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The
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Decompression Illness in Aviation

Further Impressions of American Medicine

Case Reports of the Barnes Hospital

Vol. VI JULY, 1943 No. 4

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Decompression Illness in Aviation[1]

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UNITED STATES NAVAL RESERVE[2]

Aviation medicine is a new field in medical science. It has received its biggest impetus in World War II because of the importance of the airplane at present and the possible implications of aircraft in the post-war world. The primary purpose of aviation medicine is the care and maintenance of aviation personnel. This branch of medicine encompasses the specialties of ophthalmology, otolaryngology, industrial hygiene, physiology, bio-chemistry, and internal medicine. Most fields of medicine deal with the abnormal individual in his usual environment. Aviation medicine is concerned with the normal individual in an unusual environment which affects the normal physiological state (figure 1). The physician in aviation medicine must have a fundamental and complete knowledge of the changes in environment as they occur in aviation and the effects of these changes upon the aviator.

When the advancement of aircraft design made possible ascents above 30,000 feet, a new problem entered aviation medicine. This problem is that of decompression illness, known to the aviator by many synonyms such as the "bends," "chokes," "itch," or "staggers," adopted from the descriptions of compressed air illness. The diver's body contains approximately 4800 cc of nitrogen at 100 feet below the surface of the water (4 atmospheres pressure). He must therefore allow 3600 cc of this nitrogen to be removed during ascent to sea level so that only 1200 cc of nitrogen will remain in his body at sea level. The aviator during an ascent to 35,000 feet (a comparable change in barometric pressure of 4 to 1) has to lose 900 cc of nitrogen to be in equilibrium at this altitude, and only 300 cc of nitrogen remains. It is apparent that the volume of nitrogen moved in the com-

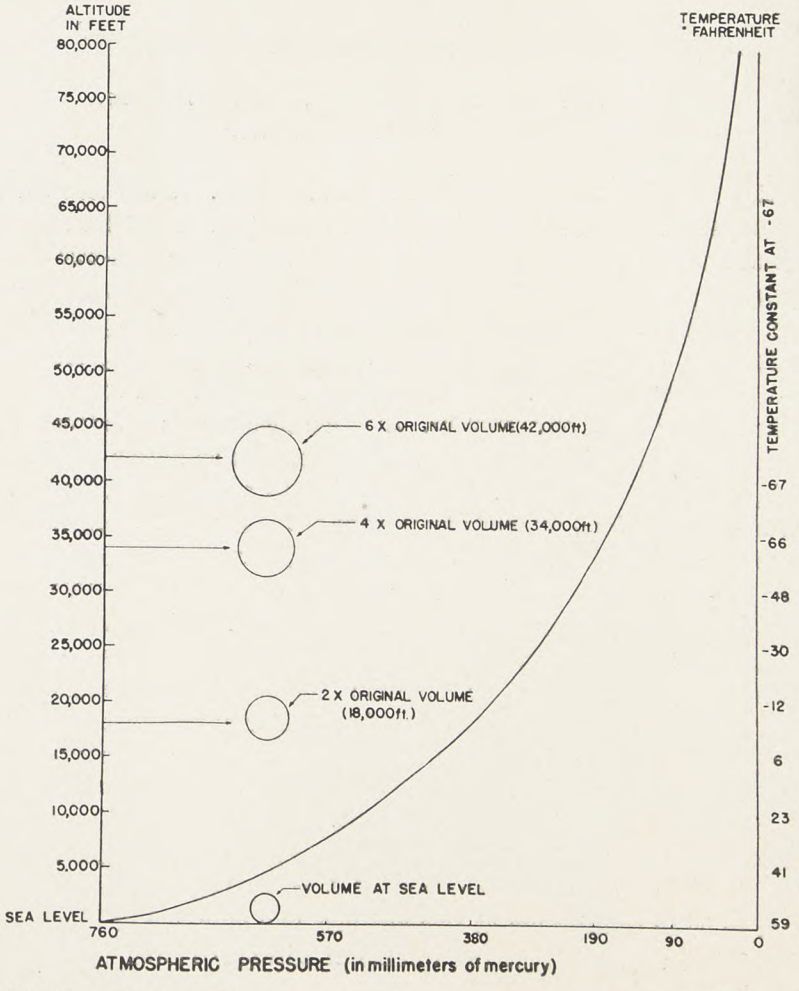
1 The views herein expressed are the opinions of the author and do not necessarily reflect the policies of the United States Navy.

2 From the School of Aviation Medicine, United States Naval Air Training Center, Pensacola, Florida.

pressed air worker is four times that which is moved by the aviator during a comparable but reverse 4 to 1 change in barometric pressure.

Definition

Decompression illness in aviation is a condition which appears during exposure to altitudes in excess of 30,000 feet. The most probable cause is the release and accumulation of nitrogen in a gaseous state in the tissues.



Relation of temperature, gas pressure and gas expansion to altitude.*

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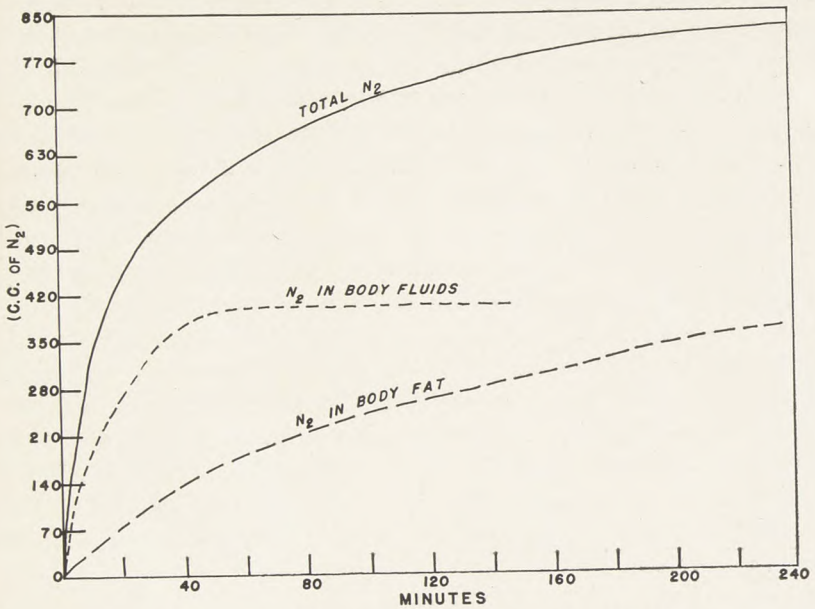

NITROGEN ELIMINATION DURING BREATHING OF OXYGEN (AFTER BEHNKE)

It is characterized by varied symptoms, mainly in the skeletal, dermal, pulmonary, and nervous systems. This condition is spoken of more formally as aeropathy, aeroembolism, or decompression illness.

Etiology

There are three major factors related to the appearance of decompression illness: (1) the altitude to which the individual is exposed, (2) the duration of exposure at this given altitude, and (3) the rate of ascent. There are other factors which may alter the development of decompression illness, and among these must be considered the individual variations in physical condition, obesity, exercise, and the inherent resistance to decompression illness.

The most widely accepted theory of the production of decompression illness in aviation is that the gasses physically dissolved in the body are liberated from their usual depots. With a rapid decrease in atmospheric pressure, the body is unable to eliminate the gas as rapidly as it is freed from solution and this gas accumulates in various areas as bubbles. Since fat takes up nitrogen five times more avidly than any other body tissue, it holds the greatest amount of nitrogen. Thus wherever fat is stored, a potential depot of nitrogen exists.

The body may be compared to a bottle of carbonated water. When the cap is secured on the bottle, a certain amount of gas is dissolved and held in the fluid by the pressure under which the gas is driven into the fluid. When the pressure is released (decompression) by removing the cap of the bottle, this physically dissolved gas is liberated from solution into the gaseous phase. This is seen when the gas effervesces after removal of the cap.

At sea level, 1000 (1) to 1400 (2) cc of nitrogen is held in physical solution in the body fluids and tissues by the partial pressure of nitrogen in the atmosphere. With a rapid decrease of atmospheric pressure, the partial pressure of nitrogen decreases and the pressure responsible for the solution of nitrogen in the body tissues is decreased. When altitudes above 30,000 feet are attained with the inhalation of 100 per cent oxygen, this decrease in partial pressure of nitrogen is so great that more nitrogen is released from solution within the tissues than can be removed at that time from the body via the blood and lungs. This produces a state of gaseous supersaturation which results in the liberation of nitrogen from body fluids. The nitrogen thus freed from solution collects as a bubble and enlarges as more gas is evolved.

Behnke (1) and Boothby (2) in their studies of elimination of nitrogen have estimated that approximately 550 cc of nitrogen can be removed from the body in the first hour and an additional 200 cc in the second hour, while breathing 100 per cent oxygen (figure 2). The first amount of gas which is freed from the body is rarely responsible for any of the serious symptoms of decompression illness. Gas diffusion from depots in the body fluids is rapid as compared with the relatively slow denitrogenation from the fat depots (figure 2). This latter source of nitrogen is thus trapped in a gaseous state in the tissues when released from physical solution by a change to low barometric pressures, and is probably responsible for the subsequent symptoms of decompression illness.

Pathologic Anatomy

The pathologic changes in decompression illness in aviation have not been studied. However, because of the similarity between decompression illness in aviation and compressed air illness, which occurs in divers, many analogies have been drawn. The changes in decompression illness are just as severe as those which occur in compressed air illness, but are more readily reversible. Gaseous emboli may obstruct the circulation dependent on the size of the bubble and the collateral vascular supply. However, if the gaseous obstruction is allowed to continue or increase in severity, ischemic necrosis of the obstructed parts may occur. Phemister and his co-workers (3) have shown that aseptic necrosis of the long bones may occur in com-

pressed air workers; this lesion has never been reported in decompression illness in aviation. Air emboli to vital organs in aviation are extremely rare. Armstrong (4) reports two cases of apparent involvement of the nervous system. One case developed chronic convulsive seizures and the other a paralysis from the waist line up. Both recovered rapidly and completely after compression to sea level.

The effects of anoxia while working at altitudes above 30,000 feet must be considered separately from the effects of decompression. Problems created by gas expansion at these altitudes must also be differentiated from the effects of decompression. The later effects of repeated exposures to low barometric pressure remain to be studied.

Symptoms

Because nitrogen may be present anywhere in the body, the symptoms of decompression illness may occur at any point in the body. The areas of predilection point to some localizing mechanism. Theoretically these areas are those of poorest collateral circulation. The severity of the symptoms are dependent upon the location of the bubble and the growth in volume of that bubble.

Paresthesias and Eruptions: The symptoms of decompression illness appear most commonly in the skin. Soon after an altitude above 30,000 feet is attained the susceptible subject will notice extensive thermal paresthesias. These usually appear suddenly and are generalized in their distribution. They may be either hot or more frequently cold. The distal portions of the extremities are more commonly and severely affected. The sensations may appear as a sudden flush or in moving waves. They usually persist as they originate, but may alternate. These early thermal paresthesias rarely persist for more than ten minutes, and they are probably attributable to the large quantity of nitrogen carried in the peripheral blood during the early phases of decompression. After a short period, which is usually free of any symptoms these early paresthesias are followed by late paresthesias. In contrast to the early changes the late thermal paresthesias are characterized by localized distribution. They are frequently accompanied by tactile paresthesias of a pruritic nature. The tactile sensations commonly occur alone, usually persisting longer than those of thermal nature and changing in location or severity with the application of pressure at the point of appearance. Occasionally subjects complain of crawling sensations of the skin together with other dysesthesias. Hypoesthesias, anesthesias or hyperesthesias occur rarely.

Three types of erythemata have been seen at high attitudes: (1) scarlatiniform eruptions of intense nature on the proximal portion of the

extremities and torso, (2) a mottled, splotchy eruption, cyanotic in color, and of a macular character usually located on the shoulders and (3) erysipeloid eruptions with sharp, raised, indurated borders on the torso.

Urticaria is found on the torso and proximal portions of the extremities. Subcutaneous emphysema occurs late and usually appears on the distal portions of the extremities. Large bubbles of gas can be felt in the regions of the joints and tendon sheaths.

Paresthesias of the conjunctivae of both thermal and tactile nature occur after about twenty minutes of exposure. The thermal sensations are usually cold, and tactile sensations are mainly pruritic in character. The lids feel irritated and gritty, with increased lacrymation. This increase in lacrymation may be due to the release of nitrogen from the lacrymal fat. A residual conjunctival injection may persist for hours after recompression. The dysesthesias of the dermal system are the earliest to disappear when the altitude is decreased. The erythemata and urticaria may last for a short period following descent.

Pain: One of the more severe symptoms of decompression illness is pain in the bones and joints. The pain is difficult to localize and appears to be articular, subperiosteal, and subperichondrial, or directly from within the bone. It may also be a referred pain from muscle or surrounding tissue, or perhaps from somewhere along the distribution of the nerve supplying that region. The pain is dull at first, and although early it may be mild and transient, it can rapidly become constant, extremely severe, and incapacitating in explosive fashion. The excruciating nature of the pain can precipitate shock and collapse with all the cardinal signs of rapid, thready pulse, cold and clammy skin, peripheral vasoconstriction, and fall in pulse pressure, followed soon by loss of consciousness. The pain may not become progressively worse, but may eventually decrease and even disappear, although it will usually return in full force. Although pain in one area is more common, it is not unusual to see multiple foci of pain in the bones and joints. There is immediate relief from pain upon descent.

"The Chokes": Pulmonary disturbances occur late in exposure to extremely high altitudes and are collectively referred to as the "chokes." There is but a short premonitory period of pulmonary discomfort. The sensations are those of "burning of the lungs," uncomfortable substernal oppression, "feeling as though there were an inflated ball within the chest," sharp sticking pain, and dyspnea. Apprehension is most marked and the subject is completely incapacitated. These disturbances are the most severe symptoms of decompression illness at high altitudes.

It is thought that these symptoms are caused by emboli or by the filtering of the circulating, gaseous nitrogen bubbles by the pulmonary capillaries.

Coughing is initiated early in the process by a tickling sensation in the throat or perhaps by an alveolar reflex. Apparently because of the extreme rarity of the atmosphere at high altitudes, coughing becomes severe, strenuous, and the individual is wracked with paroxysms. The face becomes flushed and cyanotic and then blanches as shock approaches. If the ambient pressure is not increased immediately, the patient may collapse. Relief from the pulmonary symptoms of decompression illness by increasing the barometric pressure is not as dramatic as that which occurs with the bone and joint pain. Frequently soreness in the chest may persist for hours following descent, but the distress is never as severe as that present at higher altitudes.

The "Staggers": Subjects complain occasionally of vertigo under conditions of low barometric pressure, but one cannot be certain that hyperventilation or anoxia is not a contributory state. When vertigo does occur, it is usually severe and is accompanied by a rotary nystagmus. When nystagmus, mental confusion and nausea appear at high altitudes the critical situation does not allow for the necessary time to study the cases completely. Compressed air workers refer to this involvement as the "staggers," but in my experience in aviation decompression illness, the vertigo is too severe to permit the erect posture, even if the restrictions of oxygen supply in aircraft would allow it.

Neurological Symptoms: The appearance of nitrogen in its gaseous phase in the central nervous system may occur either by liberation from a depot within the nervous tissue proper or as an embolic phenomenon. There is nothing in the clinical picture of vertigo to indicate the site of development, either peripheral or central. Hemiplegias, paraplegias, and myelitides are extremely rare and, although they have been reported (4) the cause remains to be confirmed. The dermal sensations may be a result of bubbles in the peripheral or even central nerve tracts. The erythemata may be a neurodermatitis secondary to nerve stimulation.

Mental State: The mental reactions at simulated high altitudes in the low pressure chamber are many and of wide scope. There is usually a state of mild apprehension accompanying the first exposure above 30,000 feet. This usually is aggravated by the appearance of the first symptoms in the dermal system but, after reassurance, this apprehensive state is replaced by one of complacency and later one of boredom. The mental state is slowed and there is a tendency toward day-dreaming. Headaches are not common during exposure, but often appear several hours after recompression. The headaches are frequently frontal and temporal, but may be generalized. Cortical infarction although suspected has not been proved. Those individuals in whom apprehension persists do not enjoy the exposure. Slight

symptoms may be exaggerated because of mental tension. In contrast to the adjusted individual, the pulse rate is increased when high altitudes are reached and remains so throughout the exposure. Sinus arythmias become more pronounced. There is a rapid return to the normal state upon descent.

Shock: Shock is the most serious immediate complication following the development of decompression illness under low barometric pressures. When shock develops with the more severe symptoms of decompression illness, immediate recompression must be instituted. Although at altitudes above 30,000 feet the subject is breathing 100 per cent oxygen, shock will so alter his respiratory excursions that a state of anoxia may supplement his already precarious position.

Fatigue: Fatigue following exposure to low barometric pressures is uniform. It is generalized and appears within 2 to 4 hours after descent to sea level.

Diagnosis

The diagnosis of the condition can be made in the presence of any of the symptoms of decompression illness which occur during exposure to barometric pressures below 225 mm. (30,000 feet). The dermal sensations may be similar to the parestheseas accompanying the alkalosis of hyperventilation. However, a brief check of the depth and rapidity of the respiratory excursions will resolve the problem. Anoxia may be confused with the cerebral manifestations of decompression illness. However the cyanosis of the mucous membranes and nail beds in the presence of a cause for anoxia (respiratory, poor mask fit, poor oxygen supply), together with the prompt recovery when the flow of oxygen is reinstated, establishes the diagnosis.

Abdominal symptoms of decompression illness in aviation are rare, if indeed they do occur. The abdominal discomfort encountered is usually dull and cramping and can be explained by the simple expansion of a water-saturated gas. This increase in intra-abdominal pressure frequently splints the diaphragm in a position of expiration, thus hampering diaphragmatic respiration.

Prognosis

The symptoms of decompression illness occur only at high altitudes. If the barometric pressure is increased before collapse ensues from decompression illness, the symptoms rapidly vanish. However, if collapse does occur at high altitude, the resultant effects are due to the state of shock, augmented by the danger of anoxia at the levels above 30,000 feet. If consciousness is lost a rapid drop to the lower altitudes is necessary, sometimes resulting in perforation of the tympanic membranes. The occurrence of pulmonary or central nervous system symptoms always causes concern

because of the extreme altitudes, but the rapid increase of barometric pressure promptly relieves the distress.

Treatment

The treatment of decompression illness is simple and effective. All that is required is to increase the ambient pressure above 282 mm (25,000 feet). With descent of 5,000 to 6,000 feet below the altitudes at which the symptoms occurred, the severity of the syndrome begins to decrease. At 25,000 feet marked relief is obtained, although the bone and joint pains may not disappear entirely until the pressure is raised above 350 mm (20,000 feet), and in some cases the pain may persist for a short time at sea level.

Prophylaxis

Preventive measures for decompression illness are needed only for those personnel who ascend to 30,000 feet or higher in aircraft. The basic principle of most preventive measures is to remove nitrogen from the tissues prior to the arrival at these extremely high altitudes. This removal can be accomplished at sea level or during ascent by breathing pure oxygen. The substitution of pure oxygen for air eliminates nitrogen from the inspired air and, under these conditions, the nitrogen tension in the alveoli rapidly approaches negligible quantities. Helium-oxygen mixtures have been used in compressed air work because oxygen becomes toxic at partial pressures above 1½ atmospheres. In aviation we deal with oxygen tensions less than one atmosphere so that this mixture has no advantage over pure oxygen. Pure oxygen breathed at pressures of 760 mm or below is not toxic for periods up to 17 hours. The reduced alveolar nitrogen tension allows the nitrogen carried in the blood to diffuse through the alveolar membrane into the alveoli and thus to be expired. The blood is cleared of all nitrogen in excess of its normal carrying capacity until the nitrogen tension in the blood equals that of the nitrogen in the alveoli. This reduced tension of nitrogen in the blood allows more of the tissue nitrogen to pass into the blood and in this manner denitrogenation of the tissues can occur. This rate of elimination of nitrogen is shown in figure 2.

To insure the elimination of all the symptoms of decompression illness, the amount of nitrogen to be removed must be large enough to prevent the accumulation of any bubble of nitrogen in the tissues of sufficient size to cause the symptoms of decompression illness. This amount of nitrogen varies with individuals, dependent of course upon circulation, nitrogen stores and physical fitness.

The construction of aircraft with pressure cabins for operations at altitudes above 30,000 feet will obviate denitrogenation since these cabins can

maintain a pressure sufficient to prevent the appearance of decompression illness. However, the effects of rapid decompression due to abrupt failure of the pressure equipment must be borne in mind. In explosive decompression of this sort, anoxia is the more important problem.

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Further Impressions of American Medicine

EWAN F. B. CADMAN AND MATTHEW A. OHEA[1]

Since our first impact with American Medicine, a relatively few months ago, we have constantly been asking ourselves: Can we be expected in so short a time to know the glowing milestones of its past, its hopes and fears for the future, and the almost feverish activities and far-flung interests which characterize its present? A formidable query indeed, before which more agile and penetrating minds than ours would falter and stumble, daunted by its magnitude and confused by its unending ramifications. Yet the question must be answered, for on that depends the success or failure of our brief mission here. For in the words of Pater, what we have to do "is to be forever curiously testing new opinions and courting new impressions."

The transition from pre-clinical work to medicine proper is essentially a change from booklore to personal observation. So, just as the former frequently "fills the mind with pictures, often exaggerated, often distorted, often blurred, and, even when well drawn, injurious to the freshness of first impressions," our evaluation of America should be experiential, not merely representing a change, however welcome, in the locus of our scholastic activities. Casting aside therefore, feelings which would immobilize us, we shall proceed to record those features which are upmost in our minds as we prepare to return to "those that sent us."

In summing up one's impressions of American Medical Education, one is rather tempted to short cut an analysis of the process itself and to take the finished product, namely the graduate, as the criterion of its success. In spite of the convenience of such an approach, its naivety divests it of any critical value.

The final years in both countries are similar in that they are devoted primarily to the study of clinical medicine. The amount of clinical material is generally speaking more plentiful in England but often, because of the larger size of the medical schools, the number of patients per capita of students is less than in the United States. One often hears in English teaching hospitals the expression "the commonest things are commonest" as an aid to diagnosis but this is certainly not true in most of the American University Hospitals. To quote a few remarks from the Report of the

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[1] Mr. Cadman and Mr. O'Hea, British medical students, attended Washington University School of Medicine under a fellowship of the Rockefeller Foundation. They completed all requirements in December, 1942. Their first impressions of American medicine were published in the January, 1942 number of the Quarterly.

Commission on Medical Education, the British system is "primarily practical and the aim is to produce a safe, competent general practitioner." The report goes on to say that very little effort is made in most of the British Schools to stimulate students in investigative work or to arouse their interest in the unsolved problems of medicine. There is a tendency to simplify the instruction and to emphasize features of particular importance in practice. The American education differs from the British in the above respects so that the interneship becomes more of an integral part of the training of the general practitioner. Both of us were fortunate enough to experience a short interneship and to confirm the above statements for ourselves.

Pediatrics as a specialty is one of the most outstanding features of American Medicine. It is usually stated that the infant mortality rate of a country is an index of its degree of civilization; in this therefore America can certainly count herself among the most highly developed countries of the world. Until comparatively recently Chairs in Pediatrics at British Universities have been almost nonexistent. The general public is not educated as to the desirability or otherwise of pediatricians and one of us was recently informed that a certain large English city would probably be unable to support a full-time pediatrician in private practice!

In a world at war, it may well be out of place, and indeed in some cases wistfully amusing, to mention opportunity as a feature of medicine anywhere. But it is such a striking part of American Medicine, one might even say the aim, that it demands special consideration. The young American medical man, his training completed, has before him longer vistas and broader avenues than his peers anywhere else in the world. The high degree of specialization is a manifestation of this opportunity. However, this limitation of one's labours has the tendency to permeate into the education of the student and interne; one commonly hears the suggestion that the haematologist or urologist be "called in." This is apt to lead to narrow-mindedness and the latter is only partially neutralized by the advantages of "picking the brains" of one who devotes the majority of his time to a relatively limited specialty. One is often pleaded with to view the patient as a whole but this becomes a little difficult when he is pigeon-holed under a multitude of specialties.

A glance through the pages of the Quarterly Cumulative Index Medicus will reveal the myriads of journals and periodicals published by the medical press of America. Without even looking up a single reference, one can be sure that this huge number of publications is merely an indication of the immense amount of research which is turned out on the production-lines of American medical research centres. The ultimate reason for such an

output is the extremely happy financial state in which these centres exist, as compared with those of the rest of the world.

The final analysis of any system must show its practical value. In the medical field this has to be assessed in terms of service to the people—preventative and therapeutic. If it can be said that medicine, in the United States reaches the people, then its existence is at once justified. To what extent this is so can be roughly judged by vital statistics, which compare more than favourably with those of any other country, with the possible exception of the pre-war small Scandinavian countries.

The public education in things medical of the American layman is vastly superior to that of the British man-in-the-street. The problem of which came first, the chicken or the egg has yet to be solved, but it is obvious that public education here followed the tremendous advances made by the profession in the early years of this century. It is a common political belief that to educate a people is to make them dissatisfied and hence a "vicious cycle" has developed which has helped in elevating American Medicine to its present heights. This process of elevation however, had the disadvantage that it made medicine very expensive to the general public. Indeed, it has been heard said by a prominent American physician that it is impossible to make medicine cheap because of its present-day nature and ramifications. No such scheme as the British National Health Insurance is at present in operation but this is somewhat offset by the pre-payment plan, which however fails to provide for some of the most deserving sections of the community.

On the eve of our departure from the United States, we find its medical men hard at work—in war, in practice and in the schools and laboratories. At this point in their progress, they may have been deflected from their purpose by external events, but not before they reached one of the pinnacles of achievement in any science-efficient modernity.

Finally, in a plea for further international exchange of things medical, might we be permitted to quote from the will of Cecil John Rhodes: "I desire to encourage and foster an appreciation of the advantages which will result from the union of the English-speaking peoples throughout the world and to encourage in the students of the United States of America an attachment to the country from which they have sprung without withdrawing them or their sympathies from the land of their adoption or birth."

BUY WAR BONDS AND STAMPS
THEN CONTRIBUTE TO THE ALUMNI FUND

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 21

PRESENTATION OF CASE

A 67 year old unemployed white man was admitted to Barnes Hospital on February 17 and died March 1, 1943.

Chief Complaints.—Nausea, vomiting, abdominal pain; weakness, pain in chest.

Family History.—The patient believed that his father had died from cancer. One brother had had tuberculosis. One son had tuberculosis.

Social History.—The patient lived in St. Louis most of his life. After a brief schooling he went to work at the age of 13 and his occupations in sequence were: factory worker, printer, butcher, teamster, and trucker's assistant. He had always been in moderate financial circumstances but had had an adequate diet. He used alcohol and tobacco in moderation.

Past History.—General health had been good and the patient had always worked hard. At the age of 24 he became seriously ill with "typhoid-pneumonia." He was incapacitated for 17 weeks and was told at the time that he had typhoid ulcers. Following this illness he was advised to seek employment out of doors because of the pneumonia which had left him weak and markedly underweight. There had been many attacks of tonsillitis which on one occasion was followed by an abscess of the throat. In January, 1940, he was thrown from a rapidly moving truck and seriously injured. Three vertebrae, 3 ribs, and his left leg were fractured.

Systemic History.—Hearing had been poor since the accident 3 years previously. For some years he had had nocturia, frequency and urgency of urination.

Present Illness.—Four years previous to admission the patient began to have a burning sensation in his upper mid-abdomen. Soon this became associated with a feeling of fullness and some pain of a cramping character. There was no definite history that this pain was related to meals. Belching would relieve it temporarily. Within a few months, the patient began to vomit, sometimes during the day, usually in the night. At such times, food eaten at the previous meal would be recognized. A physician, after having

examined him radiographically made a diagnosis of ulcer, gave him medicine, and placed him on a diet. No relief was secured during the following 2 months. The patient took a patent medicine which was effective for some weeks, but the symptoms returned thereafter. Another physician then treated him with injections into his hip every day for 25 days during which time the symptoms disappeared, only to return 2 weeks later. His symptoms were fairly continuous, and a few months before admission, constipation, which had not been present previously, became marked. Belching of gas gradually became more pronounced, and the amount of food vomited increased in volume. This vomitus in the first portion had a very offensive odor and consisted of a thick material. This was followed by recognizable food particles. For a year previous to admission there had been an increasing loss of weight which totaled about 30 pounds since the onset of his illness. There had, likewise, been increasing weakness.

About 6 weeks previous to admission, the patient began to complain of tingling of his fingers on exertion. If he walked but a block this symptom would occur; if he went further, a dull ache occurred in his arms, spread across his chest and up into his throat. There was, also, some pain in his legs on these occasions. Continued exertion would induce shortness of breath, dizziness and faintness, which disappeared promptly on resting.

Physical Examination.—Temperature, 37.6°, pulse 78, respiration 20, blood pressure 140/80. The patient appeared chronically ill with some undernourishment and a marked pallor of the skin. Examination of the head and neck was negative. Expansion of the chest was small and there was hyperresonance throughout the lung fields. There were basal rales which did not increase on coughing. The heart was somewhat enlarged to the left with a forceful, apical impulse. There was a soft systolic murmur over the base (one observer located this over the apex). The abdomen was soft and no abnormal organs or masses were felt. There was no tenderness or resistance. Rectal examination was negative. A neurological examination revealed vibration sense to be absent below the knees.

Laboratory Examination.—Blood count—red blood cells 2,860,000; hemoglobin 3.2 grams; white blood cells 6,450; differential count: eosinophiles 1%; basophiles 1%; juvenile forms 5%; stab forms 22%; segmented forms 40%; lymphocytes 29%; monocytes 2%; size and shape of red blood cells not unusual. Urinalysis—sp. gr. 1.022; acid; albumin—negative; sugar—negative; microscopic—negative. Stool—Appearance not unusual; microscopic—no ova or parasites; guaiac test faintly positive. Kahn negative. Blood chemistry—non-protein nitrogen 26 mg%; total proteins—5.5 grams %; albumin 3.3 gms. %; globulin 2.2 gms. %; icterus index 6. Gastric Analysis—550 cc. of thick yellowish-white material with a foul odor was

obtained on lavage. Free acid was present. Roentgenographic examination of the gastro-intestinal tract—Gastric motor insufficiency (2°) organic, due to prepyloric carcinoma. There was a gross mutilating filling defect of the prepyloric antrum. The duodenum was not visualized because of an inadequate clearance. Colonic motor delay (2°), redundency (2°) distal. Appendix was not visualized. Diagnosis—carcinoma of stomach, prepyloric obstruction, gastrectasia (1+). Electrocardiogram—On the basis of low voltage in lead I, slurring in all leads, and irregularity, an interpretation of myocardial damage and auricular fibrillation was made.

Course in Hospital.—The patient received daily gastric lavage with a recovery of decreasing amounts of stomach content until the fasting residue was 55 cc. This contained no free acid and 10° combined acid before histamine, and 20° free and 25° combined acid after histamine. A hematologic consultation revealed a hypochromic, microcytic anemia, characteristic of prolonged bleeding. A surgical consultation revealed a palpable mass to the right of the midline in the epigastrium. On the fifth hospital day the temperature began to rise, and 48 hours later attained 40.1°. The fever was attributed to a urinary tract infection, since the urine had shown many pus cells, and had yielded cultures of E. coli and non-hemolytic streptococci. The white blood count at this time was 17,100. A genito-urinary consultation indicated that the prostate gland was enlarged to twice its normal size, was adenomatous, and slightly boggy. Cystitis was present and pyelitis was suspected. Sulfathiazole was administered until the blood level was 7.3 mg% and 78 mg% of free sulfathiazole was present in the urine. Transfusions were given. The red blood count reached 3,200,000 with 6.1 grams of hemoglobin. The white blood cells remained elevated at 27,400. Under chemotherapy the temperature gradually fell during the following week. However, the non-protein nitrogen rose to a level of 77 mg% two days before death. The abdomen at times was somewhat distended and tympanitic. Stool examinations continued to show small amounts of blood and on one occasion Endolimax nana cysts were found. During the last 3 days of life the patient developed hiccough which could not be controlled. He became restless, somewhat disoriented, and then obtunded. Terminally, he developed Kussmaul breathing, and was cyanotic. The pulse became gradually imperceptible and he died quietly.

CLINICAL DISCUSSION

Dr. Harry Alexander: This patient, evidently had a large prepyloric lesion of the stomach, diagnosed as carcinoma. The symptoms, dating back four years from his death, were continuous. They were qualitatively about the same; pain and vomiting. However, they did increase in intensity dur-

ing the several months just previous to his death. If we accept this diagnosis of carcinoma of the stomach, we must inquire whether these symptoms, continuous for four years, were caused by the carcinoma, or were the result of some antecedent lesion or functional disturbance. Dr. Larimore, may carcinoma of the stomach exist with continuous symptoms for four years?

DR. JOSEPH W. LARIMORE: I think the course may continue for that length of time.

DR. ALEXANDER: Do you feel that we need not infer that there was any other lesion then? May we expect, when the organs are shown us, to find only the carcinoma?

DR. LARIMORE: We should of course consider everything we can think of. From my contact with the case, however, I should say that it was a highly characteristic infiltrative lesion, which in all probability was carcinoma.

DR. ALEXANDER: I would like to inquire whether there could have been a pre-existing gastritis or ulcer?

DR. LARIMORE: There could have been, but I have seen no reason to suspect that there was.

DR. HAROLD SCHEFF: I think carcinoma of the stomach continuing for a number of years is rare. I would suggest duodenal ulcer as a possible antecedent lesion—it is one of the commonest causes of pyloric obstruction; or the man might have had a gastritic condition of the antrum.

DR. ALEXANDER: In other words this ulcer might have come about four years before death. He would have been about 63 then. Is that not rather late for an ulcer to develop?

DR. SCHEFF: It is late, but not impossible.

DR. ALEXANDER: Do you believe the carcinoma developed independently of the duodenal ulcer? Would a gastric ulcer be more likely than a duodenal ulcer?

DR. SCHEFF: I don't think the ulcer would have had any relation to the gastric carcinoma.

DR. ALEXANDER: You would support the attitude that gastric ulcer is not relevant to gastric carcinoma. Dr. Horner, do you agree?

DR. JOHN L. HORNER: Yes, I do. This patient may well have had some other lesion, since after four years the mass in the stomach was so small as to be almost questionable. There must have been bleeding for a long time, and one would certainly expect the growth to be much larger. Therefore, it seems unlikely that this man had had a carcinoma for four years.

DR. ALEXANDER: The point about ulcer is important. Not so many years ago it was presumed by many who were authorities that all cases of gastric

ulcer were potential cases of gastric carcinoma. Now there has been a reversal of this opinion—so much so that Walter Palmer, who is an expert, emphatically states that no case of gastric ulcer develops into gastric carcinoma. The consensus today, if not quite as arbitrary as that, certainly leans toward this point of view.

DR. HORNER: Not everyone would agree with Palmer, but that is the general opinion: a benign gastric ulcer does not become malignant.

DR. ALEXANDER: In gastric carcinoma, Dr. Larimore, is a preceding atrophic gastritis usually presupposed?

DR. LARIMORE: I see no reason to presuppose that. Certainly it isn't characteristic of atrophic gastritis to form a carcinoma.

DR. ALEXANDER: Let us put it the other way. In cases of carcinoma does one commonly find gastritis?

DR. LARIMORE: Usually.

DR. ALEXANDER: Hypertrophic or atrophic?

DR. LARIMORE: I leave that to the pathologists.

DR. SCHEFF: I find carcinoma frequently associated with atrophic gastritis, certainly.

DR. LARIMORE: Does that hold for all cases of carcinoma?

DR. ALEXANDER: I do not know. I quote Ewing, who states rather emphatically, not only on his own evidence but on quoted testimony, that in carcinoma of the stomach there usually is a mucosal change and an atrophic gastritis.

DR. LARIMORE: Achlorhydric or secretive?

DR. SCHEFF: Usually achlorhydric.

DR. ALEXANDER: No one hesitates, then, to accept this diagnosis of carcinoma of the stomach. If this carcinoma has gone on for four years, what about the vomiting which usually occurred at night? Do we presuppose a pyloric obstruction on the basis of this, Dr. Horner?

DR. HORNER: It is only suggestive. If he had had pyloric obstruction four years ago, one would expect the obstruction to be complete before the present time.

DR. ALEXANDER: Does this length of time suggest any particular type of cancer of the stomach? What kind of cancer might be in keeping with this duration?

DR. HORNER: The more fibrotic type with infiltration of the walls, rather than adenomatous or polypoid. A slow-growing lesion is usually more fibrotic.

DR. SCHEFF: Do you mean scirrhous?

DR. HORNER: Yes.

DR. SCHEFF: A polypoid type that has undergone malignant degenera-

tion would explain this lesion. A scirrhous type of carcinoma in the antrum can go on for four years.

DR. ALEXANDER: Dr. Larimore, do you agree? Do some gastric cancers grow more slowly than others?

DR. LARIMORE: There is a variation in malignancy in all types. I rather suspect that the scirrhous type grows more slowly than the adenomatous type.

DR. ALEXANDER: What about the gelatinous type? Of course adenocarcinoma may occur at any age. Is there anything to the dictum that the larger the growth the fewer the metastases, or is that antiquated?

DR. SCHEFF: I think that is true.

DR. ALEXANDER: Then when this stomach is shown to us, we expect to see a large carcinoma, the type of which is not identified, at the prepyloric region. Now this patient had other manifestations. He had tingling of his fingers, pain in his chest radiating to the neck, and pains in the limbs on walking. These began some six weeks before death, and were relieved on rest. Dr. Massie, do you feel that these symptoms may be cardiac in origin?

DR. EDWARD MASSIE: I think in a 67 year old man, a history of pain on pressure in the chest with some extension to the throat is fairly typical of angina pectoris. The tingling is not typical. However, it might occur. One might wonder why this occurred six weeks prior to admission. Perhaps this man had coronary arteriosclerosis. With a normal red count his heart would be compensated, but with a red count of two million, I think his narrowed coronary arteries and the degree of oxygenation that could occur with the degree of anemia he had, would result in myocardial ischemia. Here is a man with coronary artery disease. With a normal red count he could get along—with a lowered red count we find coronary artery insufficiency. In the electrocardiogram there were Q waves present in CF-2 and CF-4. If we had had another electrocardiogram to confirm this I would speak with more certainty.

DR. ALEXANDER: Dr. Moore, is the blood count with these estimations of cell volume in keeping with a diagnosis of carcinoma of the stomach?

DR. CARL MOORE: All it suggests is that the man has been bleeding. Once in a while with carcinoma of the stomach there is a macrocytic anemia instead of microcytic, hypochromic anemia. Why this occurs is not too well known. It probably is related to the degree of infiltration in that portion of the stomach where the intrinsic factor is made.

DR. ALEXANDER: One may get a picture simulating pernicious anemia, quite the reverse of what one sees here. This man developed a urinary infection, which responded to sulfathiazole. It was acute, but his temperature did come down. The nonprotein nitrogen rose to 77 mgm. per cent, whereas

on admission it had been normal. He developed Kussmaul breathing. What are the factors that may have elevated his nonprotein nitrogen?

DR. WM. BARRY WOOD, JR.: You have to consider the infection of the urinary tract. He had been given a drug which occasionally affects the kidneys. He had been vomiting, and had probably been bleeding into the gastrointestinal tract. Each one of these factors would tend to cause a rise in nonprotein nitrogen.

DR. ALEXANDER: Are there any other comments?

DR. HORNER: It should be mentioned that there are other lesions of the stomach which can closely mimic carcinoma, particularly syphilis. However, the Kahn test was negative. About 400 cases of tuberculosis of the stomach have been described, including ulcerated cases and tuberculous infiltration from adjacent glands. It seems to me we cannot exclude these as possibilities.

DR. ALEXANDER: Is there any significance to the character of the vomitus? The first portion of it consisted of a foul-smelling material, and this was followed by foodstuffs.

DR. HORNER: It is suggestive that there is something pyogenic in the stomach. Phlegmonous gastritis superimposed on carcinoma might give that type of vomiting. A tuberculous process might give a secretion of that sort.

DR. ALEXANDER: Are there further suggestions?

DR. LARIMORE: I don't think you have to have anything but stagnation in the stomach to give that picture. The first portion of the vomitus was the fermented and stagnating material which rises to the top. Twenty years ago, when we couldn't X-ray the stomach, we would let the gastric contents stand for a time in order to diagnose the gastric lesion. This fermented material would rise to the top.

The only thing I have to say further about this case is that when we look at the X-ray picture of this lesion, we are looking at the end result. This has not been a static disease, but a progressive disease. It probably began as a lesion that interfered with the motility of the stomach. You need not go to gastritis, tuberculosis, or syphilis to explain this lesion. The man had not had the characteristic pain of duodenal or gastric ulcer. He had something interfering with gastric motility, which has been progressive.

DR. CARL MOORE: The question of free acid with carcinoma of the stomach is often discussed. About 35 per cent, according to a report I have here, do have free acid.

DR. ALEXANDER: Are there any more remarks?

DR. LARIMORE: The presence of free acid would be somewhat against there being an atrophic gastritis.

DR. SCHEFF: I disagree. A patchy type of gastritis may show minimal amounts of free acid.

CLINICAL DIAGNOSIS

Carcinoma of stomach

DR. ALEXANDER'S DIAGNOSIS

Carcinoma of stomach

ANATOMIC DIAGNOSIS

Carcinoma of the stomach with partial obstruction of pylorus
Metastatic carcinoma of peripancreatic lymph nodes
Atrophic gastritis
Arteriolar nephrosclerosis, moderate
Hypertrophy and dilatation of heart (600 grams)
Arteriosclerosis of coronary arteries, advanced
Recent infarct at apex of left ventricle
Benign hypertrophy of prostate gland
Hypertrophy and dilatation of bladder
Acute pyelonephritis, bilateral
Perinephric abscess, right

PATHOLOGIC DISCUSSION

DR. ROBERT MOORE: On the basis of anatomic changes found at autopsy we may say that this patient was a man both chronologically and anatomically old (as shown by atrophy of the skin and senile changes in the prostate), who had for some months or years before death a slight to moderate obstruction to the flow of urine from the bladder (supported by the finding of hypertrophy and diverticula of the bladder). On at least one occasion he had an attack of a mild urinary infection, which left small fibrous scars of a pyelonephritis in both kidneys. During the days or weeks immediately before death there was an exacerbation of this urinary infection, with the formation of numerous small abscesses in the kidneys and rupture of one of these into the right perinephric tissues.

In his stomach he had had for some years one of the lesions frequently found in older people: atrophic gastritis. Some months ago a carcinoma appeared in the mucosa of the pyloric part of the stomach. The presence of this tumor interfered with the ingestion of food to a moderate degree, and emaciation and slight serous atrophy of the fat were the result. Inasmuch as the carcinoma invaded the muscularis of the stomach but did not ulcerate through this layer, we may conclude that he did not have an ulcer in which a carcinoma started, but rather a carcinoma with secondary ulceration.

For some years he had hypertension. This statement is based upon the finding of a moderate degree of hypertrophy of the heart without demonstrable cause, except for arteriolosclerosis in the viscera. He did not, however, have any significant persistent cardiac failure during this period of time.

Two days before death the flow of blood through the coronary arteries to a part of the heart became inadequate and an infarct formed. At this same time or a few days before, the heart began to fail, and finally bacteria invaded the congested lung and a bronchopneumonia was the terminal event.

DR. ALEXANDER: Dr. Moore, you showed a slide of another case, wherein it was your feeling that the carcinoma was based on an ulcer. What is the statistical incidence of such an event?

DR. ROBERT MOORE: A few per cent.

DR. MASSIE: This man had 1500 cc. of fluid in his pleural cavity. Do you feel that he did have passive congestion?

DR. ROBERT MOORE: He had a greater degree of cardiac failure than one ordinarily sees in acute coronary occlusion.

DR. ALEXANDER: One more question, which perhaps cannot be answered. Is it your feeling that this carcinoma may have existed for four years?

DR. ROBERT MOORE: It is possible, but if so, it is a rare example.

DR. ALEXANDER: What type of carcinoma is this?

DR. ROBERT MOORE: Adenocarcinoma. It would be better for a gastroenterologist to answer the question of whether this man's symptoms during four years could be accounted for by atrophic gastritis.

DR. SCHEFF: I think that they could.

CASE 22

PRESENTATION OF CASE

G. S., a 57 year old minister, was first admitted to Barnes Hospital on the Surgical Service, August 25, and discharged August 31, 1935.

Chief Complaint.—Hemorrhoids.

Family History.—Entirely irrelevant.

Social History—The patient was born in a small town in Missouri where he lived until he went to college in Chicago. There he studied for the ministry. He was married soon after this and for the rest of his life was a pastor in several small town churches. He had 4 children and, although financial circumstances were always strained, there was no indication of insufficient diet.

Past History.—As a child he escaped all usual contagious diseases but contracted several of these from his children. The most significant were small pox, scarlet fever and diphtheria. At 18 he was severely injured when

a cow stepped on his back and there was extravasation of urine from the wound for several weeks. For the last 25 years of his life there were occasional periodic attacks of upper abdominal pain which at one time was diagnosed as "gall stones."

Systemic History.—The patient had a chronic, persistent cough for many years, which was not productive. He attributed this to excessive pipe smoking. While in the Army in 1918 he was operated upon for hemorrhoids. These returned in 1925 and for the year previous to admission they increased in size and there was profuse bleeding on several occasions. During this period there had been increasing constipation.

Physical examination.—Temperature 97.6°, pulse 78, respiration 20, blood pressure 110/85. The patient was well nourished and developed. The eyes were normal. Respiratory tract was normal. There was a soft systolic murmur at the apex of the heart which was otherwise negative. The abdomen showed no abnormalities. The prostate gland was moderately enlarged and slightly tender. On proctoscopic examination large hemorrhoids appeared.

Laboratory Examination.—Urinalysis and blood count were within normal limits and the blood Kahn test was negative.

Course in Hospital.—Under sacral anesthesia, several large hemorrhoids were removed. Postoperative course was uneventful. Patient was discharged on the sixth day.

Second Hospital Admission.—October 1 to October 7, 1935, on the Medical Service.

Interval History.—Shortly after his previous admission the patient developed a constant and dull aching pain involving the upper portion of the abdomen. It occurred about one hour following meals. This pain had recurred frequently. It was never very intense and was not associated with nausea, vomiting or eructation. The pain seemed to be influenced by posture. It would appear when bending over or assuming certain positions. Ever since 1916 when the patient developed his back injury there had been some pain in the lower back on undue exertion. Five weeks previous to admission this pain became more pronounced and was constant. There had been loss of weight of 10 pounds since last admission, for which the patient was unable to account.

Physical Examination.—Temperature 37.4°, pulse 80, respiration 18, blood pressure 135/90. Although the patient was well nourished, there were indications of weight loss. Other abnormal findings, since previous examination, included impaired vision in the left eye; marked tenderness on pressure over the lumbo-sacral articulation on both sides; some tenderness over the cervical spine. Bending backwards caused pain in the abdomen.

Laboratory Findings.—Red blood cells 5,940,000; hemoglobin 80%; white blood cells 7,050; differential—stab forms 8%; segmented forms 59%; lymphocytes 30%; monocytes 3%. Gastric Analysis—Fasting content showed 20° free HCl and 20° combined acid. Stool—Guaiac negative. Blood Kahn negative. Basal Metabolic Rate— – 1%. Electrocardiogram—T waves upright; leads 1 and 2 had low voltage; left axis deviation. These findings were interpreted as myocardial changes. Uranalysis—Sp. Gr. 1.010; sugar—negative; albumin—negative; microscopic—negative. Roentgenogram of the lumbar spine showed a moderate amount of hypertrophic spurring of the anterior, superior and inferior aspects of the bodies of the lumbar vertebrae. Diagnosis was hypertrophic osteoarthritis of the lumbar spine.

Course in Hospital.—On orthopedic consultation no new findings were revealed and a diagnosis of hypertrophic osteoarthritis of the spine and lumbo-sacral strain was made. Symptomatic treatment was advised and the patient was discharged.

Third Hospital Admission.—Patient was admitted on November 10 to Surgical Service and was discharged November 23, 1938.

Chief Complaint.—Difficulty in urination.

Interval History.—About 2 years previous to this admission the patient first noticed an increased frequency of urination, especially at night. For six months there had been increasing difficulty in voiding, and control was somewhat impaired.

Physical Examination.—Temperature 37°, pulse 82, respiration 18, blood pressure 145/95. The only change from previous admission was found in the prostate gland which was diffusely enlarged, firm, and very hard. Three hundred cc of residual urine were obtained on cathertization.

Laboratory Findings.—Blood count—red blood cells 4,200,000; hemoglobin 80%; white blood cells 8,000. No differential count was recorded. Urinalysis—Sp. Gr. 1.012; albumin—negative; sugar—trace; miscroscopic—few red blood cells and white blood cells. Culture—staphylococci. Blood Kahn negative, non-protein nitrogen—29 mg%.

Course in Hospital.—Under spinal anesthesia a transurethral resection of the prostate was performed and some 15 to 20 pieces removed. These, on microscopic study, showed glandular hyperplasia, fibrous tissue and evidences of old chronic inflammatory changes. No malignant cells were reported. The patient made an uneventful recovery.

Fourth Hospital Admission.—Patient reentered on the Surgical Service on November 14 and was discharged November 24, 1939.

Chief Complaints.—Frequency and burning on urination, urinary incontinence.

Interval History.—For 3 months, following the prostatic operation, the patient had felt entirely well. Then recurrence of the symptoms of the previous admission began and these increased gradually. In addition there was some incontinence of urine.

Physical Examination.—Temperature 97.2°, respiration 18, pulse 90, blood pressure 150/100. The only change from previous examination was that the systolic murmur heard previously was no longer present. The blood pressure, as noted, had risen to 150/100. The liver had enlarged and the edge was felt midway between the right costal border and the level of the umbilicus. The prostate was but slightly enlarged, very firm and no nodules were felt. Residual urine was 400 cc. On cystoscopic examination a moderate amount of coarse trabeculation in the bladder was seen. A collar type of obstruction in the posterior urethra, and some prostate hypertrophy were observed.

Laboratory Findings.—There was essentially no change from previous admission with the exception that culture from the bladder urine revealed E. coli.

Course in Hospital.—Under spinal anesthesia transurethral resection was done. The prostatic tissue removed on miscropic examination revealed no definite lesion. Convalescence was uneventful.

Fifth Hospital Admission.—The patient entered the Medical Service on February 22 and died March 15, 1943.

Chief Complaints.—Pain in the muscles and joints; swelling of the neck; loss of appetite, nausea and vomiting; weight loss.

Interval History.—In April, 1941, while hanging wallpaper, the patient fell and was severely bruised. The local physician found no broken bones. Generalized soreness of the muscles and deep pain in various bones persisted. He was treated with liniment and pills. There was no improvement for 2 months and the patient entered Jefferson Barracks Hospital, where he received many types of study and treatment. He remained one month and was definitely improved. However, in October, 1942, the symptoms increased to such an extent that his sleep was frequently interrupted because of continued soreness of his muscles, and pain in several joints. At no time was there any local swelling, redness or limitation of motion. A few months previous to this a small painless swelling had developed in the neck above the clavicle on the left side. This increased steadily in size until admission. During the autumn of 1942, the patient began to lose his appetite and had nausea and vomiting. He was treated for liver trouble without improvement. Five weeks previous to admission he began to complain of pain in the stomach after eating and vomiting shortly after meals became frequent. Character of the vomitus was not stated. His appetite

which had been poor, failed rapidly and 3 weeks previous to admission pain in his arms, chest, back and legs and feet became much worse and he was unable to rest. About this time a loss of sensation in his right hand developed. The patient became more irritable and his wife noticed personality changes. For a week before admission the patient took but little food, because of his fear of vomiting. There had been a weight loss of some 30 pounds during the last 5 weeks.

Physical Examination.—Temperature, 37.8°, respiration 20, pulse 80, blood pressure 140/95. The patient did not appear acutely ill; was alert and cooperative. A port wine discoloration over the chin, neck and part of the upper chest was noted. Tortuosity of the arteries was observed in the retinae and disc outlines were not sharp. The ears were negative. The pharynx was slightly injected. A large, firm, nodular, slightly tender mass about the size of a lime was present just above the left clavicle. There was questionable dullness on percussion over the left upper lobe. The heart was not enlarged. The rhythm was regular and sounds of good quality. No murmurs were heard. There was some tenderness on deep palpation of the upper abdomen. A sharp liver edge was felt 12 cm. below the right costal margin in the midclavicular line. The spleen was questionably palpable. The prostate was not enlarged but was very hard. Motion of the spine was unimpaired. Neurological examination was negative.

Laboratory Findings.—Blood count—red blood cells 4,990,000; hemoglobin—11 grams; white blood cells—11,000; differential count—basophiles 1 %; eosinophiles 3%; stab forms 5%; segmented forms 51%; lymphocytes 24%; monocytes 6%. Urinalysis—Sp. Gr. 1.022; albumin—negative; sugar—negative; microscopic—20 to 25 white blood cells per high power field. Stool—guaiac test slightly positive. Blood chemistry—non-protein nitrogen 31 mg%; total protein 5.8 grams %, albumin 3.4 grams %; globulin 2.4 grams %; calcium 9.9 mg%; phosphorus 3.7 mg%; phosphatase 4 Bodansky units; icteric index 6; cephalin flocculation test 24; Kahn negative. Gastric analysis—no free acid; 3° combined acid; 3 minutes after histamine 9° free, 29° combined acid, guaiac negative. Roentgenograms—The gastro-intestinal tract showed colonic motor delay. Interpretation was indeterminate. Chest film showed a large amount of peribronchial infiltration extending out from the hilar shadow in a fan-shaped fashion. Diagnosis—peribronchial infiltration and early cardiac decompensation. Films of the paranasal sinuses were clear. Open film of the urinary tract was partially obscured by considerable gas. No abnormalities were noted.

Course in Hospital.—Twenty-four hours after admission the patient suddenly became irrational and disoriented, was unable to obey commands, and talked irrelevantly. Neurological consultation revealed a slight weak-

ness of the right lower face. Lumbar puncture was done—initial pressure was 160 mm. which rose to 250 mm. on compression of each jugular. Spinal fluid was clear and there were no cells; Pandy test—negative, proteins 25 mg%, Wassermann negative, colloidal gold curve—0000000000. Subsequent urine examinations showed increased numbers of white blood cells. On culture E. coli was recovered. There was constant slight elevation in temperature. The patient was placed on sulfamerizine which attained a level of 4.0 mgm per cent in the blood. On consultation, no lesion of the ears, nose or throat was discerned. There was little change in the patient's condition other than generalized, increasing weakness, because of inability to take more than small amounts of food. A biopsy of the mass in the neck was made. This was discovered to be composed of many discrete lymph nodes. Microscopic section revealed a good deal of necrosis with an island of malignant epithelial cells forming poorly differentiated glands embedded in fibrous tissue. Nine days previous to death a complete right sided hemiplegia suddenly developed and persisted. Four days before death many moist rales appeared at the bases of the lungs. An electrocardiogram taken at this time was interpreted as possible posterior myocardial infarction of recent origin. Digitalis therapy was begun. The white blood count on that day was 18,300. The patient's course thereafter was rapidly downhill, with increasing moisture in the lungs, rapid heart beat and failing pulse volume.

CLINICAL DISCUSSION

Dr. Harry Alexander: This patient had a biopsy taken from a lymph node on the left side of the neck, and the diagnosis of adenocarcinoma was made. The first problem, therefore, is to attempt to localize the organ in which the primary carcinoma occurred, because evidently this was a metastasis. Localization can only be done by exclusion and inference. The man's head was examined by an otolaryngologist, who found nothing significant. After roentgenological examination of his lungs it was assumed that he did not have carcinoma of the lungs. The lesion, then, is presumed to have been below the diaphragm. Where may it have originated? Dr. Wood, have you any suggestions?

Dr. Wm. Barry Wood, Jr.: I would suggest the stomach, in spite of the negative gastrointestinal series.

Dr. Alexander: What inclines you to that opinion?

Dr. Wood: The patient had a great many gastrointestinal symptoms, starting with dull pain in the upper abdomen, followed later by persistent vomiting. He had a Virchow's node on the left side, which is very common in carcinoma of the stomach. Furthermore, adenocarcinoma is a common form of neoplasm of the stomach.

DR. ALEXANDER: This Virchow's node was first discovered in August, 1942. His gastrointestinal symptoms began five weeks before admission. Is it your feeling that the metastasis could have been dated August, 1942?

DR. WOOD: He must have had the carcinoma for a long time.

DR. ALEXANDER: Dr. Moore, what is the incidence of correct diagnosis of carcinoma of the stomach by gastrointestinal X-ray?

DR. SHERWOOD MOORE: It is quite accurate. I think it would be safe to say 60 per cent.

DR. HAROLD SCHEFF: It is about 90 percent accurate according to quoted reports.

DR. ALEXANDER: Do you think this man is likely to have had carcinoma of the stomach?

DR. SCHEFF: I do.

DR. ALEXANDER: Are there any other suggestions?

DR. SHERWOOD MOORE: The prostatic history is suggestive.

DR. ALEXANDER: True, there is a history of hyperplasia, which is often a forerunner of carcinoma of the prostate. Is anything else in favor of this diagnosis?

DR. EDWARD MASSIE: The pain in the bones might indicate metastases to the bones, which are common with prostatic carcinoma.

DR. SHERWOOD MOORE: That is why I wanted to show those radiographs of the lumbar vertebrae. We should have had more examination of the skeleton.

DR. ALEXANDER: Virchow's nodes do occur, not infrequently, in carcinoma of the prostate. In one series as high as 6 per cent is reported. They occur also not uncommonly in carcinoma of the uterus and testis. Here we find a history of prostatic hypertrophy, which is presumed to be a forerunner of carcinoma of the prostate, and we find metastases in Virchow's node, which is also in keeping. It is of some interest that the node was described as having a great deal of fibrous tissue, with some acinic and glandular cells—which fits in, too.

DR. WOOD: What do you think the original lesion in the prostate was? From the description it sounded more like chronic prostatitis than benign hypertrophy.

DR. ALEXANDER: Is not the glandular hyperplasia part of hypertrophy of the prostate?

DR. WOOD: But are fibrous changes of that kind observed?

DR. ALEXANDER: Perhaps not that early. Certainly, these changes may occur. I think they may exist for years, but I will defer this question to Dr. Moore. Another point, that occurs to me, is that this man had only

four Bodansky units of phosphatase in the blood. Dr. MacFarlane, how do you interpret this?

DR. WAYNE MACFARLANE: Carcinoma of the prostate may occur with normal phosphatase, or the phosphatase may be elevated. This finding would not rule out carcinoma of the prostate.

DR. ALEXANDER: May adenocarcinoma occur in any organ other than the stomach and prostate to produce these symptoms?

DR. LLEWELLYN SALE: What about hepatoma—adenocarcinoma of the liver?

DR. ALEXANDER: That is a possibility. One thing against it might be that those tumors do not readily metastasize.

DR. SALE: They do invade the vessels.

DR. ALEXANDER: They may invade the blood vessels of the liver or surrounding vessels, but remote metastases are uncommon—about 20 per cent. However, it might have occurred here. What other organs must be considered?

DR. MASSIE: What about the possibility of explaining some of the symptoms on the basis of cerebral metastatic lesions?

DR. ALEXANDER: You think he might have had a primary tumor in his head that went down the left cervical duct?

DR. MASSIE: I would like to suggest hypernephroma.

DR. ALEXANDER: Certainly that is an adenocarcinoma. Or to be safe you might call it adenocarcinoma of the clear cell type. That is a possibility, but with that tumor, metastases are very common. In this case there is no evidence of metastases other than those in the neck and in the liver.

DR. MASSIE: There was a question of an enlarged spleen, which might have been the kidney on the left side.

DR. ALEXANDER: He might have had an adenocarcinoma of the kidney of the usual granular cell type, or a hypernephroma. Metastasis to the lung might be expected in that case.

DR. SHERWOOD MOORE: I would like to suggest adenocarcinoma of the rectum.

DR. ALEXANDER: A good suggestion, for if a gastrointestinal X-ray series is taken, that is one type of carcinoma that may be missed, is it not? Carcinoma of the rectum or of the sigmoid colon could metastasize.

DR. EDWARD REINHARD: It may not have been mentioned, but proctoscopy was performed on this patient, and nothing was found.

DR. ALEXANDER: Then that rules out carcinoma of the rectum?

DR. SCHEFF: It rules out carcinoma of the rectum, and makes carcinoma of the sigmoid unlikely.

DR. CARL MOORE: I believe you could eliminate hypernephroma, because the cells of the metastases of hypernephroma are highly characteristic, and would have been noticed on biopsy.

DR. ALEXANDER: What other organs might have been the primary seat of an adenocarcinoma?

DR. SCHEFF: The small intestine?

DR. ALEXANDER: That is unlikely. Very unlikely.

DR. CARL HARFORD: Can you eliminate the bronchus entirely?

DR. SHERWOOD MOORE: I think so.

DR. LAUREN ACKERMAN: I think it should be mentioned that the tail of the pancreas may give rise to adenocarcinoma.

DR. ALEXANDER: That is true. In this case the carcinoma probably could not be in the head of the pancreas because there was no jaundice. It was brought out in a case we had some weeks ago that pain is a constant symptom in carcinoma of the pancreas, and this man had no such pain.

Of the possibilities suggested we may say, then, that carcinoma of the rectum is unlikely, hypernephroma is unlikely, carcinoma of the liver is less unlikely; leaving carcinoma of the stomach and of the prostate as the most probable. Is adenocarcinoma of the stomach a slow-growing or rapidly-growing tumor?

DR. SCHEFF: It is usually a rather rapidly-growing tumor.

DR. ALEXANDER: I think we shall have to leave it at that and go on to discuss other aspects of this case. Dr. Massie, you suggested that the cerebral lesions may have been metastases to the head. How would the tumor get there?

DR. MASSIE: It might have been blood-borne.

DR. ALEXANDER: That brings up the question as to whether tumor cells go through lung capillaries.

DR. MASSIE: I would hesitate to discuss that question.

DR. ALEXANDER: Dr. Sale, do epithelical cells, carcinoma cells, go through lung capillaries? In lymphosarcoma that is the method of spread but those are smaller cells. I am inclined to think that a blood-borne invasion of the brain through the lungs would be very rare. Clinically it is uncommon to see metastases to the brain from carcinoma of the stomach or prostate.

DR. SALE: Don't the vertebral veins offer a pathway for metastases or emboli?

DR. WOOD: When the vertebral veins are involved the metastases usually go from the venous side into the spine.

DR. BARRETT TAUSSIG: I believe that carcinoma of the lung not infrequently metastasizes to the brain. Could not there be a pathway from the

mediastinal lymph nodes or Virchow's node that would by-pass the lung and reach the brain?

DR. ALEXANDER: I do not see how there could be a pathway from Virchow's node without pulmonary involvement.

DR. TAUSSIG: It might invade the carotid artery and go up.

DR. ALEXANDER: Dr. Rioch, this man three weeks before admission began to notice a weakness in his right hand. On admission a slight weakness of the right side of his face was noticed, and nine or ten days after admission he developed hemiplegia. If these signs are all part of one process, it must have been a slowly-developing process. What lesions do you suggest?

DR. DAVID RIOCH: The numbness of the right hand and the metastasis of the lymph glands of the neck might suggest that the brachial plexus was involved.

DR. ALEXANDER: It was on the left side of the neck.

DR. RIOCH: There was probably more, or else it was a metastasis to the spine. The hemiplegia I regard as metastatic.

DR. ALEXANDER: This man presented some evidence of arterial disease: elevated blood pressure and possibly arteriosclerosis. Do you think there could have been a slowly developing cerebral thrombosis?

DR. RIOCH: He probably had more than one attack. One attack may have caused the facial weakness, and a terminal attack may have occurred sometime before death.

DR. ALEXANDER: Is there any other discussion about this point? Either this was a metastasis which reached the brain, or an arteriosclerosis giving rise to a slowly developing thrombosis.

Did this man finally develop signs of cardiac failure? The X-ray picture was interpreted as cardiac decompensation. Dr. Massie, what do you think we may find?

DR. MASSIE: It is likely that the patient had disease of the coronary arteries caused by arteriosclerotic changes. We may also find hypertrophy of the left ventricle. I do not think he had myocardial infarcts. If we had a record prior to the administration of digitalis we could better interpret the electrocardiographic changes. Rapid digitalization, such as this patient had, will sometimes produce such changes.

DR. ALEXANDER: This man was paralyzed. He had been in bed for weeks. Does something have to happen to cause such a man to die of myocardial failure in four days? Was he unconscious, Dr. Reinhard?

DR. REINHARD: I don't remember. He was obtunded, but I don't know whether he was comatose.

DR. ALEXANDER: Does decompensation occur in a man lying quietly in bed, without any accident?

DR. MASSIE: It is frequently seen in people who are in the terminal stage of another disease. Certainly cardiac failure is the mode of exitus of the majority of patients who die, regardless of cause.

DR. ALEXANDER: Are there other suggestions?

DR. LAUREN ACKERMAN: At the State Cancer Hospital we often play this game of trying to find out where the tumor came from. Several things in this case are interesting. In the first place it would have been of great value to have had an acid phosphatase test, for if it were elevated it could be stated that the bones were involved, which would point to carcinoma of the prostate. If you assume that the large liver is the result of metastasis rather than of congestive failure, carcinoma of the prostate is unlikely, for it seldom metastasizes to the liver. If the patient had had a hypernephroma one would have expected the pathologist to make the diagnosis, and if the cerebral lesion is a metastasis there would be large metastatic foci in the lungs. Although the film was interpreted as showing congestive failure, the changes in the lung might also be caused by lymphangitic metastases, which can contribute to heart failure. This can occur in both carcinoma of the stomach and of the prostate. I think in this case there is more evidence for the stomach as the primary seat of the cancer than for any other organ.

DR. ALEXANDER: I want to remind you that the liver was felt on his fourth admission in 1939. At that time the liver was enlarged, although not as much as on his last admission. Perhaps that may affect your opinion?

DR. ACKERMAN: Carcinoma of the prostate can metastasize to the liver, but carcinoma of the stomach does so more often. The trouble with a malignant neoplasm is that it never acts as you expect it to. On the basis of probabilities, carcinoma of the stomach is most likely here.

DR. ALEXANDER: We must adhere to clinical observations. You are not disturbed by the fact that this man had had a Virchow's node in August of the year before—six months before admission—and that his recent symptoms occurred only five weeks before admission, and that therefore the lesion of the stomach had been there for more than six months?

DR. ACKERMAN: Almost anything is possible with tumors. A small lesion in the stomach may be there over a considerable period of time. Mallory and Stout have described very early carcinomas of the stomach which remained localized for some time. We cannot always rely on Virchow's node as indicating carcinoma of the stomach. We have had many cases in which it was absent, with metastases elsewhere.

DR. ALEXANDER: I still think carcinoma of the prostate is very probable. According to clinical observations of this case, then, we may say that the

diagnosis lies between carcinoma of the stomach and carcinoma of the prostate.

DR. WOOD: Which of the two do you favor, Dr. Alexander?

DR. ALEXANDER: I was almost persuaded by Dr. Ackerman, but I do favor carcinoma of the prostate. I cannot account for this man's digestive symptoms being only five weeks old. The fact that the X-ray diagnosis is probably 90 per cent accurate leaves only a 10 per cent chance for carcinoma of the stomach, but it may exist.

DR. WOOD: Do you think the man had metastases in the brain?

DR. ALEXANDER: No. It would be extremely unusual clinically to have metastases in the brain and no lesion of the lung. It is much more likely that the brain lesion was a thrombus.

CLINICAL DIAGNOSIS

Carcinoma, site of primary undetermined

DR. ALEXANDER'S DIAGNOSIS

Carcinoma of the prostate
Cerebral arteriosclerosis

ANATOMIC DIAGNOSIS

Adenocarcinoma of prostate gland with extension to trigone of bladder (history of 2 transurethral resections for urinary obstruction)

Metastatic carcinoma in iliac, para-aortic, peripancreatic, portal hepatic mediastinal, and pulmonary lymph nodes and in lungs, and pleura.

Metastatic carcinoma of lumbar vertebrae with myelosclerosis

Vegetative endocarditis of aortic valve (Hemolytic Staphylococcus aureus)

Infarcts of kidneys and spleen

Healed infarct of interventricular septum

Arteriosclerosis of aorta and of the coronary arteries, advanced, and of pulmonary, renal, splenic and cerebral arteries, moderate.

Encephalomalacia in parietal lobes of brain

Hypertrophy and dilatation of the heart

Infarct in lower lobe of left lung.

PATHOLOGIC DISCUSSION

DR. ROBERT MOORE: Here is a 57 year old white man who sometime in the summer and fall of 1935 developed a disturbance of his cardiovascular renal function, that led to hypertension, demonstrated first in October 1935, but apparently not present in August 1935. In the intervening years between 1935 and 1943 there were changes in the tissues usually associated with hypertension—arteriosclerosis and hypertrophy of the heart, which

weighed 380 gm. at the time of autopsy. The body length was 168 cm. and the maximum normal weight of the heart for this body size is 330 gm. At the same time arteriosclerosis of the major arteries began, and at some period perhaps in the fall of 1935, when he complained of constant and dull pain in the upper abdomen, the blood supply to the interventricular septum was restricted and an infarct formed. By 1943 this was completely healed and we found no other lesion—cholelithiasis or healed peptic ulcer—to account for these symptoms.

Some time before 1938 a carcinoma of the prostate formed in the extreme posterior lobe. This caused some dysuria, but when the biopsy of the vesical neck was taken in November, 1938, no tumor was found, probably because it had not yet invaded this part of the prostate. I do not believe that this man at any time had benign hypertrophy because there is no evidence of it in the specimens removed by biopsy or in the autopsy specimen. The carcinoma continued to grow and there was again sufficient urinary obstruction to bring him into the hospital in November 1939. By 1941-42 the carcinoma had metastasized to the vertebrae and perhaps was responsible for some of the deep pain in the bones of which he complained. By the fall of 1942 it had metastasized to a supraclavicular node and probably to the lung.

During these same years he was developing an osteoarthritis of the spine and possibly of other joints.

The arteriosclerosis of the coronary and cerebral vessels was also progressing. There was increasing impairment of the blood supply to these organs and by the fall of 1942 there were what were described as "personality changes," and by the winter of 1942-43 some failure of the heart—supported by the finding of subchronic passive congestion of the lungs and liver. This passive congestion interfered with the flow of the blood in the veins of the extremities and thrombi formed. Some of these thrombi broke off and as emboli lodged in the pulmonary arteries, with resultant infarction in the left lower lobe, some time in January or February, 1943.

In February 1943 staphylococci entered the body, possibly by a pneumonic process about the infarct, possibly from the infection of the lower urinary tract. Organisms localized on a previously damaged aortic valve, and an acute vegetative endocarditis was established. Pieces of these vegetations were displaced and lodged in the branches of the arteries to the kidney and spleen, and infarction of small parts of these organs resulted.

Nine days before death either an embolus from the vegetation on the aortic valve, or an autochthonous thrombus in the sclerotic middle cerebral

artery occluded it and a large part of the left parietal lobe was deprived of blood supply.

The heart showed increasing failure and the man apparently died of cardiac failure, based on arteriosclerosis of the coronary arteries. During the terminal illness his state of nutrition was reasonably well maintained and the storage of fat and protein in the liver was adequate, but the carbohydrate reserves were somewhat depleted.

At the moment of death some selected enzyme systems in the liver were still active but there was presumably some damage because of the observation of an elevated acid and alkaline phosphatase and a depression of catalase and possibly of the QO_2.

It would have been of interest to know the state of the chemical constituents and enzyme systems in the cardiac muscle.

DR. ALEXANDER: Is there evidence that tumor cells may go through the pulmonary capillaries?

DR. ROBERT MOORE: It has been stated in the literature that it occurs. The statement is based on the old observation that the pulmonary capillaries are larger than the systemic capillaries. I think, however, that if one examines the problem carefully, comparing the incidence of metastasis of carcinoma of the bronchi with all other carcinomas, one finds no difference just as soon as the other carcinomas metastasize to the lung. There is metastasis to the lung first, and then metastasis from metastasis.

DR. ALEXANDER: Is this particular type of carcinoma different from the usual adenocarcinoma? And how about the statement frequently made that carcinoma of the prostate is preceded by prostatic hypertrophy?

DR. ROBERT MOORE: In answer to your first question I would say that it differs histologically, but not enough to justify any separate category clinically. With regard to your second question, there is a great deal of difference of opinion as to whether or not benign hypertrophy of the prostate becomes malignant, by the hyperplastic cells changing into malignant cells. In my opinion it is extremely rare, since when a carcinoma is found in a prostate in which there is also benign hypertrophy, both small, the hypertrophy is usually in the lateral lobes and the carcinoma in the posterior lobes.

DR. WOOD: I think you should be congratulated, Dr. Alexander, on your diagnosis. I would like to reiterate that the diagnosis would have been much easier for all of us if we had done an acid phosphatase test.

BUY WAR BONDS AND STAMPS
THEN CONTRIBUTE TO THE ALUMNI FUND

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March - May, 1943

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News from the Medical School and Affiliated Hospitals

The Chancellor announced the following gifts to the School of Medicine between April 1 and May 31, 1943: from Lederle Laboratories, $2,400 in support of research on experimental pneumonia under the direction of Dr. Wood in the Department of Internal Medicine; from an anonymous donor, $2,500 in support of research work carried out in the Department of Internal Medicine by Dr. Kountz; from The Commonwealth Fund, $9,360 in continued support of studies on hemorrhage, burns, and shock, under the direction of Dr. Elman in the Department of Surgery; from The John and Mary R. Markle Foundation, $3,000 annually for two years in support of Dr. Carl V. Moore's hematological studies; from Dr. Kiyoshi Inouye, an alumnus of the School, a gift of $500; from Dr. Nathan Womack and Dr. Carl Lischer, a gift of $150 to be used for supplies for the surgical pathology museum.

New appointments to the School of Medicine include: Dr. Herbert Breyfogle as Instructor in Pathology; Mrs. Dorothy Ritzmann as Assistant in Anatomy; Dr. Richard W. Maxwell as Assistant in Clinical Medicine and in Clinical Radiology; Drs. Drew Peterson and Franz Steinberg as Assistants in Clinical Medicine; Dr. William G. Reese as Assistant in Neuropsychiatry; Drs. Parker Beamer, William Callahan, Fred Schweitzer, Harlan Firminger, Jean Boyle Dehlinger and Edwin Edwards as Assistants in Pathology; Drs. Harold E. Eisele, Edward O. Kraft, Frederick W. Klinge, Gordon S. Letterman, Charles E. Lockhart, C. Barber Mueller, D. Elliott O'Reilly, Mordant E. Peck and Chester A. McAfee as Assistants in Surgery; Dr. Aquiles Lentino as Fellow in Chest Surgery; Dr. Ernest Sachs, Jr. as Fellow in Neurological Surgery.

Leaves of absence from the staff of the School of Medicine for military duty have been granted to the following: Dr. James L. O'Leary, Associate Professor of Neuroanatomy and Assistant Professor of Neurology; Dr. Dudley R. Smith, Assistant Professor of Clinical Obstetrics and Gynecology; Dr. Raymond F. Holden, Jr., Instructor in Clinical Medicine; and Dr. Frank W. Stevens, Assistant in Neuropsychiatry.

At the Spring Meeting of the Texas Branch of Society of American Bacteriologists held in Dallas, Dr. S. Edward Sulkin, instructor at Wash-

ington University School of Medicine spoke on the subject, "The Role of the Laboratory in the Diagnosis of Virus Diseases."

Resignations from the staff of the School of Medicine include: Dr. Robert Royce, Assistant in Anatomy; Dr. Alexander Langsdorf, Jr., Assistant Physicist to the Radiological Institute.

Dr. Ernest Sachs, Professor of Clinical Neurological Surgery, was this year (1942-43) President of the American Neurological Association. The annual meeting was held in New York City on May 6 and 7. The entire meeting was devoted to various phases of neurology pertaining to the war. Dr. Sachs's presidential address was on "The Contributions of War to Medicine." In order to make the sessions of special value, Dr. Sachs arranged with the British and Russian Embassies to bring over representative men for the meeting. Great Britain was represented by Professor Geoffrey Jefferson, who has been doing special research work on peripheral nerves at the Nuffield Institute at Oxford. Russia was represented by Professor Vladimir Lebedenko, who is the Professor of Surgery and head of the surgical clinic at the University of Moscow. The presence of these foreigners added greatly to the interest of the meeting.

The Joint Medical Board recommended to the appropriate Boards the following appointments to the staffs of the hospitals: Dr. Michael Karl Assistant Physician to the Barnes Hospital; Dr. Edward Reinhard, Assistant Physician to the Barnes Hospital; Drs. Anne C. Tompkins, Samuel P. Martin and Charles Huguley, Assistant Residents in Medicine on the ward service of the Barnes Hospital; Drs. Frances M. Sullivan, Claude S. Wright, and Warren B. Mills, Assistant Residents in Medicine on the private service of the Barnes Hospital; Dr. Cyril J. Costello, Assistant Resident in Surgery to the Barnes and St. Louis Children's Hospital; Dr. Robert Godwin, Resident in Otolaryngology to the Barnes, McMillan, and St. Louis Children's Hospital; Dr. Carl Cori, Pharmacologist to the Barnes Hospital; Dr. George H. Bishop, Biophysicist to the Barnes Hospital; Dr. Richard Maxwell, Voluntary Assistant in Radiology to the Barnes Hospital; Dr. Howard Slaughter, Resident in Ophthalmology to the Barnes, McMillan, and St. Louis Children's Hospitals; Dr. Claude C. Gray, Assistant Resident in Ophthalmology in the Barnes, McMillan, and St. Louis Children's Hospitals; Drs. Benjamin Milder and Virgil A. Toland, Assistant Ophthalmologists to the Barnes, McMillan and St. Louis Children's Hospitals; Dr. Harold K. Roberts, Assistant Physician to the Barnes Hospital; Dr. G.

O'Neil Proud, Assistant Otolaryngologist to the Barnes, McMillan and St. Louis Children's Hospitals; Dr. Donald Bottom, Resident in Radiology; Dr. Robert Alvin Rix, Jr., Fellow in Neurosurgery to the Barnes and St. Louis Children's Hospitals and Dr. Florence Heys, Theron Catlin, Fellow to the St. Louis Children's Hospital.

Mrs. Mary Keith resigned as Superintendent of the St. Louis Maternity Hospital on May 10, 1943, to accept the superintendency of the St. Louis County Hospital.

Miss Kittie McKelvey has been appointed Superintendent of the St. Louis Maternity Hospital.

Resignations in the School of Nursing include: Ruth Bouchard, Instructor in Physical Education and Social Director.

The following members of the hospital staffs have joined the armed forces: Major Dudley R. Smith, Assistant Obstetrician and Gynecologist; Capt. Milton Smith, Assistant Physician; Lt. Carl Lischer, Assistant Surgeon; Lts. C. Barber Mueller, Fred Klinge, Edward O. Kraft, and D. Elliott O'Reilly, Assistant Residents in Surgery; Lts. Glenn Turner and Harold Roth, Assistant Residents in Medicine; and Lt. Harry H. Baird, Interne in Medicine.

Appointments in the School of Nursing include: Gladys Ruth Hill, Instructor in Physical Education; Betty E. Ricklefs, Instructor in Nursing; Florence Hicks, Assistant in Nursing and Infirmary Supervisor; and Pearl A. Jones, Assistant in Nursing and Supervisor of Infant Nutrition to St. Louis Children's Hospital.

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News of Alumni

1881

The two survivors of the class of 1881 of the old St. Louis Medical College, now the School of Medicine of Washington University, met for a dinner March 3. Dr. Willis Hall, 5219 Delmar Boulevard, entertained Dr. and Mrs. James Dickson, 5883 Julian Avenue, to celebrate the sixty-second anniversary of their graduation.

A bill changing the name of the St. Louis City Hospital to the Max C. Starkloff Hospital in honor of the late surgeon and first health commissioner of St. Louis, who served 30 years under five mayors, was adopted unanimously March 19 by the Board of Aldermen. Dr. Starkloff received a medical degree from the university in 1881.

1885

Dr. G. W. Tidwell writes: "Am now rounding out my 58th year of medical practice. A few loyal patients are still helping me to carry on." Dr. Tidwell's address is: 56 Walnut Street, Rutherford, New Jersey.

1906

Colonel Wm. H. Smith is Commanding Officer of LaGarde General Hospital, New Orleans, La. Prior to coming to New Orleans, he organized the Station Hospital at Camp Shelby, Mississippi. Colonel Smith's daughter, Dr. Judith D. Smith, graduated from Women's Medical College, Philadelphia, Pennsylvania, in June, 1942, and is interning at Charity Hospital, New Orleans, Louisiana.

1920

Hiram S. Liggett, Beaumont Building, St. Louis, Mo., Class Secretary.

Major William N. Jenkins has been overseas since December, 1942.

Colonel Harvey L. White is post surgeon at the 183rd Station Hospital, A.P.O. Seattle, Washington.

1921

Richard Paddock, 4500 Olive, St. Louis, Mo., Class Secretary.

Captain William B. Lewis is stationed at Fort Lewis, Washington.

1922

Armin C. Hofsommer, 639 Lee, Webster Groves, Mo., Class Secretary.

G. L. Chamberlain writes that his son, Gilbert, is a first year medical student at Washington.

Lieutenant Commander Irl George Tremain is stationed at the Marine Corps Base, San Diego, California.

Dr. Curtis H. Lohr, Superintendent of the St. Louis County Hospital, has been called into active service with the 88th General Hospital, Longview, Texas.

1924

Dr. T. K. Brown, 630 S. Kingshighway, St. Louis, Mo., Class Secretary.

Captain E. H. Christopherson's address is: Office A.U.S. Coordinator Inter-American Affairs, Caixa Postal 1530, Rio de Janeiro, Brazil.

Lieutenant Commander Val B. Satterfield is stationed at the Bremerton, Wash., Navy Yard Hospital.

1925

Myron Davis, 3720 Washington, St. Louis, Mo., Class Secretary.

Lieutenant Commander George L. Drennam is in charge of the dispensary of the 12th Naval District Medical Office, San Francisco, California.

Major James L. Knott is now serving in Brazil. His present address is: Caixa Postal 201, Victoria, Esperito Santo, Brazil.

1926

Alvah G. Heideman, Metropolitan Bldg., St. Louis, Mo., Class Secretary.

Captain Vernon Alfred Vesper is with the Medical Corps in North Africa.

1927

Major Franklin E. Walton, 0203-8874, 21st General Hospital, A.P.O. 685, c/o Postmaster, New York City, Class Secretary.

Captain Arthur C. Fortney is Medical Director, North Dakota State Selective Service, Fraine Barracks, Bismarck, N. D.

Captain William C. Goodlett is stationed at Ashburn General Hospital, McKinney, Texas.

1928

Lieutenant Commander Paul R. Rollins is stationed at the 13th Naval District Dispensary, Seattle, Washington.

Dr. Axel N. Arneson now has the rank of Major. He recently visited St. Louis to attend the wedding of Dr. Louis H. Hempelmann, Jr., class of '38, Major Arneson's address is: Station Hospital, A.A.F.T.T.C., St. Petersburg, Fla.

1929

L. C. Drews, Metropolitan Bldg., St. Louis, Mo., Class Secretary.

Alumni of the university now stationed at Bushnell General Hospital, Brigham City, Utah, met for a reunion March 21 at the apartment of Lieut. Col. and Mrs. Frank Queen, in Brigham City. "Like all army folks, constantly on the move," Mrs. Queen writes in a letter to the Alumni Bulletin, the Queens have a minimum of euipment, "partly furnished by the landlord and partly acquired at various 10-cent stores. But we managed to rustle up 13 chairs and by using cooking spoons and paring knives everyone finally had enough table service to eat the spaghetti supper. Rationing went unnoticed in the hubbub of reminiscences of the 'good old days.' Military representatives attending the party, all of whom have medical degrees from the university included: Col. Robert M. Hardaway, '10, commanding officer of the post ("Incidentally we consider Col. Hardaway tops among commanding officers," writes Mrs. Queen); Lieut. Col. Frank Queen, '29, chief of the Laboratory Service; Maj. Norman Q. Drey, '36, chief of the Out Patient Department; Maj. Jerome Levy, '25, chief of Gastro-Intestinal Section; Capt. Robert S. Smith, '33, of Surgical Service; and Capt. Joseph R. Rebillot, '32, executive officer of the 34th General Hospital. With the wives of the officers, other guests were Dr. W. R. Merrell, M.D. '27, a practicing physician in Brigham City, and Mrs. Merrell.

Commander Craig Byron Johnson is in the Navy Medical Corps overseas.

Captain Thomas P. Wilson is with the Air Corps Engineers overseas.

1930

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

Captain Cleo R. Catley is stationed at the Pyote, Texas, Army Air Base.

Lieutenant Colonel George Conrad Mayfield is in the Medical Corps, North Sector General Hospital, overseas.

Lt. Col. Stanley L. Harrison and Captain Joseph Gitt are among the 17 St. Louis doctors who are members of the staff of the 21st Station Hospital in the Middle East. This hospital has been cited three times in official communications as the best conducted U. S. Hospital over in Palestine. It is a 1000 bed hospital.

1931

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

Max Magnes is stationed at Fort Hamilton, N. Y.

Rolla Boyd Wray is in the armed forces, stationed at Rochester, Minn.

1933

Lieutenant Frank Krenning Bosse is at the March Field Station Hospital, Riverside, California.

Major Truman Guthred Drake, Jr., is acting executive officer of 21st General Hospital now in Africa.

Richard Y. Sakimoto, practicing in Honolulu, Hawaii, writes that he is doing well in his line and averaging about twenty-five deliveries per month. Thus far he has had no operative mortality or maternal deaths in obstetrics.

Lieutenant William Wolf Herman is stationed at the Branch School of Aviation Medicine, Nashville, Tenn.

Major Robert T. Terry is with the 29th General Hospital at Fort Meade, Md.

1934

Stanley M. Leydig, 1652 S. Grand, St. Louis, Mo., Class Secretary.

Dr. Fred Curtis Reynolds reports that his new address is: 32nd General Hospital, Comp Bowie, Texas.

1935

Lieutenant Colonel Paul C. Sheldon, MC, formerly commander of the 62nd Med. Tng. Bn., has been appointed Director of Schools in MRTC. Colonel Sheldon has just recently returned to camp after attending the Command and General Staff School, at Fort Leavenworth, Kansas.

After completing a course in Thoracic Surgery at Barnes Hospital, Major Sam William Downing is now stationed at the Fitzsimmons Hospital in Denver, Colorado.

Captain Elmer G. Graul is in Australia, and his present address is: Officers' Candidate School, A.P.O. 923, c/o Postmaster, San Francisco, California. He writes: "I've been a lot of places, seen and done quite a bit in a small way, orthodox and otherwise and now have become somewhat of a schoolmarm. Washington University Alumni are quite well represented on this side of the world, though we haven't gotten around to a reunion as yet. . . . Recently enjoyed articles by J. B. Brown, McDowell and Frederick Jostes. It has been a source of amazement to me how many of our faculty are well known, quoted, and followed here. Professor Graham certainly did some excellent missionary work here for Washington University a few years ago."

Major Bert Bradford's new address is: APO No. 402, Nashville, Tenn.

1936

Captain Ralph Kenneth Earp is with the 41st General Hospital overseas.

Lieutenant Lewis Edwin Rector is in the Navy Medical Corps.

Captain Robert William Elliott, Chief of the Medical Service at the Station Hospital, Army Air Field, Warrensburg, Missouri, recently visited Barnes Hospital and the Medical School on his way to Chicago where he is scheduled to take the examinations for the American Board of Internal Medicine. He states that he is among the fortunate who are able to practice medicine in the Army just as he did in civilian life.

1937

Interest was aroused when two 50-franc notes were received from Major D. R. Roberts, who is somewhere in North Africa. Duff Allen and Rogers Deakin bought the notes from the Alumni Association to keep as souvenirs. Major Robert's present address is: 356509, 47th Inf., APO No. 9, c/o Postmaster, New York City, N. Y., U. S. Army.

1938

Captain John William Shuman, Jr., is overseas.

1939

Captain Bart M. Passanante, Secretary-Treasurer of the Class of '39 has been in North Africa since the first of the year where he is in charge of the surgical ward service of the 91st Evacuation Hospital. He writes: "I believe it is not censorable to say that I got the first casualty admitted to our young organization." . . . The officers' quarters are called pup tents, and each have a special name. Captain Passanante calls his "Bedside Manor." His present address is: 0-380997, APO 700, 91st Evacuation Hospital, c/o Postmaster, New York City, New York.

Carl A. Brakel reports that he enjoys his activities in the Medical Corps not only in the field of medicine and surgery, but also as a qualified parachutist and cross-country skiier. He has also done his bit in solving the man power problem by announcing the birth of an eight pound son. Mother and child are feeling fine.

Arthur Waite Bohne is with a general hospital at Fort Meade, Mo.

1940

Mary McFayden Bishop writes that after a successful convalescence at Koch Hospital, she now has a part-time position in the Municipal Tuberculosis Sanitorium at Rockford, Illinois. Her husband, Marion Dale Bishop is company commander at Camp Grant, Illinois. His address is: Captain Marion D. Bishop, Co. B, 26th Bn., Camp Grant, Rockford, Illinois.

Lieutenant Herbert A. Iknayan is in North Africa with the Medical Corps.

William Douglas Love is a surgeon lieutenant in the Royal Canadian Naval Volunteer Reserve. After twenty-eight months overseas, he is now in Canada on duty.

1941

Lieutenant Bruce Canaga, Jr., is with the Fleet in the Pacific.

Garland Frederick Smith has been promoted to the rank of captain.

First Lieutenant William Leon Topp is flight surgeon at Roswell Advanced Flying School, Roswell, N. M.

Lieutenant Judson Chalkley's new address is: 513 Parachute Infantry, Fort Benning, Ga.

D. M. Bramwell writes: "I was wondering if any of my classmates who are not in the service or who do not have residencies, or who are finishing one year residencies this year in June, might be made available to work in the First Aid Unit, Swan Island Shipyard, Henry Kaiser Company, Inc., Portland, Oregon. . . . Of course I would consider doctors from any classes. . . . However, there is one stipulation—we cannot use doctors over fifty years of age, and they must be in good health and fair physical condition."

We recently received the following letter from Lieutenant Peter D. Fleming (APO 503, c/o Postmaster, San Francisco, California) who is somewhere in New Guinea: "Have been running into fellow alumni repeatedly. It is the one touch of the states here. New Guinea is really a charming place —beautiful, bizarre, and treacherous. Part of my work is concerned with the treatment of the native population (who are blacks, not Polynesians). I therefore have been able to see some of the tropical diseases about which we read about in the good old days."

"Communication with the natives is somewhat difficult because they have numerous dialects and so I have learned a little Pidgin English by necessity. If I took down a history in the exact words of the native, it would sound something like this: (I wonder how it would look in the clinical chart

at Barnes?) Chief Complaint: (1) Me no savvy pek-pek, one feller week. (2) Bell' belongs me pen too much—one day. Present Illness: Me go finish one feller week, no savvy pek-pek (constipated one week). Yes'day, today me no savvy ki-ki (anorexia) me trow out (vomiting). Bell' belonga me pen too much (abdominal pain). Head fire-up (fever). Yes'day me takim salts ki-ki (cathartic). Bell' belonga me pen too much, me no savvy workim (unable to work). Pen, pen too much, by um by me go dead finish (pain is killing me.)—That was an acute appendix."

"My popularity with the native is very great—not due to my ability nor even my bedside manner, but due to the fact that I am more generous with my bandages and the brilliant colored antiseptics. Gentian Violet is very much coveted. Iodine does not show up and is not thought to be too good. My dressings serve many purposes. After the sore or wound is healed; they become necklaces or hair pretties Four inch gauze makes a nice G-string for a favorite pickaninny boy. One of the boys who works for me wants me to give him the 'calico fixim glass' —in other words the Medical Department towel which has a brilliant red stripe on it."

"Although my native practice is interesting and humorous, I am really working with the RAAF on a crash boat which picks up casualties from naval and air catastrophes that occur in certain sections, which is not unexciting."

"The seven o'clock 'jam session' has started. This is our term for the variety of jungle noises starting at about this time. So many insects are being attracted to my kerosene lantern that writing is quite difficult. I've just been told we have caught another rat—our 32nd in three days. I'm off to pronounce him dead and see he's buried deep enough."

We would like to express our appreciation to Joseph J. Pontier, '37, Edward Grose, '33, Captain A. L. Magnelia, '27, and O. W. Reinders, M '91 for their most generous gifts to the Alumni Association.

Student News

Elrie P. Rodgers and Dorothy Ann Llewellyn were married June 26, in Morgantown, West Virginia. Dorothy has been accepted at Washington University Medical School with the class entering in January, 1944. Mr. Rodgers is a member of the junior class.

Ralph R. Luce and Geraldine R. Brodin were married June 11. Mr. Luce is a member of the sophomore class. Miss Brodin is the daughter of Mr. and Mrs. Fred O. Brodin of Cheney, Washington.

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In Memoriam

Brice X. Corbin, '98, Tacoma, Wash.; aged 72, died March 1.

John V. Dillman, Mo. '99, Louisville, Ill.; aged 73, died February 11.

William F. Eimbeck, '96, New Haven, Mo.; aged 71, died April 10.

Bert Edward Greer, Mo. '00, Dallas, Texas; aged 66, died February 25.

Thomas F. Miller, Mo. '99, Lamar, Mo.; aged 64, died January 10.

Clarence Quinan, '97, San Francisco, Calif.; aged 72, died December 9.

Solomon Thomas Shelly, Mo. '83, Mulvane, Kansas; Aaged 86, died January 17.

WASHINGTON UNIVERSITY

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

Walter E. McCourt, A.M., Assistant Chancellor[1]

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.

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