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The The

WASHINGTON UNIVERSITY MEDICAL ALUMNI QUARTERLY



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Malignant Nephrosclerosis

Case Reports of the Barnes Hospital

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Vol. IX

OCTOBER, 1945

No. 1

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Malignant Nephrosclerosis

THEODORE GREINER*

Richard Bright first recognized the occurrence of renal lesions with cardiac hypertrophy, but it remained for Volhard and Fahr to differentiate essential hypertension with, and without, renal insufficiency, and to recognize the corresponding pathological changes.¹² Klemperer and Otani completed the pathological picture of malignant nephrosclerosis,¹² and Goldblatt,⁷ Wilson,¹⁷ and others designed experiments to explain the pathogenesis of it.

Malignant hypertension is a syndrome of high blood pressure with a severe and rapidly progressive clinical course. Most cases are the primary form which Fishberg calls the malignant phase of essential hypertension. Rare cases of hypertension from renal lesions, such as glomerulonephritis or renal infarct; from lead poisoning; or from endocrine disturbances, as adrenal gland tumors, may terminate in malignant hypertension.³ From 4 to 7 per-cent of patients with essential hypertension develop malignant hypertension.

Malignant nephrosclerosis is the renal end-stage of malignant hypertension, with classical pathological changes in the kidney and death in uremia. However, in some cases of malignant hypertension death may intervene before the renal end-stage. MacMahon and Pratt¹³ estimate that 60 per-cent of patients with malignant hypertension die from renal failure, 20 per-cent from cerebral hemorrhage, and 17 per-cent from heart failure.

The clinical recognition of malignant hypertension is important because of its hopeless prognosis. The patients with it are younger than those with benign hypertension. The average age of onset is 42 years, and 90 per-cent of patients are less than 60 years old. There is an unusually high, sustained

^{*} Submitted in partial fulfillment for credit in undergraduate pathology, class admitted January 1944.

blood pressure. Almost always the diastolic blood pressure is over 130 mm. of mercury.⁵ The heart is enlarged. Frequently there is precordal pain, or decompensation with rales, tender liver, and dyspnea on exertion.

Increased intracranial pressure gives severe headaches, and sometimes nausea and vomiting, and convulsions. The spinal fluid pressure usually ranges from 250 to 350 mm. of water. A typical hypertensive neuroretinopathy is consistently found.⁵ It consists of marked papilledema, which may be as great as 6 diopters, general spastic constriction of the arterioles, arterio-venous compression, general edema surrounding the disk, areas of hemorrhage, and "cotton-wool" patches.

Renal insufficiency occurs considerably later than the retinopathy. It is the terminal stage in those patients who are not killed sooner by heart failure of cerebral accident. The urine contains albumin, casts, and red blood cells for some time preceding death, but there is no anemia until near the end. In the early stages of the disease the concentrating power and phenolsulfonephthalein excretion of the kidney, and the non-protein nitrogen of the blood are normal. As the disease progresses the concentrating power is impaired, giving a fixed specific gravity of about 1.010; there is more and more retention of phenolsulfonephthalein; and the non-protein nitrogen level of the blood rises gradually, leading to a typical uremic death.

The differential diagnosis of the early stage of malignant hypertension from benign hypertension is largely by the marked papilledema. This may lead to confusion with a space-filling lesion in the cranial cavity. However, the high blood pressure, cardiac enlargement, and albuminuria cannot be accounted for on this basis. Malignant hypertension which is first seen in the stage of renal failure can sometimes be differentiated from chronic glomerulonephritis by the less severe anemia, but usually they can be separated only by the rapidly progressive course of renal failure. Klemperer and Otani found the duration of renal failure in malignant hypertension averaged 32 days. 12

The course of malignant hypertension is short and rapidly progressive. The condition of the retina is the best guide to the clinical course. The prognosis, excepting the two rare cases mentioned later, is hopeless. Death comes in about eight months to a year after the appearance of symptoms; few live longer than two years.

In studying cases of malignant hypertension it is extremely important to locate those caused by renal infarct or adrenal tumor. In these two rather rare instances surgical removal will give complete remission of symptoms and save the patient's life. No known treatment will favorably influence the fatal course of all other malignant hypertensions. However,

two aspects must be kept in mind to keep the patient comfortable. Heart failure is very disturbing, and may lead to fatal pulmonary edema. Rest and digitalis will usually control it, although morphine and venisection may be required in severe grades. Second, the increased intracranial pressure gives headaches which salicylates do not completely relieve. Lumbar puncture and hypertonic intra-venous injections are reported to be of value.⁴ Morphine should be given for convulsions, and in the amount necessary to control terminal distress.

Autopsy on patients expiring in renal insufficiency as the end-stage of malignant hypertension shows the classical findings of malignant nephrosclerosis.¹² Gross findings are:

1. Hypertrophy of the heart to about 600 grams, mainly the left ventricle.

2. A kindney normal or slightly reduced in size, firm, red, with prominent petechiae. The surface may be diffusely granular. Cross section shows petechiae, thick, gaping vessels, and hemorrhages in the pelvis.

Microscopic sections show widespread lesions of the arterial tree. 13

These are especially marked in the kidneys, somewhat less intense in the other splanchnic visera, and to a slight degree in the skeletal muscle. Arteries show hypertrophy of the media and proliferation of the connective tissue of the intima and adventitia. The arterioles show two changes:

- 1. Productive endarteritis, starting with mucoid material deposited under swollen endothelial cells. The intima organizes by proliferation of cells, formation of fibrils, and deposition of collagen. The resulting fibrosis narrows the lumen, and may become hyaline. Thrombi frequently form, occluding the vessel.
- 2. Necrotizing arteriolitis with destruction and splitting of the wall, and necrosis of the muscle. Red blood cells and fibrin are found in the wall and surrounding stroma, but there is no infiltration with leucocytes. These lesions are found in the state of repair, indicating that they are not all terminal.¹³

In addition to the arteriolar lesions, the kidneys show other microscopic changes. Focal glomerular lesions of the same nature as the arteriolar lesions are present: fibrosis, hyaline formation, and hemorrhage. There is fatty degeneration of the tubules, which may proceed to necrosis. The connective tissue stroma is slightly increased, and there is focal lymphocytosis.

Since the syndrome of malignant hypertension may be interrupted by death before its inevitable conclusion in uremia, its pathological picture will not always be the classical one described above. Kimmelstiel and Wilson¹¹ suggest that the renal lesions vary with the extent and severity of the disease, and that they fall into three stages:

- 1. Fulminating disease with no renal involvement. This form is very rare.
- 2. Death in which renal failure played little part in the final picture. The blood non-protein nitrogen and the kidney concentrating power are nor-

mal. Careful histological study shows scant focal glomerular lesions and necrotizing arteriolitis. Productive endarteritis may or may not be present. If it is, it will be found when there is sufficient duration of hypertension. About 40 percent of cases of malignant hypertension are in this group.

 Classical death in uremia. The blood non-protein nitrogen is high, and the kidney concentrating power is impaired. There is conspicuous necrotizing arteriolitis, focal glomerulitis, and proliferative endarteritis.

The pathological background of the symptoms is suggested by several men. Klemperer and Otani¹² say that the vascular alterations give parenchymal atrophy of the kidney which leads to the renal insufficiency. Kimmelstiel and Wilson¹¹ more specifically emphasize that the renal failure closely parallels the focal glomerular lesions. Fishberg explains the group of symptoms associated with the brain by assuming that the cerebral arterioles do not constrict as strongly as the arterioles in the rest of the body. As a result the high blood pressure is transmitted into the cerebral capillaries giving edema of the brain. This increases the intracranial pressure, which is responsible for the papilledema, headaches, nausea and vomiting, and convulsions.

Many theories have been advanced to explain the cause and pathogenesis of malignant hypertension since Volhard and Fahr postulated a toxic factor superimposed on atherosclerosis of small vessels.¹² But as yet, the cause is completely unknown, and the pathogenesis uncertain, although experimental work on animals, if it can be applied to man, suggests possibilities.

It seems certain that malignant hypertension is a progressive vascular disorder, for there is no renal involvement until late in the disease, and the arteriolar lesions appear before there is any failure of renal function. Hypertension, either essential or from any other cause, in which there is an unusually high diastolic blood pressure may go to the malignant phase. Fishberg says that the diastolic pressure must be 130 mm. of mercury or over for a year to become malignant, and that when the blood pressure fluctuates the average pressure is usually well above 130. But why does the diastolic pressure go up in a small percent of people with hypertension? Possibly the relative youth of patients with malignant hypertension is a factor. Over 90 percent of all cases of malignant hypertension occur before the age of sixty, whereas the majority of the cases of benign hypertension occur after that age.

Derow and Altschule³ mention the possibility that infections, toxins, and mental stress are factors causing benign hypertension to enter the malignant phase, but state that the evidence is inconclusive. Since Mac-

Mahon and Pratt¹³ were not able to find a common exogenous or endogenous factor preceding the malignant phase, they suggested the possibility of a constitutional factor which favored the transformation into malignant hypertension.

Many thoughtful men have tried to reason out the pathogenesis of malignant nephrosclerosis. Kimmelstiel and Wilson¹¹ probably best explained the development of productive endarteritis. They think that it is a result of vasospasm all over the body, and that it is found mostly in the splanchnic organs because the arterioles there are more sensitive to the vasospastic agent. Klemperer and Otani¹² say that the necrotizing arteriolitis is not inflammatory, and point to the absence of significant white blood cell infiltration. They think that accelerated arteriosclerosis in the kidney arteries and arterioles gives ischemia, which in turn may cause the renal lesions. They support this viewpoint by citing the similarity of the glomeruli in malignant nephrosclerosis to the ischemic glomeruli found next to renal infarcts. Kimmelstiel and Wilson¹¹ extend this idea by saying that ischemia causes the necrotizing arteriolitis and focal glomerulitis, and that these lesions directly cause the renal failure.

The work of Goldblatt^{6, 7} and of Wilson and Byrom¹⁷ forms the best experimental basis for the explanation of the pathogenesis of malignant nephrosclerosis. The crucial point in evaluating their results is deciding whether the lesions produced by experiments on animals are analogous to those of malignant nephrosclerosis in man.

Goldblatt⁷ found that by severe constriction of both renal arteries of dogs, or by severe constriction of one renal artery when the other kidney was removed, he could produce a clinical picture closely resembling malignant hypertension. The dogs developed sustained high blood pressure, followed in short order by renal insufficiency.

At autopsy of his dogs Goldblatt found gross petechiae in the splanchnic viscera. Microscopic study showed arteriolar lesions which resembled those of malignant nephrosclerosis in man in appearance and distribution. They were extensive in the splanchnic viscera, especially the gastro-intestinal tract, and scattered in skeletal muscle. The kidneys did not show them; in the lungs they were in the bronchial arterioles, but not in the pulmonary arterial system. The lesions were focal or concentric hyalinization in the intima, in some cases leading to complete obliteration, and necrosis of the arteriolar wall with hemorrhage into the tissue. No blood pigment was found, indicating that the lesions were recent or terminal. Goldblatt noted the following points about the pathogenesis of these lesions:

1. Elevated pressure in the arterioles must be one factor, for the lesions were not found in the pulmonary system where the pressure was low,

nor in the kidneys which were protected from the pressure by the arterial clamps.

- 2. Ischemia could not be involved, for the ischemia of benign hypertension is as severe as that of malignant hypertension, with no necrosis of the arterioles.
- 3. Renal failure must be of direct concern, for the lesions were produced only when renal failure occurred.

Goldblatt therefore postulated that malignant nephrosclerosis was produced by a combination of high intra-arterial tension and a substance accumulated in the blood from renal insufficiency.

Wilson and Byrom¹⁷ directed their work toward proving that the kidney changes in malignant nephrosclerosis could be caused solely by hypertension. They argued that Goldblatt produced malignant nephrosclerosis in dogs only when the constriction of the renal artery was severe enough to give renal insufficiency also. Thus he did not show the effect of hypertension on the intact kidney.

They found that they could produce hypertension in 65 percent of rats by partial occlusion of the renal artery of one kidney, leaving the other kidney intact. This hypertension took one of two main courses:

- 1. In one group the blood pressure rose gradually, and remained at a constant high level for months, during which time the animals appeared in normal health.
- 2. But in about one-third of the hypertensive rats the blood pressure ran an irregular course. There were weakness, wasting, and coma. The blood urea was within normal limits. On removal of the clamped kidney the blood pressure quickly returned to normal.

The pathological picture in this second group was similar to that of malignant nephrosclerosis. The clamped kidney showed only diffuse tubular atrophy. In the other kidney they found hyaline swelling in the arterioles; necrosis of all coats of the vessels, and hemorrhage into the wall; and cellular proliferation of the intima. The glomeruli showed focal necrosis, hyaline formation, and hemorrhagic infarcts. In the plate accompanying their paper they paired these lesions in the rat kidney with corresponding sections of malignant nephrosclerosis in man, and demonstrated a marked similarity. However, in man the lesions are widespread, while in the rat experiments, the pathological changes are scattered, and most of the kidney tissue is normal. But the relatively short duration of the hypertension in the experiments might account for this. In addition, similar arterial lesions were found in other organs, in a distribution similar to that of malignant nephrosclerosis.

If these lesions are analogous to those of malignant nephrosclerosis, some factors are immediately excluded from the pathogenesis of that disease.

Renal insufficiency could not be concerned because only one kidney is involved. This conclusion is supported by the normal blood urea. Likewise, the role of ischemia and of toxins is discounted, for the lesions were found only in the unclamped kidney. Vasoconstriction due to a circulating vasopressor substance could not be the sole cause, for it would give lesions in both kidneys.

They concluded that the lesions are related to the increased intra-arterial pressure from which the ischemic kidney is protected. The absolute level of duration of the hypertension does not seem to be a factor. Having found that similar renal lesions result from the focal intense arterial spasm caused by the injection of the drug vasopressin, they decided that the determining factor of arteriolar necrosis is "A sudden strain imposed on the vessel wall by the combination of severe vasoconstriction and the resultant rapid rise in blood pressure."

This experimental evidence supports the belief that malignant hypertension is a phase of essential hypertension, and that malignant nephrosclerosis is but the terminal manifestation. Further, that malignant hypertension is the direct cause of the renal lesions, and that these lesions in turn cause the renal insufficiency.

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Case Reports of the Barnes Hospital

Clinical and Postmortem Records Used in Weekly Clinicopathologic Conference at Barnes Hospital, St. Louis

W. Barry Wood, Jr., M.D., and Robert A. Moore, M.D., Editors CASE No. 73

Presentation of Case

I. K., a 77 year old retired business man, entered Barnes Hospital on April 12 and died on April 14, 1945.

Chief Complaints: Weakness and abdominal discomfort.

Family History: Two brothers had diabetes.

Past History: The patient had never been robust. At the age of 40 he was presumed to have tuberculosis and was sent to a sanitarium, but the diagnosis was never proven. He had malaria many years ago. For several years he had had some nocturia and frequency, and occasional urinary incontinence. There was a long history of "colitis," occasional diarrhea, and recurrent episodes of abdominal pain relieved by passing flatus. Six years previous to admission sugar was found in the urine but apparently the diabetes was mild and was controlled by diet alone.

Present Illness: About one month previous to admission the patient began to feel tired and listless. There was a return of his abdominal symptoms which became more marked with a considerable amount of generalized pain and flatulence and frequent eructations. No nausea or vomiting occurred. He had alternating constipation and diarrhea but no blood was noted in the stools. During the present illness he developed a constant dull pain in his lower back. Two weeks before admission he consulted a physician who found a high blood sugar. He was hospitalized and given 20 units of insulin daily. His health gradually deteriorated; he lost his appetite almost completely, and became exteremely weak and lost from 6 to 8 pounds in weight.

Physical Examination: Temperature 35.6 C., pulse 90, respirations 18, blood pressure 130/70. The patient was poorly developed and emaciated. He appeared asthenic and pale. His speech was slightly slurred. There were a few papillomatous lesions of the skin of the trunk, and several café au lait spots. The skin was atrophic. The fundi showed second degree selerosis of the arteries. The mouth was edentulous, and the mucous membranes were pale. The chest showed a markedly increased anteroposterior diameter, and the breathing was chiefly costal. Expansion was poor. The

percussion note was hyperresonant throughout. The breath sounds were diminished. No rales were heard. The diaphragm appeared to be low and moved poorly. The heart borders could not be percussed. The rhythm was regular; sounds were distant; and no murmurs were recorded. The radial and brachial arteries were markedly thickened. The dorsal pedis and posterior tibial arteries could not be felt. The abdomen was distended and tympanitic. The liver was palpable about four fingerbreadths below the right costal margin. It was firm, tender, and the surface was definitely irregular and nodular. Shifting dullness was present in the flanks. There was a direct reducible inguinal hernia. A fine tremor of the hands was noted and the tendon reflexes were hypoactive. Slight edema appeared over the ankles.

Laboratory Findings: Blood count—red cells 3,340,000, hemoglobin 12.3 gms., white cells 15,900, differential count—eosinophiles 1 per cent, stab forms 31 per cent, segmented forms 60 per cent, lymphocytes 8 per cent. Urinalysis: albumin 1+, sugar trace, many granular casts. Stool examination—negative. Kahn reaction—negative. Blood chemistry—sugar 205 mgms. per cent, non-protein nitrogen 45 mgms. per cent, acid phosphatase 5.3 units, cephalin flocculation test 3+, total proteins 4.4 gms. per cent, albumin 2.3, globulin 2.1. Electrocardiogram: ST segments slightly depressed in leads I and II, T waves low upright in lead I. Interpretation—myocardial damage. Roentgenogram of the chest indeterminate.

Course in Hospital: An abdominal paracentesis was done on admission and 500 cc. of amber fluid were removed. No attempt was made to take off a great deal more which seemed to be present. The cell count was 25 per cu. mm., the culture was negative, non-protein nitrogen 45 mgms. per cent, total proteins 1.2 gms. per cent. The specific gravity was not stated. The day following admission the patient appeared confused. He ate practically nothing. A few crepitant rales appeared at both bases. The venous pressure was 130 mm. NaCl, circulation time 30 seconds (?) (patient uncooperative). That evening he became comatose. The sclerae seemed somewhat icteric. The icterus index was 50; the prothrombin time was 52 per cent of normal. Urinalysis showed a heavy trace of albumin with many granular casts. No tyrosine crystals were seen. Lanatoside C was given intravenously. Two days after admission the white blood cell count rose to 21,000 with 26 per cent stab forms and 66 per cent segmented forms, although there was no fever. The platelets were 41,000. After treatment with insulin the blood sugar dropped to 74 mgms. per cent. The patient remained comatose and became incontinent. Jaundice was obvious. Chevne-Stokes respiration developed. The blood pressure gradually fell to 55/35; the pulse became imperceptible; and death came quietly.

Clinical Discussion

DR. HARRY ALEXANDER: That this patient was diabetic is quite evident from his history before admission to the hospital and also from the fact that the blood sugar was 205 mgms. per cent on admission. According to the physical examination in the hospital and according to the laboratory reports the emphasis seems to be on involvement of the liver. Dr. Moore, do you agree with that?

DR. CARL V. MOORE: The fact that the liver was large, tender, and nodular and that there was evidence of hepatic dysfunction with the cephalin cholesterol test would indicate involvement of the liver.

DR. ALEXANDER: What do you think was wrong with the liver?

Dr. C. V. Moore: That is a difficult question to answer. As a part of the diabetes this patient could have a considerable amount of fat infiltration of the liver which could account for the enlargement. He had ascites and his liver was nodular, observations that suggest cirrhosis. There is also the possibility that there were metastases in the liver. I think it is probable that this man had metastases in his liver with fat infiltration as a result of the diabetes.

DR. ALEXANDER: With metastases to the liver, where are the commonest sites of the primary lesion, Dr. Scheff?

DR. HAROLD SCHEFF: I think the stomach would be the commonest, followed by the large bowel, biliary tract, and pancreas.

Dr. Alexander: Dr. Moore, I note that you combine this patient's large, presumably fatty, liver with metastases to account for the dysfunction. Would a secondary carcinoma of the liver itself give dysfunction?

DR. C. V. MOORE: Yes, it could. The clearest evidence on that subject has been presented by Dr. Wade. He found that isolated metastases to the liver do not usually give a three or four plus cephalin cholesterol, but do frequently produce a disturbance in bromsulfalein excretion or the intravenous hippuric acid test. The cephalin cholesterol reaction ordinarily is one or two plus. However, secondary carcinoma of the liver does certainly give dysfunction if the carcinomatous metastases are sufficiently extensive.

DR. ALEXANDER: It is true that the liver must be substantially destroyed before there is dysfunction. If this patient had a primary carcinoma of the liver, what type do you think it would be?

Dr. C. V. Moore: Primary carcinoma of the liver is certainly possible, but it is a comparatively rare disease and it is difficult to diagnose.

DR. ALEXANDER: That is true. The reason that I mention it is that carcinoma of the liver cell type gives enormous lesions compared to that

originating in the biliary system. Dr. Moore, how widely will primary liver cell carcinoma metastasize beyond the lymph nodes?

Dr. Robert A. Moore: Metastases are not widespread in this type of carcinoma.

Dr. Alexander: Naturally the lesion would have to be extensive, if no other factors were added, to give a positive cephalin flocculation test, low plasma proteins, and a prolonged prothrombin time such as were observed in this case. This patient had signs of insufficiency in liver function which implies a widespread destruction or replacement.

Dr. William Olmsted: I think it should be pointed out that this patient died with typical symptoms and signs of hepatic death.

Dr. Alexander: Do you believe that death from a liver carcinoma, with no other factor in so far as the liver is concerned, would be rare?

DR. OLMSTED: Yes, that would be rare.

Dr. Alexander: Dr. MacBryde, will you discuss the factor of a fat infiltration of the liver in association with diabetes?

Dr. Cyril MacBryde: It is true that patients who have poorly controlled diabetes over a period of time do have fat infiltration of the liver and the liver may be somewhat enlarged. However, I think it is unlikely that that was an important cause of this patient's liver disturbance. It is evident that his diabetes was not severe; the duration of the condition is unknown; and the type of diet is not known. Those are all important factors. If his diet had been one with liberal protein and carbohydrate over a long period of time it is not probable that there would be much fat infiltration. An inadequate diet, without liberal amounts of protein and carbohydrate, would bring about fat infiltration. This patient's age is also an important factor. It is more unusual to find large, fatty livers in individuals of this age than in younger diabetics.

DR. ALEXANDER: Dr. Olmsted, since you observed this patient, will you comment on his diet?

Dr. Olmsted: This patient adhered strictly to his diet which was sufficiently liberal in protein and carbohydrate.

Dr. Alexander: Do you agree that this type of diabetic patient does not usually develop a fatty liver?

Dr. Olmsted: Yes, I would agree with Dr. MacBryde on that point.

Dr. R. A. Moore: Autopsy observations, Dr. Alexander, support the opinion of Dr. Olmsted and of Dr. MacBryde. We rarely see fat infiltration in controlled diabetes.

Dr. Alexander: Is it possible to have cirrhosis and a large fatty liver without the existence of carcinoma, Dr. Moore?

DR. C. V. MOORE: That is quite possible and is a definite possibility in this case. I should like to know how large the nodules were in this patient.

DR. OLMSTED: The liver was quite hard and the edge was unusually hard. It was agreed that the surface of the liver was uneven and most of the observers thought that they could feel rather large nodules.

Dr. C. V. Moore: Under those circumstances I think cirrhosis is a less likely cause of the nodules. When large nodules exist the first thing to consider is a metastatic tumor.

Dr. Scheff: Dr. Alexander, is it not unusual to see cirrhosis in a patient seventy-seven years of age? In a recent report on cirrhosis most of the patients were between fifty and sixty years of age.

Dr. Alexander: It has been shown that this man had diabetes and it was presumably not the type to produce a fatty liver. Because the nodules were large, it may be assumed that there was a primary cancer of the liver which was sufficiently extensive to reduce the liver function. Is that correct, Dr. Wood?

Dr. W. Barry Wood: That is correct unless you postulate that he had both cirrhosis and carcinoma, which is a possibility.

DR. ALEXANDER: It is true that carcinoma is frequently associated with cirrhosis.

Dr. R. A. Moore: The percentage in liver cell carcinoma is eighty or ninety per cent and in bile duct carcinoma, sixty per cent.

Dr. Alexander: Then we need not assume that this man had metastatic carcinoma. Instead we may assume that he had primary liver cell carcinoma.

Dr. C. V. Moore: I thought that the most probable diagnosis was secondary carcinoma. On the basis of chance, secondary carcinoma of the liver is more common than primary. No primary site of the carcinoma was found in this patient. The prostate was described as being normal. However, the acid phosphatase of 5.3 Bodanski units is slightly elevated and is suggestive of a small carcinoma of the prostate which was overlooked at the time of examination and which had metastasized to the liver and perhaps to the vertebral column.

Dr. ALEXANDER: That is all true, but how are you going to account for the amount of liver dysfunction that this patient had? On the basis of a secondary carcinoma, the greater part of the liver would have to be destroyed to bring about this dysfunction. It would be much easier to account for the dysfunction on the basis of cirrhosis with primary carcinoma.

Dr. C. V. Moore: Yes, it could be cirrhosis, plus fat infiltration, plus secondary carcinoma. I do not think any of those are mutually exclusive.

Dr. Wood: Dr. Alexander, this patient had a constant pain in his back. I think Dr. Moore would be interested in this point in relation to his suggestion of carcinoma of the prostate.

Dr. Olmsted: Dr. Alexander, would it be pertinent to discuss the relation of carcinoma and secondary metastases in the diabetic as compared to that in the non-diabetic?

Dr. Alexander: It would indeed be pertinent. Please continue.

Dr. Olmsted: The relation between carcinoma and secondary metastases is entirely different in the two groups, the diabetic and the non-diabetic. In the diabetic group the pancreas is more frequently the site of primary carcinoma.

Dr. Alexander: Dr. Moore, how commonly does carcinoma of the prostate metastasize to the liver?

Dr. R. A. Moore: It may metastasize to the liver, but in the presence of a large liver I would look for some primary site other than the prostate. I would emphasize what Dr. Olmsted has said. Carcinoma of the pancreas is at least three or four times more common in diabetics than in non-diabetics.

Dr. Wood: Whenever it is thought that a patient has cirrhosis of the liver in the presence of diabetes, hemochromatosis should be considered. The spots that this patient had were not characteristic of hemochromatosis, but it has been pointed out by those who have studied the disease that there may be no pigmentation of the skin. That is of course rare and I think is unlikely in this case, but it should be mentioned.

Dr. Alexander: This patient had a large amount of albumin, casts, and a non-protein nitrogen of 45. Is there any relation between what we have already discussed and the renal disturbance, Dr. Bulger?

Dr. Harold Bulger: Of course this patient was an elderly individual with vascular disease and it is almost certain that he had arteriosclerosis. In view of a possible primary pancreatic insufficiency I think choline deficiency should be mentioned. This would eventually involve the liver, and bring about cirrhosis and involvement of the kidney.

Dr. Alexander: In other words, he might have a deficient pancreatic function which would produce a large fatty liver. Dr. Moore, is diabetes selective of the islands of Langerhans or can there be a lesion of the pancreas so extensive that it would involve the entire pancreas—such as chronic fibrosis or pancreatitis?

Dr. R. A. Moore: That has been reported, but it is rare.

Dr. Alexander: Dr. Wood, what do you think of the possibility of a hepatorenal syndrome?

DR. Wood: That should be considered.

DR. ALEXANDER: Dr. MacBryde, what is your opinion?

Dr. MacBryde: I think it is quite possible that this patient will show a hepatorenal syndrome in spite of the fact that he does not have everything that goes with the picture. He had albuminuria, low plasma protein, chronic diarrhea, etc.

Dr. Alexander: Do you think he had nephrosis?

Dr. Wood: He had some late jaundice which may have induced a bile nephrosis.

DR. ALEXANDER: In summing up this discussion it is thought that the patient's large liver was not due to fat or to cirrhosis. What would make this liver large and still cause it to fail to the point of liver death if it was not fat infiltration or cirrhosis?

Dr. Wood: I would like to ask Dr. Moore if he has ever seen a large liver in a patient dying a hepatic death of cirrhosis.

DR. ALEXANDER: Dr. Moore, in cirrhosis is it not true that as a liver begins to become cirrhotic there is hypertrophy of liver cells which are perfectly functioning cells?

Dr. R. A. Moore: That is not always true. The answer to Dr. Wood's question depends upon the definition of "hepatic death." There is a type of disease called subacute cirrhosis which has all the signs and symptoms of cirrhosis and in which the liver is large.

Dr. Alexander: Are those patients usually jaundiced?

Dr. R. A. Moore: There is usually subacute hepatitis associated with it, but I do not remember accurately whether jaundice is the usual thing or not.

Dr. Alexander: But is it usually a rapid process?

Dr. R. A. Moore: Yes.

Dr. Alexander: Do you recall any example of death from cirrhosis, uncomplicated by fat or tumor but with a large liver?

Dr. R. A. Moore: I do not know of any such cases, with the exception of subacute cirrhosis.

Dr. Alexander: Then this patient either had fat or a tumor if we exclude the possibility of subacute cirrhosis.

DR. Wood: It is probably tumor in this particular case.

DR. ALEXANDER: If it is tumor, would you postulate a primary or a secondary tumor?

DR. Wood: I would postulate that it is a primary tumor of the liver on the basis of the marked liver dysfunction. This patient probably had cirrhosis with primary liver cell carcinoma.

Dr. Alexander: I agree with that viewpoint. It would be difficult to put it on any other basis unless some factor such as fat infiltration or sub accute cirrhosis is present to account for the large liver.

Dr. C. V. Moore: I would like to know how significant Dr. Bottom thinks the radiographic interpretation of peritoneal irritation is?

Dr. Alexander: Dr. Bottom, is it possible to see peritoneal irritation with ascites on a radiograph?

Dr. Donald Bottom: The shadow could be from ascites or it could be from infiltration of the mesentery by tumor.

Dr. Scheff: Would it be possible to have a carcinoma of the body or the tail of the pancreas that would metastasize massively to the liver without a large liver and signs of cirrhosis?

Dr. Alexander: I do not know, but I presume that is possible.

Dr. C. V. Moore: I think we are placing too much emphasis on the evidence of hepatic insufficiency in this patient. What evidence of this insufficiency exists except the three plus cephalin cholesterol? I do not think the low protein can necessarily be attributed to the liver.

Dr. Alexander: That is true. However, the low protein could be attributed to the liver because it does occur. The prothrombin time was prolonged and the man died what seemed to be a liver death, which is suggestive of liver insufficiency. It is quite possible that this was not a liver death, but it does not seem to be uremia. The sweetish breath of "new mown hay" is a characteristic sign of hepatic insufficiency. Dr. Olmsted, was this factor checked?

Dr. Olmsted: No, I do not think it was checked.

STUDENT: Would the low specific gravity and the low protein content of the ascitic fluid have any bearing on whether this was a fluid from carcinoma of the mesentery or a transudate?

Dr. Alexander: The low specific gravity and the low protein would imply a transudate from cirrhosis.

Dr. Scheff: How do you explain the marked leukocytosis?

DR. ALEXANDER: The patient had rales in his chest, and he was quite debilitated. Is there anything further? It is generally agreed then that this man had cancer of the liver, with probable cirrhosis. However, it has not been decided whether this is primary or secondary cancer. Dr. Harford, do you agree?

Dr. Carl Harford: If there is also cirrhosis, then it should be postulated that the cancer is primary.

Anatomic Diagnosis

Carcinoma of the tail of the pancreas.

Metastatic carcinoma in the porta hepatic and periportal lymph nodes.

Metastatic carcinoma in the liver with almost complete replacement of the left lobe by permeation through portal veins.

Acute interstitial pancreatitis.

Fat necrosis of pancreatic fat.

Ascites (1800 cc.).

Icterus, generalized.

Arteriosclerosis of the aorta and of the iliac arteries, advanced.

Thrombus partially occluding the aorta, 2 cm. proximal to the bifurcation, and completely occluding the left common iliac artery (history of cold left foot and absence of popliteal and dorsalis pedis pulsation, 10 days).

Arteriolar nephrosclerosis.

Hyalination of islands of Langerhans, moderate (history of diabetes mellitus—6 years).

Pathologic Discussion

Dr. Robert A. Moore: On the basis of the gross examination, this patient had a primary carcinoma of the tail of the pancreas with metastases to the liver. The metastases to the liver were unusual in that the entire left lobe was replaced by carcinoma. In the right lobe of the liver there were isolated metastases and foci of congestion representing circulatory disturbance to that part of the liver. The reason for the latter was permeation of the portal venous system by carcinoma inside the liver. This man had lost a third of his liver by actual replacement by carcinoma and he had a serious circulatory disturbance in the remainder of the liver In addition there were metastases to the lymph nodes.

There was not sufficient replacement of the pancreas to explain the diabetes. As was pointed out by Dr. Olmsted, it is most unusual for a carcinoma to cause diabetes and when it does occur the pancreas is almost entirely replaced by carinoma. Therefore, some other anatomic lesion in the pancreas must be found to explain the diabetes.

This man did have advanced arteriosclerosis of the abdominal aorta and the iliac arteries with a thrombus occluding the aorta and left iliac artery. It is evident that the thrombus in the aorta had been present only a few days as shown by the lack of organization.

Microscopically it is seen that the carcinoma in the tail of the pancreas is a highly anaplastic tumor, a type frequently seen in the pancreas. Acini

are present occasionally; there is a great deal of proliferation of connective tissue; and there are occasional cells which contain globules that appear to be mucus although the carcinoma is not a mucinous type. Throughout the pancreas there are small foci of fat necrosis as well as an increase of connective tissue throughout the parenchyma which is not involved by carcinoma. There is also an acute interstitial pancreatitis as shown by numerous polymorphonuclear leukocytes, plasma cells, and lymphocytes throughout the interstitial tissue.

The small blood vessels of the pancreas have thickened walls, loss of nuclei, and hyalination. I would associate the arteriolar disease with the interstitial fibrosis in that a close correlation exists between those two lesions. This patient also has arteriolar disease in other organs so that he has diffuse vascular disease affecting the small blood vessels as well as the large ones.

A significant number of the islands of Langerhans show hyalination. Hyalination is the most common anatomic lesion in diabetes in patients at this age. Hyalination of the islands and interstitial fibrosis of the pancreas are the lesions of the pancreas found most frequently in diabetic patients over the age of forty years.

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First Year Class — September, 1945

| | | . , , , |
|--|--|---|
| ARKANSAS | Home Address | University or College Attended |
| Smith, Kathleen (C) Stull, Fred E. (N) | Fayetteville Jonesboro | University of Arkansas Tulane University of Louisiana |
| CALIFORNIA | | 1 |
| Hebert, William M. (C) Settle, B. Janet (C) Zinn, Marion A. (C) | Fresno Oakland Fullerton | Fresno State College Stanford University Pomona College |
| COLORADO | | |
| Hayhurst, Dale W. (A) Munro, Gordon (N) Sugioka, Keneth (C) | | Stanford University University of Colorado University of Denver |
| CONNECTICUT | | |
| Byrne, Richard C. (A) | Hartford | Missouri School of Mines |
| GEORGIA | | |
| McChesney, John A. (A) | College Park | University of Georgia |
| HAWAII | | |
| Au, Man Hing (C) Ohtani, John M. (C) | Honolulu Honolulu | University of Hawaii University of Hawaii |
| IDAHO | | |
| Kurahara, Louis (C) | Hunt | Kenyon College |
| ILLINOIS | | |
| Bond, R. Jerry (C) Hermann, William (C) Hughes, Robert (A) Kadlub, Edwin D. (A) Kilian, Duane J. (C) London, Stanley L. (N) Meyer, Walter L. (N) Minnihan, Robert W. (A) Peterson, Shirley (C) | Mt. Vernon Highland Chicago Chicago Wood River Springfield Jacksonville Dixon Park Ridge | Washington University Washington University University of Illinois New Mexico School of Mines University of Michigan De Pauw University Miami University University of Missouri University of Chicago |
| IOWA | | |
| Moore, Janice S. (C) Smiley, Gertrude H. (C) | Sioux City Grinnell | Oklahoma City University Grinnell College |
| MASSACHUSETTS | | |
| Page, Lot B. (A) Raso, Anthony J. (A) | West Newbury East Boston | Middlebury College Northeastern University |
| MICHIGAN | | |
| Goodhue, Gerald A. (A) Korn, Robert (A) | Holton Detroit | Western Michigan Coll. of Ed. State University of Iowa |
| MINNESOTA | 3" | |
| Fischer, John R. (C) Vandergon, Keith G. (A) | Austin Delano | University of Arizona University of Missouri |
| MISSISSIPPI | | |
| Jernigan, James A. (N) Slatter, John A. (A) | Tupelo Natchez | Mississippi College Colgate University |

| MISSOUPI | Home Address | University or College Attended |
|---|--------------------------------|--|
| MISSOURI Berg, Leonard (C) | St. Louis | Washington University |
| Bond, Marcus B. (N) | Enon | Westminster College |
| Breidenthal, Ward B. (A) | Kansas City | St. Louis University |
| Bruns, Kenneth (N) | St. Charles | Central College |
| Bumgarner, Roger W. (N) | Odessa | University of Missouri |
| Cannon, William J. (N) | Elsberry | Central College |
| Davis, Dorothy J. (C) | Webster Groves | De Pauw University Washington University |
| Downen, John V. (N) | St. Louis | Washington University |
| Fingerhood, Marvin G. (N) Fischer, George W. (A) | Webster Groves | Iowa State University |
| Gray, Margaret B. (C) | Independence | University of Kansas City |
| Gray, Mary J. (C) | University City | Swarthmore College |
| Happel, Elizabeth (C) | St. Louis | Smith College |
| Heideman, Milo L., Jr. (C) | St. Louis | Harvard College |
| Hessing, John W. (N) | St. Louis | Westminster College |
| Innes, Robert C. (N) | Fayette | Central College |
| Jaeger, Joseph N. (C) | Jackson | Southeast Mo. St. Teachers Coll. Washington University |
| Jick, Sidney (C) Kelly, Patrick (C) | University City Springfield | Southwest Mo. St. Teachers Coll. |
| Larson, Ralph T. (C) | Kirkwood | Washington University |
| Levitt, Joseph (A) | St. Louis | University of Missouri |
| Lipschitz, Ervan (C) | St. Louis | Washington University |
| Lund, Robert H. (N) | University City | Miami University |
| Luten, John G. (N) | St. Louis | Westminster College |
| McGinty, Charles P. (A) | Cape Girardeau | Southeast Mo. St. Teachers Coll. |
| Moore, Robert (C) | Kirksville | Northeast Mo. St. Teachers Coll. Oberlin College |
| Roseran, Marvin (N) Schiele, Herbert (A) | University City St. Louis | Dartmouth College |
| Stein, Marvin (C) | St. Louis | Washington University |
| White, Laurens P. (N) | St. Louis | Westminster College |
| Wood, James (N) | University City | Westminster College |
| Yaeger, Gerald A. (N) | University City | Northwestern University |
| MONTANA | | |
| Greaves, Donald C. (C) | Great Falls | Montana State University |
| Rothwell, Leonard E. (C) | Billings | Montana State University |
| | | |
| NEBRASKA Hawkins, James C. (A) | Lincoln | University of Nebraska |
| Hult, John E. (A) | Wahoo | Southwest Mo. St. Teachers Coll. |
| | Trumos | |
| NEW JERSEY | Nowfoundland | University of Misseyri |
| Sargent, Harold (A) | Newfoundland | University of Missouri |
| NEW MEXICO | | |
| Laurain, Alan R. (A) | Roswell | University of Minnesota |
| NEW YORK | | |
| Norsa, Luigia (C) | Schenectady | Vassar College |
| Pfeffer, Robert I. (N) | White Plains | Miami University |
| Rokaw, Stanley (C) | Brooklyn | Coll. of City of New York |
| OHIO | | |
| Benes, Doris M. (C) | Cleveland | Western Reserve University |
| Livingston, Harry H. (C) | Warren | University of Kansas City |
| Pietrykowski, Felicia (C) | | University of Toledo |
| Schulz, Dale M. (C) | Middleton | Miami University |
| OKLAHOMA | | |
| Felton, Warren L. (N) | Bartlesville | University of Texas |
| Forrester, Ralph H. (N) | Tulsa | Southeast Mo. St. Teachers Coll. |
| Stalnaker, Robert (A) | Konawa | University of Oklahoma |
| Temple, Gene H. (C) | Okmulgee | Northeastern State College |
| 0 | | |

| OREGON Bauman, Wiliam F. (N) | Home Address Medford | University or College Attended Willamette University |
|--|-------------------------|--|
| PENNSYLVANIA Whitmore, J. Stewart (N) | Bridgeville | Westminster College |
| WASHINGTON Thornberry, Robert D. (C) | Everett | State College of Washington |
| WISCONSIN Kubinek, Roland W. (A) Streeter, Ralph T. (A) | Eagle River Superior | University of Wisconsin Baylor University |
| PUERTO RICO | | |

Third Year Class — July, 1945

Soto-Franco, Alfonso (C) Santurce University of Puerto Rico

| ALABAMA | Home Address | University or College Attended |
|--|--|--|
| Alexander, Jack W. (A) Koffler, Irving E. (A) Shamblin, John L. (N) | Fayette Mobile Tuscaloosa | University of Alabama University of Alabama University of Alabama |
| COLORADO | | |
| McLin, Leonard D. (N) | Fort Lyon | University of Alabama |
| MISSOURI | | |
| Davis, Jack M. (C) Gregory, Jack A. (A) King, John D. (N) McNeel, Lee A. (A) Perry, William D. (N) Regan, William W. (N) Schwartz, Ely (A) Stallard, Donald J. (A) Tanner, Robert H. (C) Thomas, Miles E. (A) Wallace, Robert N. (N) Wevgandt, Glenn R. (N) Willoughby, James W. (N) | Wheeling Kansas City St. Louis Webb City Mound City Columbia Rolla St. Joseph Jefferson City Kansas City Ava Joplin Norborne | University of Missouri |
| NORTH CAROLINA | | |
| Fanney, Grafton C., Jr. (A Smith, Roger A. (N) Worth, Winfield A., Jr. (N | Raleigh | University of North Carolina University of North Carolina University of North Carolina |
| SOUTH DAKOTA | | |
| Deer, William C., Jr. (A) Gibson, Glenn G. (A) | Pierre Sioux Falls | University of South Dakota University of South Dakota |
| VIRGINIA | | |
| Dunckel, Wiliam C., Jr. | Tibitha | Harvard University |
| WEST VIRGINIA | | |
| Morgan, Margaret A. (C) Stutzman, Leon (C) | Beckley Shepherdstown | West Virginia University West Virginia University |
| | 0 | |

News from the Medical School and Affiliated Hospitals

The following gifts to the School of Medicine were announced between July 1 and September 5, 1945: from The John and Mary R. Markle Foundation, \$7,000 to Dr. Carl Moore to continue for two years his studies of iron metabolism in humans and animals with radioactive isotopes of iron and the effect of high oxygen tensions on the production of red blood cells by bone marrow; from The Squibb Institute for Medical Research, \$2,400 for the establishment of a fellowship for the study of experimental syphilis under the direction of Drs. Barry Wood and John Smith; from The Rockefeller Foundation, \$2,500 toward the researches of Dr. Shaffer in the field of biochemistry; from The Rockefeller Foundation, \$7,000 in continued support of Dr. Carl Cori's research in carbohydrate metabolism; from the Office of Scientific Research and Development, a contract under the direction of Dr. Wood; from Dr. Louis F. Aitken, \$500 to the Thekla Aitken Fellowship Fund in the Department of Internal Medicine; from the Eli Lilly Company, \$1,200 in continued support of Dr. MacBryde's work on insulin mixtures in the Department of Internal Medicine; from the Winthrop Chemical Company, \$1,200 in continued support of Dr. MacBryde's work in synthetic estrogenic compounds in the Department of Internal Medicine; from the William S. Merrell Company, \$600 to re-establish a fellowship for the study of "TACE" under the supervision of Dr. Willard Allen; from Mrs. Ina C. Urbauer, \$5,000, from Mr. Franklin W. Olin, \$5,000, from Mr. Arthur B. Baer, \$2,000, and from Mr. Roy B. Jones, \$1,000, for the study of degenerative diseases under the direction of Dr. Kountz.

New appointments to the staff include: Dr. Conrad Sommer as Assistant Professor of Clinical Psychiatry; Dr. Sidney F. Velick as Research Associate in Pharmacology; Mrs. Anne E. Royer as Instructor in Medical Psychology; Dr. Thomas W. Kemper as Assistant in Clinical Orthopedic Surgery; Dr. Leon Fox as Assistant in Clinical Orthopedic Surgery; Dr. V. J. Verda as Assistant in Surgery; Dr. William B. Shalleck as Research Fellow in Biophysics.

Dr. I. D. Kelley Jr., Assistant Professor of Clinical Otolaryngology, has been granted a leave of absence for naval service.

The following have resigned from the staff: Dr. Arda A. Green, Assistant Professor of Biological Chemistry and Research Associate in Pharmacology;

Dr. Mary Miller, Research Associate in Pathology; Dr. Julius W. Vieaux, Instructor in Obstetrics and Gynecology; Mr. Leonard M. Board, Instructor in Public Health; Dr. Charles A. Stone, Assistant Professor of Clinical Orthopedic Surgery.

Dr. Richard G. Scobee is now back as Director of the Post-Graduate Courses in Ophthalmology and is also on the staff of the department. He was a captain in the Army Medical Corps.

Dr. M. G. Seelig, Professor of Clinical Surgery, has been reappointed by Governor Donelly to the State Cancer Commission. Dr. Seelig was originally appointed to succeed the late Dr. Fred Taussig who was associated with Dr. Ellis Fischel in the establishment of the State Cancer Commission and the Ellis Fischel State Hospital.

Lieutenant Colonel Oscar P. Hampton, Consultant in Orthopedic Surgery in the Mediterranean Theater of Operations, who has been on temporary duty at the Office of the Surgeon General, recently visited twenty-three general hospitals in this country where he studied fracture and orthopedic cases which had received initial and reparative surgery in the Mediterranean Theatre of Operations to determine the effectiveness of the procedures used overseas in the light of end results obtained here. Col. Hampton went overseas originally as Chief of the Orthopedic Section of the Twenty-first General Hospital.

A. H. Meyer, a former member of the house staff of Barnes Hospital in Surgery, is now a lieutenant, junior grade, in the Navy. He is aboard the USS Philip (DD 498), c/o Fleet Post Office, San Francisco, California, and during the month of July was busy near the Philippine Islands.

Leonard T. Furlow, Associate Professor of Clinical Neurological Surgery, now in the Navy, has been promoted from the rank of commander to that of captain.

The fact that the war is over, is increasingly evident about the medical school and hospitals. Dr. Henry Schwartz is back as full-time Associate Professor of Neurological Surgery. Sam Harbison, resident in surgery just before the war, is in civilian clothes. Sam Gollob, who had an appointment in pediatrics but was unable to accept it, spoke at the monthly staff meeting at the Children's Hospital. He has several medals, including the Purple Heart. He said that he was most proud of the small button in

the label of his civilian coat. His most trying experiences were at the Anzio Beachhead. Robert Ansheutz, who was appointed intern in surgery on July 1, 1941 and was called up July 7, 1941, has returned to keep his internship. Ben Senturia is now full-time in otolaryngology. He spent most of the war at the research center for aeromedicine in Texas. His work on areo-otitis was standard in the Air Forces.

With a view to the future development of the field of physical medicine, the University and the Missouri Association for Occupational Therapy have signed a new contract. The unversity will accept full responsibility for the conduct of the school in the future.

So far as can be determined at the present time, the allocation of the interns and residents in civilian hospitals by Procurement and Assignment will continue for at least another year. Discharged veterans who accept residencies do not count against these quotas.

The Department of Pediatrics has established an eight-week refresher course. The first four weeks will be spent in a general review of pediatrics and the last four weeks in supervised clinical work. Registration is limited to six persons.

The graduate courses in otolaryngology and ophthalmology which have been given for many years and were discontinued during the war, have been started again. These courses last eight months. The mornings are spent in the laboratory in the study of the pertinent basic sciences and the afternoons in the clinics and hospitals. For the course beginning about October 1, 1945, there are seven registrations in otolaryngology and ten registrations in ophthalmology.

On the evening of September 24, 1945, Colonel Howard Rusk of the Office of the Air Surgeon spoke before the Barnes Hospital Society on "Model Civilian Rehabilitation Service and Center; Its Relation to the Medical School and Public." The outlook for physical medicine and rehabilitation seems most promising.

Alumni News

1885

Ben Hudson's new address is 615 South Second, Springfield, Illinois.

1886

Benjamin F. Stockett has moved to 4258 Vallejo, Denver 11, Colorado.

1891

P. H. Morrison's address is now 7062 Washington Blvd., University City 5, Missouri.

1894

Guthrie E. Scrutchfield, retired army major, has written us that his address is #412 Forsyth Apt., Savannah, Georgia.

1895

Robert J. Curdy wrote a letter regretting his inability to attend the fiftieth anniversary reunion of his class, but he gives as reason that "having acquired the inevitable collection of squeaks and rattles incident to seventy-seven years of existence, travel has become something of a hardship, even so moderate as to St. Louis."

1896

Floyd Stewart again has his office in the Chemical Building, 721 Olive Street, St. Louis 1, Missouri.

1897

R. M. Cowan's address is 446 South Grant, Springfield, Missouri.

1899

Louis Ostrom writes that at the last reunion of his class he was delegated to find out what became of E. B. Williams, also a member of the class. He has found him well and very active with a large practice in Montezuma, Iowa. Dr. Ostrom writes that "in spite of lack of nurses and assistants, I saw him perform an appendectomy (spinal anaesthesia) during my visit and do a fine job, too."

1904

John Beckert is now at the hospital in Alton, Illinois.

1905

Robert C. Strode has moved to 122 S. Burgess Street, West Branch, Michigan.

George M. Park's new address is Post Office Box 6, Eureka, Misscuri.

1907

G. H. Reinhardt can be reached at the Mira Loma QM Depot, Mira Loma, California.

1908

Leland P. Viley's address is 5011 Walnut Street, Kansas City 2, Missouri.

1919

W. G. Wander has moved to 1171 East Grand Blvd., Detroit 11, Michigan.

1922

Colonel Lee D. Cady, commanding officer of the Twenty-first General Hospital in France, sponsored by Washington University, was decorated with the Croix de Guerre by the French Government "for exceptional services during the liberation of France." The ceremony took place in Paris at Napoleon's Tomb.

1923

Lieutenant Colonel James Barrett Brown, chief of plastic surgery at Valley Forge General Hospital, Phoenixville, Pennsylvania, was promoted recently to the rank of colonel. He went to the hospital in May 1943 after serving for a year as senior medical consultant in plastic surgery and burns in the European Theater of Operations.

1925

Major James Knott has written us that he has been receiving the Quarterly at his overseas address. He is now back in the United States at a temporary duty address; therefore, he has given us his home address of 3401 Pershing Drive, San Diego 4, California.

1926

Lieutenant Commander Charles G. Johnston can be reached at 1512 St. Antoine, Detroit 26, Michigan.

1927

Sol Londe's new address is 9200 N. W. 4th Ave., Miami 38, Florida.

1928

Colonel Wilford F. Hall is with Hqrs. 9th Air Force at APO 696, c/o Postmaster, New York, New York. The Headquarters of the 9th Air Force, which is the occupational air force of Germany, is located in Bad Kissingen, Germany (Bayaria).

Brigadier General Earl Maxwell is surgeon of the South Pacific Base Command. He entered the Medical Department of the Army as a first lieutenant in the Medical Reserve in 1928, graduating from the Army Medical School in Among other places, he has served at the Gorgas Hospital at Ancon; as chief of Eye, Ear, Nose, and Throat Service and assistant base and flight surgeon at the station hospital, Barksdale Field, Louisiana; as laboratory officer, Station Hospital, Fort Leavenworth, Kansas; and as base surgeon and sanitary inspector, Army Air Base, Savannah, Georgia. He is a graduate of the Medical Field Service School, Carlisle Barracks, Pennsylvania, and the School of Aviation Medicine, Randolph Field, Texas, and has a flight surgeon rating. He has been awarded the Air Medal and the Legion of Merit and attained his present rank in January, 1944.

John B. Harter has written that, beginning October 1, 1945, his address will be c/o State Tuberculosis Sanatorium, Bluegrass Avenue, Louisville 9, Kentucky.

1929

Lieutenant Commander Guerdan Hardy can now be reached at his home address, 7460 Stratford Avenue, University City 5, Missouri.

Jacob S. Fishman is a captain in the Medical Corps overseas.

On July 5 Charles H. Appleberry was promoted from the rank of major to lieutenant colonel. He is on Mindanao in the Philippines and has been commanding officer of the 2nd Field Hospital since March. While on Mindoro he was awarded the Bronze Star for bravery and devotion to duty under enemy shell fire.

Major A. W. Freshman is with the 172nd General Hospital at APO 689, New York, New York.

Major Lawrence C. Ball's new address is 116th Evacuation Hospital, APO 758, New York, New York.

Major W. C. Finn has written us that his correct address is 373rd General Hospital, APO 1057, c/o Postmaster, San Francisco, California. He also says that he has seen both Dr. Higgins and Dr. Whittaker near his base.

1930

Francis G. Pipkin's address is 601 W. 61st Terr., Kansas City 2, Missouri.

Stanley L. Harrison has been promoted to the rank of colonel. He is chief surgeon of the Persian Gulf Command Headquarters and can be reached by addressing mail to 8023 Crescent Drive, Clayton 5, Missouri.

Leon A. Taylor is now a commander and is stationed at the U. S. Naval Hospital, Memphis, Tennessee.

1931

Sam A. Bassett is at the Naval Air Facility, Columbus 3, Ohio.

Major Henry C. Barber came into the Alumni Office recently to tell that he had just returned from overseas and could be reached at his home address of Normal, Illinois.

1932

Lieutenant Benjamin I. Allen is with the 3rd General Hospital at APO 3922, c/o Postmaster, New York, New York.

Lieutenant Colonel Joseph R. Rebilott has APO 14499, c/o Postmaster, San Francisco, California.

Lieutenant Francis W. Aubin's address is 57th Field Artillery Battalian, APO 7, c/o Postmaster, San Francisco, California.

1933

The Bronze Star was recently awarded to Lieutenant Jesse John Wimp, formerly of Kirksville, Missouri. The citation accompanying the award read "for meritorious service as medical officer attached to a Marine Battalion in the Empress Augusta Bay Area, Bougainville, Solomon Islands, from November 7, 1943 to January 20. 1944. With the assistance of one junior officer and a small enlisted staff. Lt. Wimp established four organizational sick bays and rendered medical assistance not only to his own battalion but to all other troops in the area. Although suffering from a painful illness during the entire period, he worked tirelessly, treating or supervising the treatment of more than 300 sick and wounded a day despite the ever present danger from constant attacks by enemy bombers and artillery. His courageous devotion to duty and inspiring leadership were in keeping with the highest traditions of the United States Naval Service." Lt. Wimp entered the service September 7, 1942.

Promotion of Reber M. Van Matre from the rank of major to colonel has been announced by the commanding general of the Chinese Combat Command. Col. Van Matre has been in China since September, 1942, and in January, 1945, was awarded the Grand Star of China Medal by the Chinese Government for his services to the Chinese Army.

Martin J. Hurst's address is 4543 Washington, Kansas City 2, Missouri.

Captain Albert A. Loverde has returned from service outside the continental United States. He served 25 months as a medical officer in the European Theater of Operations and was awarded the European campaign ribbon with three battle stars. He is now being processed through the Army Ground and Service Forces Redistribution Station in Miami Beach, Florida.

Lieutenant Commander Henry Allen spent a few days' leave in St. Louis recently. He is laboratory officer of the Naval Hospital in Norfolk, Virginia, where he has been since returning from the Southwest Pacific.

1934

Carl P. Birk has moved to 1120 Oakland Court, Decatur 33, Illinois.

Lieutenant Charles H. Talbott can now be reached at 7918 Kingsbury, Clayton 5, Missouri.

Major Stanley F. Hampton's new address is Regional Hospital, San Antonio District, AAF PDC, San Antonio, Texas.

Major James G. Telfer came into the Alumni Office recently to report his return from the Philippines where he had the task of reorganizing the Division of Communicable Diseases of the City of Manila Department of Health. On the first of August, 1945, the Department was returned to the officials of the Philippine Commonwealth. Major Telfer is now on duty at the U. S. Marine Hospital, San Francisco, California.

John J. Brown is with the 758th GHQ Tank Battalion at APO 17104, c/o Postmaster, New York, New York.

John A. Saxton is at the Snodgrass Laboratory, City Hospital, St. Louis 4, Missouri.

Major W. H. Doyle is stationed at the Fitzsimmons Hospital E-4, Aurora, Colorado.

1935

Ellen S. Loeffel's address is 3720 Washington Avenue, St. Louis 8, Missouri.

Lieutenant Colonel Kenneth V. Larsen, division surgeon of the Ninety-first "Powder River" Division in Italy, has been decorated with the Bronze Star for meritorious service with the Fifth Army.

Lieutenant Colonel Bert M. Bullington is overseas as chief of medical service of a general hospital. His home address is 305 East Washington Street, Urbana, Illinois.

Reese H. Potter has been transferred to the Rehabilitation Center, Jefferson Barracks 23, Missouri.

Captain Max Goldenberg can be reached at 7547 Wellington Way, Clayton 5, Missouri.

1936

Captain Stephen S. Ellis' adress is 382nd Station Hospital, APO 14252, c/o Postmaster, San Francisco, California.

Captain Saul Dworkin, who was captured by the Germans last December 19 during the Battle of the Bulge in Belgium, has been reported liberated.

Major Alexander Silverglade is now with the U. S. S. Comfort, c/o Fleet Post Office, San Francisco, California.

The Bronze Star was awarded to Lieutenant Commander Wallace E. Allen "for distinguishing himself by heroic and meritorious conduct as Senior Medical Officer aboard a carrier in her action against a Japanese . . . Force in the Southwest Pacific Area. While his ship was under . . . enemy shellfire, Lt. Comm. Allen planned and supervised the vital medical activities, rendered treatment to the wounded and encouraged all personnel by his splendid endeavors. Because of his prompt medical attention many men were saved from painful suffering and long hospitalization. His conduct throughout distinguished him among those performing duties of the same character."
Paul D. Fleming has moved to 3810
Fannin, Houston, Texas.

1936

Robert H. Rutherford's address is 423 South Broad, Carlinville, Illinois. Robert J. Mueller is with the 1560th S. C. U. C. R. S. at Wakeman Convalescent Hospital, Camp Atterbury, Indiana.

1937

Captain Robert C. Kingsland can be reached at R. R. 4, Prospect Hill, Frederick, Maryland.

James W. Burke, Jr., diplomate of the American Board of Dermatology and Syphilology, has announced the opening of his office at Suite 607-609 Maison Blanche Building, New Orleans, Louisiana.

Lawrence E. Mendonsa's new address is 504 North Central Avenue, University City 5, Missouri.

Captain Russell J. Vaughn was graduated June 23 from the Army Air Forces School of Aviation Medicine, Randolph Field, Texas.

Charles E. Martin is stationed at the Station Hospital, SM AAF, San Marcus, Texas.

1938

Horace E. Jones has moved to 1322 W. 5th Street, Anderson, Indiana.

Captain Reuben R. Harris is at the Camp Rucker, Alabama, Station Hospital after serving 31 months in India and Burma.

Captain Howard A. Steiner is with the 31st General Hospital, APO 70, c/o Postmaster, San Francisco, California.

Lieutenant Colonel Eugene F. Melaville has received a new assignment. He is now a division surgeon, and his address is Hdqrs. 93rd Infantry Division, APO 93, c/o Postmaster, San Francisco, California.

Captain Joshua E. Jensen's new address is 105th Station Hospital, APO

427, c/o Postmaster, New York, New York.

Lieutenant Colonel Harry G. Moseley has been transferred to Hq. AAF TAS, Washington, D. C.

Captain William G. Baker has as his new address 432 Milton, Salt Lake City 4, Utah.

Captain Malvern T. Bryan wrote on August 14 that he was on his way home from the European Theater of Operations where he had accummulated a total of 126 points. He was overseas for 42 months. He also said that he hoped to be able to drop in and say "hello" at the Alumni Office very soon. His address will be 244 North Garland Street, Memphis 4, Tennessee.

1939

Major Miles Everett Foster, Jr., is stationed at the U. S. Army General Hospital, Camp Carson, Colorado Springs, Colorado.

Major Mark J. Brockbank has been assigned to the Station Hospital at Camp Beale, California.

Major Herman F. Inderlied has moved to 115 Empire Street, Grass Valley, California.

William B. Hildebrand's new address is 1722 Arlington Avenue, Bessemer, Alabama,

Major Wilson Brown has just returned from Europe. Shortly after V-E Day he was transferred from the 21st General Hospital and made commanding officer of a medical unit which was to go to the Pacific. As they neared the Panama Canal, V-J Day was announced; and the ship changed course and landed on the east coast. New orders have not yet been issued.

1940

Captain Joseph S. Summers has written us that he has just returned from overseas. His permanent home address is 209 Boonville Road, Jefferson City, Missouri.

Major Howard T. Robertson can be reached at 802 Willow, Concordia, Kansas.

William C. MacDonald has his office at 539 North Grand Blvd., St. Louis 3, Missouri.

Captain Paul Guggenheim has reported to the AAF Regional and Convalescent Hospital, Fort George Wright, Spokane, Washington, where he will be one of the personal physicians to whom returned airmen are assigned for consultation during their convalescence.

Betsy G. Wootten's address is 90 Woodacre Drive, San Francisco, California.

Lieutenant Sydney T. Wright now has APO 782, c/o Postmaster, New York, New York.

Robert L. Merrill, who spent 26 months as a flight surgeon overseas, is now stationed at the Camp Davis, North Carolina, hospital.

Harold A. Budke is a captain in the Medical Corps overseas. His address is 15th Gen. Disp., APO 465, c/o Postmaster, New York, New York.

Captain Roland Cross's overseas address is 132nd Evacuation Hospital, APO 758, c/o Postmaster, New York, New York.

Captain Robert Anschuetz was stationed at the Medical Staging Service, Camp Stoneman, California. (See p. 32)

1941

A daughter was born to Bernice Albert Torin on July 2, 1945.

William F. McGinnis is with the Third General Hospital in France.

Charles L. Yarbrough is in the Army in the European Theater of Operations.

Captain Barney W. Finkel, attached to the medical corps, was awarded the Silver Star for gallantry in action in the invasion of the Philippines.

Captain Henry H. Caraco is now overseas. His address is 123rd Evacuation Hospital, APO 18081, c/o Postmaster, New York, New York.

1942

Russell L. Herdener is in South America and has 2623-29th Avenue, West, Seattle 99, Washington as his address temporarily.

Caroline Kreiss Pratt is at the School of Tropical Medicine, San Juan, Puerto Rico.

Lieutenant Frederick W. Klinge has been assigned to the McCaw General Hospital, Walla Walla, Washington.

Herman T. Blumenthal has been promoted to the rank of captain. His address is 29th Medical Laboratory, APO 465, c/o Postmaster, New York, New York.

Lieutenant Samuel T. Ellis recently returned from 11 months in France and Germany with the 15th Tank Battalion of the Sixth Armored Division.

Captain Edwin E. Devereux has been transferred to the Marana Army Air Field in Arizona.

Lieutenant George A. Daman is with a field hospital overseas.

Captain Elbert H. Cason can now be reached at 9218 Gravois, St. Louis, Missouri.

Captain William G. Reese's new address is 98th General Hospital, APO 403, c/o Postmaster, New York, New York,

Lieutenant Edward Henry Jones, Jr., recently suffered second degree burns of the face and arms in the Pacific. He is expected to return to this country shortly.

Captain John R. Showalter, whose address is 1562nd AAF. B. U., PD, ATC, APO 920, c/o Postmaster, San Francisco, California, sends greetings from Biak in the New Guinea group of islands. He writes that when he was on Guam, he ran into McAfee, a classmate, and that they had a big discussion about old times and old friends.

Captain Joseph L. Ponka writes that he has been in Manila since May 1. He says that the city was thoroughly wrecked, and that the natives are suffering from all sorts of diseases. It rains every day since the rainy season started, unfortunately, as soon as he arrived. Capt. Ponka sends best wishes to all his classmates. His address is 312th General Hospital, APO 75, c/o Postmaster, San Francisco, California.

March, 1943

William A. Seidler, Jr., is stationed at M. C. Hq. Comdt., LAPF, Wilmington, California.

Captain Parker Beamer has been assigned to a special laboratory unit in Puerto Rico. His address is Medical Laboratory, APO 851, c/o Postmaster, Miami, Florida.

James Read can be reached at 693 Chalmers, Detroit 15, Michigan.

Lieutenant Sigmund Gundle is now with the Regional Hospital, Fort Bragg, North Carolina.

Captain Edward H. Dunn is a member of a unit which is part of the Army of Occupation in Germany. His address is 777 AAA AW BN (SP), APO 403, c/o Postmaster, New York, New York.

David Feldman has moved to Apt. 318, 5220 Drexel, Chicago, 15, Illinois. 3425 Lawn Avenue, St. Louis 9, Missouri is Richard T. Odell's new address.

Captain George R. Aufderheide stopped by the School on his return from Europe wearing the Purple Heart and the ETO ribbon. He participated in the wild ride to bolster our forces on the southern side of the Ardennes Bulge. Capt. Aufderheide is now on the staff of the O'Reilly General Hospital, Springfield, Missouri.

December, 1943

Lieutenant Harl W. Matheson is with the Medical Detachment of the 34th Infantry, APO 25, c/o Postmaster, San Francisco, California.

Daniel F. Sullivan can be addressed

c/o Mrs. C. M. Sullivan, 14 Hilltop, R. R. 5, East St. Louis, Illinois.

Lieutenant Martin P. Meisenheimer writes us that his address is 7th Evacuation Hespital, APO 70, c/o Postmaster, San Francisco, California. He also says that he met a classmate, Harl Matheson, who was running a dispensary at a replacement depot only 20 miles from his own base.

Lieutenant Walter J. Kennedy, Jr., can be reached at 612 South 11th Street, Yakima, Washington.

Julius K. Neils' engagement to Miss Gloria Mary Peck was announced at a tea given July 7. The marriage will take place in September. Dr. Neils is a resident physician at St. Louis Children's Hospital, St. Louis 10, Missouri.

Reinhold Engleman is at the Robert Koch Hospital, Koch, Missouri.

September, 1944

Irvin Birenboim is a lieutenant at the Army-Navy General Hospital in Hot Springs, Arkansas. His home address is 520 East 61st Street Terr., Kansas City, Missouri.

R. B. Hunt is now in Clendenin, West Virginia.

John J. Rupp has gone to the Albany Hospital, Albany, New York.

Burford H. Burch is a lieutenant in the Medical Corps at Thayer General Hospital, Nashville 5, Tennessee.

June, 1945

Richard M. Strong has become engaged to Miss Vina Isach. Dr. Strong is interning at St. Luke's Hospital, Kansas City, Missouri. Miss Isach is a graduate of the Washington University School of Nursing and is on the nursing staff of Barnes Hospital.

Student News

Mrs. William F. Cheaney, the former Miss Mary Davis, was married to Dr. Norbert J. Bublis on Saturday morning, July 7, 1945. The bride is a senior in the Medical School.

Pfc. Robert Counts, a junior, has become engaged to Miss Jane Weightman Jones of Webster Groves. The marriage will probably take place this winter.

In Memoriam

Charles B. Leslie, Mo. '97, Meade, Kans., died August 8, aged 72. James B. Overton, '31, Talmage, Calif., died March 28, aged 40. William H. H. A. Vogt, Mo. '97, St. Louis, Mo., died June 17, aged 68. R. E. Wilson, '97, St. Louis, Mo., died July 14.

WASHINGTON UNIVERSITY

Arthur Holly Compton, Ph.D., Sc.D., LL.D., Bridge Chancellor

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

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

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

The School of Business and Public Administration Isaac Lippincott, Ph.D., Acting 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., 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., B.S., A.M., Director

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

University College
Willis H. Reals, Ph.D., 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: Complete information about any of the schools listed above may be obtained by writing to the Dean or Director concerned.