The
WASHINGTON UNIVERSITY
MEDICAL ALUMNI
QUARTERLY

PUBLISHED IN THE INTEREST OF
THE UNIVERSITY AND THE ALUMNI

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The Medical Social Service Department
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Conservation in the Hospital Laboratory
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Case Reports of the Barnes Hospital
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Vol. VII  OCTOBER, 1943  No. 1
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Office of the Washington University Medical Alumni Quarterly, 602 South Euclid Avenue, St. Louis 10, Missouri

Published quarterly by Washington University School of Medicine, St. Louis, Mo. Entered as second class matter December 14, 1937, at the Post Office at St. Louis, Mo., under the act of August 24, 1912.

* In the Armed Forces.
McMillan Hospital
1943
What the Clinical Staff May Expect of the Medical Social Service Department

LLEWELLYN SALE
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The treatment of the sick individual has undergone many changes, has been a gradual development through the ages. Some of these changes have improvement in underlying philosophy, in attitudes and, of course, in diagnosis and the application of therapeutic measures. These have been retained and have been incorporated in the every day practice of medicine. Some of the changes have been tried and found wanting. These were discarded and are now of interest mainly to the medical historian.

The physicians' attempts to be of service to the sick and inadequate individual have gone far beyond the stage of administering obnoxious draughts brewed from herbs and the wielding of the scalpel. The patient is envisaged, as a whole, as a unit. He is considered, however, not only as an isolated unit, but as a member of many groups, of society in the last analysis. Our present day society with its complexities, its speed, its competitive character, creates for the individual many problems. His ability to cope with these problems, to make his adjustment in the face of many difficulties depends on many factors, but finally on his adequacy or inadequacy. There are periods in the life of the larger group—the community, the state, the nation, even the world—when the adequacy of the individual and of large groups of individuals is severely tested. In such an era we now find ourselves. A world at war now constitutes a milieu in which inadequacies thrive. The number of individuals whose defense breaks down increases, the number of those who are able to assist diminishes.

We are living, then, in a world in which it becomes increasingly more important not to treat symptoms, but to treat the individual as a whole "to work for his basic adjustment." It is not surprising that early medical

social service should have laid special emphasis on the psychiatric patient. This obviously maladjusted individual needed the support that he could get from the psychiatric worker. Since the beginning of medical hospital service, in 1904, there has been a large growth in the medical social work field. It is now recognized that not only those patients whose difficulties are purely functional, but that those with organic changes need the support that can come from those qualified to help him understand and to try to combat the problems that come with personality difficulties, economic stresses, and the burdens of our largely urban, high speed, mercilessly competitive life.

Medical social service at first met with considerable resistance on the part of the physician. This is particularly true of social service to a patient in the hospital. Physicians have, however, within a comparatively short time recognized the value of this service to the patient and to the physician himself in his attempts to treat and, wherever possible, to rehabilitate the invalid. Social service has taken its place with roentgenology, the laboratory and other diagnostic and therapeutic measures as an important adjunct in the treatment of the patient.

The medical staff has a right to expect the medical social service workers to show an interest in the purely medical—scientific if you will—aspects of the patient, their client. This the average social service worker is not only willing, but eager to do. In so far as time permits they should attend history meetings, clinical and pathological conferences. This broadens the base of their understanding of the problems involved and strengthens the sense of cooperation and coresponsibility. It has seemed to me that joint medical and social service ward rounds should be made at regular intervals and this is done at some institutions. The fallacy of the oft misquoted statement that “a little learning is a dangerous thing” is in this instance again exposed. The medical social worker should attend such meetings not only for her edification, but should join in the discussions and make comments and suggestions that come out of her studies. These will often be of practical aid in treatment and may at times help to clarify a diagnostic problem.

It goes without saying that the medical staff expects the social service department to make a complete social study of the patient with all that that implies. The pertinent data must then be assembled and from them a social diagnosis, correlated with the medical diagnosis, is the basis for a plan for medical social treatment.

The medical staff may expect the social admitting officer to make a careful analysis of the patient’s financial resources to determine his eligibility for free care. This protects the hospital against being imposed upon and
limits hospital abuse. This should not be the sole, nor even the most important aim of the admitting officer. I am convinced that the vast majority of people prefer to pay for medical care. Some of my friends tell me that I am much too gullible. The Anglo-Saxon principle that it is better to allow a hundred guilty men go free than to have one innocent man punished—if applied to hospital admissions expresses my feeling in this matter. I would rather see several patients admitted to the ward who could pay for their care, than to see one deserving patient denied access. This might sound dangerous from the administrator's point of view, but my social service friends tell me that hospital abuse is almost negligible.

Social service workers, because of their training, are aware of the desirability of having prospective patients maintain their self-respect and independence. This motive justifies a careful financial investigation; the protection of the hospital against abuse should, it seems to me, be secondary. Such a study involves a knowledge of the patient's income as related to his obligations and responsibilities. Hospital care should not involve expenditures which will handicap other members of the family and deprive them of the necessities for decent living. This is unfair to the members of the family, it is a source of worry to the already handicapped and harassed sick individual, and is surely neither good social nor medical practice. The nature of the illness, its probable duration, the cost for special examination—such as x-ray, electrocardiogram, basal metabolism rate determination, bio-chemical studies and other laboratory procedures—must be taken into account in determining eligibility for free or part time care.

The prognosis, especially as to the patient's ability to continue at his work, is an important consideration in attempting to arrive at a conclusion on a wise and fair social evaluation.

If and when the patient is sufficiently well to leave the hospital the medical staff may expect the medical social worker to supervise and help him plan his further course. If he goes to another institution she will probably have no further obligation. If he goes home and needs further care she has an important function to perform. She is already familiar with the content of his social history, the medical diagnosis, the prognosis and post-hospital therapeutic needs. If the patient is permanently handicapped, he and his dependents may need relief from a private or public agency. It may be important to seek new work for him that comports with his physical disability. His treatment may be dietetic and the social worker must attempt to see to it that this is provided within the limits of the family budget. If further observation and clinic treatment is important and the patient does not report, she must learn why he has not done so and try to have him follow instructions. She must, in a word, carry on in the
interest of his medical and social welfare. This, it seems to me, is not the least of her responsibilities.

I have attempted to call attention to some of the things that the clinical medical staff may expect from the medical social service department in their cooperative endeavor to rehabilitate the patient. What the medical social service department may expect from the clinical staff is another important subject for a discussion. Each may expect from the other mutual respect and cooperation. Each may expect an intelligent interest in the objectives of the other and some familiarity with the techniques employed by the other. Each must make the patient's welfare the sole beneficiary of his thinking and doing. Each will thus make a contribution to the sick individual and to the community of which he is a part.
Outline of Program for Conserving Resources of the Hospital Laboratory

F. R. Bradley
Superintendent, Barnes Hospital

Hospital administrators always have realized their responsibility for economy, and long ago they put conservation programs into effect. Depression years accelerated the introduction and increased the scope of these programs. But the problem of dealing with today’s priorities, rationing and personnel shortage makes what has been done in the past seem quite minimal indeed. Any effective conservation program requires that the hospital department head and his workers have an appreciation of the need to conserve, plus the willingness and ability to act. We are creatures of desire. What we want to do or like to do, we accomplish.

The degree of accomplishment in a hospital conservation program is governed by three factors: Adequate training of employees, adequate supervision, and adequate tools and ideas. If the employee is genuinely interested in conservation, he will create many of the ideas that a department head can put into effect.

Although this article is concerned especially with conservation in the hospital laboratory, it is perhaps well to consider a few of the general principles of employee cooperation. One approach is based on the observation that most people think in generalities but live in detail.

These generalities are symbolized by such slogans as: Reduce breakage . . . Reduce usage . . . Reduce waste . . . Substitute.

The problem is to reduce such material to practical suggestions. An idea contest sometimes brings surprising results.

From such a contest came several practical suggestions from the laboratory which are presented here.

Clinical Microscopy, Blood Bank

Use plastic or no coverslips in looking at urinary sediments.
Wash and re-use all slides and glass coverslips.
Substitute trisodium phosphate in cleaning glassware and rubber tubing. This eliminates the use of cleaning solution which consists of concentrated sulfuric acid and potassium dichromate for most laboratory glassware except that used for special blood chemistry procedures.
Use specially cut coverslips in setting up crossmatches for blood trans-
fusions. These coverslips are approximately one millimeter thick and do not break when dropped. They also are easier to clean with less breakage. Cover the foot of the beds in the blood drawing room with castoff pieces of rubber sheeting; cover the pillows with small hand towels; and place eighteen-inch square pieces of rubber sheeting under the donor's arms. This protects and conserves linens.

Store rubber tubing, rubber bulbs and rubber bands in sealed metal containers in the ice box.

Place wide rubber bands around blood counting pipettes before placing them in the shaking machines. This protects the tips and ends of the pipettes against being cracked or broken.

Use cleansing tissue for cleaning all parts of microscopes except the objectives or oculars where lens paper is still employed.

Use less alcohol in running up tissues and in cleaning blood counting pipettes. Water and acetone are used to clean the pipettes in place of water, alcohol, and ether.

Use extra heavy test tubes for special routine work, such as in running urinalyses. This cuts down breakage.

Use the Somogyi method of determining quantitative urine sugars in place of Benedict's quantitative sugar method. The Somogyi method requires only sodium carbonate and test tubes and a Bunsen burner. This avoids the use of more expensive glassware such as burettes and the more expensive and complicated reagents.

**Bacteriology Laboratory**

Wash coverslips used in pneumococcus typing and re-use. Wash by boiling, and clean with xylol and alcohol.

Conserve the use of mice for pneumococcus typings so that typings are made only when use of specific sera is intended. The army requirements for mice are 95 per cent of the supply, so they are difficult to obtain for civilian use.

Use discarded surgical instruments for autopsies on guinea pigs and mice used in laboratory.

Use discarded greens from kitchen to feed laboratory animals.

**Blood Chemistry Laboratory**

Make a job analysis. It may be possible to rearrange space and operations so that work may proceed on an "assembly line" technique.

Reduce unnecessary motion to a minimum. Arrange for all specimens to enter the "assembly line" at the same place.

Use various types of apparatus designed to facilitate rapidity of measure-
ments. The automatic burette which finds its own zero point is an example.

Blood sugar determinations are made in great numbers and can serve as an illustration of procedure.

All blood specimens enter the line at the same place; movement and space arrangement from left to right or vice versa:


While one chemist makes titrations, an assistant makes calculations. When a series of calculations is completed, the first chemist checks the calculations of the second.

General

The broken end of pipette tops and tips whose calibrations are not altered may be reground.

Broken flasks and graduates may be salvaged, provided the calibrations are not destroyed, by chipping the sharp edges with a copper wire Bunsen burner screen. (Hold the glass vessel in one hand and chip with downward sweeping motions of the screen held in the other hand so that any glass particles chipped off will be thrown away from the eyes and face.)

Grinding of pipettes, flasks, and graduates may follow shearing, if desired. The grinding may be done on a small emery grinding machine with a rheostat attachment so that it runs at a slow rate. The technician must use safety goggles in this procedure.

Glass instruments, such as burettes, whose calibrations will not be harmed by repair, should be taken to a glass blower. Often new stop-cocks can be blown on, so that a $6 burette can be repaired for $1.50.

Mercury used in Van Slyke equipment may be recovered and cleaned. Perhaps the simplest way is to strain the mercury, which is picked up from desk tops and the floor, through a chamois. Pressure, by twisting the chamois and squeezing it with the fingers, is necessary. In addition, mercury may be cleaned by running hot and cold water alternately through the mass, and then bubbling compressed air through the mercury during the night.

Use Pyrex glass. Generally, pyrex glass lasts much longer than softer glass. One exception has been found to be glass funnels.

Recover permunit after absorption of ammonia from urine in urea determinations as originally described by Folin.

The last suggestion is an important one, for it tersely describes a philos-
ophy of teaching institutions which are exemplified by our modern hos-
itals.

"Divide laboratory work among the technicians so that each one has a
different range of laboratory work each week (allocation of work). Rotate
the technicians at one-week intervals, so that each can learn the other's
work, does not become bored and get stale on the job, and each can pick
up where the other left off."

By reducing breakage, waste, usage, by substitution and increasing effi-
ciency, we gain more than the cost of supplies. The money value of sup-
plies saved is not the entire saving. With fewer supplies to handle, the
efficiency of the hospital is improved.

One must be careful, however, not to reduce the amount of useful sup-
plies to the point where delay and borrowing occur. Shortage of essential
diagnostic and therapeutic apparatus in any hospital means inefficiency
and many harried moments.

An example may be that of blood pressure apparatus. Several years ago
the taking of routine blood pressure was unusual and done by the intern.
In those days, one blood pressure apparatus to two or three nursing divi-
sions was adequate. Today the nurses take routine blood pressure readings,
often every fifteen minutes, especially for postoperative neurosurgical pa-
tients, and we can well imagine what would happen when there were not
at least one blood pressure apparatus on each floor.

Shortage not only causes borrowing and the loss of equipment for hours,
due to forgetfulness, but there are many unhappy moments when two pa-
tients need blood pressure reading simultaneously. To reduce the supply
of such critical apparatus is like having an army without rifles.

(Grateful acknowledgement is made to the personnel of the various
laboratories who so kindly contributed data for this article.)
Case Reports of the Barnes Hospital
Clinical and Postmortem Records Used in Weekly Clinicopathologic Conferences at Barnes Hospital, St. Louis
W. Barry Wood, Jr., M.D., Robert A. Moore, M.D., Editors

CASE 27

PRESENTATION OF CASE

L. H., a 37-year-old single laborer, entered Barnes Hospital on March 23 and died April 25, 1943.

Chief Complaints.—Pain in the lower back, pain in the right lower abdomen, weight loss, asthma, and expectoration of blood stained sputum.

Family History.—Irrelevant.

Social History.—Patient was born in Illinois. He finished the eighth grade in school and then was a coal miner for 10 years. After that he worked as a day laborer for a contracting firm on a government project. His smoking, drinking and eating habits were moderate. He knew of no contact with cases of tuberculosis.

Past History.—Other than the usual childhood diseases there was no record of any significant illness or operation.

Systemic History.—Not significant.

Present Illness.—For one year the patient had complained of some weakness and fatigue in contrast to his apparently excellent previous health. On November 24, 1942, he fell from a truck and injured his right testicle, which immediately became painful and swollen. It did not respond to treatment prescribed by a local physician and the swelling progressed. One month later the patient entered a Marine Hospital where the right testicle and epididymis were removed through an abdominal incision. These weighed 115 grams. On section the testis was replaced by a white, fairly firm tumor, which encroached slightly upon the epididymis. On microscopic examination the parenchyma had been replaced by sheaths of tumor cells with large chromatic nuclei, and prominent nucleoli. Mitotic figures were numerous. The tumor was very vascular. A diagnosis of embryonal carcinoma of the testis was made. The patient was relieved of symptoms following this operation and was discharged from the hospital on January 30, 1943. Shortly after this, pain in the lower back developed which was constant and not affected by movement. This was followed by pain in the right lower abdominal quadrant which was intermittent, and cramping in nature. For these symptoms he was given 16 deep roentgen-ray treatments
at a local hospital with little, if any relief. During the last month there had been a weight loss of 25 pounds.

For 4 years before admission the patient had had periodic attacks of asthma, appearing at intervals of every 4 weeks. These attacks apparently were not severe. They were worse during the late summer. No specific treatment had been given for them. In addition to asthma, there had been frequent colds during which the patient coughed and expectorated blood tinged sputum. For the last 2 years he had produced such sputum at increasing intervals. He had not been aware of having had fever.

While at the Marine Hospital a positive Wassermann was discovered. He was given several injections into his hip and these were followed later by intravenous injections at a government clinic.

**Physical Examination.**—Temperature was 37.5°, pulse 100, respiration 18, blood pressure 120/84. The patient was well developed but poorly nourished. He appeared to be chronically ill and complained of pain in his back. The skin over the lower abdomen, back and buttocks was discolored a deep brown and small areas of desquamation were present. The skull revealed no masses or tenderness. The pupils reacted to light and accommodation. The retinal arteries appeared normal. The pharynx was diffusely reddened. The trachea was in the midline. There was moderate enlargement of several cervical lymph nodes on either side and there was an enlarged supraclavicular node on the left. There was considerable flattening of the thoracic wall at both apices and intraclavicular regions, particularly on the left, where expansion was diminished. The percussion note was unchanged. Breath sounds were impaired over the entire left chest and fine rales were heard over this area. A few rales were heard over the right apex. The heart showed no abnormalities. The abdomen revealed a surgical scar in the right lower quadrant. There was moderate tenderness and some resistance over this area. The liver apparently extended to the level of the umbilicus but the edge was not well defined. On rectal examination an indefinite resistance, high on the right side, was palpated, over which there was moderate tenderness. The right testicle was absent; the left felt normal. No penile scar was observed. Neurological examination was negative.

**Laboratory Findings.**—Blood Count—red blood cells 4,460,000; hemoglobin 11 grams; white blood cells 5,350; differential count: basophiles 1%, eosinophiles 7%, stab forms 7%, segmented forms 62%, lymphocytes 9%, monocytes 9%. Urinalysis—Sp. Gr. 1.030, albumin negative. Stools—guaiac negative. Sputum—purulent and blood tinged, acid fast bacilli seen. Blood Kahn 4 plus. Wassermann—cholesterinized antigen 1 plus. Friedman test—weak type 1 reaction. Roentgenograms—dis-
tributed throughout each lung field were multiple, discrete areas of infiltration varying in size from 2 or 3 to 5 or 6 mm. in diameter. The right leaf of the diaphragm was held to the lateral chest wall by a large adhesion. Diagnosis—miliary tuberculosis? miliary carcinomatosis? Lower lumbar spine and sacrum—no evidence of bony change. Laminograms of the lungs showed a large cavity in the apical portion of the left lung.

Course in Hospital.—For the first 2 weeks the patient had a mild fever. The pain in the back was severe and required almost constant sedation. On April 9 a supraclavicular lymph node on the left side was removed. On microscopic section the architecture was completely destroyed by infiltration of malignant squamous epithelial cells. On April 15 a lumbar puncture was done. The dynamics were normal. The spinal fluid was clear with 5 cells, Pandy test negative, protein 37 mg%, Wassermann negative, colloidal gold curve 0012110000. Toward the middle of April the patient's condition gradually deteriorated in that he was under almost constant sedation for pain. The temperature gradually became higher. He complained of frontal headache, intensified by changing position. He talked incoherently at times and was unable fully to comprehend questions. The white blood cell count at that time was 15200 and the differential count remained essentially that on admission. On April 22, marked swelling of the eyelids developed and soon spread over the left side of the face and involved the upper lip. There was slight extension to the right face. The swollen areas were red, hot and slightly tender. On the following day the lesion continued to spread and showed a raised advancing edge. Erysipelas was suspected and sodium sulfadiazine was given intravenously. The inflammation improved somewhat. At this time the liver was more distinctly felt. The edge was rounded, firm and nodular as was the surface. Signs in the chest present on admission became accentuated. The patient then gradually became comatose, the blood pressure fell and respirations became shallow and increased. He expired quietly.

CLINICAL DISCUSSION

Dr. Barry Wood: There seems to be little doubt about the primary diagnosis in this case. The patient evidently had a malignant tumor of the testicle. He also had pulmonary tuberculosis, and terminal erysipelas of the face. There are certain other features in the history which are of interest. First, the patient was a coal miner; second, he gave a history of asthma; and third, he had a positive serological reaction for syphilis. There are three problems on which we might focus our attention in the clinical discussion: 1) what was the nature of the pulmonary lesion? 2) what were the extent and location of the metastases from the testicular tumor? and
3) did this patient have syphilis? Dr. Moore, you saw this patient in the hospital. What type of tumor of the testicle do you think this patient had?

Dr. Carl Moore: We saw only a report from the Marine Hospital. It described the tumor as an embryonal carcinoma, and we had no reason to doubt that interpretation.

Dr. Wood: Do you believe that all embryonal carcinomas of the testis are teratomatous in origin?

Dr. Carl Moore: Dr. Ewing states that they are, and that about sums up my opinion.

Dr. Wood: There is a fairly well-founded theory that all testicular carcinomas arise from teratomata. The diagnosis of embryonal carcinoma would seem to be logical in this case. May I ask, Dr. Moore, why the Friedman test was done?

Dr. Carl Moore: When the patient came into the hospital we knew he had tuberculosis and a tumor of the testicle, and we wondered what caused the enlargement of the lymph nodes in the neck and what the lesion in the chest was. I was inclined to think, influenced perhaps by Dr. Sherwood Moore's report, that this was not miliary tuberculosis or disseminated tuberculosis, but a diffuse carcinomatosis of the lung in addition to tuberculosis with cavitation. In order to try to find out whether the patient had extensive metastases, the Friedman test was carried out, since in a fair percentage of cases it is positive in the presence of an embryonal carcinoma of the testis or a metastasis from such a tumor.

Dr. Wood: There are certain testicular tumors that are called choriomas, which have in them cells which are also found in the placenta. These tumors are associated with excessive amounts of chorionic gonadotropin. In the present case, however, the Friedman test gave a one plus, or first degree reaction, which, I believe, is not considered definitely positive. Dr. Moore, are embryonal carcinomas of the testicle radiosensitive?

Dr. Sherwood Moore: The true carcinoma of the testicle is extremely radioreistant. The embryo is very sensitive in some cases, and in these cases there is an immediate, striking therapeutic result from radiation. The Friedman test, incidentally, guides us in our dosage in embryoma of the testicle. The pain that this patient had in his back is a very characteristic finding in these cases. If it is carcinoma of the testicle there will be little relief after radiation, but if it is an embryoma great relief will follow radiation. I might also mention that there were no bone changes in the lumbar spine as shown in the Roentgen ray film.

Dr. Llewellyn Sale: Dr. Wood, does an increased assay of estrogens in the urine go with the positive Friedman test in cases of chorioma?

Dr. Wood: I believe not, but I will refer the question to Dr. MacBryde.
DR. CYRIL M. MACBRYDE: No, the positive Friedman test is due to gonadotropic hormone, not estrogenic hormone.

DR. SALE: I thought that in the dysgerminoma of the testicle the hormone was excreted in the urine.

DR. MACBRYDE: That is a very uncommon tumor. Immature teratomas are much more common and produce the same hormone produced by the chorion—gonadotropic hormone.

DR. WOOD: Dr. Sale, it is common for these patients to give a history of trauma. What role does trauma play in the production of the tumor?

DR. SALE: I think its role is very much like its role in carcinoma of the breast. I am inclined to think it is just coincidental, unless one assumes that it may act as a stimulus for the growth of the tumor. I believe that it merely focuses attention on a lesion that was already there.

DR. WOOD: The second point I think we should discuss concerns the location and extent of metastases. Are there any suggestions as to where this tumor has metastasized?

DR. PAUL HAGEMAN: I think that with the location of a metastatic node above the left clavicle one may assume that the tumor extended up the periaortic nodes through the abdomen and chest. From the location of the pain one may assume also that it involved the lumbar spine.

DR. WOOD: There is some question about the second of these sites, is there not? The Roentgen ray films of the lumbar spine showed nothing abnormal. How do the tumor cells get from the testicle to the left supraclavicular gland?

DR. HAGEMAN: The drainage of the testicle goes into the lymphatics of the pelvis, which drain into the lymphatic vessels following the aorta, and join the drainage into the thoracic duct—the mechanism operative in producing a Virchow's node.

DR. WOOD: It is common for tumors of the testicle to present first in the upper abdomen. There is another channel of drainage along the spermatic vein. The lymphatics running along the spermatic vein join those in the celiac axis, and the metastases in the upper abdomen result from direct extension along this route. Certainly we would expect involvement of lymph nodes in the thorax and abdominal cavity. Is there any other explanation of the pain besides a metastasis in the lumbar spine? Could the back pain be caused by direct pressure from lymph nodes in the pelvis?

DR. HAGEMAN: I doubt it.

DR. WOOD: Are there any other possible sites of metastases?

DR. EDWARD MASSIE: The liver is a likely site.

DR. WOOD: Would you be certain of that?
DR. MASSIE: The liver in this case is described as being enlarged, and on the basis of statistical evidence I think it is involved.

DR. WOOD: Any other possible metastases?

DR. SHERWOOD MOORE: There is one other thing that suggests involvement of the bone. I have seen one case which metastasized to the skull, with severe headache.

DR. CARL MOORE: I think the parenchyma of the lung should be listed as having been affected.

DR. WOOD: Why are you so certain the lung is affected?

DR. CARL MOORE: From the Roentgen ray films one gets the impression that there are so called "cannon-ball" metastases.

DR. WOOD: Has anyone else any other suggestions?

DR. LAUREN ACKERMAN: There must have been a great many tumor cells in the thoracic duct near the lung, and a great many tumor cells must have been going to the lung if the tumor metastasized along the aorta.

DR. WOOD: There are several possibilities in regard to this pulmonary lesion. We know the patient had tubercle bacilli in the sputum, so we can without hesitation make a diagnosis of pulmonary tuberculosis. There may be a tumor. Then the patient was a coal miner. Dr. Moore, I take it that you favor a diagnosis of tuberculosis with cavitation and possibly some silicosis, but no tumor.

DR. SHERWOOD MOORE: Yes, although there may or may not be some metastasis. The lesion to my mind is substantially tuberculosis.

DR. WOOD: Dr. Goldman, what is your opinion?

DR. ALFRED GOLDMAN: In regard to the silicosis, it would be necessary to know exactly how much exposure and what type of exposure the patient had had before making a diagnosis. The Roentgen ray film is compatible with pneumoconiosis, tuberculosis, or miliary carcinoma. Carcinoma of the lung and tuberculosis do not occur very often simultaneously. Our own statistics here show that. I know of only one case here in which a patient with tuberculosis acquired a carcinoma. Some reports in the literature give an incidence of as high as 15 per cent, for the association of carcinoma of the lung with tuberculosis. I think that is too high. The picture in this case could be explained as an old tuberculosis and then recently an acute miliary spread.

DR. MASSIE: I wonder if one could get some information from the cardiac configuration? With tuberculosis and asthma one might expect cor pulmonale, and since we do not see that here, might we reason that this is a case of diffuse carcinomatosis?

DR. BARRETT TAUSIG: There is one other point: the clinical history is
not particularly suggestive of miliary tuberculosis. There was no episode of fever, such as one would expect in miliary spread.

Dr. Wood: The absence of fever would seem to be important. Dr. Goldman, what about the eosinophilia and the asthma?

Dr. Goldman: He may have had a true bronchial asthma, but the silicosis which we think preceded the tuberculosis in this case would give him attacks of dyspnea. This would probably be a better explanation for the dyspnea than true asthma.

Dr. Wood: Are you in favor of the diagnosis of tuberculosis?

Dr. Goldman: I favor tuberculosis, probably with silicosis and a terminal invasion by the tumor. There could be miliary carcinomatosis. Until Dr. Carl Moore’s remarks I was leaning heavily toward tuberculosis without carcinoma.

Dr. Wood: Dr. Hageman, this patient had several positive serological tests for syphilis. Do you believe he had syphilis?

Dr. Hageman: There is no clinical history of syphilis. No findings point to it specifically, but this type of reaction—a markedly positive Kahn test and weakly positive Wassermann—is the kind we find often in false positive tests. Tuberculosis is often the cause of false positive Kahn and Wassermann reactions. The incidence of false positive tests in tuberculosis runs as high as 7 per cent. I would regard this as a false positive reaction.

Dr. Carl Moore: Would the amount of therapy this man had change the Wassermann reaction to that extent?

Dr. Hageman: There was only a three-month interval between the onset of his illness and the time he died. I doubt it.

Dr. Wood: Dr. Hageman, what is a cholesterinized antigen and why is it employed?

Dr. Hageman: The addition of cholesterol to the Wassermann antigen makes it more sensitive. A Wassermann test can be made as sensitive or as insensitive as is desired. Ours is less sensitive than the Kahn. In the various stages, we would first get a positive Kahn, then a positive cholesterinized Wassermann, and then a positive alcoholic Wassermann.

Dr. Carl Moore: I have here a sheet given out by the Army Medical School showing the conditions which cause false positive tests for syphilis. It is interesting to see the great number of diseases implicated, including both tuberculosis and carcinoma.

Dr. Wood: In this case we seem to agree that the patient had embryonal carcinoma of the testicle with metastases. We feel that this may be a false positive test for syphilis. The stumbling block seems to be the pulmonary lesions. It might be valuable in this connection to go back to the metastases and trace out the tumor spread from the primary lesion. Obviously the
supraclavicular node metastasis must have been by way of the lymphatics. However, Dr. Massie, you suggested metastasis to the liver. How would the tumor cells get to the liver if they did not go through the lung?

Dr. Massie: I was thinking of direct venous spread.

Dr. Wood: That would mean that a lymph node around the portal vein would have to erode the portal vein. Is it not more likely that tumor cells from the testicle went by way of the spermatic vein and the inferior vena cava to the lungs and thence metastasized to the liver? The spread of testicular tumor cells is often by way of the blood stream as well as by way of the lymphatics. We have evidence that there are metastases in the liver, and possibly in the brain, and it is difficult to see how these cells reached their destination unless there were metastases in the lung. We would, I believe, be justified, therefore, in saying that the pulmonary lesions include tumor, tuberculosis, and possibly silicosis.

Dr. Sherwood Moore: May I ask why this orchidectomy was done through a laparotomy?

Dr. Wood: I don’t know, unless the surgeon wanted to examine the regional lymph nodes.

Student: Dr. Wood, I would like to ask about the surgical report on the biopsy of the supraclavicular node. It was a squamous cell carcinoma. Is that a common finding in carcinoma of the testicle?

Dr. Wood: Theoretically almost any type of cell may be found, if it is assumed that the primary tumor is of teratomatous origin. In practice, epithelial metastases are not uncommon.

Dr. Ackerman: It is true, is it not, that once the tumor begins to metastasize, only one type of cell tends to metastasize?

Dr. Wood: Yes, that is true.

Student: Dr. Wood, would you ascribe this man’s death to his erysipelas?

Dr. Wood: I think the erysipelas must have influenced the situation but I do not think it was alone the cause of his death. Metastases to the brain or skull might have contributed, in addition to the pulmonary lesion and the obvious tuberculosis.

CLINICAL DIAGNOSIS

Malignant embryoma of testis with metastases to lung, liver, and lymph nodes.

Tuberculosis of lung, far advanced

Syphilis of undiagnosed site

Erysipelas of the face
MEDICAL ALUMNI QUARTERLY

DR. WOOD’S DIAGNOSIS

Embryonal carcinoma of testicle with metastases to regional, abdominal, thoracic and supraclavicular lymph nodes, lungs, liver and brain
Pulmonary tuberculosis with cavitation
Silicosis
Erysipelas, terminal
Bronchial asthma
Syphilis, latent

ANATOMIC DIAGNOSIS

Metastases of embryonal adenocarcinoma of the testicle in the abdominal, thoracic and cervical lymph nodes, in the wall of the inferior vena cava, in the liver and the intrahepatic radicles of the portal vein, and in the lungs
Chronic fibrocaseous tuberculosis of the upper lobe of the left lung with cavitation
Bronchogenic tubercles in all lobes of the lungs
Tuberculous ulcers in ileum and cecum
Miliary tubercles in the mesenteric lymph nodes, spleen, liver, and renal cortices
Syphilitic aortitis

PATHOLOGIC DISCUSSION

DR. MARGARET SMITH: The tumor as found in metastases was an adenocarcinoma. This is one of the less common, but well recognized types of malignant tumor of the testis. All of the metastases were of a similar histologic character. We were not able to examine the original tumor of the testis, so that the presence or absence of other types of tissue in the original tumor cannot be proven. However, most pathologists are in accord with Ewing in believing that all embryonal tumors of the testes are teratomas. Both the tuberculous lesions and the metastases must have been concerned in producing the physical signs in the lungs in this patient.

CASE 28

PRESENTATION OF CASE

M.S., a 64-year-old housewife was first admitted to Barnes Hospital on February 23 and discharged March 16, 1942.

Chief Complaints.—Loss of appetite, loss of weight, diarrhea.
Family History.—Irrelevant.
Social History.—The patient was born in Illinois, where she had lived all her lifetime. She had a 10th grade education, married at the age of 20, had 3 children, and had been occupied as a housewife. During her last few years, she lived in poor financial circumstances and required old age assistance.
Past History.—The patient’s general health had always been good and the only significant recorded illnesses were malaria at the age of 26, without recurrence, and a hysterectomy at 60 because of uterine prolapse. This was followed by a recto-vaginal fistula which eventually closed.

Systemic History.—The teeth were extracted many years ago and false dentures worn. Her average weight was 155 pounds.

Present Illness.—In the late autumn of 1941, the patient noticed that she was gradually losing her appetite and realized that she was unable to take as much food as formerly. Early in December of that year she suddenly developed pain in the right upper abdomen associated with a hard shaking chill, followed a day or two later by jaundice. Her skin became a deep yellow and this pigmentation lasted for about 4 weeks. During this interval there was little or no pain and no further chills. The patient was considerably nauseated, however, and vomited a few times. She had been given oral medication and a fat-free diet. During her convalescence from this illness she had frequent watery stools which were not formed and were of a light yellow color. She passed 10 to 15 such stools a day, on an average, and her movements were frequently associated with a rather severe gripping pain in the right lower abdomen. Her appetite continued to be poor and her diet consisted largely of the following foods: cereals, bread, orange juice, milk, and an occasional egg. There was a weight loss of 55 pounds from the onset of symptoms to the date of admission to the hospital.

During this illness the patient maintained that she had developed heart trouble because of shortness of breath on exertion, palpitation, and occasional swelling of the ankles.

Physical Examination.—Temperature 37.2°, pulse 84, respiration 20, blood pressure 140/70. The patient was an elderly woman who appeared to be chronically ill. There had been apparent weight loss. The skin and sclerae were slightly yellow. The eye grounds showed arteriosclerotic changes of mild degree. The tongue was not coated but was slightly reddened. There were fissures at the corners of the mouth. The lungs were clear except for a few crackles at the right base. The heart was not enlarged, the sounds were distant and there were no murmurs. There was an occasional extrasystole. The peripheral arteries were somewhat thickened. The liver descended to the umbilical level. The surface was smooth and not tender. There was moderate senile vaginitis. Rectal examination was negative. Slight edema of the ankle was present on each side.

Laboratory Findings.—Blood count—red blood cells 4,360,000, hemoglobin 12.9 grams. White blood cells 6,950, differential count: basophils 1%, "stab" forms 3%, segmented forms 66%, lymphocytes 23%, monocytes 7%. Urinalysis—specific gravity 1.005, albumin faint trace, microscopic
negative. Stool—unformed, clay colored; guaiac negative; urobilinogen 1 plus, urobilin 1 plus, much neutral fat, many fatty acid crystals; culture negative for typhoid-paratyphoid group; negative for parasites. Blood Kahn—negative. Blood chemistry—sugar 119 mg%; non-protein nitrogen 18 mg%; total proteins 5.2 grams, albumin 3.0, globulin 2.2; icterus index 10, amylase 40 units. Liver function test—hippuric acid 17% excretion of sodium benzoate. Bromsulphonphthalein test—5% retention 30 minutes and 5% in one hour. Prothrombin time 18 seconds; control 23 seconds. Electrocardiograms were essentially normal. Roentgenograms of the gastrointestinal tract—duodenal cap was very markedly dilated and somewhat elongated, and at the juncture of the first and second portions was a definite accessory pocket which retained the barium beyond 48 hours. This did not have the characteristics of a simple diverticulum and raised the question of perforation. It was stated that such an inflammatory action could well involve the common duct. The impression was that of perforation of the duodenum with accessory pocket (chronic).

Course in Hospital.—Under symptomatic treatment including high vitamin and high caloric intake, the patient improved in that her diarrhea ceased. She gained weight, stools assumed a more normal appearance, and her appetite returned. Shortly before discharge the following laboratory data were secured. Blood amylase was 28 units per cent, total blood proteins were 5.4 grams with albumin 2.9 and globulin 2.5. A gastric analysis showed no free acid either before or after injection of histamine. The hippuric acid test gave 21% excretion of sodium benzoate and the blood count was within normal limits. The patient was referred to the Gastro-Intestinal Clinic in the Outpatient Department.

Second Hospital Admission.—During the year that the patient was under observation in the Gastro-Intestinal Clinic she slowly lost ground. Soon after discharge from the hospital, loose, watery, yellowish to clay-colored stools reappeared. Likewise, griping pain on bowel movement in the right lower quadrant, sometimes about the rectum, also returned. Her appetite gradually failed. Edema of the ankles became pronounced and the patient was referred to the Cardiac Clinic where digitalis was administered without effect. In January 1943, a gastroscopic examination was performed and normal findings were reported. One week previous to admission the stools became more frequent and contained blood and mucus. These persisted in spite of medication. During her observation in the Clinic, vitamins and pancreatin had been given.

Physical Examination.—Temperature 39.2°, pulse 110, respiration 25, blood pressure 100/60. The patient was markedly emaciated; her skin was very dry and atrophic and there were numerous small dilated veins generally
distributed. The tongue showed definite atrophy of papillae although this was not marked. There were fissures at the corners of the mouth. The heart and lungs appeared to be normal. The liver was felt but one finger breadth below the right costal margin although a sense of resistance was noted in this region. The abdomen was otherwise negative. Pelvic and rectal examination were as on the first admission.

**Laboratory Findings.**—Blood count—red cells 4,400,000, hemoglobin 10.5 grams, white cells 6,150, differential count: basophils 1%, eosinophils 3%, "stab" forms 18%, segmented forms 58%, lymphocytes 19%, monocytes 1%. Urinalysis entirely negative. Stool—thin, watery, with clay colored particles and very much mucus. Microscopically there were partially digested and undigested meat fibers, many unidentified crystals, and no blood cells; guaiac negative; Sudan III, trace of free fat. Blood chemistry—non-protein nitrogen 19 mg%, total proteins 4.3 grams, albumin 2.2, globulin 2.1, amylase 23 units%, calcium 7.7 mg%, phosphorus 2.2 mg%, phosphatase 7 Bodansky units, cholesterol 36 mg%. Hippuric acid test—oral method—17% excretion of sodium benzoate; intravenous method 73% excretion. Cephalin flocculation test—negative. Blood Kahn—negative. Glucose tolerance test—fasting blood sugar 55mg%; following specimens contained 104, 142, and 113 mg% (at the end of 3 hours). Urine specimens all negative. Gastric analysis no free acid after test meal and after histamine. Duodenal drainage produced 20 cc. of yellow bile; culture—nonhemolytic streptococci and E. coli; guaiac negative; slight liquefaction of protein media after 12 hours, moderate liquefaction after 36 hours. Basal metabolic rate—plus 10. Intravenous cholecystograms showed nonvisualization of the gall bladder throughout the series. Lying in the region of the gall bladder and also in the region of the right kidney were multiple areas of calcification, the largest of which was approximately 1 cm square. A diagnosis of pathological gall bladder and intra-abdominal calcification of indeterminate nature was made.

**Course in Hospital.**—Under symptomatic treatment, the diarrhea and pain in the abdomen disappeared within a few days. The patient appeared to be improving although her appetite remained poor. A gain in weight was noted but coincident with this the ankles became more swollen and gradually fluid developed in the abdomen and in the chest. The fever noted on admission disappeared within a few days. The blood proteins gradually fell to a total of 3.3 grams with albumin 1.7 and globulin 1.6. An anemia developed with red cells of 3,200,000, hemoglobin 6.5 grams. One week before death 1,900 cc. of thin, milky, greenish-yellow ascitic fluid were removed. It had a specific gravity of 1.005 and there were 40 cells per cmm. It contained 0.8 grams per cent of protein and culture was sterile. In spite
of tube feeding and intravenous injections of plasma and whole blood, the blood proteins remained low and general anasarca gradually increased. Two weeks before death there was a temperature elevation coincident with the rise in white blood cell count to 17,600. Several days later bronchial breathing was heard over the right chest. Sulfamerizine was given without effect. The blood pressure gradually fell during the last several days to a level of 50/40. The diarrhea continued. Apathy developed during the last week and the patient died quietly.

CLINICAL DISCUSSION

DR. HARRY ALEXANDER: This patient seemed to be entirely well until late in 1941, when she had some loss of appetite, followed by the signs and symptoms of sudden obstruction of the common duct. Soon after this the presenting symptom developed which continued throughout the rest of the time she was observed—chronic diarrhea. The question arises whether this was a cause or an effect of the defect in the common duct, or whether the anorexia had any relevance. Are there any suggestions?

DR. CYRIL MACBRYDE: I think she had an acute pancreatitis.

DR. ALEXANDER: If there is an obstruction in the common duct the presumption usually is that it is above the pancreatic duct, but it is true that the bile becomes infected in these cases. In favor of a pancreatitis, then, is the fact that this patient did have gallbladder disturbances. What else?

DR. MACBRYDE: Or it may have been just the other way around—she may have had an acute pancreatitis which caused the obstruction to the bile duct.

DR. ALEXANDER: What is there in favor of this possibility?

DR. MACBRYDE: The fact that the first attack subsided and there was no recurrence of the pain. It may have been the pain of pancreatitis and not that of gallbladder colic.

DR. ALEXANDER: She had a very low amylase, did she not?

DR. MACBRYDE: That wasn’t taken until some time later.

DR. ALEXANDER: You feel that she had an acute pancreatitis that completely subsided?

DR. MACBRYDE: Yes, and that is borne out by the fact that her bulky, watery stools continued over a long period of time, associated with a different type of abdominal pain. I think she had an acute pancreatitis which subsided and was followed by a chronic pancreatitis.

DR. ALEXANDER: What evidence is there for chronic pancreatitis?

DR. MACBRYDE: The chronic diarrhea.

DR. ALEXANDER: She had diarrhea, and the description stated that the stools were frequent, loose, and watery. I don’t know that they were said
to be bulky. On the first examination there seemed to be an increase in fat in the stools. What else is in favor of pancreatitis?

DR. HAROLD SCHEFF: The muscle fibres in the stools. There appears to be an absence of trypsin.

DR. ALEXANDER: There was a test taken, however. Do you feel that this was a valid test, Dr. Taussig?

DR. ALBERT TAUSSG: We are not told at what temperature it was kept, and that is a crucial point. Ordinary incubation would allow bacterial action to produce as much digestion as there was here. A temperature of 54° C. or thereabouts is necessary to inhibit the action of most bacteria.

DR. SAMUEL MARTIN: It was incubated at 54° to 56°C.

DR. ALEXANDER: Therefore, there was some fermentation present. How would one account for the poorly digested muscle fibers in the stool in the presence of trypsin?

DR. CARL MOORE: On the mere fact of the presence of diarrhea.

DR. ALEXANDER: Again this patient had a low amylase. Dr. MacBryde, what does this mean? Where is the amylase absorbed?

DR. MACBRYDE: I don't believe anyone actually knows. We do know that the most important diagnostic use for the amylase test is the determination of high amylase, which is associated with an acute pancreatitis at the time it is operative. Immediately after this there is apt to be a fall almost to a normal level, and it may persist at normal levels for some time. There is some evidence, less conclusive, however, that low blood amylase may be associated with chronic pancreatitis.

DR. ALEXANDER: She did manufacture trypsin and she did manufacture insulin.

DR. MACBRYDE: I still think she did not have trypsin getting into her intestine because she persistently had the undigested muscle fibers and chronic diarrhea.

DR. ALEXANDER: It is very difficult to estimate fat in the stools; and I believe it has been established that one must destroy a very large amount of pancreas before there is depressed function. If this is true, the lesion as we will see it pathologically should be a marked pancreatic lesion. Do you agree Dr. MacBryde?

DR. MACBRYDE: Yes.

DR. ALBERT TAUSSG: This action of trypsin on Loeffler's medium at 54° C. is very intense. If you put a drop of diarrheal or duodenal content on the medium, in a short time it will digest a deep hole in the medium. I gather that here a good deal of the contents were put on top of the medium, yet there was only a slight digestion. I think there is good reason to be-
lieve that the greatest significance of low blood amylase is the indication of impaired hepatic function.

Dr. Alexander: What other evidence is there that hepatic function is lowered? Low blood amylase and low hippuric acid, and what else?

Dr. Taussig: Low proteins.

Dr. Alexander: But what about the ratio? This ratio is not inverted, with the proteins as low as they are. Is that to be expected or is it unusual?

Dr. Taussig: It merely indicates a failure of the compensatory function of the globulin and indicates even greater diminution of hepatic function than with compensatory rise in the globulin.

Dr. Alexander: There was an enlarged liver. Is it your impression that this patient had an organic hepatitis and an organic pancreatitis?

Dr. Taussig: Yes.

Dr. Hageman: What about a failure of absorption of material from the gastrointestinal tract?

Dr. Alexander: How much of this may be explained on the basis of an enteritis? Certainly if one ingests hippuric acid and it doesn’t enter the portal veins, then the liver cannot receive it. A low hippuric acid test was found, low calcium, low phosphorus, low proteins. What about the cholesterol?

Dr. MacBryde: It is very low.

Dr. Alexander: Low cholesterol. We may fit these findings in with pancreatitis, hepatitis or enteritis.

Dr. Wood: The diarrhea, the mucus and blood in the stool, and the pain would all fit in with enteritis.

Dr. Alexander: The Roentgenogram of the gastrointestinal tract was taken on the patient's first admission in February, 1941. She died a year later and there was no other film. With a diarrhea as intense as this for a year would you expect structural changes in the bowel, Dr. Scheff?

Dr. Scheff: Yes.

Dr. Alexander: What could have caused the diarrhea? In true pancreatitis there are large fatty stools and the fat itself causes some frequency.

Dr. MacBryde: I think the frequency of the stools is characteristic of the pancreatitis. That would explain all of the absorptive difficulties.

Dr. Alexander: It is said that in any case of unexplained diarrhea in an adult one should think of pancreatitis. There is another fact that I should like to bring to your attention: the patient had an absolute achlorhydria.

Dr. Scheff: It is common to have diarrhea with a histamine-fast achlorhydria.
Dr. Alexander: May the diarrhea in this 64 year old patient have originated because of an absolute achlorhydria?

Dr. Wood: It is very unlikely. It followed immediately the attack of chills, fever, pain, and jaundice.

Dr. Hageman: Did she receive dilute hydrochloric acid at any time?

Dr. Martin: She received it in the clinic over a long period of time to no avail.

Dr. Llewellyn Sale: What about her avitaminosis?

Dr. Alexander: What deficiency might have been caused by her diet, as described in the clinical abstract?

Dr. Schieff: Protein deficiency.

Dr. Carl Moore: It would depend upon what kind of cereal and bread she ate, but the chances are that she didn't eat whole grain cereal and bread. She had a deficiency of the B complex on that assumption. To fit in with this, she had a glossitis and a cheilitis, and a partial cessation of diarrhea when she was given vitamins.

Dr. Alexander: The diarrhea itself is a symptom of avitaminosis. The question is, did the diarrhea come from a pancreatitis, a vitamin deficiency, or possibly from a lack of acid? The important thing is that this woman eventually, I presume, starved to death. She had lots of food and vitamins, and she may or may not have absorbed them, but she certainly did not utilize them. When she came to the hospital the second time her diarrhea remained unchecked. Was it caused by liver disease or lack of absorption? Absorption has been studied in cases of sprue, pellagra, and pernicious anemia, and it has been found that there is great impairment of amino acid absorption, and even glucose is poorly digested in advanced cases. Dr. Moore, what is your feeling about this?

Dr. Carl Moore: I would like to comment first on the cephalin-floculation test which is the most sensitive of all the tests of hepatic function. It is inconceivable to me that her cephalin-floculation test could be negative with any great degree of liver damage. It is my feeling that this patient had comparatively little disturbance of hepatic function. Throughout the history it is repeatedly stated that she had clay-colored stools when she did not have clinical jaundice. It certainly ought to be emphasized that she did not have an obstructive jaundice or a hepatogenous jaundice.

Dr. Alexander: In sprue there may be light colored stools with no obstructive jaundice. May I supplement your remarks by asking if the prothrombin time is a good liver function test? Dr. Wood, what do you think?

Dr. Wood: The cephalin-floculation test probably depends upon the globulin fraction of the serum protein. Here it is not increased, which
might account for the negative cephalin-floculation test. The prothrombin time should be abnormal if there is marked involvement of the liver. I agree that there is no very good evidence that the liver is involved. I do think the pancreas is involved. I believe the diarrhea is on the basis of an acute pancreatitis, which followed an attack of biliary disease.

Dr. Sale: How do you account for the original attack which was accompanied by intense jaundice, which lasted 14 weeks?

Dr. Alexander: Dr. Bottom, on the basis of the Roentgenogram, do you think that there could be a stone?

Dr. Donald Bottom: It is a rather unusual shape for a stone, but it might be.

Dr. Carl Moore: Do you think serious consideration should be given to the suggestion that there was perhaps a diverticulum or actual rupture of the duodenum, which may have set this off? It might have been responsible for the initial pain, and the pancreatic deficiency, and the initial episode of jaundice. The pancreatic deficiency could have persisted along with the avitaminosis, which was cured; and she may have died because of the pancreatic insufficiency.

Dr. Alexander: The radiologists are puzzled about the shadow. The second observation indicated that this was calcification and not a duodenal diverticulum.

Dr. MacBryde: All experimenters know that when the pancreatic duct is obstructed it is common for the pancreas to build itself a new channel back into the duodenum. This patient might have had a fistula or a new channel.

Dr. Margaret Smith: What was the significance of the very low cholesterol?

Dr. Bulger: I do not know unless it was related to very marked gastrointestinal insufficiency.

Dr. Alexander: A chronic enteritis will prevent the absorption of amino acids. This might conceivably affect the lacteals so that no fat could get into the pancreatic duct.

Dr. MacBryde: She had a very low blood sugar.

Dr. Alexander: But when she was given glucose it went up to 140.

Dr. Scheff: This patient had jaundice without clay-colored stools for four weeks during that period of time.

Dr. Alexander: You did the gastroscopy on this patient, Dr. Scheff. Are the normal gastric findings compatible with the presence of achlorhydria?

Dr. Scheff: No, but they are occasionally found.

Dr. Wood: Dr. Alexander, what is your diagnosis?
DR. ALEXANDER: With a diarrhea so intense that there was at one time mucus and blood in the stool, cramping and pain in the abdomen without cessation, I had expected to find a chronic enteritis—either an atrophy or a marked edema such as one would find in pernicious anemia, pellagra, or chronic sprue. After Dr. Taussig’s remarks I feel that these tests were not accurate enough. I believe that most of this patient’s symptoms could be explained by a diagnosis of enteritis. What is your feeling, Dr. Wood?

DR. WOOD: I think that the pancreas is the main source of trouble. The pancreatitis may be secondary to biliary tract disease, possibly to a stone. I think that there may possibly be ulcerative lesions in the lower bowel to account for the mucus and blood—in other words, ulcerative colitis in addition to the pancreatitis.

DR. ALEXANDER: How do you account for the low proteins?

DR. WOOD: By poor absorption from the gastrointestinal tract.

DR. MACBRYDE: The liver shrank a great deal. If that is true, there should be a cirrhosis. The patient had a low grade jaundice, which might fit in with the cirrhosis.

CLINICAL DIAGNOSIS

Pancreatic fibrosis

Hypoproteinemia, of unknown causation.

DR. ALEXANDER’S DIAGNOSIS

Chronic enteritis

ANATOMIC DIAGNOSIS

Mucinous adenocarcinoma involving duodenum, common bile duct and head of pancreas, with ulceration of the superior and descending parts of duodenum.

Fistulae between duodenum and common bile duct

Dilatation of common bile duct above tumor

Obstruction of pancreatic ducts by tumor

Acinar atrophy and fibrosis of pancreas

Fatty metamorphosis of liver

Fissures at each corner of the mouth

PATHOLOGIC DISCUSSION

DR. MARGARET SMITH: This tumor, a mucinous adenocarcinoma arising in the region of the ampulla of Vater, is an unusual and rare tumor. At the time of death the tumor was so large that it was not possible to determine the exact site of origin. In one of the microscopic sections there is a direct continuity between normal duodenal mucosa and neoplastic tissue, suggesting that it was primary in the mucosa of the duodenum.
News from the Medical School and Affiliated Hospitals

The U. S. Public Health Service has made a grant to Washington University in order to finance a cooperative study by Drs. M. Trotter and V. Lanier of the Department of Anatomy and Dr. Howard McKnight of the Department of Obstetrics and Gynecology of the spinal dura mater and of the posterior surface of the sacrum in connection with the new method of continuous caudal anesthesia in childbirth.

In the July, 1943 issue of the Quarterly, the following sentence concerning the report of the meeting of the American Neurological Association was erroneously printed. "Great Britain was represented by Professor Geoffrey Jefferson, who has been doing special research work on peripheral nerves at the Nuffield Institute at Oxford." The sentence should have read: "Great Britain was represented by Professor Geoffrey Jefferson, who is the leading neuro-surgeon in England; and Dr. John Z. Young, who has been doing special research work on peripheral nerves at the Nuffield Institute at Oxford."

Dr. Meyer Wiener, professor of clinical ophthalmology, and member of the Board of Honorary Consultants to the Surgeon General of the U.S. Navy, is on the West Coast observing battle casualties in the navy hospitals and appraising treatment methods.

The following promotions in the staff have been made for 1943-44: Dr. Edward G. McGavran to Professor of Public Health Administration and Acting Head of the Department of Public Health; Dr. Margaret G. Smith to Associate Professor of Pathology; Dr. F. O. Schwartz to Associate Professor of Clinical Ophthalmology; Dr. Gerty T. Cori to Associate Professor of Research Biological Chemistry and Pharmacology; Lieut. Col. Earl H. Perry to Associate Professor of Military Science and Tactics; Drs. Carl G. Harford and John R. Smith to Assistant Professor of Medicine; Drs. Paul O. Hageman, Samuel B. Grant, Walter Fischel, Alfred Goldman, Arthur Strauss and Llewellyn Sale to Assistant Professor of Clinical Medicine; Dr. Adolph Conrad to Assistant Professor of Clinical Dermatology; Drs. William O. Russell and Paul Wheeler to Assistant Professor of Pathology; Drs. H. Rommel Hildreth and Carl Beisbarth to Assistant Professor of Clinical Ophthalmology; Dr. Hiromu Tsuchiya to Assistant Professor of Parasitology.
New appointments to the staff include: Dr. T. Dale Stewart as Visiting Professor of Anatomy; Dr. Sam Gray as Associate Professor of Pathology; Dr. Hans B. Molholm as Assistant Professor of Psychiatry; Dr. Harry Wiese as Instructor in Clinical Medicine; Mr. Alfred Buchmueller as Instructor in Social Service in Neuropsychiatry; Dr. O. A. Grueble as Lecturer in Public Health; Miss Frances Kimura as Assistant in Bacteriology and Immunology; Dr. Charles Huguley as Assistant in Medicine; Dr. William Park as Assistant in Clinical Medicine; Dr. Homer A. Sweetman as Assistant in Psychiatry; Drs. Howard E. McKnight and Seymour Monat as Assistant in Obstetrics and Gynecology; Dr. Robert W. Godwin as Assistant in Otolaryngology; Drs. Jean Dehlinger, William Klingberg, James Owen, John Wilson, Jack Burnett and Mary Ritchey as Assistant in Pathology; Drs. Jane Erganian and Joseph Rubel as Assistant in Pediatrics; Drs. Robert M. Rankin and Cyril J. Costello as Assistant in Surgery; Dr. Robert S. Reiss, Felip Alberto de la Balze, Llewellyn Sale, Jr., Reubenia Dubach, and Misses Mary Johnson and Mary Ruth Smith as Research Assistant in Medicine; Dr. Dagoberto E. Gonzalez as Visiting Fellow in Chest Surgery; Dr. Manuel E. Albarenque as Rockefeller Foundation Fellow in Neurological Surgery; Dr. Alvin Rix as Fellow in Neurological Surgery.

Leaves of absence for duty in the armed forces have been granted to the following: Dr. Mary Miller, Research Associate in Pathology; Dr. Ralph C. Pollock, Instructor in Clinical Medicine; Dr. Carl Lischer, Assistant in Surgery; Dr. D. Elliott O’Reilly, Assistant in Surgery; Dr. Frederick W. Klinge, Assistant in Surgery; Dr. Edward O. Kraft, Assistant in Surgery; Dr. C. Barber Mueller, Assistant in Surgery; and Dr. Henry Barnett, Instructor in Pediatrics.

Leaves of absence for national service have been granted to the following: Dr. Arthur L. Hughes, Consulting Physicist to the Radiological Institute.

The following members of the staff were retired on July 1 with the titles indicated: Dr. Harry W. Lyman, Professor Emeritus of Clinical Otolaryngology; Dr. Frederick J. Taussig, Professor Emeritus of Clinical Obstetrics and Gynecology; Dr. Frederick Woodruff, Associate Professor of Clinical Ophthalmology; Drs. Walter Baumgarten and Louis H. Hempelmann, Assistant Professor Emeritus of Clinical Medicine; Dr. Adolph G. Schlossstein, Assistant Professor Emeritus of Clinical Obstetrics and Gynecology; Dr. Thomas Pote, Lecturer Emeritus in Pathology.

Dr. David McK. Rioch, Professor of Neurology, resigned from the staff.
effective September 1, 1943 to become the Director of Research at the Chestnut Lodge Sanitarium, Rockville, Maryland.

The Chancellor announced the following gifts to the School of Medicine between June 1 and August 31, 1943: from the William S. Merrell Company, $1,200 for the establishment of a fellowship under Dr. Willard Allen for investigation of Tri-p-Anisyl Chloroethylene; from the Winthrop Chemical Company, $1,200 in continued support of the fellowship under Dr. Cyril MacBryde’s direction; from the Influenza Commission of the Board for the Investigation of Epidemic Diseases, of the U. S. Army, an allocation of $4,050 to Dr. Wood for a project to be conducted under his direction; from The Commonwealth Fund, a grant of $4,700 a year for three years to Dr. Wood for his studies on the mechanism of recovery in bacterial pneumonia; from the William S. Merrell Company, $700 in support of the study on the therapeutic value of Theophylline Isobutanolamine under the direction of Dr. Julius Jensen; from Mrs. Frederick E. Woodruff, $300 for hospitalization of eye patients.

The Joint Medical Board recommended to the appropriate Boards the following appointments to the staffs of the hospitals: Dr. Alfred M. Large, Assistant Surgeon to the Barnes and St. Louis Children’s Hospital and Dr. George Saslow, Assistant Psychiatrist to the Barnes Hospital.


Appointments in the School of Nursing include: Honora Wells Camden, Assistant Professor of Nursing in Charge of Public Health Nursing in the Basic Program; Virginia H. Harrison, Instructor in Nursing; Mary Helen Goodof, Assistant in Nursing and Supervisor on Medical Ward 2418 at Barnes Hospital; and Virginia T. Doyle, Assistant in Nursing and Surgical Supervisor at Barnes Hospital.

Dr. S. Albert Key is President-Elect of the American Orthopedic Association.
It is with deep regret that the Quarterly announces the death of Dr. Frederick J. Taussig, Professor Emeritus of Obstetrics and Gynecology. Dr. Taussig graduated from St. Louis Medical College in 1898 and had been on the faculty of Washington University since 1906. In addition he had for many years served as chief of the department of gynecology and chairman of the executive committee of the Medical Board of the Barnard Free Skin and Cancer Hospital. He was vice-chairman of the State Cancer Commission and active in the administration of the Ellis Fischel State Cancer Hospital.

Dr. Robert Elman and Dr. Alexis Hartman gave clinics at the Army and Navy General Hospital, Hot Springs, Arkansas on September 16. Dr. Paul Hageman and Dr. H. Relton McCarroll presented similar clinics on September 22. These clinics are a part of the War-Time Post-graduate Medical Lectures sponsored by national medical societies.

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Mary Keith formerly Superintendent of the St. Louis Maternity Hospital now Superintendent of the St. Louis County Hospital.
News of Alumni

1879
W. B. Lucas, of Mendon, Mo., writes: “I graduated from the St. Louis Medical College in 1879. I have been in active practice from that date to the present. My health is fairly good and am able to be on the go about all the time.”

“I would be glad to know if any of my class are still living, and if so, where?”

“The last meeting of this class in St. Louis there were only eight thought to be living. . . . With the best wishes for this great institution, I remain, Yours truly.”

1889
Dr. Josiah G. Moore’s new address is: 1480 S. San Antonio Ave., Pomona, California.

1906
Colonel Wm. H. Smith, M.C. announces his new address as: Army General Hospital, Auburn, California.

1909
Dr. Archibald R. Stone’s new address is: Box 1938, Columbia 16, Ohio.

1914
O. F. McKittrick, writes: “Please find enclosed check for the picture you so kindly sent to me recently. I am much pleased with it and hope some day to get a larger one which shows the growth of our school as time goes on.”

“As to your invitation to write about myself, it is difficult for me to know what information would be the most interesting to impart. However, after my return from overseas in 1919 after serving with the 78th Division in the first world war, I remained here in the east. I was fortunate in securing a very fine location in one of Harrisburg’s closest suburban towns, where from the beginning I have had more work than I could do. It has been my aim always in whatever I undertook in my work to excel, and at the same time be of service to as many of the sick as possible. This is the reason why I have stuck to the general practice of medicine and nearly every day there are new and interesting diseases and conditions in which I come in contact. My location is also near many fine clinics and specialists. Being a Missourian, and having graduated from the best medical school in the country furnished the foundation for whatever success I have had.”

“This war finds me at my work because I was asked to remain where I am. I would like to express my appreciation for the case reports you people print in the Quarterly. In the July issue of the Quarterly “Case Reports of the Barnes Hospital’ were very interesting and instructive.”

1922
We are grieved to learn of the death of Helen Curtis Fleming of West Palm Beach, Florida on July 6, 1943. She was the wife of Dr. S. Ward Fleming, Class of ’22, and a graduate of Washington University School of Nursing, Class of ’24.

1925
Major Jerome S. Levy stopped in the Alumni Office recently while visiting in St. Louis. He has been stationed at the Bushnell General Hospital, Brigham City, Utah, for the past year. He is chief of the Gastro-intestinal Sub-section in the Medical Service. Dr. Levy enjoys his work, and finds the climate in Utah delightful. He has assisted in taking care of casualties from all fronts. He brings greetings

Dr. Shigeichi Okami's new address is: P. O. Box 149, Fort Lupton, Col.

Dr. William E. Hart's new address is: Patterson Field, Fairfield, Ohio.

Rogers Deakin recently received this letter from Major E. H. Christopherson, APO 603, Miami, Florida: "Your letter of way back in April has been forwarded to me at a new station. It was very nice to hear from you and Duff. I have frequently had reports on you fellows during the years and have a fairly good idea about your behavior."

"I have been down this way about seven months now. Spent two months in Rio and about four months in the Rio Doce Valley on a general public health program. Since the first of July I have been director of the Amazon Project."

"This project is general public health and medical care of the entire valley. It covers an area over half the size of the U. S. We have 32 stations in the valley. Some are over 2000 air miles away and 4000 miles by boat. The problems are great, especially of communication and transportation. In most places it is pioneering as nothing has ever been done before. We are building a few hospitals, establishing dispensaries and doing general control measures, especially for malaria. It is undoubtedly the largest public health program ever undertaken, also the largest anti-malaria program. We have probably the world's finest malaria laboratory under Dr. Caurey, loaned from the Rockefeller Foundation. All the tropical diseases are prevalent and it is an excellent place to study them."

"There are several Washington University graduates down this way. James Knott '25, and H. V. Markham '26 are here with me. Jean Rogier '34, and John Seddon '35, are in the Rio Doce. Jean Starkloff '29 is in Rio at the moment. Of eleven doctors from the states in the outfit, six are from Washington University. I guess they recognize good men when they see them."

"This is a big job and with lots of difficulties to overcome. We work pretty hard, and it is rather hot, but we are making out fine. I believe I can speak for the rest when I say we'll all be glad to get back."

1928

William M. Brewer's new address is: Station Hospital, Camp Gruber, Oklahoma.

1929

L. C. Drews, Metropolitan Bldg.,
Class Secretary.

Lt. Col. James H. Forsee's new address is: 2249 Ivy Street, Denver, Colo.

Dr. Estella E. DeFreitas's new address is: 1372 East Front St., Plainfield, New Jersey.

1930

Clyde E. Kane, 706 Walton Ave.,
St. Louis, Mo., Class Secretary.

Dr. J. W. Tidwell's new address is: 500 Thorn Place, Marion, Illinois.

1931

Sam Bassett, 1200 Big Bend Road, Richmond Heights, Mo.,
Class Secretary.


1932

Dr. Louis T. Byars, 607 N. Grand, St. Louis, Mo., Class Secretary.

Captain Ralph Stuck is now in the European theatre of war. Any communications to him should be sent to the following address: 516 Franklin St., Geneva, Ill.

Lt. Col. Wm. R. Lovelace, surgeon
and air forces expert, who attended Washington University School of Medicine during the years 1930-31 and 1931-32, made a parachute jump from 40,200 feet—highest on record in this country—to convince himself and everybody else that the emergency oxygen equipment furnished to Army airmen is all that laboratory tests indicate.

The leap of more than seven and one-half miles was his first jump. His only injury was the freezing of his left hand. The jerk of his opening parachute flipped off his heavy glove in a 50-below temperature. Otherwise Col. Lovelace suffered no discomforts, as the heavy regulation clothing, including oxygen mask, goggles and helmet, protected even his face from the cold.

Lovelace jumped from an Army bomber near Ephrata, Washington. The oxygen equipment which he used was developed under his supervision for the Office of the Air Surgeon.

1934
Stanley M. Leydig, 162 S. Grand, St. Louis, Mo., Class Secretary.
Dr. Mary M. Schmeckebier’s new address is: 936 Leavenworth St., San Francisco 9, California.

1935
Dr. M. K. O’Heeron’s new address is: 112 Station Hospital, APO 3334, New York, N. Y.
Major John S. Poe’s new address is: 1512 N. Hancoek Road, Arlington, Va.
Lt. Comm. R. V. King’s new address is: Naval Hospital, Philadelphia, Pa.
Any communications to Major Bert M. Bullington should now be addressed: 3761 North Meridan Street, Indianapolis 8, Indiana.

1936
Dr. N. W. Drey’s new address is: Camp Williams, Utah.

1938
Dr. John R. Lionberger, Barnes Hospital, St. Louis, Mo., Class Secretary.
Captain Harry Baers is home visiting after having been on duty as flight surgeon for the U. S. Army Air Corps in the South Pacific. He spent most of his time in the Hawaiian Department in Honolulu. During the battle of Midway he had the opportunity to be in a flying fortress that took part in the battle. He also was among the group to fly over Munda during the Solomon invasion.

Captain Baer’s wife, Donna Norwood, is a former nurse from Washington University and Barnes Hospital. While in Honolulu she was a nurse at Queens Hospital. She and the captain are the proud parents of two children.

The Captain thinks that Hawaii is an excellent place to be on foreign duty. Meat is plentiful and the only restrictions are blackouts and no night life. “Tropical weather is moderate compared with the sultry heat of St. Louis,” says Dr. Baers.

1939
Lt. Heinz E. Cron is now stationed at Camp Barkeley, Texas.

1940
Lt. Carl T. Buehler’s new address is: Governors Island, New York.
Lt. Robert Koch recently visited St. Louis while on furlough. He is stationed at Winfield, Kansas, where he is chief of the Medical Section.

Lt. Seymour Brown’s new address is: Great Lakes Naval Training Station, Great Lakes, Ill. Lt. Brown recently completed an advanced course in anaesthesia at the Lahey Clinic and visited in St. Louis on his way to Great Lakes. His Pacific Medal has five stars on it. Battle of Midway, Battle of the Coral Sea, Occupation of Guadalcanal,
Victory at Guadalcanal, and the Raid on Tokyo.

Lieutenant Commander William Love has spent the past two and one-half years in the Canadian Navy. He was recently sent to St. Louis by his Medical Director General to attend clinics at Washington University. He has now been assigned to a hospital where he is chief medical officer. His address is: H.M.C.S. St. Hyacinthe, St. Hyacinthe, P. Q.

1941

Lt. John P. Lee, APO No. 41, c/o P. M., San Francisco, California, writes: "At the present I am in New Guinea where I have been for quite some time. There are quite a few other Washington men here. Many I have contacted personally or heard of through other medical officers. Here is the list of those I recall at the moment: Lt. Col. Alvah Miller '34, Capt. Frank Robinson '35, Lt. Peter Fleming '41, Lt. Lawrence Kotner '38, Lt. Barney Finkel '41 (in Australia), Lt. Robert Elman '41 (S. W. Pacific area), Lt. S. Jackson '41 (in Australia)."

Lt. Chas. Williams's new address is: Ft. Jackson, S. C.

Captain and Mrs. Frank K. Tatum, 1809 Oak St., Abilene, Texas, announce the birth of a daughter, Julie Ann, July 14, 1943.

1942

Lt. Glen O. Turner, who has been stationed at Camp Grant, Illinois, visited St. Louis while on a three-day furlough. He has just completed a course in general military training prior to being assigned to a permanent station.

Lt. Jackson Neavles has completed his training as a medical officer with paratroops at Camp Benning.

Student News

Laboratory partners in the sophomore class, Claire Conerly and Robert H. Ramsey were married Friday, August 13 in the Third Baptist Church in St. Louis. The class presented the newlyweds with a set of dishes. The bride is the daughter of Mrs. W. L. Conerly, of Jackson, Alabama, and the groom is the son of Mr. and Mrs. J. W. Ramsey of Fort Smith, Arkansas.

The medical school resembles a military academy since the induction of the majority of students. Of the 397 students, 265 are in the army, 76 in the Navy, and 56 (including the women) are civilians.

Miss Anne Peggs became the bride of Robert McKay Scott Saturday, September 11. Mr. Scott, a member of the junior class, is the son of Mrs. and Mr. Lewis L. Scott, of Glendale, Mo. The bride is the daughter of Mr. and Mrs. Joseph W. Peggs, University City, Mo.

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Recent Acquisitions by the Library

Possession does not imply approval


Association for research in nervous and mental disease. The role of nutritional deficiency in nervous and mental disease. New York, 1943. (Research publications, v. 22.)

Ballenger, W. L. and Ballenger, H. C. Diseases of the nose, throat and ear, medical and surgical. Philadelphia, 1943. 8th ed.


Beck, S. J. Personality structure in schizophrenia; a Rorschach investigation in 81 patients and 64 controls. New York, 1938.


Cohn, E. J. and Edsall, J. T. Proteins, amino acids and peptides as ions and dipolar ions. New York, 1943.


Dake, H. C. and deMent, Jack. Ultra-violet light and its applications, including a description of the numerous practical applications found for ultra-violet light and fluorescence in the industries, sciences, and art. Brooklyn, 1942.


Schiff, Fritz and Boyd, W. C. Blood grouping technic, a manual for clinicians, serologists, anthropologists and students of legal and military medicine. New York, 1942.


Weiss, E. and English, O. S. Psychosomatic medicine, the clinical application of psychopathology to general medical problems. Philadelphia, 1943.

Publications by the Staff of the School of Medicine, Washington University

June - August, 1943

Alexander, H. L., Goldman, A., Moore, C. V., et al. Tuberculosis pericarditis; tuberculosis of the superficial part of the myocardium; partial fibrous obliteration of the pericardial sac; fibrocaceous tuberculosis of the anterior mediastinal lymph nodes; milliary tubercles in liver and spleen; thrombi in the branches of the pulmonary arteries; infarcts of the lower lobes and left upper lobe of the lungs. (Barnes case 20) J. Missouri M. A. 40: 199-204, July, 1943.


Alexander, H. L., Moore, C. V., Taussig, B. L., et al. Subacute bacterial endocarditis involving aortic valve and ventricular surface of anterior leaflet of mitral valve (Actinomycyes graminis); bacteremia (Actinomycyes graminis); focal embolic glomerulonephritis; acute arteritis of small cerebral vessels; hemorrhage into right cerebral hemisphere; partially healed infarct of spleen; chronic endocarditis of mitral and aortic valves, slight; hypertrophy and dilatation of the heart, 550 grams; bronchopneumonia of upper and lower lobes of the lungs (pleomorphic bacterium morphologically similar to Actinomycyes). (Barnes case 18) J. Missouri M. A. 40: 176-180, June, 1943.


Bradley, F. R. Outline of program for conserving resources of the hospital laboratory. Hospitals, 17: 87-88, August, 1943.


Heinbecker, P., Rolf, D. & White, H. L. Effects of extracts of the hypophysis, the thyroid and the adrenal cortex on some renal functions. Am. J. Physiol. 139: 543-548, August, 1943.


Lischer, C. E. & Elman, R., with the technical assistance of Harry Riedel. Experimental burns. II. Effect of elastic pressure applied to a burned area. War Med. 3: 482-483, May, 1943.


MEMBERs OF THE STAFF OF
WASHINGTON UNIVERSITY SCHOOL OF MEDICINE
IN THE ARMED FORCES

AGRESS, HARRY, MAJOR—Assistant in Clinical Medicine and in Pathology—A. P. O. 362, c/o Postmaster, New York, N. Y.

ALLEN, HENRY C., LIEUTENANT—Instructor in Pathology and Assistant in Clinical Medicine—Navy 156, c/o Fleet Postmaster, San Francisco, California.

ALLEN, HUBERT L., CAPTAIN—Assistant in Clinical Obstetrics and Gynecology—2nd Army Headquarters, Memphis, Tenn.

ALVIS, EDMUND B., CAPTAIN—Assistant in Clinical Ophthalmology—A. P. O. 362, c/o Postmaster, New York, N. Y.

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BARTLETT, ROBERT W., MAJOR—Assistant Professor of Clinical Surgery and Instructor in Anatomy—B. T. C. No. 5, AAFTC, Kearns, Utah.

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HOLDEN, RAYMOND F., JR., LIEUTENANT—Instructor in Clinical Medicine—Station Hospital, Army Air Base, Alliance, Nebraska.

HUGHES, ARTHUR L.—Consulting Physicist to Mallinckrodt Radiology Institute—Address unknown.

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MARCUS, MORRIS D., CAPTAIN—Assistant in Clinical Otolaryngology—Camp Carson, Colo.

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MOUNTJOY, PHILIP S., CAPTAIN—Assistant in Clinical Otolaryngology—Base Hospital Will Rogers Field, Oklahoma City, Okla.

MUeller, C. Barber, LIEUTENANT, J. G.—Assistant in Surgery—Address unknown.

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ROULHAC, George—Assistant in Surgery—Address unknown.

ROULETTE, Avery P., Lt. Colonel—Assistant Professor of Clinical Surgery—A. P. O. 600, c/o Postmaster, New York, N. Y.

RUSK, Howard, Major—Instructor in Clinical Medicine—Office of Air Surgeon, Air Force Europe, Washington, D. C.

Russi, Simon, Lieutenant—Assistant in Pathology—Lawson General Hospital, Atlanta, Ga.

Satterfield, Val B., Major—Assistant Professor of Clinical Psychiatry—U. S. Navy Hospital, Bremerton, Washington.

SCHWARTZ, Henry G., Major—Assistant Professor of Clinical Neurological Surgery—A. P. O. 362, c/o Postmaster, New York, N. Y.

SCHWARTZMAN, Bernard, Captain—Assistant in Clinical Pediatrics—Station Hospital, P. A. A. B., Topeka, Kansas.

SCHWARZ, Henry II, Lieutenant—Assistant in Surgery—A. P. O. 795, c/o Postmaster, New York, N. Y.

ScHOTT, Wendell G., Lt. Commander—Assistant Professor of Clinical Radiology—Medical Special Group 82, USNR, San Diego, Calif.

Seddon, John W., Lieutenant—Instructor in Clinical Medicine—A. P. O. 603, c/o Postmaster, Miami, Fla.


Sendina, Ben H., Captain—Instructor in Clinical Otolaryngology—School of Aviation Medicine, Randolph Field, Texas.

Shefts, Lawrence—Fellow in Chest Surgery—Address unknown.


Smith, Dudley R., Major—Assistant Professor of Clinical Obstetrics and Gynecology—7th Service Command Unit, General Hospital, Topeka, Kansas.

Smith, Edward, Lieutenant—Instructor in Pathology—Laboratory of Second Service Command, 90 Church Street, New York, N. Y.


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Stevens, Frank W.—Assistant in Neuropsychiatry—Address unknown.

Thornton, Robert L.—Physicist in Charge of the Cyclotron of the Radiological Institute—Address unknown.

Tureen, Louis L., Major—Assistant Professor of Clinical Neurology—A. P. O. 302, c/o Postmaster, New York, N. Y.

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WASSERMAN, HELMAN C., CAPTAIN—Instructor in Clinical Obstetrics and Gynecology—Station Hospital, A.A.F. B.F.S., Newport, Arkansas.

WEBIG, JOHN, CAPTAIN—Assistant in Clinical Medicine—Address unknown.

WEICHSELBAUM, THEODORE E., LIEUTENANT—Research Associate in Bacteriology—c/o General Delivery, Hattiesburg, Miss.

WHITE, BART N., LT. COLONEL—Assistant in Medicine—A. P. O. 795, New York, N. Y.

WHITE, HARVEY LESTER, COLONEL—Associate Professor of Physiology—A. P. O. 942, Seattle, Washington.

WRIGHT, SYDNEY, LIEUTENANT—Assistant in Medicine—A. P. O. 795, c/o Postmaster, New York, N. Y.

WULFF, GEORGE J. L., MAJOR—Instructor in Clinical Obstetrics and Gynecology—A. P. O. 182, c/o Postmaster, Los Angeles, California.

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Miss Estelle Claiborne,
Superintendent of the St. Louis
Children’s Hospital
In Memoriam

John Barstow Coryell, St. L. '85, St. Louis, Mo., aged 73, died May 31.
Fred L. Gibbs, '13, St. Louis, Mo., aged 52, died June 4.
Clarence E. Grim, '01, Kirksville, Mo., aged 69, died May 26.
Archibald G. Henderson, Mo. '99, Leonardville, Kansas, aged 78, died March 14.
Frank B. Lansdowne, Mo. '97, Kenosha, Wis., aged 67, died April 19.
William V. Lindsay, Mo. '98, Winona, Minn., aged 69, died April 24.
Francis W. Mann, Mo. '89, Wellington, Mo., aged 79, died March 11.
John H. Simon, Mo. '90, St. Louis, Mo., aged 74, died May 24.
Alvin H. Sippy, Mo. '85, Clayton, Mo., aged 79, died April 13.
Edward R. Stewart, Mo. '87, Blair, Neb., aged 81, died May 1.
Washington University School of Nursing and the St. Louis Children's Hospital
WASHINGTON UNIVERSITY

George R. Throop, Ph.D., LL.D., Bridge Chancellor

Walter E. McCourt, A.M., Assistant Chancellor

The College of Liberal Arts
  William G. Bowling, A.M., Dean

The School of Engineering
  Alexander S. Langsdorf, M.M.E., Dean

The School of Architecture
  Alexander S. Langsdorf, M.M.E., Dean

The School of Business and Public Administration
  William H. Stead, Ph.D., Dean

The Henry Shaw School of Botany
  George T. Moore, Ph.D., Director

The School of Graduate Studies
  Richard F. Jones, Ph.D., Dean

The School of Law
  Warner Fuller, B.S., LL.B., Acting Dean

The School of Medicine
  Philip A. Shaffer, Ph.D., Dean

The School of Dentistry
  Benno E. Lischer, D.M.D., Dean

The School of Nursing
  Louise Knapp, R.N., A.M., Director

The School of Fine Arts
  Kenneth E. Hudson, B.F.A., Director

The University College
  Willis H. Reals, Ph.D., Acting Dean

The Summer School
  Frank L. Wright, A.M., Ed.D., Director

Mary Institute, a preparatory school for girls, located at Ladue and Warson Roads, is also conducted under the charter of the University.

Note: Those desiring information concerning any of the divisions listed above should write to the Dean or Director concerned.

1 Deceased May 30, 1943.