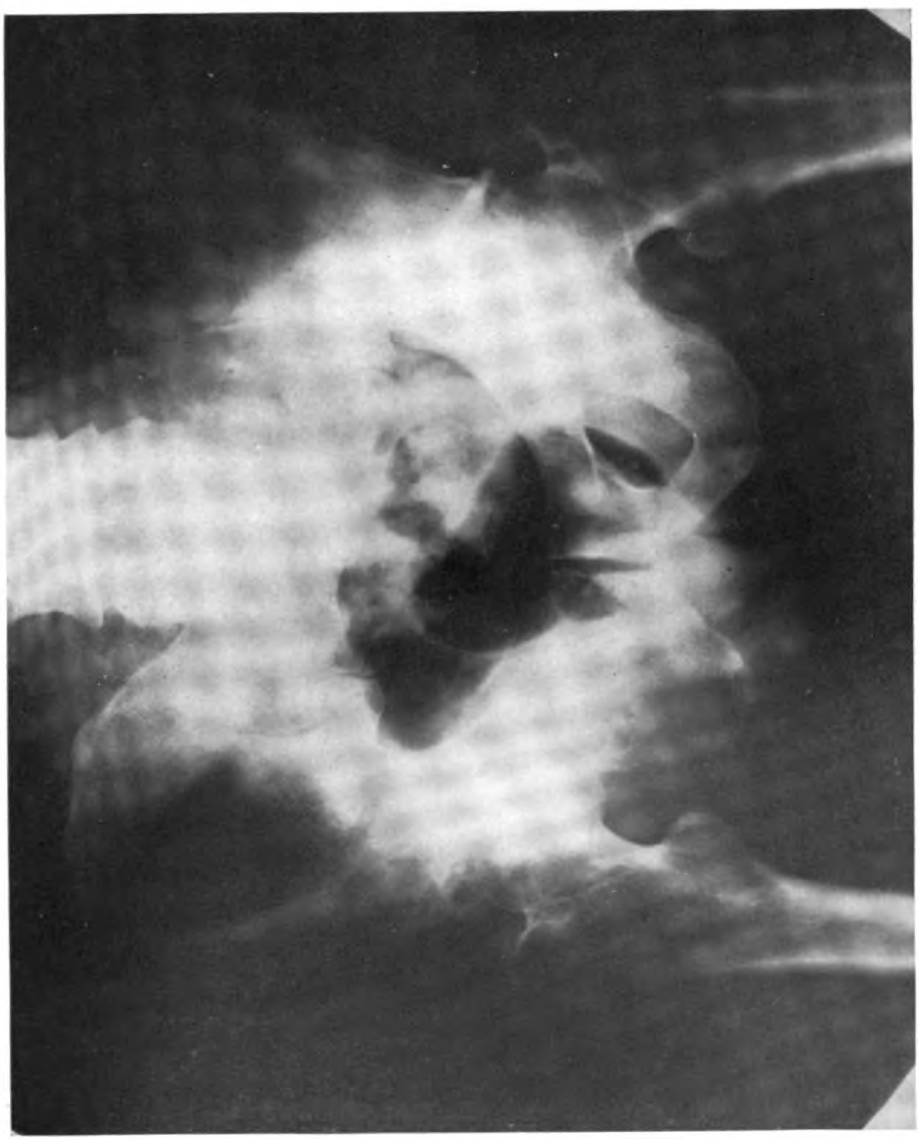

FIGURE 19.—CASE 7, A. R. OSTEOLYTIC PROCESS IN LEFT ISCHIAL RAMUS.

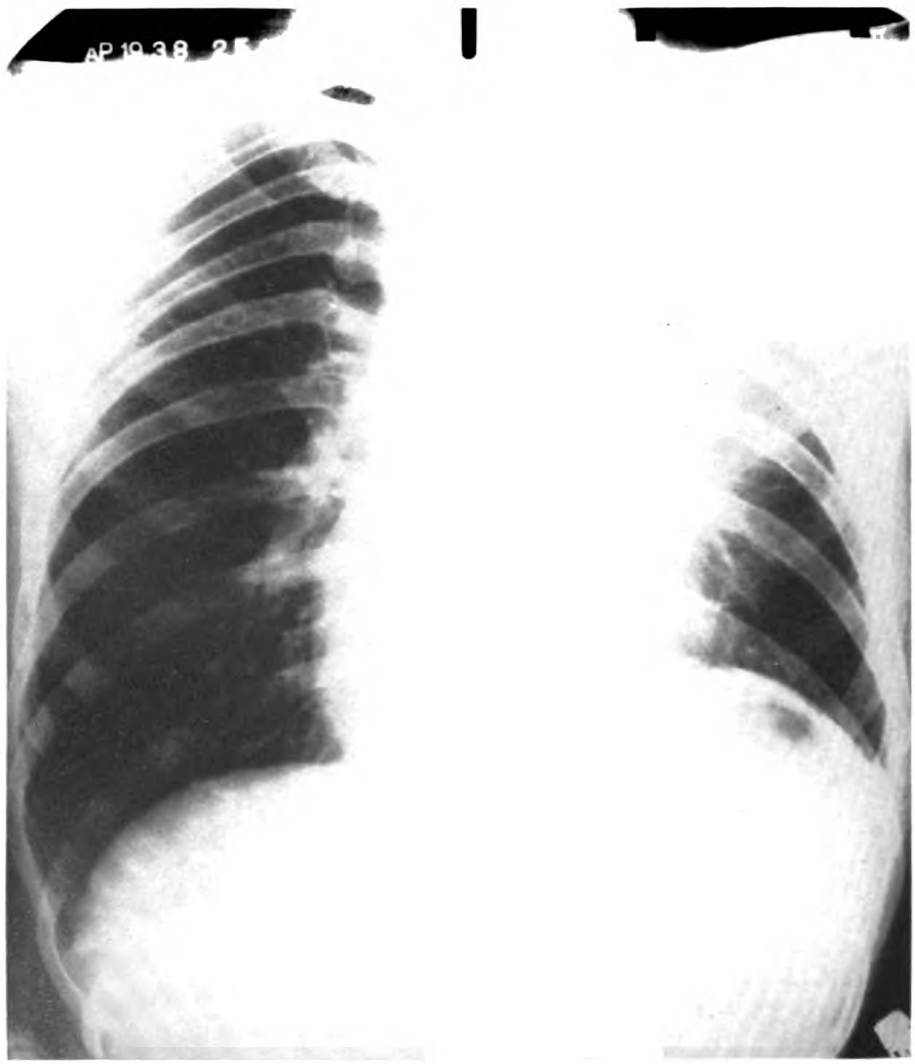

FIGURE 20.—CASE 14. E. W. TUMOR NODULE UPPER LEFT HILUM AND ALMOST COMPLETE ATELECTASIS LEFT UPPER LOBE.

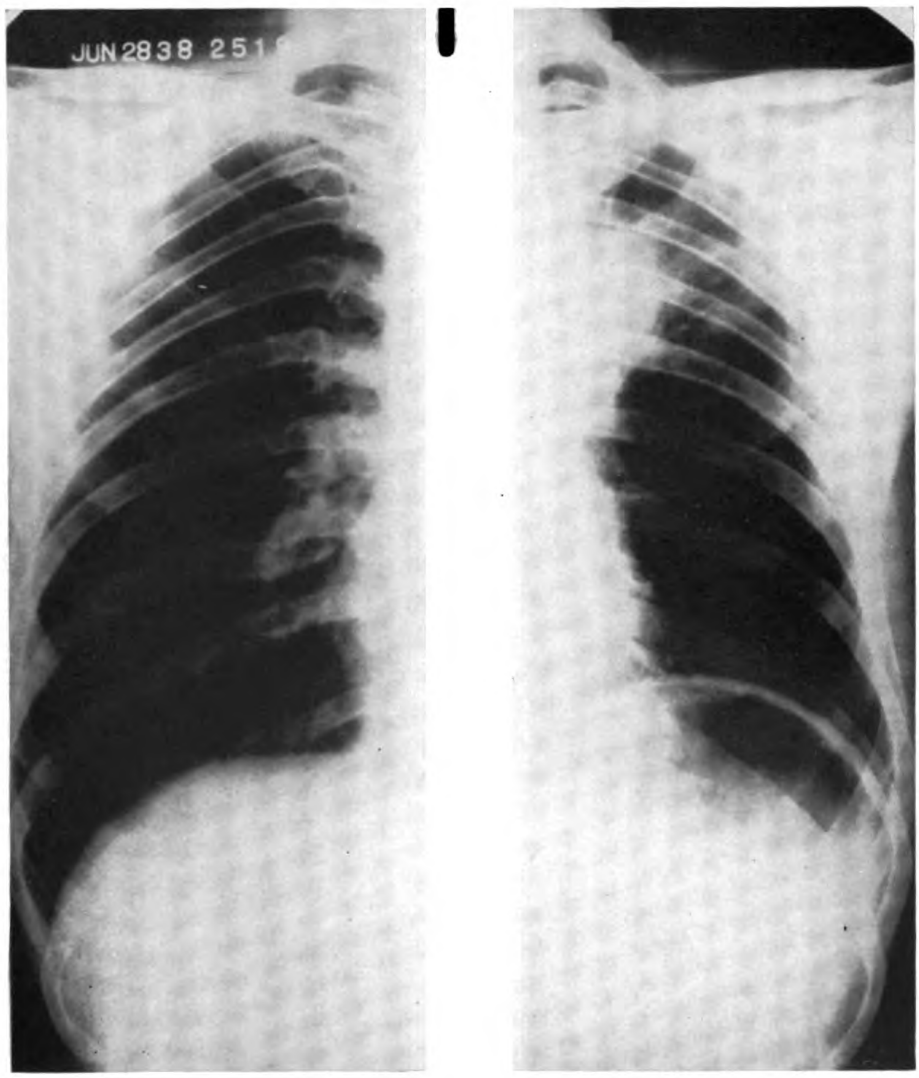

FIGURE 21.—CASE 14, E. W. TUMOR NODULE MORE APPARENT AFTER ROENTGEN THERAPY.

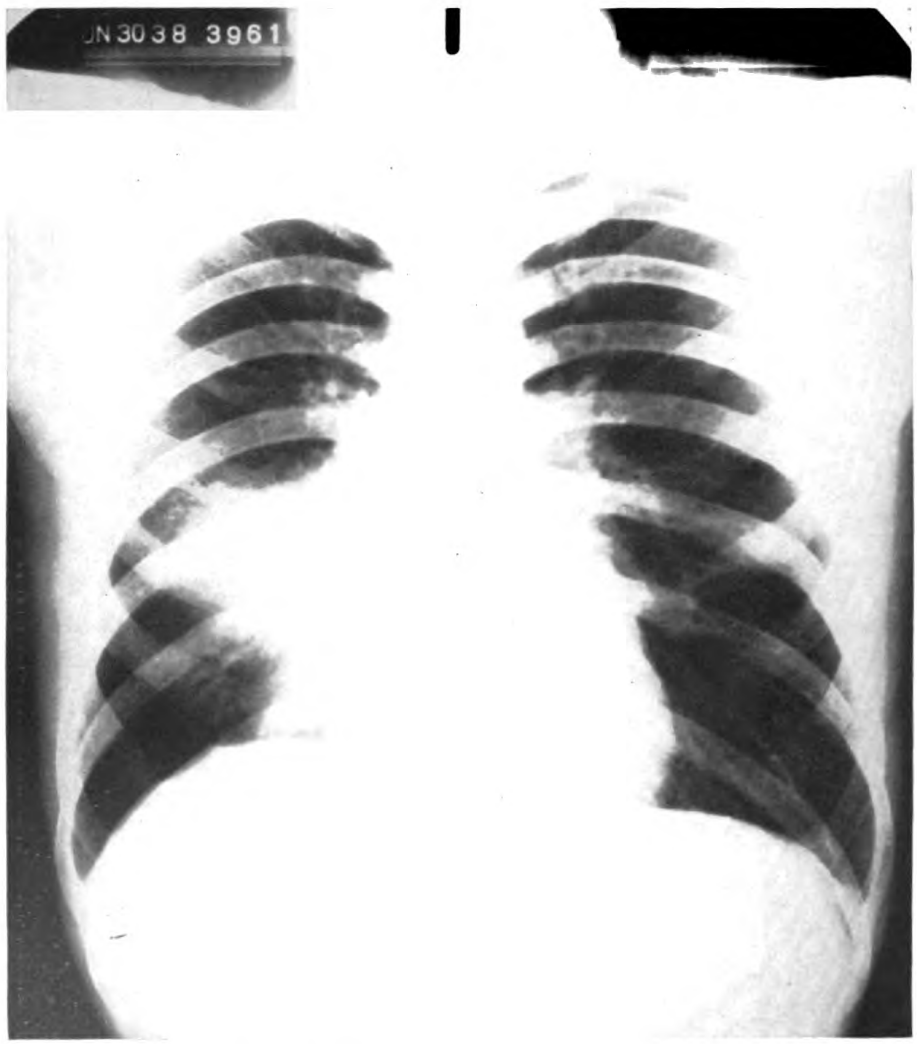

FIGURE 22.—CASE 18, M. B. ATELECTASIS RIGHT MIDDLE LOBE OF THE LUNG

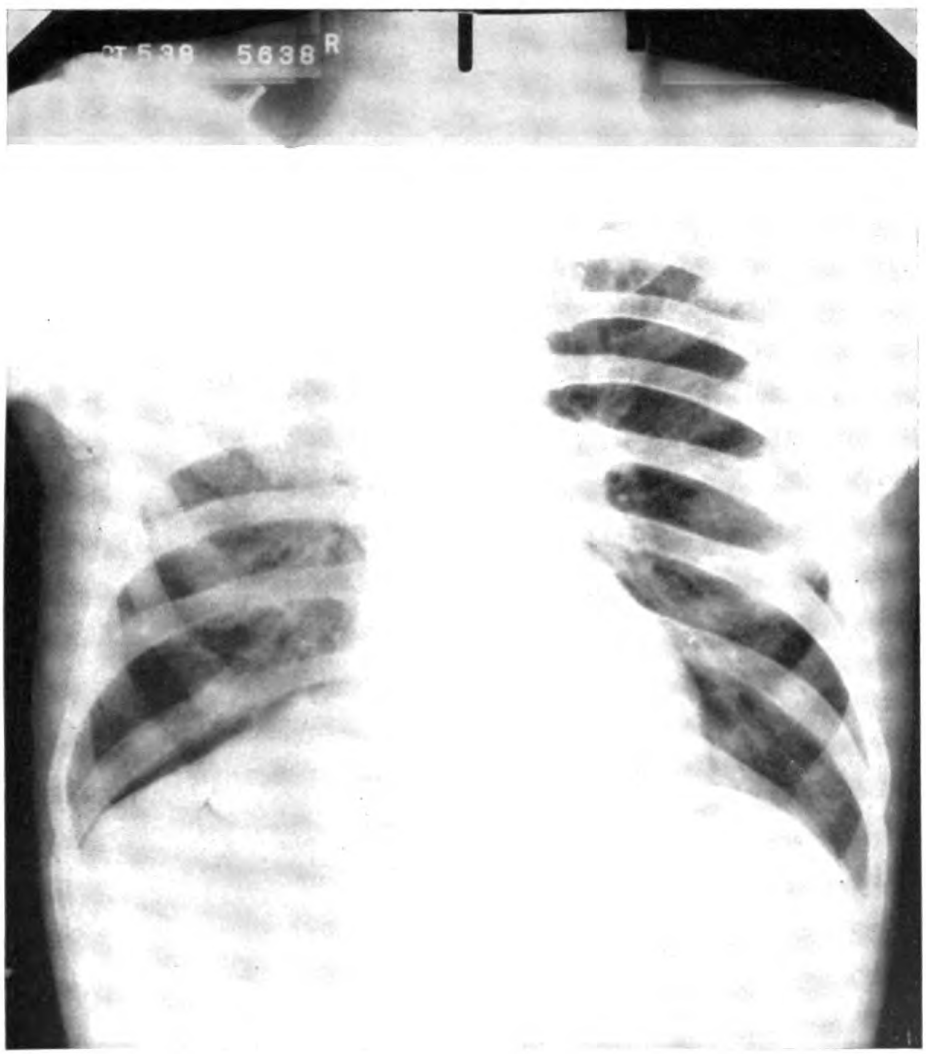

FIGURE 23.—CASE 20, J. McG. TUMOR NODULE UPPER RIGHT HILUM AND COMPLETE ATELECTASIS RIGHT UPPER LOBE.

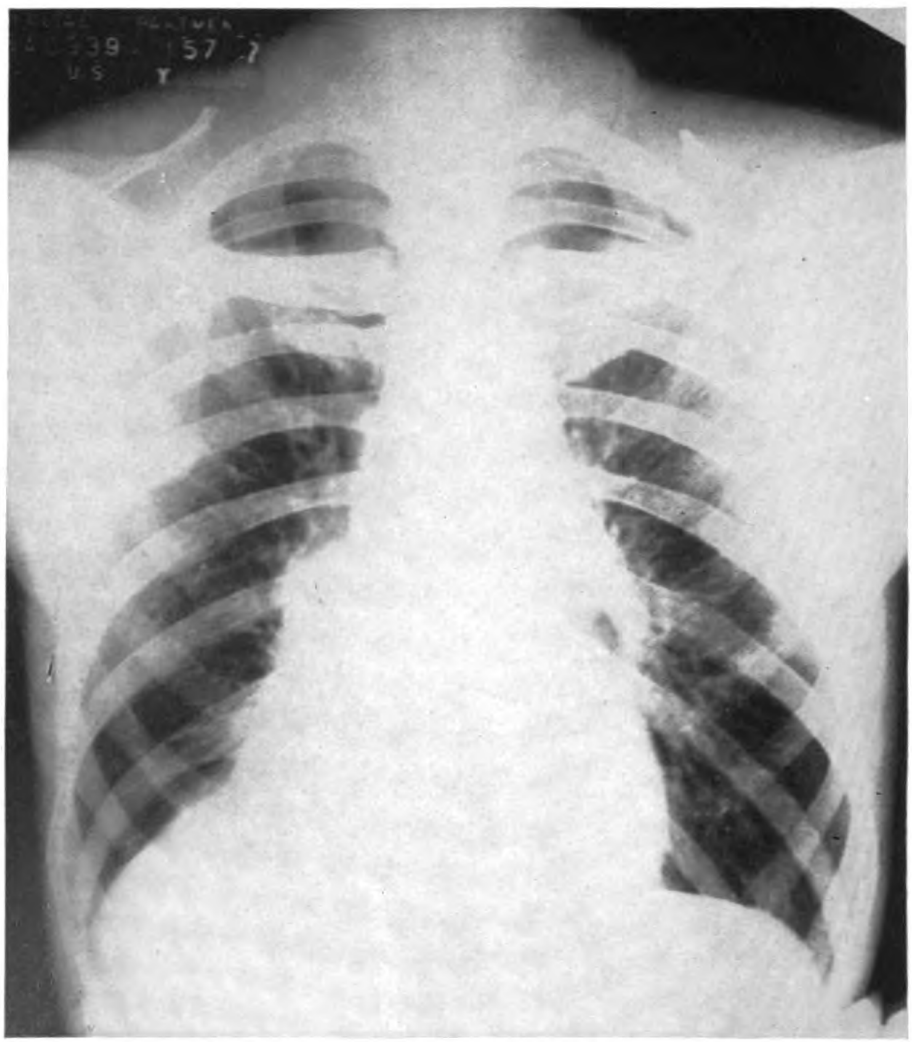

FIGURE 24.—CASE 22, W. W. TUMOR NODULE LOWER RIGHT HILUM AND PARTIAL ATELECTASIS RIGHT LOWER LOBE.

the left hip, a biopsy was performed after the roentgenogram revealed the destructive lesion in the pelvis. The microscopic examination revealed metastatic adenocarcinoma, probably from a bronchus. There were no symptoms referable to the chest. The roentgenogram of the chest was negative. Autopsy (September 24, 1937) revealed a small primary tumor nodule, 1 centimeter in diameter, in a secondary bronchus to the left lower lobe. Microscopic examination proved it to be a primary adenocarcinoma.

Case 14.—E. W. Roentgenogram (fig. 20) shows the left border of the aorta and the heart obscured by a generalized opacity which fills the upper two-thirds of the left upper lobe. The tumor nodule is barely visible in the upper hilum region. The intercostal spaces on the left are narrowed and the left dome of the diaphragm is markedly elevated. The trachea is displaced to the left. Five months prior to admission this patient had noticed pain under the left scapula and slight cough. Slight hemoptysis occurred 2 months later. One month before admission hoarseness had set in. The laryngoscopic examination revealed paralysis of the left vocal cord. The bronchoscopic examination showed the left upper bronchus flattened slightly in the anteroposterior diameter. No tumor was seen. Later an enlarged cervical gland was removed. Microscopic section showed carcinoma, medullary type, metastatic. From April 26 to May 12, 1938, he received 1,860 roentgen units to each anterior and posterior field.

Roentgenogram (fig. 21) was taken 5 weeks after roentgen therapy was completed. The tumor nodule is now fairly well circumscribed. The atelectatic process has largely disappeared. The intercostal spaces have widened somewhat but the left dome remains elevated probably because the left phrenic nerve is compressed by the enlarged left hilar glands. The mediastinal contents are in the middle line. Temporary improvement occurred following roentgen therapy but the patient by August 3 showed evidence of widespread metastases and died August 21, 1938. No autopsy was obtained.

Case 18.—M. B. Roentgenogram (fig. 22) reveals a triangular area of increased, fairly homogeneous density which extends outward from the lower right hilum region. The apex of this density is directed toward the lateral chest wall. There is no visible tumor nodule. There is a slight shift of the heart to the right but little, if any, change in size of the right hemithorax. The shadow was produced by atelectasis of the middle lobe. Because of the persistent cough and discomfort in the chest, a bronchoscopic examination was done. A bleeding fungating tumor mass was found almost completely obstructing the right main bronchus below the level of the carina. This neoplasm had a ball-valve action. Microscopic examination proved it to be a primary epidermoid, or squamous, carcinoma, grade 1. Pneumonectomy was successfully carried out. This patient is well with no evidence of recurrence 22 months after operation.

Case 20.—J. McG. The roentgenogram (fig. 23) shows a dense homogeneous area in the right upper lobe region which extends from the apex to a level with the sixth rib posteriorly. The apex is contracted and the trachea is displaced to the right by the lobar atelectatic process. The lower margin of the shadow shows a concave line at the outer half due to atelectasis, while the inner half presents a convex line due to the large tumor nodule. This S-shaped line is considered pathognomonic, and was first described by Ross Golden of New York. This patient had noticed dull pain in the right upper chest for 2 months prior to admission. No cough or hemoptysis. He lost 20 pounds in 2 months. The bronchoscopic examination showed no gross neoplasm. The right vocal cord was paralyzed. Pneumonectomy was performed on December 8, 1938. The patient died December 14, 1938, of empyema and pleural fistula. The right upper lobe

revealed a large squamous cell carcinoma. The tumor was beyond the reach of the bronchoscope.

Case 22.—W. W. The roentgenogram (fig. 24) reveals a fairly well circumscribed tumor nodule in the lower right hilum area and an area of increased, homogeneous density at the right cardiodiaphragmatic angle. The heart is displaced moderately to the involved side and the inner half of the right dome of the diaphragm is elevated. The small area of atelectasis confined to a portion of the lower lobe probably accounts for the absence of narrowing of the intercostal spaces on the right. This patient had an harassing cough, hemoptysis, weakness, and night sweats for 1 year. He had lost 14 pounds of weight. In 1919 his physician had made a diagnosis of pulmonary tuberculosis. The bronchoscopic examination showed the right lower bronchus occluded by a fungating mass. The right middle lobe bronchus was somewhat compressed. The microscopic examination of the bronchoscopic specimen revealed an undifferentiated bronchogenic carcinoma, grade 3. A thoracotomy was performed but pneumonectomy was not carried out because the tumor showed extension to the mediastinum. Roentgen therapy was instituted and the patient received 7,200 roentgen units. He was ambulatory and apparently in fair health when last seen on August 2, 1940.

BRONCHOSCOPIC EXAMINATION

The bronchoscopic examination is invaluable. It is reported that 75 percent of the lung field is within the reach of the endoscopic examination in the hands of the well-trained worker. This type of examination was performed in 23 cases.

There were positive endoscopic findings in 14 cases. Microscopic examination of the biopsied sections showed an undifferentiated type of carcinoma in 7 instances and a squamous carcinoma in 5. There were only 2 cases with adenocarcinoma.

TABLE 13.—*Bronchoscopic examination in 23 cases*

	Cases
Undifferentiated carcinoma	7
Squamous	5
Adenocarcinoma	2
Neoplastic biopsies in a total of	14
Inflammatory tissue	4
Blood exuding into bronchus but no tumor	2
Paralysis of the left vocal cord	2
Paralysis of the right vocal cord	1
	9

Percentage of positive findings with biopsies, 60.7 percent.

LABORATORY EXAMINATIONS

The laboratory examinations on admission showed the red cell count was above 4 million per cu. mm., in 15 cases. This finding was in keeping with the heatlhy appearance of a large percentage of these patients. The red cell count was below 4 million per cu. mm., in 11 cases.

The white cell count was below 8,500 cu. mm., in 12 cases and above this number in 17 cases. The latter group had an average white count of 12,500 per cu. mm. The patients with the elevation of the white count showed more signs of infection in the lung fields than those with counts normal in number or below.

The blood sedimentation rate was in the neighborhood of 25 mm./60 min. in 17 cases and below 12 mm./60 min. in 1 case. The high sedimentation rate was consistent with the degree of chronic infection in the parenchyma distal to the bronchostenosis.

The Kahn blood test for syphilis was negative in 19 cases and positive in 4. Two of the latter had positive spinal fluid changes characteristic of syphilis.

The sputum was negative for tubercular bacilli in 23 cases and it was positive in none.

In case 28, the diagnosis of bronchial carcinoma was made at another hospital from the microscopic examination of sanguineous pleural fluid.

TABLE 14.—*Summary of histological types in 30 cases*

Type of carcinoma	Number	Percentage
Squamous	11	36. 7
Adenocarcinoma	6	20. 0
Undifferentiated	13	43. 3
	30	100. 0

AUTOPSIES

Autopsies were performed on 17 cases. The kidneys, liver, brain, adrenal glands, and the skeleton showed a relatively high percentage of metastatic involvement. The kidneys were involved in 41 percent, the liver in 35 percent, the brain in 29.5 percent, the adrenals in 23.6 percent, and the skeleton in 24 percent. These figures were considerably higher than those reported in cases with cancer of other organs.

SURGICAL THERAPY

Early surgical treatment seems to offer the only hope at present. Five pneumectomies were performed with four fatalities.

Case 18, the only survivor in this group of five cases, had his right lung removed at this hospital on August 9, 1938, for a squamous carcinoma, grade 1. He was living and well when last seen in June 1940.

Three thoracotomies were performed but the carcinoma in each case was too far advanced for surgical excision. These cases had "frozen" mediastina.

ROENTGEN THERAPY

Nine of the 30 cases received deep x-ray therapy. Of these, 5 received what was considered adequate dosage.

The daily dose was 200 roentgens measured in air without back-scatter, using the following technic: 200 K. V. P.; 0.5 mm.; Cu. and 1.0 mm. Al. filter; 15 by 20 cm. fields; 50 cm. skin-focal distance; 25 milliamperes. Size of the fields varied with the case.

TABLE 15.—*Roentgen therapy*

Case No.	Initials	Roentgens	Fields	Duration after administration	Type	Response
				Months		
1	E. G.	800	A. and P.	2.25	Undifferentiated carcinoma	Poor.
6	W. W.	1,500	A., P., and L.	5.5	Undifferentiated carcinoma	Do.
8	E. D.	1,500	A. and P.	10.0	Undifferentiated carcinoma	Fair.
9	T. D.	1,860	A. and P.	7.0	Undifferentiated carcinoma	Do.
14	E. W.	1,860	A. and P.	4.0	Undifferentiated carcinoma	Poor.
21	A. C.	2,100	A. and P.	8.0	Adenocarcinoma	Do.
22	W. W.	2,400	A., P., and L.; living Aug. 1940.	3.0	Undifferentiated carcinoma	Good.
26	F. M.	1,860	A. and P.	1.5	Adenocarcinoma	Poor.
28	C. H.	(1)		1.5	Undifferentiated carcinoma	Do.

[1] Unknown, treated elsewhere.

CONCLUSIONS

Bronchogenic carcinoma seems to be on the increase. Perhaps more and better diagnostic procedures make this increase more apparent than real.

Occupation did not seem to be a factor in our group.

There is a rapid and extensive metastatic dissemination which may fool the clinician as to the true nature of the primary lesion.

The symptoms and physical findings in the early stages are not pathognomonic. X-ray examination of the chest is a valuable aid. Atelectasis should be the warning figure when present. A negative chest, however, is not absolute evidence bronchogenic carcinoma is not present. Other types of tumors, pleurisy, pleurisy with effusion and lung abscess may confuse the picture. A lung abscess may be due to carcinoma. Increased trunk markings if followed serially may later show an atelectatic area, which was the first evidence of the nature of the lesion. We feel that a marked shift of the mediastinal contents may be a valuable sign for early surgical measures as patients with this finding were less liable to have mediastinal fixation.

Early and repeated bronchoscopic examinations are invaluable. If the lesion is in a favorable location positive tissue examinations clinch the diagnosis quickly. Indirect bronchoscopic findings also help in determining the nature and extent of the involvement. In selected cases bronchography with lipiodol may be of value. If these methods

fail aspiration biopsy, artificial pneumothorax or exploratory thoracotomy may be employed to establish a diagnosis. In the early stages of the lesion other laboratory procedures may not give much aid. If the chest contains bloody fluid, microscopic examination may show cancer cells.

It must be remembered that the radiographic appearances are momentary pictures. The hilar type may rapidly become converted into the lobar form due to atelectasis. The parenchymatous nodular carcinoma may excavate due to central nerosis and develop an appearance not unlike a lung abscess. The atelectatic type may go on to bronchiectasis or empyema in a few weeks.

Early and complete removal of the involved lung seems to offer the only hope for cure at present. Deep x-ray therapy is of value in alleviating dyspnea and pain.

SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

THE FEASIBILITY OF BILATERAL PHRENIC PARALYSIS COMBINED WITH PNEUMOPERITONEUM

By Commander E. G. Brian, Medical Corps, United States Navy, and Lieutenant E. Ricen, Medical Corps, United States Navy

Bilateral phrenic nerve paralysis is by no means an unknown procedure. Sauerbruch and Jehn as early as 1914, and Kroh in 1921, had performed bilateral phrenicotomy for the purpose of applying artificial respiration in cases of diaphragmatic spasm caused by tetanus. More recently both temporary and permanent phrenic paralysis have been used successfully in the treatment of persistent singultus.

To Curti in 1924 goes the credit for first using bilateral phrenicotomy and phrenic exeresis in the treatment of 6 cases of pulmonary tuberculosis. In 5 of his cases simultaneous bilateral phrenicotomy was the operation of choice. Schwatt in 1933 collected 5 additional cases from the foreign literature to which he added one of his own. Duryea reported a case in which the procedure was employed immediately postpartum to offset the effect of the sudden descent of the diaphragm following delivery. Potter, Berry, and Bortone have also reported 1 case of consecutive bilateral phrenicotomy. Cohen and Williams in 1938 reported a series of 11 cases of bilateral operation, the largest series reported in the literature to date. A careful review of the literature mentioned above reveals a total of 25 cases of bilateral phrenic operation used in the treatment of pulmonary tuberculosis, reported to date. Joannides and Schlack have suggested the use of bilateral phrenic paralysis combined with pneumoperitoneum for cases in which bilateral artificial pneumothorax is indicated but found to be impossible. As yet they have published no results.

In previous publications the authors have emphasized the desirability of combined phrenic paralysis and pneumoperitoneum. Our

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observations have demonstrated that the pulmonary compression resulting from pneumoperitoneum is greatly enhanced by a preliminary phrenic paralysis. The combined procedures of pneumoperitoneum and phrenic paralysis accomplish two definite objectives. The elevated paralyzed diaphragmatic leaf compresses the lung and the respiratory diaphragmatic excursions are eliminated. The direct result of this compression is the drainage of inflammatory exudates from pulmonary cavities and the bronchial tract. The phrenic paralysis results in immobilization and functional rest of the lung. The end result of this compression and immobilization is eventually absorption and fibrosis.

Up to the present writing a series of 9 cases of bilateral pulmonary tuberculosis have been treated with consecutive temporary phrenic paralysis. It is our purpose to present a careful analysis of these cases, discussing the immediate and late results and the feasibility of this procedure.

INDICATIONS

1. Cases in which bilateral artificial pneumothorax is indicated but impossible to carry out.
2. Far advanced bilateral cases in which the vital capacity is reduced to such a degree as to make bilateral artificial pneumothorax an unsafe procedure.
3. When bilateral pneumothorax has been ineffective in closing cavities.
4. Preliminary to major surgical collapse procedure. Improvement may occur to such an extent as to make the patient a more suitable risk for future surgery.
5. As a palliative measure. It is of distinct value in the utterly hopeless case by alleviating the toxemia and making the patient more comfortable in addition to the marked beneficial psychological effect on those patients for whom previously nothing whatsoever had been done.
6. Bilateral basilar disease.
7. Pulmonary tuberculosis complicated by bilateral basal bronchiectasis.
8. Unilateral phrenic paralysis and contralateral pneumothorax, when the latter can no longer be maintained.
9. Unilateral phrenic paralysis with contralateral spread, when an obliterative pleuritis exists on the contralateral side.

COMPLICATIONS

Complications are fortunately rare. They may be classified under two heads. Complications due to pneumoperitoneum and those due to bilateral phrenic paralysis. Some of the more important ones due to the former are as follows:

Air embolus, although a rare occurrence, must be borne in mind. However, with the exercise of ordinary care in inserting the needle into the peritoneal cavity, it can be almost completely avoided. Subcutaneous emphysema although more common is not very serious as the air is usually absorbed fairly rapidly. Massive atelectasis of the lung may occur but this again is one of the rarer complications. Other less serious sequels are obliteration of the peritoneal space and free fluid in the peritoneal cavity. The accumulation of small amounts of fluid in the peritoneal cavity does not contraindicate the continuance of pneumoperitoneum. In one instance pneumoperitoneum was discontinued because of the appearance of hernia. One case of air embolus developed, but recovery was complete without sequel.

Complications resulting from bilateral phrenic paralysis are likewise fortunately rare. Among the more important are gastro-intestinal symptoms. These may vary from mild to severe. Slight nausea and vomiting are usually the rule, persisting from a few days to a few weeks. In all our cases recovery was complete. Other gastro-intestinal symptoms noted were loss of appetite, gastric distension, regurgitation, and feeling of fullness. These symptoms were likewise of a transitory nature. Additional gastro-intestinal symptoms mentioned in the literature are acute dilatation of the stomach and interposition of the colon.

Pulmonary complications are exceedingly rare. A transitory dyspnea was the only distressing symptom occurring in our cases. In all cases dyspnea was present for a few days only, gradually disappearing as compensation took place. Gradual improvement in the respiratory function takes place as functional improvement develops in the cardiorespiratory muscles due to the alleviation of the tuberculotoxic symptoms. In no instance was dyspnea severe enough to necessitate the use of the oxygen tent.

No effect on cardiocirculatory function was noted as a result of bilateral phrenic paralysis although theoretically at least this complication is possible. Cohen and Willauer report one case of severe right heart dilatation complicating this procedure.

DISCUSSION

Considerable difference of opinion exists among phthisiologists regarding the comparative merits of temporary versus permanent phrenic paralysis: Alexander states that over a 5-year period of time approximately 95 percent of his initial phrenic operations were of the temporary type. He believes the temporary operation should be used on those cases in which reasonable doubt exists as to the end result, especially when further unilateral or bilateral collapse measures may be required that might too greatly reduce the vital capacity if phrenic paralysis were of a permanent character. Among the indications

which may be listed for permanent phrenic paralysis are: Unilateral pulmonary parenchymal cavitation in which pneumothorax collapse has failed; as a preliminary to a thorocoplasty; and lastly preliminary to the discontinuance of artificial pneumothorax, when the function of the healed lung has been impaired by fibrosis.

A similar controversy arises over the matter of simultaneous compared to consecutive phrenic interruption. Although some of the early operations were of the simultaneous type, today we believe the consensus of opinion favors the consecutive procedure as a more rational and safer method.

In our series, consecutive temporary interruption of the phrenic nerve was the operation of choice in all cases. In both the initial and the consecutive operation the technic consisted of exposure and simple crushing of the phrenic nerve, after first injecting with 1-per-cent procaine. Dunner recommends an interval of several weeks between each operation. Schwatt advises not less than 1 month and longer if no urgency exists.

Cohen and Willauer in their series have allowed intervals of from 5 to 40 months to elapse between the two operations. In our own cases the shortest interval between operations was 13 days, the longest 7 months. In all cases the choice for the initial operation was on the side of the most advanced and most active lesion. No arbitrary time limit can be set for the interval between the initial and the consecutive operation. As soon as dyspnea disappears and the respiratory and cardiac rates indicate adjustment has occurred it is safe to proceed with the next phrenic paralysis. This period of adjustment following the initial phrenic interruption may vary from a few days to a few weeks, occasionally it may take several months. In certain cases sufficient improvement will occur in the contralateral lesion to eliminate the necessity for bilateral paralysis. In all our cases the consecutive phrenic operation was attempted at a time when the contralateral diaphragm was still paralyzed. Following the bilateral paralysis, some dyspnea occurred but it was likewise of a transitory nature. Clinical improvement occurred rapidly in all of the nine cases. Reduction in temperature soon followed. Marked diminution of cough, increased appetite and general improved feeling of well-being likewise resulted. In addition there is a marked beneficial tonic or psychic effect even in the most advanced cases.

Bed rest was utilized to the fullest extent in all of our cases in connection with the pulmonary collapse therapy administered.

Case Reports

Case 1.—F. C. Guamanian male, age 22 years, admitted August 29, 1939, with tuberculosis, pulmonary, chronic, active, far advanced, bilateral.

Chest film on admission (fig. 25) revealed a caseous pneumonic exudate infiltrating right apex and upper five right anterior interspaces. Cross infection

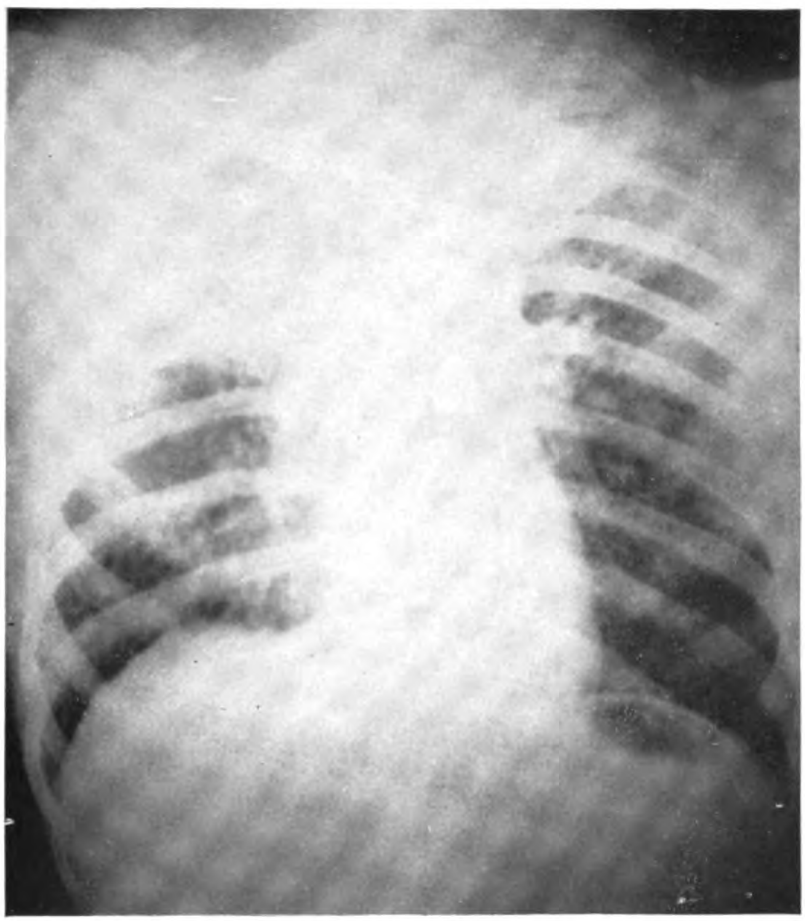

FIGURE 25.—CASE 1. F. C. BEFORE OPERATION.

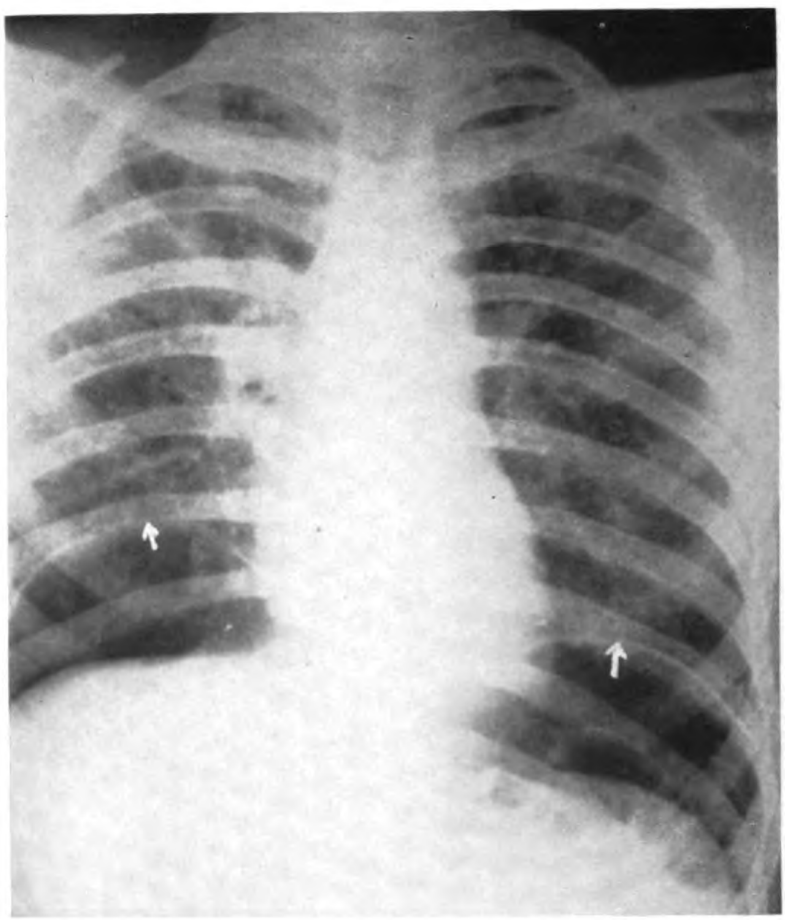

FIGURE 26.—CASE 1. F. C. AFTER OPERATION.

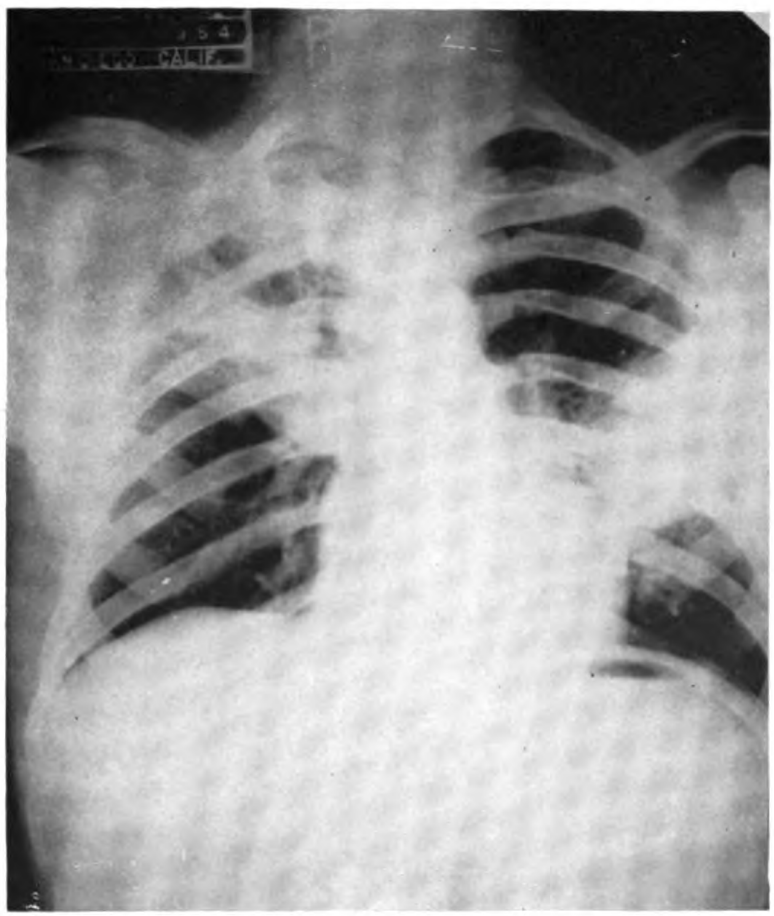

FIGURE 27.—CASE 3, R. T. BEFORE OPERATION.

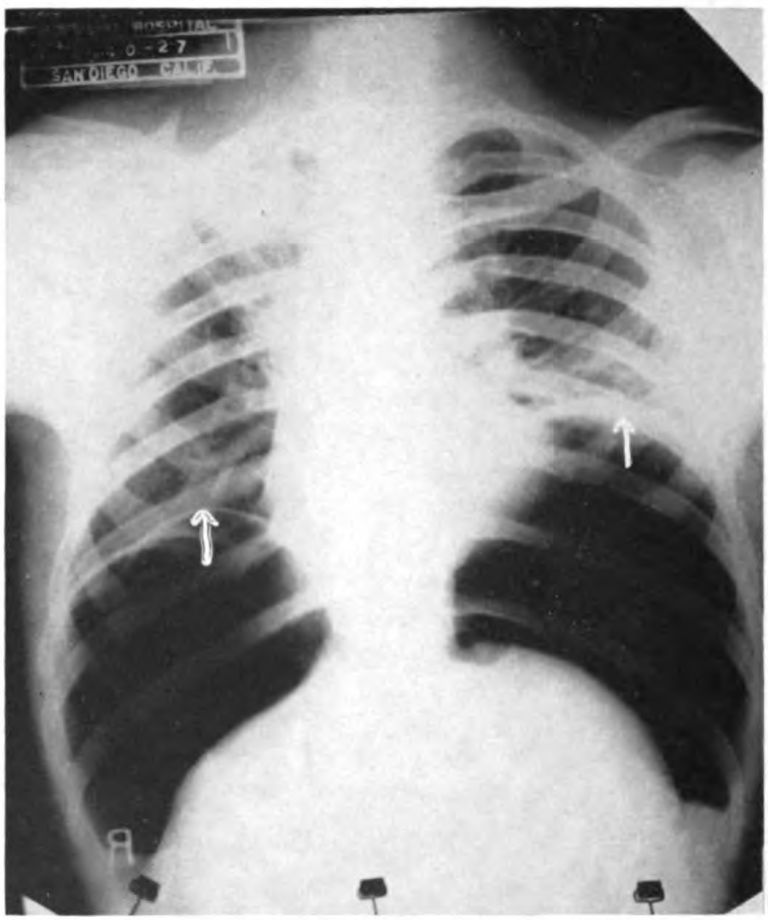

FIGURE 28.—CASE 3, R. T. AFTER OPERATION.

has occurred resulting in a pneumonic exudate infiltrating left apex and upper four anterior interspaces. Cavities are noted in the first and third right anterior interspaces

Sedimentation index on August 29 was 23 mm. Sputum was positive for *M. tuberculosis hominis* (*M. b. t.*) on admission.

Right and left artificial pneumothorax were both unsuccessfully attempted. A right phrenic crush was done September 22. A left phrenic was done October 5. Pneumoperitoneum was induced September 2, and has been continued with weekly refills of 1,000 cc. of air.

So little cardiac and respiratory embarrassment resulted from the initial phrenic paralysis that the second operation was performed 13 days later. Again cardiac and respiratory function became adjusted in a few days. Bilateral phrenic paralysis combined with pneumoperitoneum was intended as a palliative procedure in this case due to the extensive nature of the disease.

After 8 months of hospitalization the results are most promising. Clinical improvement has been evidenced by normal temperature, pulse and respiratory range. Cough has diminished and appetite has improved. Weight has remained constant. Sedimentation index on April 3, 1940, was 16 mm. Sputum examination on April 9, was negative for *M. b. t.*

Chest film April 1, 1940 (fig. 26): The cavity in the right upper lobe is considerably smaller and there is clearing of the infiltration in this region. Clearing is also noted in the left upper chest. Each diaphragm is elevated considerably by pneumoperitoneum.

This case is of especial interest because the treatment administered was intended solely as a palliative measure due to the extensive pulmonary involvement. After 8 months both clinical and radiographic evidence of healing has been noted. This is still too short a period to make any definite prediction of cure. However, it is reasonably certain that without the treatment instituted, this case would not be alive today.

Case 2.—A. B. adult, white male age 21 years, admitted December 15, 1939, with diagnosis of tuberculosis, pulmonary, chronic, active, far advanced, bilateral.

Chest film on admission reveals an infiltrative process involving the periphery of the left upper lobe, the left base and the central portion of the right lower lung field. The lesion in the bases are bronchiectatic in type. The findings are those of active pulmonary tuberculosis of unusual distribution.

Sedimentation index December 15 was 17 mm. Sputum on admission was positive for *M. t. h.*

A left artificial pneumothorax was unsuccessfully attempted. A right phrenic crush was done January 4, 1940. A left phrenic crush was done January 22. Pneumoperitoneum was initiated January 9, and has been continued with weekly refills of 1,000 cc. of air.

After 4 months of hospitalization, sedimentation index April 17, is 8 mm. Sputum is still positive for *M. t. h.* Weight has remained constant.

Chest film April 1: Both diaphragmatic leaves are forced upward by a large amount of air in the peritoneal cavity. The lesion in the left apex appears to be more sharply outlined and shows evidence of healing.

Following the initial phrenic interruption, cardiac and respiratory function adjusted rapidly. This was followed by the second operation 18 days later. Clinically, improvement as evidenced by normal temperature, pulse and respiratory range soon followed. Diminished cough was also noted. Although the period of 4 months is far too short to prognosticate ultimate cure, definite clinical and radiographic evidence of healing has been demonstrated. The feasibility of even the far advanced case to carry on cardiac and respiratory function despite

combined bilateral phrenic paralysis and pneumoperitoneum has, we believe, been demonstrated by these two cases.

Case 3.—R. T. adult, white male age 22 years, admitted April 27, 1939, with a diagnosis of tuberculosis, chronic, active, far advanced, bilateral.

Chest film (fig. 27) on admission reveals a caseous pneumonic exudate infiltrating the right upper lobe. There is a cavity 2 by 3 cm. at the level of second anterior interspace. There is also a pneumonic exudate infiltration involving the third, fourth, and fifth left anterior interspaces.

On admission sedimentation index was 25 mm. Sputum was positive for *M. t. h.*

A right phrenic crush was done May 2. A left phrenic crush was done October 5. Pneumoperitoneum was initiated October 3, and has been continued with weekly refills of 1,000 cc. of air.

After 1 year of hospitalization, the sedimentation index is 5 mm. Sputum is negative for *M. t. h.* Weight has remained constant.

Chest film (fig. 28) April 21, 1940: The left diaphragm is at the level of the fifth posterior interspace. The pneumonic infiltration involving the third, fourth, and fifth left anterior interspaces has undergone complete absorption. The process in the right upper lobe has likewise cleared considerably and shows evidence of fibrosis. The cavity previously mentioned is no longer outlined.

In this case an interval of months was allowed to elapse between the initial and the consecutive phrenic paralysis. No distress was noted after either operation. Clinical improvement paralleled radiographic evidence of healing. As soon as function of either diaphragm returns, the phrenic paralysis will be repeated.

Case 4.—A. F., adult white male, age 42, admitted August 5, 1938, with diagnosis of tuberculosis, pulmonary, chronic, active, far advanced, bilateral.

Chest film on admission: In the right lung field from the apex to the level of the second rib viewed anteriorly, is an infiltration, bronchopneumonic in type. There are numerous small ring shadows in this area. There has been a cross infection to the left lung field and there is an infiltration of lesser density but of approximately the same extent as on the right side.

Sedimentation index on admission was 19 mm. Sputum was positive for *M. t. h.*

Right and left artificial pneumothorax were unsuccessfully attempted. A right phrenic crush was done January 10, 1939, and a left phrenic crush was done July 6. Pneumoperitoneum was instituted June 6, and has been continued with weekly refills of 1,000 cc. of air. February 8, 1940, the right phrenic crush was repeated due to resumption of function of the right diaphragmatic leaf.

Sedimentation index April 1, 1940, was 12 mm. Sputum is still positive for *M. t. h.* There has been a gain of 9 pounds in weight since admission.

Chest film April 1, 1940: Both diaphragmatic leafs are elevated considerably by pneumoperitoneum. Considerable improvement has occurred in the processes previously described in the left upper lobe and in the right chest.

This case is of considerable interest because on admission the pulmonary tuberculosis was complicated by advanced laryngeal tuberculosis. In addition to the healing which has occurred in the pulmonary parenchymal lesions the laryngeal tuberculosis has responded most favorably. To date this is our only case of laryngeal tuberculosis, associated with pulmonary tuberculosis, in which the laryngeal lesion has been altered favorably. This case also demonstrates the feasibility of repeating the phrenic paralysis once diaphragmatic function has been resumed. This procedure will be repeated again if the indication recurs.

The next four cases of consecutive bilateral phrenic paralysis are mentioned briefly for the purpose of record only. In all instances the

bilateral phrenic paralysis combined with pneumoperitoneum has been in effect too short an interval to draw any definite conclusions regarding clinical and radiographic evidence of healing. It is contemplated at a later date to report the results in these cases.

Case 5.—B. G., adult Negro male, age 24, admitted March 14, 1940, with a diagnosis of tuberculosis, pulmonary, chronic, active, far advanced, bilateral.

Chest film on admission: Extensive infiltrative processes involve the entire right chest and the left apex; some portions of the right chest lesions are soft and miliary in type. There is a cavity in the base of the right upper lobe about 2 cm. in diameter.

Sedimentation index was 26 mm. Sputum was positive for *M. t. h.*

Right pneumothorax was unsuccessfully attempted. A left phrenic crush was done April 2. A right phrenic crush was done April 23. Pneumoperitoneum was initiated April 6, and has been continued with weekly refills of 1,000 cc. of air.

Case 6.—H. S., adult white male, age 26 years, admitted February 10, 1940, with a diagnosis of tuberculosis, pulmonary, chronic, active, far advanced, bilateral.

Chest film on admission: There is an active infiltrative process in the right chest and the left apex. Several small bronchiectatic areas are noted in the central portion of the right upper lobe and there are some fibrotic bands extending to the periphery of the right upper lobe opposite the first and second interspaces at the axillary line.

Sedimentation index was 20 mm. Sputum was positive for *M. t. h.*

Right pneumothorax was unsuccessfully attempted. A right phrenic crush was done March 26, 1940. A left phrenic crush was done April 23. Pneumoperitoneum was initiated March 30, and has been maintained with weekly refills of 1,000 cc. of air.

Case 7.—R. S., adult white male, age 29 years, admitted February 24, 1940, with diagnosis of tuberculosis, pulmonary, chronic, active, far advanced.

Chest film on admission: There is a moderate amount of fluid in the right base. Extensive infiltrative processes are seen in both upper lobes and a suggestion of cavitation is noted in the periphery of both these lobes. Chronic fibrotic changes are seen in the central lung fields.

Sedimentation index was 27 mm. Sputum was positive for *M. t. h.*

Right and left artificial pneumothorax were unsuccessfully attempted. A right phrenic crush was done April 1. A left phrenic crush was done April 23. Pneumoperitoneum was initiated April 25, and has been continued with weekly refills of 1,000 cc. of air.

Case 8.—W. G., adult white male, age 32 years, admitted February 26, 1940, with a diagnosis of tuberculosis, pulmonary, chronic, active, far advanced, bilateral.

Chest film on admission: Extensive active infiltrative lesions are seen in both upper lobes and a cavity about 4 cm. in diameter is outlined in the right opposite the second interspace at the axillary line. Several large cavities are noted in the left upper lobe.

Sedimentation index was 21 mm. Sputum was positive for *M. t. h.*

Right and left artificial pneumothorax were unsuccessfully attempted. A left phrenic crush was done March 7. A right phrenic crush was done April 11. Pneumoperitoneum was initiated March 5, with weekly refills of 1,000 cc. of air.

The following case of extensive bilateral disease is one in which bilateral phrenic paralysis combined with pneumoperitoneum was used purely as a palliative measure. In this sense the procedure has succeeded. Clinically, temperature has been reduced, cough dimin-

ished, and a general feeling of well-being induced despite radiographic evidence of progression.

Case 9.—J. B., adult white male; age 22; admitted November 10, 1939, with a diagnosis of tuberculosis, chronic, active, far advanced, bilateral.

Chest film on admission: There is an extensive infiltrative lesion involving the entire left upper lung fields and the right lower lobe. A large cavity is outlined in the right lower lobe.

Sedimentation index was 24 mm. Sputum was positive for *M. t. h.*

A right phrenic crush was done November 20. A left phrenic crush was done January 8, 1940. Pneumoperitoneum was initiated November 14, 1939, and has been continued with weekly refills of 1,000 cc. of air.

Sedimentation index April 3, 1940, was 22 mm. Sputum, April 14, was positive for *M. t. h.* There has been 10 pounds loss in weight since admission.

Chest film April 20: Further cavities appear to be developing in both upper lobes. A cavity about 2 cm. in diameter is seen in the periphery of the right upper lobe. One in the left opposite the second anterior rib is about 2 by 4 cm. and another at the level of the third is about 3 cm. in diameter.

We believe the procedure in this case to be justified in spite of radiographic evidence that the lesions have been growing more extensive. Clinically, improvement is noted, the tuberculous toxemia alleviated, and the patient made definitely more comfortable. The results obtained from this procedure even as a purely palliative measure merit its adoption.

SUMMARY

Nine cases of far advanced bilateral pulmonary tuberculosis, summarized in table 1, have been presented in which the collapse therapy has consisted of consecutive bilateral phrenic paralysis combined with pneumoperitoneum. The feasibility of this procedure has, we believe, been demonstrated in the case reports. In cases 1 and 9 the treatment was instituted purely as a palliative measure. In case 9 the clinical improvement and reduction of the tuberculous toxemia justified the procedure. Unexpected results were obtained in case 1. Clinical improvement was quite marked. Sputum has become negative for *M. t. h.* Sedimentation index has been reduced from 23 to 16 mm. In addition, definite radiographic evidence of pulmonary parenchymal cavity closure, absorption, and fibrosis occurred.

In case 4 complicated by laryngeal tuberculosis definite improvement has occurred in both the pulmonary and laryngeal lesions. There has been a gain of 9 lbs. in weight. Sedimentation index was reduced from 19 to 12 mm.

TABLE 1.—*Summary of treatment and results*

Case	Initial phrenic crush	Duration of paralysis as of May 1, 1940	Period between operations	Consecutive phrenic crush	Duration of paralysis as of May 1, 1940	Duration of pneumo peritoneum as of May 1, 1940	Results		
							Clinical	Sputum	Roentgenogram
1 F. C	Right	7 months	13 days	Left	5 months	7 months	Improved	Negative	Improved.
2. A. B	do	4 months	18 days	do	3 months	4 months	do	Positive	do.
3. R. T	do	12 months	5 months	do	6 months	5 months	do	Negative	Marked improvement.
4. A. F	Left	10 months	7 months	Right	3 months	11 months	do	Positive	Improved.
				Left	2 months				
5. B. G	do	1 month	3 weeks	Right	1 week	3 weeks	do	do	No change.
6. H. S	Right	do	33 days	Left	do	1 month	do	do	do.
7. R. S	do	do	3 weeks	do	do	1 week	do	do	do.
8. W. G	Left	2 months	5 weeks	Right	3 weeks	2 weeks	do	do	do.
9. J. B	Right	5 months	6 weeks	Left	3 months	5 months	do	do	Process has extended.

Case 3 presents both clinical and radiographic evidence of healing. Sputum has become negative, for *M. t. h.* Sedimentation index has been reduced from 25 to 5 mm.

In cases 5, 6, 7, and 8, clinical improvement has occurred. The length of time under treatment has been too short to demonstrate any radiographic evidence of healing. It is contemplated at a future date to report the late results in these cases.

It is to be emphasized that combined phrenic paralysis and pneumoperitoneum were used in these cases, after other methods of inducing collapse had failed or had been found inadvisable. Unfortunately, all of the cases when first seen were far advanced. In most of them an obliterative pleuritis was present. Bilateral phrenic paralysis with pneumoperitoneum offered the only means possible of inducing collapse at the time it was attempted. Despite the fact that although this procedure was in many instances adopted as a last resort or as a palliative measure the results have been most promising.

CONCLUSIONS

1. Consecutive bilateral phrenic paralysis combined with pneumoperitoneum offers a means of inducing bilateral collapse therapy of pulmonary tuberculosis after other methods have failed or are deemed inadvisable.

2. Far advanced bilateral disease may respond with both clinical improvement and radiographic evidence of healing.

3. Its use as a palliative measure is justified in hopelessly advanced cases. Clinically symptoms may be alleviated despite anatomical extension of the lesion.

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TREATMENT OF GONOCOCCAL INFECTION WITH SULFAPYRIDINE

SEVENTY AMBULATORY CASES TREATED ABOARD SHIP

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INTRODUCTION

The outstanding results reported by Van Slyke [1] in the use of sulfapyridine in the treatment of gonorrhea prompted us to make a study in the use of this drug to determine its applicability and efficacy, with margins of safety on ambulatory cases, aboard ship, without interfering with the performance of routine duties of men treated. Van Slyke reported apparent cures of 82.5 percent in a series of 114 cases of gonococcal infection in males by the use of sulfapyridine.

A review of the literature indicated its apparent worthiness for use in the fleet, provided certain factors to be outlined were anticipated by appropriate laboratory procedures and remedial measures established for their control at the time of their appearance.

ANALYSIS OF CASES TREATED

Seventy cases of gonorrhea diagnosed by finding Gram negative intracellular diplococci in urethral smears were treated. Forty-five cases had been previously treated as bed patients with an average dosage of 520 grains of sulfanilamide over a 10-day period. Ten of these 45 cases received no other treatment during the 10-day period and the remainder had received anterior irrigations in addition to the sulfanilamide. The proportion of complications among the two groups was greater among those receiving anterior irrigations thus confirming the observations of Morse and Hirsch.[2]

Complications had been treated routinely. Fifteen cases received no treatment excepting bed rest with forced fluids and alkalinization for 10 days preceding their sulfapyridine. Ten cases were started immediately on sulfapyridine at the onset of infection.

TABLE 1.—*Clinical classification of cases*

Anterior urethritis	20
Posterior urethritis	21
Anterior and posterior urethritis	13
Posterior urethritis and prostatitis	16

TABLE 2.—*Interval between onset of infection and institution of therapy*

1–10 days	16
11–20 days	12
21–90 days	25
91 or more days	17

[1] Van Slyke, C. J., Sulfapyridine treatment of gonococcal infection of hospitalized males. Reprint from publication No. 11 of the American Assoc. for Advancement of Science, pages 120–124.

[2] Morse, C. E. and Hirsch, F. G., Treatment of acute gonorrheal urethritis, Mil. Surg. 86:53, Jan. 1940.

The exact number of days between onset of infection and institution of sulfapyridine therapy in each case is shown in the following charts.

METHOD

All cases were examined before treatment was started to determine the nature of the existing complication and to rule out any pathology that might be considered as making the individual unsuitable for this form of chemotherapy. The cases, all males, averaging 21.5 years of age, were in excellent physical condition.

Transcriptions of reactions were made from ward records of those having had sulfanilamide to act as a basis for comparative study of possible reactions to sulfapyridine.

Laboratory work consisting of hemoglobin, red-cell count, white count, differential, sedimentation time, and routine urinalysis was done at the commencement of the course and again at the termination. Hemoglobin, white count and urinalysis for albumen and blood were made on alternate days during the treatment. All men were inspected daily and questioned for untoward results. Daily 2-glass tests were done.

TREATMENT

Three grams of sulfapyridine were given with 4 grams of sodium bicarbonate in 3 equally divided doses the first day. One and five-tenths grams of the drug with 2 grams of sodium bicarbonate in 3 equally divided doses were given for the succeeding 9 days. No form of local treatment was used. Fluids were not restricted, in fact patients were encouraged to use liberal quantities. In eight cases it was deemed advisable to continue the therapy over the 10-day period.

DISCUSSION OF TOXICITY AND REACTIONS

Various workers have stressed the importance of repeated laboratory procedures in order to properly evaluate the toxicity and the blood level of the drug.

Nausea and vomiting is the most frequently stressed toxic manifestation of the drug. In our series 75.7 percent experienced nausea the first 2 days and 7.1 percent vomited. In severe cases, according toPepper,[3] these unpleasant symptoms may be controlled by stopping the administration of the drug, forcing fluids, and giving nicotinic acid 100 mgm. t. i. d. The drug if restarted may be mixed with water, fruit juices or milk (30 mgm. soluble in 100 cc. of water at 27° C.).

Small amounts of sodium bicarbonate or aluminum hydroxide solution may be given after the drug to lessen its toxicity. The use of barbiturates and chloral hydrate has been emphasized by other

[3] Pepper, O. H.

workers. The nausea and vomiting in our series was not sufficiently severe to necessitate any form of therapy to control.

A mild cyanosis was observed in all of our cases and its appearance does not contraindicate the continuance of the drug. In cases of moderate cyanosis the use of methylene blue, 2 grains orally q. i. d., has been advocated; in more severe cases, methylene blue 1 percent, 1 to 2 mgm. per kilo body weight, may be given intravenously. If the drug is restarted after its discontinuance methylene blue, 1 grain, may be given with each dose of the sulfapyridine. This form of medication, however, was not necessary in any of our cases.

The appearance of skin rashes has been stressed by some workers and was experienced in one of our cases. This followed exposure to the sun during inspection of the crew and disappeared 7 hours after its appearance. The drug was discontinued for 2 days and restarted with no recurrence of the measly appearing rash.

Hematuria has been reported in some instances and was thought to be due to the formation of acetyl sulfapyridine calculi in the renal tubules and pelvis. In a personal communication, Van Slyke[4] mentioned that what was viewed as hematuria occasionally was in reality hematoporphyrinuria and not considered an indication to suspend treatment but merely a temporary manifestation not linked with toxicity. Our microscopic and benzidine tests done on all cases on alternate days routinely, revealed no evidence of hematuria in any case.

Elevation of temperature has been considered by some as an indication to discontinue treatment. If the elevation of temperature is due to the drug, medication should be stopped and continued only with great caution. We had one case that developed a temperature of 103° which we considered to be due to an epididymitis that he exhibited on the third day of his course. He was made a bed patient, the drug was continued, the temperature dropped the second day, and the epididymis returned to normal on the seventh day.

RESULTS

Upon the completion of the course of treatment all men who were apparently cured were given prostatic examinations and smears of prostatic secretion were stained. Two glass urine findings were recorded. On the following day sounds were passed to the bulbomembranous junction and the canal massaged over it.

Men discharged as cured were cautioned regarding proper prophylaxis and the use of condoms in the future and that injudicial use of alcohol, if accompanied by intercourse, would bring out any existing infection. While not encouraged to resort to these provocative meas-

[4] Van Slyke, C. J. Personal communication, May 20, 1940.

ures, men were instructed to report them for record purposes, should they occur.

TABLE 3.—*Incidence of reported provocative measures*

Provocative measures	Percent
Alcohol	90
Sexual intercourse	75. 7
Alcohol and sexual intercourse	71. 4

Of the apparently cured, all of whom received sounds, the day following their prostatic massage, none developed a urethral discharge afterward or any subsequent symptoms of prostatitis as evidenced by the 2-glass test.

The following charts show results of treatment in individual cases.

TABLE 4.—*Group treated 1–10 days after onset of illness*

Case	Reaction and day of occurrence		White blood count		Sedimentation rate		Urine		Urethral discharge		Prostate		Total dose in grains	Total treatment days	Day of illness therapy was begun	Subjective results	Apparent clinical cure
	Type	Day	Before	After	Before	After	Before	After	Before	After	Before	After					
M. C.	N	1	9,200	4,500	8	4	Sh, Cl	Cr, Cr	Tk, y-w	No	Nor	Nor	254	10	2	Gd	Yes.
T. V.	N	2	8,600	5,000	10	5	Sh, Cl	Sh, Cl	Tk, y-w	Tk, W	do	do	254	10	2	Ner	No.
W. P. K.	N	1	8,400	6,400	10	5	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	3	Gd	Yes.
A. E. C.	N	1	8,500	6,000	5	4	Sh, Cl	Cr, Cr	Tk, y-w	Tk, W	do	do	408	15	8	Gd	No.
C. C. H.	N	2	7,900	4,400	4	3	Sh, Sh	Cr, Cr	Tk, y-w	No	do	do	254	10	8	Gd	Yes.
R. D.	N	1	7,000	4,200	6	4	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	9	Gd	Yes.
H. B.*	N	1	7,200	5,000	6	4	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
R. B.*	N	1	6,600	4,200	10	5	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
E. B. H.*	N	1	6,000	4,000	7	5	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Ner	Yes.
R. E.*	N	1	7,600	5,000	8	4	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
E. K. M.*	O	0	9,200	5,000	6	4	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
K. P.*	N	1	7,800	5,400	12	6	Cl, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
S. P. S.*	N	1	6,700	4,400	7	5	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
R. S.*	N	1	7,000	4,800	6	6	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
M. V.*	N	1	6,400	5,000	8	4	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.
J. S. W.*	N	1	6,700	5,400	7	4	Sh, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	10	Gd	Yes.

* Bed rest and alkalinization for 10 days prior to institution of treatment.

Key:
N —nausea
R —rash
Cl —cloudy
Cr —clear
Fat—fatigued

Key:
Gd —good
Ha —haze
Ner—nervous
Nor—normal
Sh —shreds

Key:
Tk —thick
Tn —thin
W —white
Y —yellow
4 plus—abundant pus

TABLE 5.—*Group treated 11–20 days after onset of illness* [a]

Case	Reaction and day of occurrence		White blood count		Sedimentation rate		Urine		Urethral discharge		Prostate		Total dose in grains	Total treatment days	Day of illness therapy was begun	Subjective results	Apparent clinical cure
	Type	Day	Before	After	Before	After	Before	After	Before	After	Before	After					
C. A. A.*	N	1	7,200	5,000	12	5	Cl, Cl	Cr	Tk, y-w	None	Nor	Nor	254	10	12	Gd	Yes.
J. B.*	N	1	7,000	4,600	14	6	Cl, Cl	Cr	Tk, y-w	None	do	do	631	20	12	Gd	Yes.
R. C. P.*	N	1	6,200	5,000	10	2	Cl, Cr	Cr	Tk, y-w	None	do	do	603	23	12	Gd	Yes.
F. D. P.*	N	1	9,000	6,000	15	5	Sh, Cr	Cr	Tk, y-w	None	do	do	254	10	12	Gd	Yes.
W. P.*	N	1	7,000	8,400	8	5	Cl, Cl	Cl	Tk, y-w	Tk, Wh	do	do	761	27	13	Gd	No.
R. S. F.	N	1	7,500	4,800	3	3	Ha, Ha	Cr	Tk, y-w	None	do	do	254	10	14	Gd	Yes.
S. L. H.	N	1	8,000	5,500	6	3	Cl, Cl	Cr	Tk, y-w	None	do	do	400	14	14	Gd	Yes.
L. A. S.	N	1	8,400	4,200	11	7	Cl, Cl	Cl	Tk, y-w	Tk, W	do	do	693	24	14	Gd	No.
J. E. T.	N	1	8,400	4,000	12	4	Cl, Cl	Cr	Tk, y-w	None	do	do	254	10	19	Gd	Yes.
R. H.	N	1	8,000	5,000	10	4	Sh, Cl	Cr	Tk, y-w	None	do	do	254	10	20	Gd	Yes.
M. M. M.	N	2	9,000	6,200	5	5	Cl, Ha	Cr	Tk, y-w	None	do	do	254	10	20	Ner	Yes.
F. J. N.	N	2	10,000	3,400	8	5	Cl, Cl	Cl, Cr	Tk, y-w	Tk, W	do	do	488	17	20	Gd	No.

* See table 4 for key to symbols.

TABLE 6.—*Group treated 21–90 days after onset of illness* [a]

Case	Reaction and day of occurrence		White blood count		Sedimentation rate		Urine		Urethral discharge		Prostate		Total dose in grains	Total treatment days	Day of illness therapy was begun	Subjective results	Apparent clinical cure
	Type	Day	Before	After	Before	After	Before	After	Before	After	Before	After					
C. H. B.	O	0	9,100	4,500	15	7	Sh, Cl	Cr, Cr	Tk, Y	No	Nor	Nor	254	10	21	Gd	Yes.
D. G. K.	N	1	8,000	7,000	4	3	Cl, Cr	Cr, Cr	Tk, y-w	No	do	do	254	10	23	Gd	Yes.
R. H. A.	O	0	8,100	6,700	11	5	Sh, Cr	Sh, Cr	Tk, y-w	Tk, y	do	do	474	18	24	Gd	No.
F. R. H.	N	1	8,500	6,500	8	8	Cl, Cl	Cr, Cr	Tk, y-w	No	do	do	254	10	24	Gd	Yes.
S. J.	N	1	6,500	4,500	6	4	Ha, Cl	Cr, Cr	None	No	4 pl	do	254	10	35	Gd	Yes.
H. F. B.	N	1	6,800	6,200	3	2	Cl, Cl	Cr, Cr	None	No	4 pl	do	254	10	38	Gd	Yes.
D. E. L.	N	1	9,000	7,000	13	2	Sh, Cl	Cr, Cr	Th, W	No	4 pl	do	254	10	38	Fat	Yes.
J. M.	N	1	12,500	4,200	17	8	Ha, Ha	Cr, Cr	Th, W	No	4 pl	do	254	10	38	Ner	Yes.
W. D. P.	N	1	7,100	5,400	5	3	Ha, Ha	Cr, Cr	Tk, W	No	Nor	do	254	10	41	Gd	Yes.
A. C.	O	0	12,000	7,500	6	3	Cl, Cr	Cr, Cr	None	No	4 pl	do	254	10	42	Gd	Yes.
T. S. L.	N	1	9,100	5,100	3	3	Ha, Ha	Cr, Cr	Tk, y-w	No	Nor	do	254	10	47	Gd	Yes.
T. S. S.	N	1	7,000	4,400	14	3	Cl, Cl	Cr, Cr	None	No	4 pl	do	254	10	55	Gd	Yes.
W. B. B.	O	0	8,100	7,000	5	3	Cl, Cr	Cr, Cr	None	No	4 pl	do	254	10	59	Gd	Yes.
C. C. H.	N	1	8,000	6,400	2	3	Ha, HA	Cr, Cr	TK, Y-W	No	4 pl	do	254	10	61	Ner	Yes.
R. K. M.	N	1	8,200	8,000	3	2	Cl, Ha	Cr, Cr	None	No	4 pl	do	254	10	61	Gd	Yes.
K. C. D.	O	0	6,500	4,200	5	2	Sh, Cr	Cr, Cr	Th, W	No	4 pl	do	254	10	64	Gd	Yes.
W. W. W.	O	0	7,000	4,850	12	2	Sh, Cr	Cr, Cr	Th, W	No	4 pl	do	254	10	71	Gd	Yes.
C. C. M.	R	4	9,200	7,000	17	3	Ha, Ha	Cr, Cr	Th, W	No	4 pl	do	254	10	74	Gd	Yes.
F. D. L.	N	1	6,400	5,200	4	4	Sh, Cr	Cr, Cr	None	No	4 pl	do	254	10	75	Gd	Yes.
W. J. E.	N	1	6,100	5,200	2	2	Sh, Cr	Cr, Cr	None	No	4 pl	do	254	10	78	Fat	Yes.
J. J. B.	N	1	7,500	5,300	15	6	Cl, Cl	Cr, Cr	Th, W	No	4 pl	do	254	10	85	Gd	Yes.
W. N. M.	O	0	7,500	4,200	25	8	Sh, Cr	Cr, Cr	None	No	4 pl	do	254	10	85	Gd	Yes.

[a] See table 4 for key to symbols.

TABLE 7.—*Group treated 91 or more days after onset of illness* [a]

Case	Reaction and day of occurrence		White blood count		Sedimentation rate		Urine		Urethral discharge		Prostate		Total dose in grains	Total treatment days	Day of illness therapy was begun	Subjective results	Apparent clinical cure
	Type	Day	Before	After	Before	After	Before	After	Before	After	Before	After					
H. A. H.	N	1	6,500	5,200	15	3	Cl Cr	Cr Cr	No	No	4 pl	Nor	254	10	92	Gd	Yes.
D. A. R.	N	1	7,500	5,000	14	3	Cl Cl	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	97	Gd	Yes.
M. W. B.	N	1	9,100	5,200	12	3	Cl Cr	Cr Cr	No	No	4 pl	Nor	254	10	99	Gd	Yes.
H. C. E.	N	1	7,800	3,850	13	3	Cl Cl	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	108	Gd	Yes.
R. E. H.	N	2	8,000	4,000	8	6	Cl Cl	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	109	Gd	Yes.
H. C. B.	N	1	7,300	5,000	10	3	Cl Cl	Cr Cr	No	No	4 pl	Nor	254	10	113	Gd	Yes.
C. H. H.	N	1	8,400	6,400	6	3	Cl Cl	Cr Cr	No	No	4 pl	Nor	254	10	114	Gd	Yes.
J. M. H.	N	1	7,400	5,900	12	5	Ha Cl	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	131	Gd	Yes.
H. A. M.	N	1	7,250	8,000	8	5	Ha Ha	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	131	Fat	Yes.
J. N. P.	N	1	10,200	5,500	10	4	Cl Cr	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	144	Gd	Yes.
H. E. S.	N	1	9,000	4,600	7	5	Ha Cl	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	147	Gd	Yes.
L. E. C.	N	1	6,700	3,400	15	3	Sh Cr	Cr Cr	No	No	4 pl	Nor	254	10	154	Ner	Yes.
W. W. P.	N	1	5,600	4,600	5	3	Ha Ha	Cr Cr	Th W	No	4 pl	Nor	254	10	216	Gd	Yes.
H. E. F.	N	1	6,500	5,200	5	3	Sh Cr	Cr Cr	Tk y-w	No	4 pl	Nor	254	10	239	Gd	Yes.
B. D. M.	N	1	9,750	6,000	3	3	Cl Cr	Cr Cr	Th W	No	4 pl	Nor	254	10	306	Gd	Yes.
V. F. T.	N	1	6,800	5,000	13	2	Sh Cr	Cr Cr	Th W	No	4 pl	Nor	254	10	331	Fat	Yes.
B. M. M.	N	1	6,900	5,000	3	2	Cl Cr	Cr Cr	No	No	4 pl	Nor	254	10	352	Gd	Yes.

[a] See table 4 for key to symbols.

DISCUSSION

In comparing each individual's reaction to sulfapyradine with his reaction to sulfanilamide, it is apparent that in the dosage used in this series of cases, sulfapyradine, is less toxic. By comparing apparent cures elicited with the two drugs sulfapyridine is definitely superior to sulfanilamide in the treatment of gonococcal infections.

The previous treatment with sulfanilamide evidently had no effect on the results obtained with sulfapyridine.

It appears that there is a time interval necessary for active immunity to be established before maximum results with sulfapyridine may be obtained as demonstrated by the lower percentage of cures in the 20-or-less-day group. It is the treatment of this interval period that should be studied to determine the most efficacious method of handling the cases during this time.

However, it is debatable in the opinion of the writers, whether or not all acute cases should not immediately be given sulfapyridine therapy, as the results in the small number of acute cases (28), (1–21 days) showed only 17.85 percent failure, which is far superior to any results obtained in any other form of treatment in a 10-day period of time. In a military organization where man power is vital, it is believed, in accordance with above opinion that therapy should be started immediately, rather than waiting for a possible active immunity to develop.

Of the 70 cases treated, nine failed to respond at the end of 10 days therapy. Continued therapy with increased dosage as noted on charts 4, 5, and 6 was carried out on 8 of these cases with successful results on 3.

It was interesting to note the 2-glass tests as daily specimens changed rapidly from many shreds to a hazy filament which on subsequent days disappeared.

SUMMARY OF TABLES 4, 5, 6, AND 7

1. 75.7 percent experienced nausea.
2. Average drop in white count during course of treatment, 2,514.
3. The drug had little or no effect on hemoglobin.
4. Average drop in sedimentation time, 4.74 mm. The greatest fall occurred in the cases having chronic infections.
5. 5.07 percent felt weak or fatigued.
6. 8.57 percent experienced anxiety and nervousness.
7. 1 case developed an epididymitis during treatment.
8. 1 case developed a drug rash which persisted for 7 hours.
9. 3 cases of stricture were met with in men who had previously been under local treatment.

10. Average total dosage of drug received, 263.2 grains.

11. Average number of days treated, 10.4.

12. 91.44 percent of the series were apparently cured.

Of the entire series, when onset of illness preceded treatment by: (*a*) 1–10 days, 87.5 percent were cured. (*b*) 11–20 days, 75 percent were cured. (*c*) 21–90 days, 96 percent were cured. (*d*) 91 or more days, 100 percent were cured.

The blood levels of sulfapyridine maintained and the level reached with dosage used, unfortunately, are not known, due to the inadequate facilities aboard ship for carrying out these determinations.

CONCLUSIONS

1. 70 cases of gonorrhea were treated with sulfapyridine with 91.44 apparent cures.

2. Sulfapyridine may be given in ambulatory cases of gonorrhea with a far more reasonable margin of safety if carefully supervised, than sulfanilamide.

3. Sulfapyridine is superior to sulfanilamide in the treatment of gonorrhea.

4. Sulfapyridine has a definite place in the treatment of gonorrhea aboard ship.

5. Ambulatory doses as given apparently can maintain the blood sulfapyridine at a level sufficiently high to effect a cure.

6. The toxic symptoms experienced in our cases were negligible. A method of treating them has been outlined.

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.

ARTIFICIAL PNEUMOTHORAX, Its Practical Application in the Treatment of Pulmonary Tuberculosis. *Contributions by Saranac Lake Physicians to the Studies of the Trudeau Foundation, Editorial Committee, Edward N. Packard, M. D.; John N. Hayes, M. D.; Sidney F. Blanchet, M. D.;* 300 pages, illustrated with 85 engravings. Lea and Febiger, Philadelphia, 1940. Price, $4.00.

This latest addition to the Trudeau Foundation Studies is a handbook on the practice of artificial pneumothorax. The use of artificial pneumothorax in the treatment of pulmonary tuberculosis has become widespread in this country since 1912. Today, this form of treatment is used so extensively that there is a real need for such a handbook. In recent years, the literature on the subject of artificial pneumothorax treatment has become so abundant and expresses so many conflicting opinions that a practical clarification of the whole subject has become necessary. This monograph fulfills this purpose in an excellent manner. It is based on the individual practical experience of a group of specialists having sanitarium as well as private practice. The proper selection of cases for this form of therapy, the indications and contraindications to its use, the technic of the operation and the complications encountered and their treatment are thoroughly and clearly discussed. Other forms of collapse therapy such as pneumolysis, oleothorax, phrenic nerve operations and thoracoplasty are briefly considered.

The subject matter is well presented and the illustrations are excellent. This book maintains the high scientific purpose of the Trudeau Foundation. Every physician who is called upon to treat pulmonary tuberculosis will find this little book an invaluable guide to this form of therapy.

TREATMENT OF WAR WOUNDS AND FRACTURES, by *J. Trueta, M. D., late director of the department of surgery, General Hospital of Catalonia, Barcelona.* With a foreword by *H. Winnett Orr, M. D., F. A. C. S.*, 146 pages and 48 illustrations. Published by Paul B. Hoeber, New York, 1940. Price $2.50.

This interesting little book reports the results of the application of the Orr treatment to war surgery in Spain. In Catalonia where 20,000 wounded patients were treated by excision of devitalized tissues

and immobilization in plaster, the incidence of gas gangrene and other infections was markedly reduced.

In the early days of the Spanish War there was an organization of first-aid posts which delayed arrival of casualties at hospital and a resulting high amputation and death rate. After a time, the first-aid posts were abolished and from then on both rates fell. The author believes that in all patients received within 6 hours after injury the wounds should be treated by excision and the application of closed plaster. If a fracture is present it should be reduced at the time surgery is done. Gunshot wounds should practically never be sutured after debridement. He prefers packing of wounds with sterile dry gauze rather than vaseline gauze and changes casts only when the foul odor becomes too offensive. He places great stress on the time element in the treatment of war wounds and thinks competent surgical treatment should be available at the first possible moment. This at the front should be the casualty-clearing station.

His experience leads him to believe that antitetanic serum should be used prophylactically but that antigas gangrene serum is of little value in prevention. He does, however, use it in the active treatment of gas gangrene.

Dr. Trueta presents statistics on his 1,073 personally treated cases which resulted in only 6 deaths and 91 percent of good or satisfactory results.

The Orr treatment has been slow in gaining favor among industrial surgeons chiefly due to the offensive odor of dressings and the fear of gas gangrene developing in the covered part. Dr. Trueta has demonstrated that by early excision of devitalized tissue and complete rest by immobilization in plaster, gas gangrene seldom occurs. It is believed that this work will do much toward placing the Orr treatment on the high plane that it deserves.

DIAGNOSIS AND MANAGEMENT OF DISEASES OF THE BILIARY TRACT by *R. Franklin Carter, M. D., F. A. C. S., associate clinical professor of surgery, New York Post-Graduate Medical School, Columbia University, director of surgery, Gouverneur Hospital, New York City; Carl H. Greene, Ph. D., M. D., F. A. C. P., associate clinical professor of medicine, New York Post-Graduate Medical School, Columbia University, New York City, etc.;* and *John Russell Twiss, M. D., F. A. C. P., assistant clinical professor of medicine, New York Post-Graduate Medical School, Columbia University, assistant physician, O. P. D., New York Hospital, New York City.* Octavo, 432 pages, illustrated with 84 engravings with 6 plates. Published 1939. Lea and Febiger. Price $6.50.

Ten years ago the clinic for the study of diseases of the liver and biliary tract, of the department of medicine and surgery, New York Post-Graduate School and Hospital, Columbia University, was established. This work now is the result of the experience gained by the staff of this clinic in collaboration with other distinguished specialists.

At the onset of the work the authors laid down the following

objectives: (*a*) A better understanding of the basic factors which are responsible for gall-bladder disease, (*b*) improvement in the methods of the detection of these basic factors, (*c*) a more specific medical therapy based upon the findings in individual cases, and (*d*) more conservative surgical management in patients with chronic cholecystitis and a functioning gall-bladder that contains no stones.

Much emphasis is placed on diagnosis and medical management and the need for cooperation between the physician, surgeon, roentgenologist, biochemist, and dietitian in handling these cases is pointed out. The indications for, and the technic of, the various liver function tests are given in detail.

The chapter on functional disorders of the biliary tract (biliary dyskinesia) is both interesting and enlightening. The authors feel that in many of these patients the formation of stones can be prevented by proper medical management. From their experience surgery has little to offer in dyskinesia except in those patients in whom reflex causes are discovered and can be removed by surgery.

The chapter on acute cholecystitis brings forth the old argument of immediate operation versus delayed operation and it leaves one with the idea that the lowest mortality and morbidity in acute cholecystitis is found in the patients operated upon within 4 days of the onset of the present illness and after a period of 6 to 24 hours in the hospital in which to prepare them properly.

A good bibliography follows each chapter. The text is well illustrated and the index, although not large, appears adequate. This work represents a vast amount of study on a large number of cases and it is safe to predict that it will soon become a favorite of both the physician and the surgeon.

DISEASES OF THE GALLBLADDER AND BILE DUCTS, by *Waltman Walters, B. S., M. D., M. S. in Surgery, Sc. D., F. A. C. S. head of section in division of surgery, The Mayo Clinic; professor of surgery, The Mayo Foundation (University of Minnesota)* and *Albert M. Snell, B. S., M. D., M. S. in Medicine, F. A. C. P. head of section in division of medicine, The Mayo Clinic; professor of medicine, The Mayo Foundation (University of Minnesota.)* 645 pages and 342 illustrations on 195 figures. Published by W. B. Saunders Company, Philadelphia and London. 1940. Price $10.00.

Coming from the Mayo Clinic this excellent book is based upon the accumulated experience of a large staff over a period of more than 50 years.

The work is divided into five parts, the first of which deals with anatomy, physiology, and pathology, and the second with diseases of the gallbladder. Part three covers diseases of the bile ducts; part four medical and surgical management, and part five preoperative and postoperative care.

Although the chapters on diagnosis and medical management are complete and very well written one cannot help but feel that the

book's greatest value appears in its handling of surgical problems of the bile ducts, and especially those associated with jaundice. The surgeon who only occasionally has to deal with these knotty problems will find much to assist him in these chapters.

The book reads well and the illustrations are outstanding. An extensive bibliography follows each chapter and the index is complete.

This book will be a welcome and useful addition to any physician or surgeon's library.

INJECTION TREATMENT of Hernia, Hydrocele, Ganglion, Hemorrhoids, Prostate Gland, Angioma, Varicocele, Varicose Veins, Bursae and Joints by *Penn Riddle, B. S., M. D., F. A. C. S. assistant professor of clinical and operative surgery, Baylor University, College of Medicine; director of the varicose vein clinic, Parkland Hospital, Dallas, Texas.* 290 pages with 153 illustrations. W. B. Saunders Company, Philadelphia and London. 1940. Price $5.50.

This excellent little book completely covers all of the conditions for which injection treatment is commonly used. As might be expected the greater part of the work is given over to the treatment of hernia, varicose veins, and hemorrhoids. The author is to be commended upon the impartial manner in which he has presented the subjects. This is especially true in regard to the injection treatment of hernia, a subject that during the past few years has caused a great deal of controversy. He discusses freely the indications and contraindications for injection of hernia and points out the pitfalls encountered. He leaves one with the impression that, although the injection treatment of hernia in some respects has many advantages over surgery, still it is inferior to surgery as a curative agent.

In the chapters on varicose veins he discusses the classification and etiology and gives the various tests for determining the condition of the superficial and communicating valves and the patency of the deep venous circulation. Many excellent illustrations add to the usefulness of the book and it is certain to prove of great value to those doing office practice.

GYNECOLOGY, MEDICAL AND SURGICAL, by *P. Brooke Bland, M. D., F. A. C. S. professor emeritus of obstetrics, The Jefferson Medical College, Philadelphia; consulting obstetrician to The Jefferson College Hospital, Philadelphia; consulting obstetrician to the Philadelphia Lying-In Hospital; formerly associate professor of gynecology in the Jefferson Medical College and visiting gynecologist to St. Joseph's Hospital, Philadelphia, etc.* Assisted by *Arthur First, M. D., associate in obstetrics, The Jefferson Medical College Hospital, Philadelphia; associate in obstetrics, Mt. Sinai Hospital, Philadelphia; assisting gynecologist, Stetson Hospital, Philadelphia.* Third revised edition, 843 pages, 445 illustrations, mostly original, with 31 full-page plates in color. F. A. Davis Co., Phila. 1939. Price $8.00.

The third edition has been almost completely rewritten to embrace the new discoveries in this specialty. The text is simplified in such a manner as to serve both the student and the general practitioner. The illustrations are very good, new ones having been added and old ones replaced.

Of special interest are the chapters on endocrine therapy, endometriosis and gynecological radiology, with special consideration of the estrogen phase of the menstrual cycle as contrasted with the progestin phase, endocrinopathic causes of amenorrhea and the chemotherapeutic measures used in treatment of various gynecologic conditions.

Many of the standard surgical procedures are rather fully discussed and illustrated, so that the surgical as well as the medical aspects of this specialty are well covered.

PRACTICAL OBSTETRICS, edited by *P. Brooke Bland, M. D., emeritus professor of obstetrics, Jefferson Medical College; consulting obstetrician, Jefferson Medical College Hospital, Philadelphia, Pa.;* and *Thaddeus L. Montgomery, M. D., clinical professor of obstetrics, Jefferson Medical College, Philadelphia, Pa.* Third revised edition, 877 pages, with 502 engravings including 27 colored plates. F. A. Davis Co., Philadelphia. 1939. Price $8.00.

The authors have revised this edition in such a manner that it is practical, comprehensive and is a distinct and valuable contribution to obstetrical literature.

Prenatal supervision, clinical record, the toxemias of pregnancy, complications, normal and abnormal labor and the puerperium are dealt with realistically and rationally. The presentation of the different tests of pregnancy is excellent, and to the point. Obstetrical analgesia and anaesthesia are discussed in detail with indications and contraindications for their use.

The authors still advise packing the uterus following curettage for incomplete abortion, although statistics show an increased morbidity and mortality with this technic in infected cases.

A MANUAL OF OTOLOGY, RHINOLOGY, AND LARYNGOLOGY, by *H. C. Ballenger, M. D., F. A. C. S., assistant professor of otolaryngology, Northwestern University School of Medicine, Chicago, Ill.*, 302 pages, illustrated with 90 engravings and 4 color plates, Lea & Febiger, Philadelphia, publishers, 1940. Price $3.75.

This text of 300 pages was primarily written for the undergraduate medical student. The author has accomplished his objective. Most texts written for the undergraduate are too voluminous for the time alloted in the study of ear, nose, throat, and laryngeal diseases.

The otolaryngologist must avail himself of a more extensive text than this for reference. Surgical technic has been omitted. The naval medical officer on independent duty would also prefer a more detailed text which deals more with treatment.

This is, however, a handy reference book for the naval medical officer in a hospital not on the nose and throat service. It is likewise a splendid text for those nurses and hospital corpsmen in the nose and throat clinics.

The diagrams are splendid and that part devoted to anatomy is well done. Hearing tests are concisely described as well as the methods for the detection of simulated deafness.

In brief this is a good compend dealing with otology, rhinology and laryngology.

PROCTOLOGY FOR THE GENERAL PRACTITIONER, by *Frederick C. Smith, M. D., M. Sc. (Med.); F. A. P. S., proctologist to St. Luke's and Children's Hospital Philadelphia; formerly associate in proctology, Graduate School of Medicine, University of Pennsylvania.* First edition, 386 pages, illustrated with 142 half tones and line engravings and 3 color plates. F. A. Davis Company, Philadelphia, 1939. Price $4.50.

The text is an excellent treatise on rectal diseases for the general practitioner. Some of the illustrations are especially good, particularly those on local anaesthesia, while others are not so clear.

The subject matter is presented in a clear and precise manner, necessarily brief to cover practically the entire field of proctology in one volume.

SHOCK. Blood Studies as a Guide to Therapy, by *John Scudder, M. D., Med. Sc. D., F. A. C. S.* From the *surgical pathology laboratory of the College of Physicians and Surgeons, Columbia University,* and the *department of surgery, the Presbyterian Hospital, New York City.* First Edition. 315 pages, 55 illustrations. J. B. Lippincott Company, Philadelphia, Montreal, and London. Price $5.50.

The author has divided the book into four parts. The first part deals with the historical and experimental work on shock. The second part deals with the varieties of shock, their analysis and treatment. The third part deals with historical development and bibliography, and the fourth part is a short laboratory manual.

Although there are other laboratory tests to measure hemoconcentration, the author describes four simple tests. The determination of cell volume by hemocrit, specific gravity of whole blood and plasma, and plasma protein determination. These tests can be rapidly accomplished and early treatment instituted.

The author presents the laboratory findings in 28 cases of shock from various causes; postoperative shock, shock due to trauma, shock due to trauma complicated with hemorrhage, shock due to hemorrhage, shock due to burns, shock due to perforated duodenal ulcer and primary shock due to paraganglioma of adrenal tissue.

The treatment of these cases is given in detail. The maintaining of body heat, adequate fluid administration, and control of pain are important factors. The efficacy of transfusion is demonstrated. The author outlines the importance of cortical hormone from the adrenal gland in the treatment of shock. It was used in the treatment of 27 cases. The dosage used depended on its effect in raising blood pressure. The results of the treatment employed by the author were very good.

The role that potassium plays in shock has proven to be of great interest. In the author's cases there were no recoveries in those

cases having an increase in plasma potassium over 100 percent or a decrease of over 25 percent.

The bibliography is extensive and covers 533 articles.

This is an excellent book and the author is to be commended on this work. This book is highly recommended to those who are called upon to treat cases of shock from whatever cause.

SYNOPSIS OF PRINCIPLES OF SURGERY by *Jacob K. Berman, A. B., M. D., F. A. C. S., assistant professor of surgery, Indiana University School of Medicine, Indianapolis.* 615 pages. 274 illustrations. C. V. Mosby Company, St. Louis, Missouri, publishers. 1940. Price $5.00.

The author has performed an excellent piece of work in boiling down the principles of surgery so that they could be presented in a small compend of this size.

In this concentrating process the essentials have not suffered but due to the lack of available space discussion on the various subjects has been limited. This at times makes the author appear rather dogmatic which probably adds to the value of the book as it was primarily written for the medical student. To compensate for this brevity of discussion the author gives a large number of selected references at the end of each chapter for those seeking further information.

This book, which measures 8 by 5 by 1 inches in size, is refreshingly new, very well written, and exceptionally well illustrated. The index is adequate.

IMPROVISED EQUIPMENT IN THE HOME CARE OF THE SICK. By *Lydia M. Olson, R. N., superintendent of nurses, Kahler Hospital, Rochester, Minnesota.* Third edition. W. B. Saunders Company, Philadelphia. 1939. Pp. 264. Ill. 419. Price $1.50.

During the era of "little wars" which occupied the first quarter of the present century, navy doctors used to pride themselves in their ability to do good professional work with the minimum of equipment. Out in the bosque, with nothing except a few pills, a pocket case, and what could be picked up in a native hut, they were at their best. Here is a book designed to help in just such emergencies of sickness for which there is inadequate preparation. The author has gathered together all the *ersatz* tricks known to housewives, nurses, and doctors, and condensed them into a small volume, profusely illustrated, which should be required reading for all our doctors and nurses who are going for duty in primitive places.

DISEASES OF THE FOOT, by *Emil D. W. Hauser, M. S., M. D., assistant professor of bone and joint surgery, Northwestern University Medical School; attending orthopedic surgeon, Passavant Memorial Hospital, Chicago.* 472 pages, 173 illustrations. W. B. Saunders Co., Philadelphia, 1939. Price $6.

Books have been written on diseases of the foot, but nothing so comprehensive has previously come to the attention of the reviewer. The author's vast experience permits him to write in a manner that

inspires confidence and challenges the reader to find a fault in his work.

Interesting chapters are found on hygiene and care of the feet, fitting of shoes, training in walking, and postural disturbances and their relation to the foot. The diagnosis and treatment of diseases of the skin, nails, bones, joints, nerves, and vascular system are covered in a highly satisfactory manner, as are fractures, sprains, and other traumata of the foot.

Much space is given over to pes valgoplanus and after one has finished reading the chapter, he is apt to possess a much more optimistic view as to the possibility of relief for his flatfooted patients.

To the naval medical officer whose daily task aboard ship brings him in contact with patients having foot complaints, this book will be of inestimable value. It should occupy a prominent place in the medical library.

COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION. Volume 31, 1939. W. B. Saunders Co., Philadelphia. 1940. Pp. 1000. Ill. 235. Price $11.50.

The thirty-first volume shows no recession from the high standards set by its predecessors. It is a matter of regret that the intensely practical articles that have appeared in Medical Clinics of North America, and Surgical Clinics of North America are omitted from the annual volume. To the practitioner, these were the most valuable of all. Limitations of space have also made necessary the inclusion of many papers by title and reference only, while others appear in abstract or abridgement.

Photographs of William J. Mayo and Charles H. Mayo recall the great loss that the medical profession of the world has sustained in the departure of these distinguished leaders.

ROENTGEN DIAGNOSIS OF THE EXTREMITIES AND SPINE by *Albert B. Ferguson, M. D., director of roentgenology, New York Orthopaedic Hospital, instructor in orthopaedic surgery, College of Physicians and Surgeons, Columbia University.* Illustrated. Paul B. Hoeber, Inc., publisher, 1939. Price $12.00.

This volume, the latest addition to the series of monographic atlases from the Annals of Roentgenology presents a contribution well worthy of attention and careful study.

The author introduces his subject with a dissertation on description and definitions. He speaks strongly against the usual manner of roentgenological description as being of little diagnostic utility and recommends a briefer mode directed toward diagnostic essentials. Rather extreme examples are given to illustrate his point but one cannot help but be in accord with the general proposition that description should be brief, analytic, and devoted to points of importance to differential diagnosis. As an aid he offers a number of descriptive terms such as "active destruction," "atrophic loss of substance,"

"reactive periosteal calcification," "inert periosteal calcification," "pyogenic density," "luetic density," etc. In the opinion of the reviewer, however, certain of these terms do not appear desirable for the simple reason that they represent conclusions rather than description. They are apt to mean either too little or perhaps too much to any but those expert in roentgenological interpretation. In other words, I feel in accord with the general viewpoint but have little enthusiasm for some of the terms.

As the book unfolds we find that the general orientation is chiefly though not entirely along pathological lines, with the clinical entities being brought in incidentally to the pathological appearances in the bone. This gives the book an excellent value in those cases which happen to present difficult problems of interpretation. It also makes the book worthy of careful study for better comprehension of the fundamental principles underlying roentgenological diagnosis. On the other hand for routine general reference one will probably find other works more convenient. It must be said, however, that an excellent index and good cross references do much to increase its value as a work of general reference.

The first sections of the book are devoted to calcareous matters in general, with thorough consideration of the various types of bone and their pathology. There is also a systematic account of calcifications in soft tissues. This is a very gratifying feature, though one rather regrets that the consideration of the frequently encountered and quite important condition, most often seen in the shoulder joint, and variously designated as chronic bursitis (*e. g.* subdeltoid bursitis), paraarthritis or peritendonitis calcaria, is so rudimentary. Tumors receive a brief but penetrating analysis; one wishes however there were more of it. The same holds good for fractures.

A number of chapters are devoted to the arthritides. Degenerative and rheumatoid varieties are very well illustrated. Tuberculosis of the joints receives a separate chapter as befits its importance, and the nontubercular infectious arthritides of various types are adequately described in another.

In the final chapter special consideration is given certain spinal conditions. Under "Wedging round back," the author treats of the wedging as due primarily to abnormally wide grooves in infancy, and apparently considers the so-called epiphysitis of adolescence a secondary manifestation, as it possibly may be. The etiology of the disturbances of the epiphyses usually listed as osteochondritis or osteochondrosis is not yet on a very firm footing. Scoliosis and anomalies particularly of the lumbosacral region are well described. Disk protrusion is given little more than bare mention, the reader being invited to look up recent articles on this subject. The author regards roentgenological demonstration of protrusion of the disks

with little favor due to fear of irritation from injected material. Much work, however, is being done along such lines and it will probably soon be evident whether or not such fears are justified.

Illustrations are, of course, copious as befits an atlas. They are in general adequate and often excellent. Clear and pertinent notes accompany the illustrations and in each instance a brief case history is appended at the end of the chapter.

OUTLINE OF ROENTGEN DIAGNOSIS; An Orientation in the Basic Principles of Diagnosis by the Roentgen Method, by *Leo G. Rigler, B. S., M. B., M. D., professor of radiology, University of Minnesota, Minneapolis, Minn.*, Atlas edition and students edition, 254 illus. shown in 227 figures, presented in drawings and reproductions of roentgenograms. Figures 6 to 51 and 55 to 72 are drawings in an original technic by Jean E. Hirsch. J. B. Lippincott Co., publishers, 1938, Price $3.

This excellent work really lives up to its title and presents a concise account of the fundamentals of roentgen diagnosis. It is of course, as the author himself states in his preface, a synopsis rather than a work of reference and as such its greatest use is as a teaching guide and handy volume for general review.

Under general principles the author makes the excellent point that x-ray findings should be regarded in the same light as gross pathological findings. He also warns us of inherent limitations. Certain features of the roentgenograph and a few technical factors are then briefly mentioned. The technic of x-ray photography is dismissed casually as something that can be learned with relative ease. This seems rather an underestimation of the matter and I imagine the author has been well favored by having excellent, permanently placed technicians. The normal and abnormal distribution of gas is next given, followed by lists of diseases and organs susceptible to x-ray diagnosis, and finally definitions and roentgenological terms.

Bones and joints are in general well considered. However, a lengthier and more complete presentation of fractures and dislocations would be helpful especially since this aspect of roentgenology appears deceptively easy to the uninitiated. Diseases and tumors are excellently presented. The arthritides are described along conventional lines. The spine and skull are adequately reviewed and up-to-date notations are included with reference to myelography and encephalography.

The thorax receives ample treatment and tuberculosis is especially well considered. The diagnostic place of roentgenology in regard to this disease is given very good evaluation. On the technical side, however, the author's statement that stereoscopic films are imperative is rather extreme.

The chapter on the abdominal organs leaves little to be desired except that one could wish for more lengthy consideration of urography and also of gynecological roentgenology.

In the Atlas Edition, illustrations are grouped at the end of the volume. These are not especially numerous but are well chosen and are technically good reproductions. Clear explanatory notes accompany them. Especially noteworthy is an excellent group of drawings illustrative of the various diseases and injuries of the bones and joints.

In general these volumes are worthy editions to the teaching literature of roentgenology.

TEXTBOOK OF NERVOUS DISEASES. By *Robert Bing, professor of neurology, University of Basel, Switzerland.* Translated and enlarged by *Webb Haymaker, assistant clinical professor of neurology, University of California, San Francisco.* From the fifth German edition. Pp. 838, with 207 illustrations. St. Louis: C. V. Mosby Company, 1939. Cloth. Price $10.

This volume is a condensation of a massive amount of material and is the English translation of Professor Bing's Lehrbuch der Nervenkrankheiten which was first issued in 1913 and is now in its fifth edition. Dr. Haymaker in his liberal and splendid translation has rearranged the material from lecture form by putting it into various chapters. He has also added considerable new data in regards to anatomy, physiology and modern concepts of treatment which add a great deal to the book for English and American readers.

The book is comprised of 30 chapters; each is followed by numerous references which include the fundamental concepts and up-to-date views on the subject. The illustrations are profuse and beautifully done; a number are taken from Professor Bing's widely read Compendium of Regional Diagonsis in Lesions of the Brain and Spinal Cord. The therapeutic discussions are unusually full and practical. The section on the psychoneurosis is dealt with in a simplified and concise manner with most stress being laid upon the clinical aspects.

We have no hesitancy in unreservedly recommending Dr. Haymaker's enlargement of Bing's Textbook of Nervous Diseases. It is one of the great modern textbooks of neurology and should be available to every student and practioner of neurology.

REGIONAL DIAGNOSIS IN LESIONS OF THE BRAIN AND SPINAL CORD. By *Robert Bing, professor of neurology, University of Basel, Switzerland.* Translated and enlarged by *Webb Haymaker, assistant clinical professor of neurology, University of California, San Francisco.* Eleventh edition. Pp. 292, with 125 illustrations. C. V. Mosby Company, St. Louis, 1939. Cloth. Price $5.

Dr. Robert Bing has long been known to English speaking physicians. His original book of 200 pages issued 30 years ago has been widely used by medical students and neurologists. The present excellent translation by Webb Haymaker has increased the size of the volume to 275 pages in keeping with recent advances in regional diagnosis, but it remains a concise, clear and exceptional work. There is probably no way to an easy approach to knowledge of the structure and function of the nervous system but it may be added that this volume comes closer to offering that approach than any other.

The book is comprised of 15 chapters. The first 3 deal with localization of spinal cord lesions in the transverse plane. The next 3 chapters deal with anatomic and physiologic basis of localization of spinal cord lesions in terms of segments. The following chapters are devoted to the localization of lesions of the brain stem, cerebellum, cerebrum, basal ganglia and the hypophysis. The material is well arranged and presented in such a way as to make its reading and understanding easy. One is not burdened with excessive descriptive material. The illustrations, with liberal legends, are mostly drawings by Dr. Bing and are admirably done.

The localization of tumors of the spinal cord by use of air myelography is not mentioned though mention is made of the use of radiopaque oil in the subarachnoid space.

On the whole, this work is probably the finest of its kind and one cannot commend too highly this exceedingly valuable and still compact book. It is especially useful for beginners in clinical neurology, but has much to offer the more advanced neurologist.

TEXTBOOK OF NEURO-ANATOMY AND THE SENSE ORGANS. By *Olof Larsell PhD. professor of anatomy, University of Oregon Medical School, Portland.* Pp. 316 with 232 illustrations. D. Appleton-Century Co., New York. 1939. Price $6.

Dr. Larsell has gathered his material from monographs, journals, textbooks of anatomy and physiology and has written an excellent textbook for first-year medical students. His arrangement and organization of the material are well done. He presents the fundamentals of neuro-anatomy including the most important microscopic, physiologic, and embryologic features in a concise manner. The student is guided in the anatomic, physiologic and pathologic methods of studying the nervous system.

The illustrations are numerous and well selected. A lucid style of writing renders the reading easy and interesting. Two excellent tables are given; one on the fiber tracts of the spinal cord and another on the principal visceral efferent or autonomic pathways, which are exceedingly useful to students of neurology. Concluding most chapters there is a discussion of clinical interpretation. A list of important references follows each chapter.

The book is well written and splendidly printed. It is a definite contribution to neuro-anatomy and should be of great value to any one who desires to orient himself in the fundamentals of this basic science.

LEGAL MEDICINE AND TOXICOLOGY by *Thomas A. Gonzales, M. D., acting chief medical examiner of the city of New York; associate professor of forensic medicine, New York University College of Medicine: lecturer on criminological medicine, New York Police Academy; Morgan Vance, M. D., assistant medical examiner of the city of New York; assistant professor of forensic medicine, New York University College of Medicine; lecturer in forensic medicine, College of Physicians and Surgeons,*

Columbia University; lecturer on criminological medicine, New York Police Academy; and *Milton Helpern, M. D., assistant medical examiner of the City of New York; assistant professor of forensic medicine, New York University College of Medicine; lecturer in legal medicine, Cornell University Medical College; lecturer on criminological medicine, New York Police Academy.* First Edition. 754 pages. Illustrated. D. Appleton—Century Co., Inc., New York, London, 1937. Price $10.

While this book has been off the press for some time, it is of sufficient merit to be called to general attention as it is one of the very few complete and authoritative works on legal medicine and toxicology.

The authors have derived their material from an experience which has included the investigation of more than 260,000 deaths, including over 25,000 toxicological cases. With such a background they can speak authoritatively on practically every conceivable manner of death and on all kinds of medico-legal problems

The book is divided into two sections. A partial list of subjects covered in the first part are: The medical examiner and the coroner, the Government and the physician, the identification of the living and the dead, the technic of autopsy, the signs of death, the determination of the multitude of causes of death, and practically every other medico-legal subject.

The second part of the book deals with almost all of the known poisons, their symptoms and toxicologic analysis, and with the various medico-legal and toxicologic aspects of the occupational diseases.

The volume is profusely illustrated by excellent pictures of actual observed cases. It will be found invaluable to the coroner, toxicologist, pathologist, criminologist, the legal profession, and physicians having to do with occupational hazards.

MARIHUANA—America's New Drug Problem—A Sociologic Question With Its Basic Explanation Dependent on Biologic and Medical Principles. By *Robert P. Walton, professor of pharmacology, School of Medicine, University of Mississippi,* with an introduction by *E. M. K. Geiling, professor of pharmacology, University of Chicago,* and a chapter by *Frank R. Gomila, commissioner of public safety, New Orleans, La.,* and *M. C. Gomila Lambou, assistant city chemist.* First Edition—223 pages with complete nomenclature associated with the hemp plant and drugs. J. B. Lippincott Co., Philadelphia. Price $3.

This excellent book is concerned with the problems of the marihuana evil and its sociological and physiological implications. Since there is danger that the menace may engulf the home, this book becomes of vital interest not only to parents but to teachers, clergymen, and leaders in social reform.

Marihuana is a drug of long and ignoble history. The rapid spread in the smoking of the *weed,* chiefly in the form of cigarettes, has become a threat to our national life. This evil affects all parts of the country and takes its toll from every stratum of society.

An important contribution to a fuller understanding of the subject is the discussion of the therapeutic applications of cannabis and its

pharmaceutical and chemical considerations. The chapter on word equivalents for the hemp plant or the crude drug is both interesting and informative.

The problem of marihuana will be solved only when the public becomes fully aware of its danger and takes appropriate measures to eradicate the evil. It is a well-written book and is recommended to the layman and professional reader.

HEADACHE AND HEAD PAINS: A Ready Manual for Physicians. By *Walton Forest Dutton, M. D., director, Medical Research Laboratories, Amarillo, Tex.* Pp. 301 with 6 illustrations. F. A. Davis Philadelphia, publishers, 1939. Cloth—Price $4.50

The clinician meets the problem of diagnosis and treatment of headache probably more frequently than any other symptom. The etiology of headache and its permanent relief are unfortunately often difficult to solve. The subject of headache encompasses nearly the entire field of human disease. Dr. Dutton has attempted to formulate a handy reference book dealing with the causes and treatment of various types of headache. The need for such a reference book is obvious, for heretofore one was forced to seek many sources for information included in this book.

In the introduction, neurophysiology, etiology of headache, analysis of causal factors, method of obtaining the history and the relief of pain are discussed briefly under separate subdivisions.

Two hundred and five affections causing headache are listed in alphabetical order which facilitates ready access to the desired information. Each entity is defined and described briefly and written prescriptions are given. Next follows in order of sequence remedies for diseases causing headache and head pains, index of remedies for headaches and head pains, and last, the general index.

The list of conditions giving rise to headache is fairly complete though obviously the discussion of each is very limited. The prescriptions listed under the various entities will be found useful, though a few drugs such as codeine sulfate, acetophenetidin (phenacetin) and acetylsalicylic acid are found repeatedly.

The volume is concisely written and the material well arranged. It will prove a handy reference book for the physician who does not have the time to refer to a standard system of medicine for the information desired.

THE DIVISION OF PREVENTIVE MEDICINE

Commander C. S. STEPHENSON, Medical Corps,
United States Navy, in Charge

TOXIC EFFECTS OF ARSENICAL COMPOUNDS

AS ADMINISTERED IN THE UNITED STATES NAVY IN 1939 WITH SPECIAL REFERENCE TO ARSENICAL DERMATITIS

By Commander C. S. Stephenson, Medical Corps, United States Navy, W. M. Chambers, Chief Pharmacist's Mate, United States Navy, and Laura T. Anderson, Senior Clerk, Bureau of Medicine and Surgery

For the past 15 years medical officers of the Navy have been required to submit to the Bureau of Medicine and Surgery monthly reports of the number of doses of arsenicals administered and the reactions therefrom. This information has been compiled and published in the following United States Naval Medical Bulletins:

September 1925.	October 1933.	October 1937.
January 1927.	October 1934.	January 1938.
January 1929.	January 1935.	October 1938.
July 1930.	October 1935.	January 1939.
October 1931.	January 1936.	October 1939.
October 1932.	October 1936.	January 1940.
April 1933.	January 1937.	

In table 1 is shown the number of doses of each arsenical administered in the year 1939, the reported reactions which occurred, and similar data for the 15-year period 1925–39. It is noted that in 1939 there was 1 reaction to 2,586 doses and 1 death to 32,324 doses. For the 15-year period 1925–39 there was 1 reaction to 1,470 doses and 1 death to 31,246 doses.

TABLE 1.—*Arsenicals, U. S. Navy, 1939 and 1925–39—type of drug, reaction, 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 1939:							
Bismarsen	457	0	0	0	0	0	0
Mapharsen	65,399	9	5	1	15	4,360	65,399
Neoarsphenamine	57,376	21	11	3	35	1,639	19,125
Sulpharsphenamine	943	0	0	0	0	0	0
Tryparsamide	5,120	0	0	0	0	0	0
Total	129,295	30	16	4	50	2,586	32,324
15-year period 1925–39:							
Acetarsone[a]	971	1	0	0	1	971	0
Arsphenamine	41,558	27	14	1	42	989	41,558
Bismarsen[b]	2,904	0	0	0	0	0	0
Mapharsen[c]	121,689	14	8	1	23	5,291	121,689
Neoarsphenamine	1,301,913	618	301	48	967	1,346	27,123
Silver arsphenamine[d]	586	0	1	0	1	586	0
Sulpharsphenamine	29,438	17	8	0	25	1,178	0
Tryparsamide	63,176	3	1	0	4	15,794	0
Total	1,562,235	690	333	50	1,063	1,470	31,245

[a] First administered during the year 1932.
[b] First administered during the year 1929.
[c] First administered during the year 1935.
[d] First administered during the year 1931.

TABLE 2.—*Proportion of reactions of various types, 1929–39*

Classification	Number of reactions	Percent of total reactions
Vasomotor phenomena	346	41.84
Arsenical dermatitis	308	37.24
Blood dyscrasias	42	5.08
Liver damage	35	4.23
Table reactions	26	3.14
Jarisch-Herxheimer	20	2.42
Reactions of minor importance	17	2.06
Gastro-intestinal	16	1.93
Hemorrhagic encephalitis	8	0.97
Optic neuritis	3	0.36
Arsenical neuritis	2	0.24
Acute renal damage	1	0.12
Border line, hemorrhagic encephalitis	1	0.12
Liver damage (doubtful reaction)	1	0.12
Vascular damage (probable adrenal hemorrhage)	1	0.12
Total	827	100.00

ARSENICAL DERMATITIS

Dermatitis in some form was observed in 22, or 44 percent, of the total reactions in 1939, as compared with 62.5 percent in 1938. The type of lesion was erythematous in 3 instances, exfoliative in 12, macular in 2, maculopapular in 4, and urticarial in 1. The reactions were classified as 12 mild, 9 severe, and 1 fatal.

A brief clinical history of each reaction follows:

MILD REACTIONS

The 12 mild reactions occurred after the following number of injections: 3 after the third injection, 1 each after the fourth, sixth, eighth, and ninth, 2 after the sixteenth, and 1 each after the thirty-seventh, forty-seventh, and sixty-fourth.

The interval between injection and appearance of symptoms varied from 15 minutes to 11 days. The length of time required for complete recovery varied from 1 hour to 33 days.

NEOARSPHENAMINE

(*1–1939*) A patient exposed to infection May 31, 1939, developed a primary lesion on the corona of the penis which was positive for *Treponema pallidum*.

Arsenical treatment began with a 0.4 gram injection of neoarsphenamine on June 2, 1939, followed by 0.8 gram injections on June 9 and 13.

Approximately 10 hours after the injection of neoarsphenamine on June 9 the patient developed a rash over the entire body. One gram of sodium thiosulphate was given intravenously, and the rash disappeared in 48 hours. Due to mild respiratory symptoms at onset, and the presence of catarrhal fever on board, it was thought that this was the probable diagnosis.

Two hours after the last injection the patient developed a generalized erythema, nausea, and vomiting. Temperature, 103°; urinalysis, 4-plus albumin; WBC, 10,150; polys, 75; bands, 3; lymphs, 20; eosins, 1; immature, 1.

Eight 1-gram injections of sodium thiosulphate were administered. Recovery in 6 days.

(*2–1939*) Following exposure to infection on December 22, 1938, this patient developed a penile lesion which was positive for *Treponema pallidum*. A Kahn blood test was 4-plus.

From February 12 to March 28, 1939, he received 8 injections of neoarsphenamine, a total of 6.40 grams.

The second course of arsenical treatment began on May 16, 1939, and consisted of 8 injections of neoarsphenamine, a total of 6.40 grams.

Twelve hours after the last injection on July 3 the patient developed an itching, macular rash on the dorsum of the hands and feet, and antecubital areas. He was given eight 1-gram injections of sodium thiosulphate intravenously. Recovery in 9 days.

(*3–1939*) A patient who was exposed to infection on September 4, 1938, developed a primary lesion immediately posterior to the coronary sulcus of the penis which was positive for *Treponema pallidum*. A Kahn blood test was 4-plus. He received 0.06-gram injections of mapharsen on December 22 and December 27, 1938, and a 0.6-gram injection of neoarsphenamine on January 3, 1939. Four injections of bismuth were given as concurrent treatment.

Two hours after receiving the injection of neoarsphenamine the patient became chilly, developed a temperature of 102.6°, and complained of pain and muscular soreness throughout the body and limbs. Examination revealed slight coryza, red and injected pharynx, and slightly edematous uvula. Two days later a fine petechial maculopapular rash was noted over chest and abdomen.

Antiluetic treatment with mapharsen was continued with no evidence of reaction. Recovery in 7 days.

(*4–1939*) After exposure to infection on October 15, 1938, this patient developed a penile lesion and general lymphadenopathy. A Kahn blood test was positive.

The first course of arsenical treatment consisted of 9 injections of neoarsphenamine, a total of 4.35 grams. After the seventh injection the patient developed a mild skin reaction, and the amount of neoarsphenamine for the eighth and ninth injections was reduced one-half. Erythema developed after the ninth injection on January 4, 1939, and remained for 24 hours. The symptoms reappeared on January 10 in a more severe form.

Examination revealed a mild exfoliative dermatitis and injected conjunctiva. The skin of the face was scaly, moist, and cracking in spots; the arms and forearms showed mild scaling; and there was a generalized redness of the thighs and legs on which were interposed many small papules.

The skin condition improved following colloidal baths and local applications of bland oil. The patient returned to duty 27 days after onset of symptoms.

On June 3, 1939, the second course of arsenical treatment began with a 0.03-gram injection of mapharsen, and 5 hours after the injection erythema of the skin and swelling of the wrists were noted. He was given daily injections of sodium thiosulphate for 3 days.

Recovery occurred in 4 days.

(*5–1939*) After exposure to infection on September 1, 1933, this patient devel oped a penile lesion which was positive for *Treponema pallidum*.

Arsenical treatment was instituted, and from October 18, 1933, to July 3, 1939, he received 31.85 grams of neoarsphenamine (number of injections not recorded) and 3 injections of mapharsen, a total of 0.18 gram; and from November 28, 1933, to August 22, 1939, 120 mercury rubs and 62 injections of a bismuth compound.

The course of treatment during which reaction occurred began on August 22, 1939, with a 0.6-gram injection of neoarsphenamine, followed by 0.8-gram injec-

tions on August 29, September 5, 12, 20, 25, October 4, and 10. After the injection on October 4 the patient complained of nausea and 48 hours after the injection on October 10 he reported with itching papular lesions under arms, on penis, and scattered over trunk. Because of history of exposure to scabies this was diagnosed scabies and sulphur ointment was applied daily for 3 days. The papular lesions enlarged and itching increased. The sulphur ointment was discontinued and calamine lotion applied. A differential smear on October 14 showed—polys, 63; lymphs, 23; bands, 1; eosins, 13; and granulation of polys.

The itching continued and papules spread to palms of the hands. Though a patch test was negative and eruption was complicated by applications this was considered a probable multiforme erythema resulting from neoarsphenamine. One gram of sodium thiosulphate was given intravenously twice daily for a period of 6 days. The rash and itching subsided rapidly and recovery occurred in 13 days.

(*6–1939*) This patient developed a penile ulcer after exposure to infection on September 19, 1939. A series of darkfield examinations were negative. A diagnosis of syphilis was made because of three positive Kahn blood tests and bilateral nontender inguinal lymphadenopathies.

Arsenical treatment began on October 5, 1939, with a 0.3-gram injection of neoarsphenamine, followed by 0.6-gram injections on October 10, 17, and 24. Four 0.13-gram injections of bismuth subsalicylate were given as concurrent treatment. Eleven days after the last injection of neoarsphenamine the patient developed a generalized maculopapular rash over entire body, including the face. There were lesions on edematous bases. Edema of the face and eyes was present. The temperature ranged from 99° to 103° for 4 days. General malaise for 2 days. Albuminuria was noted once during period of reaction.

Treatment consisted of 1,000 cc. of 5 percent glucose intravenously, twice daily for 9 days; sodium thiosulphate intravenously daily for 10 days; and 10 percent calcium gluconate daily for 10 days. Lesions faded gradually after 1 week and disappeared on November 17. Arsenical treatment was resumed with mapharsen and no adverse symptomatology was noted. Recovery in 16 days.

(*7–1939*) After exposure to infection in April 1939 this patient developed a primary penile lesion. Darkfield examinations of the lesion were negative. On June 8, 1939, a gland puncture was positive for *Treponema pallidum.*

Arsenical treatment began with a 0.4-gram injection of neoarsphenamine on June 6, 1939, followed by 0.8-gram injections on June 13, 20, and 27, a 0.2-gram injection on July 3, and a 0.3-gram injection on July 11.

Due to severe itching of the skin following the third and fourth injections, the fifth and sixth were reduced in size. Itching continued. No eruption noted. One-gram injections of sodium thiosulphate were administered on July 12, 13, and 14. Recovery in 3 days.

(*8–1939*) Exposure to infection occurred on December 7, 1938, and 1 month later this patient developed an ulcer in the pubic region, typical of lymphogranuloma venereum. A Kahn blood test was 3-plus on January 16, 1939.

A 0.3-gram injection of neoarsphenamine was administered on January 24, 1939, and a 0.6-gram injection on January 31. Six days after the last injection the patient developed a rash on the abdomen and back, and a sore throat. These symptoms cleared 1 week after onset. A 0.1-gram injection of neoarsphenamine was administered on February 14 and the rash reappeared the following day. This condition persisted for 4 days and quickly subsided with no further complications. Recovery in 6 days.

(*9–1939*) This patient developed a penile lesion and marked glandular enlargement after exposure to infection on February 17, 1938. A darkfield examination was positive for *Treponema pallidum.*

From April 30, 1938, to May 11, 1939, he received 34 injections of neoarsphenamine, a total of 19.35 grams, and from June 27, 1938, to July 14, 1939, he received 46 injections of bismuth compounds.

The fourth course of arsenical treatment began with 0.6-gram injections of neoarsphenamine on July 6, 13, and 20, 1939. Fifteen minutes after the last injection the patient developed urticarial wheals in small patches over the body. The veins were dilated and prominent, the conjunctiva was injected, and itching of the skin was evident. One gram of sodium thiosulphate was given intravenously. Recovery in 1 hour.

MAPHARSEN

(*10–1939*) One month after exposure to infection in 1938, this patient developed a penile lesion which was positive for *Treponema pallidum.*

From August 28 to October 15, 1938, he received 8 injections of neoarsphenamine, a total of 3.75 grams. Two days after the last injection he developed an itching rash which gradually developed into a severe exfoliative dermatitis. (See case 20–1938, October, 1939, Bulletin.)

From December 17, 1938 to June 13, 1939, he received 8 injections of bismuth, a total of 2.34 grams. The second course of arsenical treatment was instituted on August 22, 1939, with a 0.015-gram injection of mapharsen. Six hours after this injection the patient developed a rash on the body, arms, and legs, macular in type and morbilliform in appearance and distribution. One gram of sodium thiosulphate was administered intravenously on August 22 and 23. Complete recovery in 5 days.

(*11–1939*) This patient developed a penile lesion following exposure to infection on August 31, 1938. A darkfield examination of the lesion was positive for *Treponema pallidum.*

He received 11 injections of neoarsphenamine (total grams not recorded) from November 16, 1938 to January 10, 1939, and 10 injections of iodobismitol from December 5, 1938 to February 6, 1939.

The second course of arsenical treatment began with a 0.03-gram injection of mapharsen on January 24, 1939, followed by 0.06-gram injections on February 1, 7, 14, and 21. Following the last two injections the patient complained of itching but no skin eruption was noted.

Six days after the last injection physical examination revealed the patient to have a few reddened, scaly areas scattered over the surface of the body. The eruption became more extensive after a few days, and he was admitted to the sick list and arsenical treatment discontinued. He was given four 1-gram injections of sodium thiosulphate, intraveneously. The skin condition gradually developed into a mild exfoliative dermatitis which showed steady inprovement under treatment. Recovery in 10 days.

(*12–1939*) This patient was exposed to infection in April 1938. Darkfield examinations of a lesion which developed on the shaft of the penis were negative for *Treponema pallidum.* One month after exposure a Kahn blood test was 4-plus.

From May 17, 1938 to February 7, 1939, he received 20-injections of neoarsphenamine, a total of 11.55 grams; 10 injections of mapharsen, a total of 0.68 grams, and 30 injections of bismuth as concurrent treatment.

The eighth course of arsenical treatment began with a 0.06-gram injection of mapharsen on March 30, 1939, followed by a 0.06-gram injection each week for 7 weeks.

Three days after the last injection of mapharsen the patient developed an erythematous papular rash on the sides of the neck and along the inner sides of the upper arms. The rash was treated locally with an acne lotion. On May 26, the patient consulted a civilian physician who gave him three intramuscular injections

of "Toxok."[1] On May 29, he reported with a generalized erythematous papulopustular dermatitis. A mild edema about the face and eyelids and a slight elevation of temperature were noted. A white blood count was 16,400. He was given three 1-gram injections of sodium thiosulphate intravenously. An exfoliative dermatitis developed which showed steady improvement under treatment. Recovery in 33 days.

SEVERE REACTIONS

The 9 severe reactions occurred after the following number of injections: 2 after the sixth; 1 each after the seventh and eighth; 2 after the ninth; 1 each after the tenth, fifteenth, and twentieth. The interval between the injection and appearance of symptoms varied from 5 hours to 6 days. The length of time required for recovery varied from 27 to 132 days.

NEOARSPHENAMINE

(*13–1939*) Three weeks after exposure to infection on September 17, 1939, this patient developed a primary lesion on prepuce of penis. A Kahn blood test was 4-plus.

Arsenical treatment was instituted on October 4, 1939, with a 0.2-gram injection of neoarsphenamine, followed by 7 weekly injections of 0.45-gram each. Three days after the last injection of neoarsphenamine the patient developed a dermatitis over chest, back, and arms. This condition developed into a painful and itching exfoliative dermatitis over the entire body, exfoliating in large flakes. In addition, there was a marked edema of the face, neck, and upper and lower extremities. The conjunctivae were markedly injected and all mucous membranes were quite inflamed. A generalized lymphadenopathy persisted and tachycardia and a decreased urine output were manifest. The temperature for 2 weeks ranged from 99° to 103°. On December 7 the WBC was 20,600; RBC, 5,400,000; hgb. 100; lymphs, 26; monos, 7; bands, 9; eosins, 5; segs, 53. The urine was negative for albumin.

Treatment consisted of dextrose 1,000 cc. of 10 percent solution intravenously twice daily from December 5 to December 10, and once daily thereafter until December 31; sodium thiosulphate 10 cc. intravenously once daily from December 6 to December 25; and calcium gluconate 10 cc. intravenously once daily from December 6 to December 31. Cod liver oil ointment applications were used externally. The skin condition terminated by desquamation. Symptoms gradually subsided. Recovery occurred in 34 days.

(*14–1939*) After repeated exposures to infection this patient developed a superficial ulcer in coronal sulcus. A darkfield examination on October 11, 1939, was positive for *Treponema pallidum*.

Arsenical treatment began on October 12, 1939, with a 0.3-gram injection of neoarsphenamine, followed by 0.5-gram injections on October 19, 26, November 2, 9, 16, and 24. Several hours after the last injection the patient reported to the sick bay with lower lip enlarged and weeping, and a rash on forearms consisting of small itchy papules. Since the patient felt well and the dermatitis was not severe enough to incapacitate, he was allowed to continue duty.

December 5: The rash now involves the neck and the area of posterior belt line and sacrum. The rash is more itchy and that of the neck is raised in red wheals after scratching. A 0.5-gram dose of sodium thiosulphate was administered. The right forearm was swollen and some tenderness of antecubital space

[1] Used in treating poison ivy.

was evident. There was no leakage of injection. Patient was put to bed and arm elevated. He was given 180 international units of betalin; 6 ampules of liver extract; and 3 tablespoonfuls of cod liver oil daily.

December 11: Swelling of arm has subsided. A definite exfoliation of arms and neck with some moist areas is noted.

December 12: WBC, 13,000; bands, 14; segs, 45; lymphs, 19; eosins, 18.

December 22: All areas are nearly cleared up. Patient is allowed to be up and about.

December 30: Patient returned to duty 36 days after onset of first symptoms.

(*15–1939*) A patient, exposed to infection on June 12, 1939, developed a penile lesion which was positive for *Treponema pallidum*.

Arsenical treatment began on July 17, 1939, with a 0.3-gram injection of neoarsphenamine, followed by 9 weekly injections of 0.6 gram each. Twenty-four hours after the last injection, administered on September 13, a very mild rash developed on the arms. He was given sodium bicarbonate and instructed to drink plenty of water. Ten days later the patient was hospitalized with an acute generalized dermatitis and examination revealed edema of the face to such an extent that the eyes and external auditory canals were closed. The skin was macerated and weeping, extending over scalp, arms, hands, legs, and feet. Only patchy areas of erythema appeared on the trunk. Mucous surface of the mouth was reddened and inflamed. Temperature 103°, pulse 100, respiration 20.

Treatment consisted of colloidal baths b. i. d., application of equal parts of lime water and olive oil to entire body, and 1-gram injections of sodium thiosulphate daily.

October 2: Acute weeping of extremities and head has subsided. Temperature normal. Skin involvement is gradually extending over trunk.

October 9: Edema about the head, face, and upper and lower extremities has subsided and the skin is desquamating profusely. Skin of the abdomen has become edematous, acutely inflamed, and weeping. Ears are much improved.

October 16: All areas desquamating. Generalized erythema gradually subsiding.

October 23: Extensive exfoliation has been completed. Branny desquamation continues.

Blood picture

Date	RBC	WBC	Bands	Segs	Lymphs	Juvs	Eosins	Monos
Sept. 23, 1939	4,300,000	14,400	13	60	8	1	7	11
Sept. 29, 1939	4,500,000	16,100	5	57	24	.....	11	3
Oct. 20, 1939	4,700,000	12,300	.....	52	28	.....	14	6
Nov. 2, 1939	4,680,000	9,550	3	57	28	.....	11	1
Dec. 4, 1939	4,920,000	10,300	14	54	26	.....	.....	6

The skin condition gradually improved and antiluetic treatment was resumed with heavy metal therapy. Recovery in 43 days.

(*16–1939*) This patient was given a diagnosis of syphilis on January 21, 1939. Arsenical treatment began on January 23 with a 0.3-gram injection of neoarsphenamine followed by 0.6-gram injections on January 30, February 7, 14, 21, and 28. Two days after the last injection the patient developed a rash on arms, chest, and feet which was attributed to paint remover. The rash improved but recurred in a more severe form on March 9, when a generalized erythema of the skin with numerous superimposed maculopapular lesions was noted. The skin was dry and thickened and a fine branny desquamation was present. The eyelids and prepuce were edematous and the external auditory canals were almost closed with debris. Temperature was 103°. Urinalysis showed a slight trace of albumin.

This was undoubtedly a case of arsenical dermatitis caused by neoarsphenamine poisoning.

March 13: RBC, 4,520,000; WBC, 10,900; hgb, 85 percent; segs, 43; lymphs, 27; monos, 4; eosins, 26.

March 23: Blood urea nitrogen, 9.7 mgms. per 100 cc. of whole blood. Improvement noted. Temperature is lower and skin is healthier, less inflamed and edematous. Considerable amount of dead skin continues to be shed.

The skin condition and other symptoms steadily subsided under treatment and the patient was returned to duty with the recommendation that the use of arsenicals be avoided and that antiluetic treatment continue with heavy metals to the limit of tolerance. Recovery in 106 days from the onset of symptoms.

(*17–1939*) This patient, exposed to infection February 11, 1939, developed a chancre on the penis which was positive for *Treponema pallidum*.

Arsenical treatment was instituted with a 0.3-gram injection of neoarsphenamine on March 21, 1939, followed by 0.6-gram injections on March 27, April 4, 11, 18, and 25, a total of 3.30 grams. Three injections of bismuth salicylate, a total of 0.39 gram, were administered as concurrent treatment.

Two days after the last injection of neoarsphenamine a dry, itching rash appeared on the forearms and hands, later extending to shoulders, face, and legs. The skin over the entire body became dry and thickened, and had a rough feeling, the result of small papules which had a tendency to coalesce. The conjunctiva and pharynx were injected and the external auditory canals were edematous, but not closed. There was a pitting edema of the hands and prepuce. The temperature ranged from 101° to 103°; WBC, 7,150; segs, 42; bands, 4; eosins, 10; lymphs, 44.

The skin condition developed into a severe exfoliative dermatitis which steadily improved under treatment. Recovery in 64 days.

(*18–1939*) Two months after exposure to infection on September 15, 1938, this patient developed a chancroid and a macular rash. A Kahn blood test was 4-plus.

From November 26, 1938 to January 11, 1939, he was given 8 injections of neoarsphenamine, a total of 4.35 grams, and 10 injections of bismuth, a total of 1.43 grams.

The second course of arsenical treatment began with a 0.3-gram injection of neoarsphenamine on January 21, 1939. Five hours after the injection the patient experienced an itching sensation about the palms of the hands. The following day small blisters appeared about the hands and spread gradually over the entire body. The skin became edematous. A low-grade erythema was present and superimposed on this were scattered small dry and scaly papular lesions. The palms of the hands and soles of the feet were covered with thickly set firm vesicles. There was a marked edema of the prepuce. The temperature ranged from 100° to 103°. On January 28 the white blood count was 18,750; segs, 44; bands, 6; juvs, 2; lymphs, 38; monos, 4. The urinalysis showed a slight trace of albumin. The skin condition developed into a severe exfoliative dermatitis with areas most involved showing serous weeping with much crust formation.

The skin conditions showed steady improvement under treatment. Recovery in 132 days.

Mapharsen

(*19–1939*) Following exposure to infection on January 13, 1939, this patient developed a penile lesion which was positive for *Treponema pallidum*.

Arsenical treatment began with a 0.3-gram injection of neoarsphenamine on January 14, 1939, and a 0.45-gram injection on January 18, followed by 6 weekly injections of 0.6 gram each.

Twenty-four hours after the last injection of neoarsphenamine the patient developed a slight cutaneous reaction. Due to this slight reaction he was given a 0.03-gram injection of mapharsen on March 8. The following day the patient reported to the sick bay with a maculopapular rash evident on the trunk and extremities. The rash gradually involved the entire body and the face. The lesions became confluent, and oozing was noted from lesions over antecubital spaces. The temperature ranged from 99.6° to 102°, and WBC was 16,870.

He was given five daily injections of sodium thiosulphate. The lesions became more extensive and severe during the first 7 days and subsided gradualy under symptomatic treatment. Recovery in 28 days.

(*20–1939*) One month after exposure to infection on December 23, 1938, this patient developed a primary lesion which was positive for *Treponema pallidum*. A Kahn blood test was 4-plus.

From January 17 to March 14, 1939, he received 13 injections of mapharsen, a total of 0.60 gram, and 18 injections of a bismuth compound as concurrent treatment.

The second course of arsenical treatment began on May 10, 1939, with a 0.3-gram injection of neoarsphenamine, followed by 0.6-gram injections on May 19, 24, June 1, and 7. Following the last injection of neoarsphenamine the patient developed itching of the skin and a generalized erythema which lasted for several days. Neoarsphenamine was discontinued.

A 0.06-gram injection of mapharsen was administered on June 14, and a 0.03-gram injection on June 21. On the day of the last injection an intertriginous lesion, resembling epidermophytosis, developed in the groin. It was treated as such and further arsenical treatment was withheld. On June 30, an exfoliative dermatitis had developed involving the entire body. WBC, 14,500; RBC, 5,980,000; hgb, 80; bands, 5; segs, 60; lymphs, 21; monos, 6; eosins, 8.

The patient was given 1 gram of sodium thiosulphate intravenously, colloidal baths, and the skin was greased with olive oil and lime water. The skin condition gradually improved under treatment. Recovery in 31 days.

(*21–1939*) After exposure to infection in June 1928, this patient developed a penile lesion. According to the patient's statement, he consulted a civilian physician at the time and was given some powder to apply to the lesion. No darkfield, Kahn, or Wassermann was done by the civilian physician.

While on the sick list blood tests revealed the patient had syphilis and diagnosis was made on January 31, 1935. Physical examination revealed a small smooth scar on dorsum of penis.

Antiluetic treatment was instituted and from February 11 to April 1, 1935, he received 8 injections of neoarsphenamine, a total of 3.4 grams; and from April 4 to July 30, 6 injections of neoarsphenamine, a total of 2.5 grams. On July 29, 1935, the patient had a reaction following an injection of neoarsphenamine and arsenical treatment was discontinued.

From April 15, 1937 to September 25, 1939, he received 40 injections of bismuth salicylate, a total of 7.32 grams.

The third course of arsenicals began October 18, 1939, with a 0.015-gram injection of mapharsen. Five days later the patient reported with a generalized erythema of skin of face, neck, body, and extremities, with desquamation beginning about the abdomen and chest. Chills and fever, swelling of the feet and ankles, and a generalized weakness were noted by the patient approximately 12 hours after the injection of mapharsen, and the following day the skin over the entire body, extremities, and head was reddened and erythematous.

He was given 1-gram injections of sodium thiosulphate daily, sodium bicarbonate solution baths, forced fluids, active catharsis, and calamine lotion for pruritis. The skin condition gradually improved. Recovery in 27 days.

Fatal Reaction

(*22–1939*) The source of infection in this case is unknown. The patient (supernumary, retired, U. S. Navy) was hospitalized on March 21, 1939, with a diagnosis of arsenical poisoning. According to the patient's statement he began antisyphilitic treatment under the care of a private physician in December 1938, and had been receiving weekly treatment of neoarsphenamine until 6 weeks prior to admission, at which time he noted a vesicular eruption on the extremities. Three weeks prior to admission the vesicular eruption involved the entire body with the exception of the face, palms, and soles, and there was some weeping, crusting, and exfoliation. On admission the patient was found to have a generalized exfoliative dermatitis, with many raw, weeping areas. Antisyphilitic therapy was intensified and given twice weekly for the next 3 weeks. Under symptomatic and supportive measures, which consisted of soothing ointments locally and sodium thiosulphate and calcium gluconate intravenously, his condition improved for about 1 week. At this time a diarrhea developed which persisted despite all treatment. Four days prior to death there was much exfoliation of the skin and new areas of weeping. The patient was semicomatose on April 3, 1939, and died at 1025 on April 4, 14 days after admission to hospital.

Autopsy findings.—Generalized exfoliation, enlarged liver, distended gall bladder, diffuse pleural adhesions on the right, irritation and congestion of the mucosa of the large bowel, and injection of the cerebral vessels.

SUMMARY

In 1939, medical officers of the Navy administered 129,295 doses of arsenicals and reported the occurrence of 50 reactions therefrom. Of these reactions 22 were arsenical dermatitis; a ratio of one case of dermatitis to 5,877 doses. Of interest in connection with the etiology of arsenical dermatitis is the number of instances in which premonitory signs were noted. These signs are repeated below and serve to indicate the necessity for careful examination and questioning of each patient before administering an arsenical.

Case 1.—A macular rash followed the second injection of neoarsphenamine. The third injection, given 4 days later, caused a generalized erythema, nausea, and vomiting.

Case 4.—A mild exfoliative dermatitis developed after the ninth injection of neoarsphenamine. An erythematous rash followed an injection of mapharsen given 5 months later.

Case 8.—A punctate rash followed the second injection of neoarsphenamine. The next injection given 14 days later caused recurrence of the rash.

Case 10.—A severe exfoliative dermatitis developed after the eighth injection of neoarsphenamine. Ten months later a macular rash followed the first injection of mapharsen.

Case 15.—A mild rash followed the tenth injection of neoarsphenamine. Nine days later a severe exfoliative dermatitis developed.

Case 19.—A slight cutaneous reaction followed the eighth injection of neoarsphenamine. A severe maculopapular rash followed an injection of mapharsen given 1 week later.

Case 20.—A generalized erythema developed after the fifth injection of neoarsphenamine. A severe exfoliative dermatitis followed the second injection of mapharsen given 14 days later.

Case 21.—Unreported erythematous rash, chills, and fever followed the first injection of mapharsen. Five days later an exfoliative dermatitis developed.

STATISTICS

HEALTH OF THE NAVY

Report for the first quarter, 1940

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of January, February, and March, 1940:

ENTIRE NAVY

Year	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1935	371	67	438	28	85	62
1936	337	49	386	30	140	42
1937	276	36	313	18	98	59
1938	387	46	434	9	128	86
1939	423	47	470	19	168	80
1940	499	49	548	21	206	90

FORCES ASHORE

1935	491	80	571	54	110	45
1936	518	50	568	59	226	26
1937	312	36	347	34	131	21
1938	447	50	497	14	195	40
1939	487	47	534	10	253	41
1940	583	43	626	26	300	49

FORCES AFLOAT

1935	312	60	372	16	72	71
1936	229	49	278	13	89	51
1937	256	37	293	8	78	78
1938	353	44	397	6	90	113
1939	390	47	437	24	125	100
1940	439	54	493	17	139	119

Common infectious diseases of the respiratory type.—A total of 9,215 admissions for these diseases was reported for the entire Navy for the first quarter of 1940, or a 55 percent increase when compared with the corresponding quarter in 1939. Catarrhal fever was responsible for 7,262 of the total admissions for respiratory diseases.

There were 5,463 admissions for these diseases reported by shore stations in the United States, 3,644 from forces afloat, and 108 from outlying naval stations. The largest number of cases were reported from the following ships and stations:

Ship or station	January	February	March	Total
Naval Training Station, Norfolk, Va	517	490	397	1, 404
Naval Training Station, Great Lakes, Ill	206	285	239	730
Naval Training Station, Newport, R. I	173	154	156	483
U. S. Naval Academy (midshipmen)	150	112	87	349
Marine Corps Base, San Diego, Calif	97	96	53	246
Marine Barracks, Quantico, Va	82	57	77	216
Naval Air Station, Pensacola, Fla	50	82	51	183
Fleet Air Detachment, Naval Air Station, San Diego, Calif	39	58	58	155
U. S. S. *Ranger*	84	31	20	135
Naval Dispensary, Washington, D. C	49	38	22	109
U. S. S. *Mississippi*	53	21	34	109
U. S. Naval Academy (other than midshipmen)	42	48	11	101
U. S. S. *New York*	51	30	16	97
Naval Air Station, Norfolk, Va	36	40	19	95
U. S. S. *Wichita*	56	30	4	90
U. S. S. *Enterprise*	8	37	44	89
N. T. S. San Diego, Calif	33	25	31	89
U. S. S. *Arizona*	36	27	25	88
Marine Barracks, Parris Island, S. C	55	26	5	86
U. S. S. *West Virginia*	34	34	16	84
U. S. S. *Pennsylvania*	46	22	13	81
U. S. S. *St. Louis*	41	29	10	80
U. S. S. *Omaha*	30	33	10	73
U. S. S. *Saratoga*	9	40	24	73
U. S. S. *Savannah*	21	27	25	73
Receiving Ship, New York, N. Y	22	36	15	73
U. S. S. *California*	37	14	19	70
U. S. S. *Maryland*	37	11	22	70
Norfolk Navy Yard, Portsmouth, Va	24	33	11	68
U. S. S. *Idaho*	46	11	9	66
Naval Hospital, Philadelphia, Pa	21	18	26	65
Navy Yard, Washington, D. C	23	28	13	64
Third Defense Battalion, F. M. F., Pearl Harbor, T. H	32	20	10	62
U. S. S. *New Mexico*	30	13	16	59
Navy Yard, Philadelphia, Pa	29	13	13	55
Norfolk Naval Hospital, Portsmouth, Va	17	20	16	53
Receiving Ship, San Diego, Calif	13	13	27	53
U. S. S. *Indianapolis*	28	22	2	52
U. S. S. *Nitro*	17	28	5	50

Other infectious diseases.—Chickenpox was reported during the quarter as follows: In January, 2 from the Naval Training Station, Norfolk, Va., and Puget Sound Naval Hospital, Bremerton, Wash., and 1 each from the U. S. S. *California*, U. S. S. *Canopus*, U. S. S. *Guam*, Naval Dispensary, Washington, D. C., and Fleet Air Base, Pearl Harbor, T. H.; in February, 1 each from the U. S. S. *Gilmer*, Naval Air Station, Norfolk, Va., and the Puget Sound Naval Hospital, Bremerton, Wash.; and in March, 2 from the U. S. S. *Yorktown* and 1 each from the U. S. S. *Gilmer* and Naval Air Station, Pensacola, Fla.

Two cases of diphtheria were admitted during the quarter, one in February from the Marine Detachment, Tientsin, China, and one in March from the Submarine Base, New London, Conn.

One case of scarlet fever was reported by the Naval Training Station, Norfolk, Va., in January.

The U. S. S. *Parrott* reported one case of smallpox in February. A ship's cook, third class, 30 years of age, with 8 years, 5 months' service, was admitted to the sick list on February 15, 1940. Probable place of infection was Amoy, China. The vaccination history of the patient is as follows: December 2, 1927, failure; December 9, 1927, failure; December 16, 1927, immune; November 27, 1931, immune; April 9, 1933, immune; and January 17, 1940, accelerated. Due to the suspicious character of the signs and symptoms and the length of the incubation period, the patient was removed to the Scott-Thresher Memorial Hospital and placed in quarantine for observation. The patient was reexamined on February 16 by the division medical officer and the diagnosis of smallpox was established. The patient was returned to duty on March 9.

A total of 155 cases of measles, 212 cases of German measles, and 85 cases of mumps were reported by forces afloat and ashore for the quarter, the majority of the cases being distributed among ships and stations, as follows:

Ship or station	Measles	German measles	Mumps
Fleet Air Detachment, San Diego, Calif	13	0	2
U. S. S. *Honolulu*	0	12	0
U. S. S. *Idaho*	0	23	0
U. S. S. *Maryland*	0	6	1
U. S. S. *Milwaukee*	21	0	7
U. S. S. *Nitro*	14	0	0
U. S. S. *Omaha*	0	20	0
U. S. S. *Ranger*	0	15	0
U. S. S. *Yorktown*	0	8	0
Naval Air Station, Norfolk, Va	0	13	1
Naval Training Station, Norfolk, Va	46	58	5
Marine Corps Base, San Diego, Calif	18	3	7
Naval Training Station, San Diego, Calif	1	1	9
Receiving Ship, San Diego, Calif	13	11	0
Marine Detachment, Peiping, China	0	0	7
4th Marines, Shaghai, China	0	0	7

Four cases of septic sore throat were reported for the quarter—two from the U. S. S. *Tennessee* and one from the naval recruiting station, Minneapolis, Minn., in January, and one from the U. S. S. *Augusta* in March.

A chief quartermaster clerk, United States Marine Corps, on duty with the Fourth Marines, Shanghai, China, was admitted to the sick list on January 19 with a mild typhus fever. The probable place of infection was Shanghai, China. There were no complications and the patient was returned to duty on March 1.

A water tender, second class, 22 years of age, with 4 years and 8 months' service, was transferred to hospital from the U. S. S. *Gannet* on February 12, 1940, with cerebrospinal meningitis. Quarantine of the vessel for a period of 10 days was instituted and special measures

of inspection and observation for all contacts were continued for the full incubation period of the disease. No further cases occurred. The patient was discharged to duty on April 16.

An apprentice seaman, 22 years of age, with 1 month's service, was admitted to the sick list at the Naval Training Station, San Diego, Calif., with cerebrospinal fever on February 9. He remained on the sick list at the Naval Hospital, San Diego, Calif., until May 7. Other than liberty in Pasadena, Calif. on February 3 and 4 this patient had been in isolation on the Station from January 9.

A sergeant, United States Marine Corps, 34 years of age, with 13 years and 6 months' service, on duty at the Marine Barracks, Quantico, Va., was admitted to the sick list on February 4 with cerebrospinal fever. He was returned to duty on March 18.

A private, United States Marine Corps, 21 years of age, with 2 months' service, was admitted to the sick list at the Marine Barracks, Quantico, Va., on February 23 with meningitis, cerebrospinal, acute, complicated by serum sickness on March 5. The patient was discharged to duty on March 23.

An uncomplicated moderately severe case of typhoid fever was admitted to the sick list at the Regimental Hospital, Fourth Marines, Shanghai, China. The patient, a private, United States Marine Corps, 21 years of age, with 2 years and 2 months' service, was admitted on March 27 and discharged to duty on April 20. Typhoid prophylaxis had been completed in 1937.

Venereal disease.—The following table of statistical data indicates the frequency of occurrence of venereal diseases during the first quarter of 1940 as compared with the previous quarter (October, November, and December 1939):

Ship	Admission rate per 1,000	
	Fourth quarter, 1939	First quarter, 1940
Battle and Scouting Forces		
Arizona	48. 99	94. 67
Arkansas	127. 78	99. 08
Astoria	30. 40	53. 81
Boise	120. 89	69. 99
Brooklyn	88. 19	95. 24
California	73. 21	66. 38
Chester	55. 21	36. 47
Chicago	63. 24	35. 49
Cincinnati	238. 66	104. 00
Colorado	119. 76	93. 62
Concord	182. 61	127. 49
Detroit	49. 73	62. 39
Enterprise	28. 33	49. 42
Helena	88. 08	170. 85
Honolulu	124. 38	92. 38
Houston	79. 77	44. 02
Idaho	89. 53	106. 80
Indianapolis	81. 76	62. 58

Ship	Admission rate per 1,000	
	Fourth quarter, 1939	First quarter, 1940
Battle and Scouting Forces—Continued		
Langley	612. 24	431. 60
Lexington	114. 37	70. 64
Louisville	147. 67	66. 47
Maryland	56. 11	52. 68
Memphis	62. 62	72. 33
Milwaukee	98. 00	149. 90
Minneapolis	49. 59	45. 13
Mississippi	70. 37	121. 84
Nashville	110. 53	96. 00
Nevada	55. 92	62. 21
New Mexico	136. 32	120. 11
New Orleans	30. 40	48. 93
New York	151. 97	118. 99
Northampton	51. 87	41. 54
Oklahoma	66. 60	55. 00
Omaha	201. 61	160. 31
Pennsylvania	61. 30	104. 71
Pensacola	94. 81	48. 71
Philadelphia	159. 32	68. 29
Phoenix	84. 51	38. 51
Portland	75. 35	64. 52
Quincy	123. 58	103. 60
Raleigh	71. 11	23. 53
Ranger	144. 39	183. 05
Richmond	82. 86	72. 20
Salt Lake City	71. 75	89. 82
San Francisco	220. 73	215. 12
Saratoga	114. 96	62. 72
Savannah	104. 35	121. 56
St. Louis	134. 89	131. 15
Tennessee	95. 97	93. 62
Texas	153. 85	108. 21
Trenton	537. 01	144. 58
Tuscaloosa	173. 65	133. 15
Utah	136. 25	79. 60
Vincennes	160. 26	197. 53
West Virginia	81. 44	53. 93
Wichita	185. 24	140. 54
Wright	53. 48	32. 97
Wyoming	158. 04	114. 81
Yorktown	126. 65	84. 10
Asiatic Fleet		
Alden	480. 00	307. 69
Asheville	828. 40	503. 35
Augusta	510. 55	597. 60
Barker	566. 93	576. 00
Bittern	131. 15	76. 43
Black Hawk	460. 80	497. 54
Bulmer	300. 00	220. 47
Canopus	318. 77	353. 44
Edsall	338. 46	512. 00
J. D. Edwards	483. 87	721. 31
Finch	312. 50	436. 36
J. D. Ford	645. 16	448. 00
Guam	771. 93	542. 37
Heron	193. 55	666. 67
Isabel	311. 69	169. 01
Paul Jones	346. 46	346. 46
Luzon	344. 09	222. 22

Ship	Admission rate per 1,000	
	Fourth quarter, 1939	First quarter, 1940
Asiatic Fleet—Continued		
Marblehead	543. 12	590. 75
Mindanao	523. 81	689. 66
Napa	230. 77	150. 94
Oahu	61. 54	358. 21
Parrott	527. 13	279. 07
Peary	349. 21	629. 92
Pecos	620. 32	886. 49
Pigeon	400. 00	676. 92
Pillsbury	393. 44	406. 78
Pope	566. 93	380. 95
Stewart	625. 00	297. 52
Tulsa	1, 000. 00	825. 81
Tutuila	758. 62	654. 55
Whipple	229. 51	603. 17

Food poisoning, U. S. S. Colorado.—An epidemic of food poisoning occurred among the enlisted men on board this vessel following breakfast on January 21, 1940. Veal hash was believed to have been the transmitting agent. No samples remained for examination. The hash was prepared, placed in baking pans, and allowed to remain on the galley table for several hours previous to cooking and serving for breakfast on January 21. There were 102 cases, 90 mild and 12 severe. The cases were scattered throughout the crew but not all divisions were affected. Every man who became ill stated that he had eaten hash. The symptoms were severe retching and vomiting of liquid mixed with food, mild diarrhea, subnormal temperature, chills, pallor, rapid pulse, weakness, and in the more severe cases marked muscle cramps in the extremities. Cramps followed severe vomiting in every case. Earliest cases were 3½ hours after eating and the last case was 12 hours. The majority of cases reported within 5 hours after eating. Duration of severe cases was 20 hours; of mild cases, 6 hours. At the end of 20 hours all cases had recovered sufficiently to assume duty.

Gastro-enteritis, acute, U. S. S. Pensacola.—On February 12, 1940, while in the Navy Yard, Pearl Harbor, the personnel of this vessel experienced an outbreak of gastro-enteritis which affected 14 percent of the crew, 14 percent of the chief petty officers, 37 percent of the warrant officers, 17 percent of the wardroom officers. The outbreak was of sudden onset and lasted 3 days. Approximately the same number of men reported to the sick bay on each of the 3 days.

All possible sources of infection were thoroughly investigated. The galley, scullery, and refrigerating rooms were inspected, the food handlers were examined, and in no instance was any laxity or negligence

apparent. A careful survey of the dietary history in each case failed to isolate any one food as the primary cause of the outbreak. No one item served on the various messes was eaten by all men affected. Samples of various kinds of sandwiches sold at the ship's soda fountain were sent to the laboratory of the naval hospital, Pearl Harbor, for examination. Samples of suspected foods, together with blood and stool specimens from some of the more severe cases, were submitted to the laboratory for examination. The report was essentially negative. A staphylococcus infection was found in some of the suspected sandwiches. However, it seems highly improbable that staphylococcus was the predisposing cause of the outbreak because only a small number of men gave history of having eaten the infected sandwiches.

Water from the yard water barge had been used. On February 12 the ship's evaporators distilled sufficient water into the ship's tanks to bring them up to proper level. On February 13 the ship again received water from the yard water barge, and on the 14th from a dock by means of a hose. This water was used for drinking, bathing, scrubbing down, etc. Successive samples were taken from the crew's drinking fountain and sent to the laboratory for culture. The report showed the specimens to be free of contamination by the Coli aerogenes group and to have a bacterial count of 600 per cc.

Laboratory, clinical, and statistical investigations failed to disclose the etiological factor of the epidemic.

Similar cases were reported among the civilian population and among families of ship's personnel who were not aboard during this time. Thus, the cause of infection may be considered to have come from some unknown outside source, rather than from aboard this vessel.

MORBIDITY

Summary for the quarter ending Mar. 31, 1940

Average strength	Forces afloat 101,074		Forces ashore 72,250		Entire Navy 173,324	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	12, 455	492. 91	11, 307	625. 99	23, 762	548. 38
Diseases only	11, 102	439. 36	10, 527	582. 81	21, 629	499. 16
Injuries and poisonings	1, 353	53. 54	780	43. 18	2, 133	49. 23
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	444	17. 57	461	25. 52	905	20. 89
(B)	3, 521	139. 34	5, 411	299. 57	8, 932	206. 13
Venereal diseases	3, 008	119. 04	894	49. 49	3, 902	90. 05

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for the quarter ending Mar. 31, 1940

The following statistics were taken from sanitary reports submitted by naval training stations.

January, February, and March 1940	Naval training station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego Calif.
Recruits received during the period	3,072	1,980	2,511	2,102
Recruits appearing before Board of Medical Survey	48	4	22	(*)
Recruits recommended for discharge from the Service	27	5	21	(*)
Recruits discharged by reason of medical survey	30	0	26	(*)
Recruits held over pending further observation	16	8	(*)	(*)
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	0	30	(*)	38

*Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the survey were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess, periapical	1	Compression, brachial plexus	1
Absence, acquired, kidney	1	Conjunctivitis, follicular	1
Absence, acquired, teeth	11	Constitutional psychopathic inferiority without psychosis	4
Absence, acquired, parietal bone (surgery)	1	Constitutional psychopathic state, emotional instability	3
Adhesions, abdominal	1	Constitutional psychopathic state, inadequate personality	11
Arthritis, chronic	2	Curvature, spine	1
Asthma	7	Deafness, bilateral	4
Astigmatism, compound, myopic	2	Deafness, unilateral	1
Astigmatism, compound, hyperopic	1	Deformity, acquired	11
Astigmatism, mixed	1	Deformity, congenital	9
Atrophy, right arm	1	Defective physical development	1
Atrophy, left hand	1	Dementia praecox	2
Blepharitis	1	Diabetes insipidus	1
Blindness, unilateral	1	Effort syndrome	4
Bronchiectasis	2	Enterocolitis, chronic	1
Cardiac arrhythmia, paroxysmal tachycardia	1	Enuresis	26
Caries, teeth	15	Epilepsy	24
Cicatrix, skin	2	Extensive and numerous unsatisfactory restorations by fillings	1
Color blindness	2	Fistula, abdominal	1
Colitis, chronic	1		

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Flat foot	15	Psychoneurosis, anxiety neurosis	1
Foreign body, traumatic, rt. chest (pistol bullet)	1	Psychoneurosis, hysteria	4
Fracture, simple	2	Psychoneurosis, neurasthenia	4
Gastritis, chronic	1	Psychoneurosis, situational	1
Genu valgum	1	Psychoneurosis, traumatic	2
Hallux valgus	1	Psychosis, intoxication, alcoholic	1
Headache	1	Rhinitis, atrophic	1
Hernia, inguinal, indirect	8	Sclerosis, disseminated	1
Hernia, femoral	1	Sinusitis, ethmoidal	1
Hernia, ventral	2	Somnambulism	3
Hypertension, arterial	1	Splanchnoptosis	1
Hydrocele, tunica vaginalis	1	Sprain	4
Hyperthyroidism	1	Stenosis, pylorus	1
Malocclusion, teeth	7	Strabismus	1
Mental deficiency, moron	1	Strain, muscle, lumbar	1
Metatarsalgia	1	Stuttering	1
Migraine	3	Synovitis, chronic	1
Myopia	8	Syphilis	1
Myositis, chronic	4	Tuberculosis, pulmonary, chronic, active, moderately advanced	1
Narcolepsy	1	Tuberculosis, pulmonary, chronic, arrested, moderately advanced	1
Nephritis, acute	1	Ulcer, duodenum	5
Nephritis, chronic	10	Valvular heart disease, aortic and mitral	1
Neurosis, intestinal	1	Valvular heart disease, mitral insufficiency	4
Osgood-Schlatter disease	2	Valvular heart disease, mitral stenosis	2
Osteochondritis dissecans	1	Varicocele	1
Otitis media, chronic	13		
Pansinusitis	1		
Paradentosis	3		
Paralysis, ocular muscle	1		
Paralysis, pudendal	1		
Perforation, ear drum, bilateral	1		
Pes cavus	2		
Prostatitis, chronic (NV)	1		

DEATHS

During the quarter ending March 31, 1940

Cause of death		Navy			Marine Corps		Nurse Corps	Total
Principal	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		10,804	2,236	132,918	1,514	25,413	439	173,324
Diseases								
Abscess, penis	Lymphangitis, abdomen.			1				1
Abscess, peritonsillar	Cellulitis, neck			1				1
Adenocarcinoma, rectum	Abscess, pelvic			1				1
Alcoholism, acute	None			1				1
Anemia, aplastic	do					1		1
Angina, Ludwig's	Pneumonia, broncho			1				1
Do	Thrombosis, coronary artery.	1						1
Angina pectoris	None	1						1
Appendicitis, acute	Peritonitis, general, acute.			2				2
Appendicitis, chronic	Peritonitis, general, acute.			1				1
Carcinoma, colloid, neck	None			1				1
Carcinoma, epiglottis	do			1				1
Carcinoma, stomach	do	1						1

Cause of death		Navy			Marine Corps		Nurse Corps	Total
Principal	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Diseases—Contd.								
Hemorrhage, cerebral	Hypertension, arterial	1						1
Hemorrhage, subarachnoid	None			1				1
Malaria, benign, tertian	do	1						1
Myocarditis, chronic	do			1				1
Obstruction, intestinal (Meckel's diverticulum).	Peritonitis, general, acute.	1						1
Pneumonia, broncho	Embolism, pulmonary			1				1
Pneumonia, lobar	None			2				2
do	Nephritis, acute			1				1
Rheumatic fever	Pneumonia, lobar			1				1
Sarcoma, fibroblastic, omentum.	None			1				1
Thrombosis, coronary artery.	do			1				1
Tuberculosis, general miliary.	do			1				1
Tuberculosis, pulmonary, chronic.	do	1		3				4
Ulcer, duodenum, perforated.	Peritonitis, general, acute.			1				1
Valvular heart disease, aortic and mitral.	Endocarditis, acute			1				1
Valvular heart disease, aortic insufficiency.	None			1				1
Total for diseases		7		26		1		34
Injuries and poisonings								
Drowning	None			4		1		5
Fracture, simple, pelvis	Rupture, traumatic, bladder.			1				1
Fracture, simple, vertebra, cervical.	Compression, cervical cord.			1				1
Injuries, multiple, extreme	None	2		9		1		12
Intracranial injury	do			3		2		5
Rupture, traumatic, kidney.	Hemorrhage, kidney					1		1
Strangulation, respiratory	None			1				1
Wound, gunshot, abdomen	do			1				1
do	Cerebral embolism	1						1
Wound, gunshot, chest	None	1		1		1		3
Wound, gunshot, head	do	2				1		3
Poisoning, acute, lysol	Psychosis, unclassified	1						1
Total for injuries and poisonings.		7		21		7		35
Grand total		14		47		8		69
Annual death rate per 1,000:								
All causes		5.18		1.41		1.26		1.59
Diseases only		2.59		.78		.16		.78
Drowning				.12		.16		.12
Poisonings		.37						.02
Other injuries		2.22		.51		.94		.67

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