1941

UWOMJ Volume 11, No 2, 1941

Western University

The University of Western Ontario
MEDICAL JOURNAL
VOLUME 11 NUMBER 2

Present Concepts of Nephritis and Allied Conditions

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IN a monograph on diseases of the kidney published in 1827, Richard Bright was the first to associate dropsy, albuminous urine and diseased kidneys. He also made other observatons, some of which are still held true a century later. Since that time, many new laboratory and clinical methods of investgating the kidneys have been developed, so that today the diseases of the kidneys are classified into three major groups: nephritis, nephrosis, and nephrosclerosis.

Embryologically, the kidneys arise from the mesoderm of the intermediate cell mass and during ontogeny there are three successive sets of excretory organs, the pro-, meso- and meta-nephros. The pro- and meso-nephros are transient except for embryological rests, while the meta-nephros persists as the human excretory organ. The first organ to have Bowman's capsules and to show differentiation into secretory and collecting parts is the meso-nephros. In the meta-nephros, the tubulo-glandular and small collecting parts originate separately, the former from the nephrogenic bud and the latter from the meso-nephric bud, while the ureter, pelvis and terminal collecting tubules arise from the meta-nephric bud. Development in the meta-nephros simulates that in the meso-nephros, but results in a much more intimate association between secretory and excretory parts.

Anomalies such as polycystic disease, horseshoe-kidney and unilateral kidney are frequently seen as are ureteric anomalies. Occasionally, kidney tumours occur and according to the view now accepted, they are renal carcinomata, being derived from adult tubules or from islets of nephrogenic tissue which have persisted within the parenchyma.

The kidneys are retro-peritoneal organs, lying one on either side of the vertebral column in loose areolar and fatty tissues which forms a protective bed. They lie between the upper border of the 12th thoracic vertebra and the third lumbar vertebra, the right being slightly lower than the left.

Blood supply is derived from the abdominal aorta by the renal arteries which divide into four or five branches before entering the kidney. In the kidney substance these branches further subdivide and their final ramifications supply the glomeruli and tubules. An important feature of the blood supply of the nephrone is that each arteriole supplying a glomerulus also supplies the corresponding tubule.

The kidney receives its nerve supply from the renal plexus. This plexus is formed mainly by fibres from the coeliac and aortic plexuses, but also receives a few fibres from the splanchnic and vagi nerves.

The functional unit of the kidney is the nephrone, of which there are from three to four million. A nephrone consists of (1) malphigian corpuscle, consisting of Bowman's capsule and the invaginated glomerular tuft; (2) proximal and distal convoluted tubules, lined with striated cubical epithelium; (3) loops of Henle — the descending portion lined with flattened cells and the ascending part, with a smaller lumen, lined with larger cubical cells; (4) collecting tubules, lined with cubical cells and opening at the apex of the pyramids.

It is now fairly well accepted that the glomerulus and capsule which act as a filter are composed of a semi-permeable membrane through which certain constituents of the blood pass. The filtration depends mainly upon the blood pressure in the glomerular vascular loops and the osmotic pressure exerted by the plasma proteins. The capsular fluid is a protein-free filtrate resembling blood plasma without the proteins. It is made up of high, low, and non-threshold substances. High threshold substances such as glucose, chlorine, sodium, potassium, calcium and amino acids are re-absorbed with water in large quantities; uric acid, urea, phosphates and sulphates are low threshold substances, while creatinine is a non-threshold substance since it is not re-absorbed. The proximal convoluted tubules re-absorb all the glucose and 20 per cent of the water. The remainder of the water, the bases, and the haemoglobin in solution are re-absorbed by Henle's loops and the distal convoluted tubules. Best and Taylor[2] are of the opinion that tubular excretion of such substances as urea, creatinine, hippuric acid, ammonia, phosphates and dyes is normally of very little importance but probably is a functional inherited relic, the importance of which is more vital in chronic kidney disease involving the glomeruli.

The functions of the kidney can be summarized as follows: (1) It helps to regulate the blood volume and fluid content of the body. This is carried out by a selective action of the tubules on the absorption of water. For some reason, the tubules are able to absorb water or let it pass through, depending upon the body's needs. This may be due to a sensitivity of the tubular epithelium to changes in the blood, or on the other hand, to decreased pituitrin secretion resulting from the dilution of the blood. (2) It aids in eliminating the basic and non-volatile acid

radicals. As far as possible, the kidney eliminates the acid and retains the base, so that while the ratio Na H_2 PO_4 to Na_2 HPO_4 is one to four in the plasma, the kidney excrets Na H_2 PO_4 and Na_2 HPO_4 in the ratio of nine to one. By a combination of this and the first mentioned function, the osmotic relationships in the blood and tissues are regulated, and also, the reaction of the blood is preserved within narrow limits. (3) Another of the important functions is the excretion of unwanted materials. These are the nitrogenous and sulphur-containing waste products as well as artificially introduced toxic substances, *e.g.*, azo dyes. (4) Finally, there is the secretory function of the kidney, about which there is still considerable doubt. There is, however, some evidence that ammonia, hippuric acid and phosphates are formed by the tubules.

ACUTE NEPHRITIS

Etiology

Essentially, acute nephritis is the result of an infection in some part of the body. Almost every known micro-organism has been blamed, but since the disease occurs most commonly following upper respiratory tract infections and as a complication of scarlet fever, the streptococci are still thought to be the most important causative organisms. Less commonly it follows such conditions as pneumonia, rheumatic fever, impetigo, sub-acute bacterial endocarditis, syphilis, toxic agents and even gonorrheal septicaemia. These conditions or their toxins may simply prepare the tissues for streptococcic invasion and may not in themselves produce nephritis.

Pathology

Acute nephritis is simply the local picture of a generalized reaction. The present concept is that nephritis is caused by an organism of low virulence, the antigen of which sensitizes the glomerular tufts and the vascular endothelium; this allergic reaction is manifested as a proliferation of endothelial cells (endotheliosis) which has been called an inflammation. If this theory be correct, then the pathology is not primarily an inflammatory one but an allergic response. This generalized endotheliosis produces an arteriolar narrowing, which increases the peripheral resistance and thus accounts for the primary rise in blood pressure in acute nephritis. The formation of intracapillary hyaline material plus the endotheliosis further decreases the lumen of the capillaries in Bowman's capsule. As a result of this glomerular obstruction, the corresponding tubules become ischaemic and show varying degrees of degenerative changes. The presence of polymorphonuclear leucocytes in the glomerular tufts and in the surrounding tissues suggests an inflammatory change. The capsular fluid contains an albuminous exudate, leucocytes and fibrin.

Usually both kidneys are enlarged, the capsule is tense, and the

glomeruli can be seen as red or grey dots, depending upon the degree of vascular congestion.

The general architecture of the kidney in acute nephritis is preserved. In order to obtain a clear concept of the pathology one should consider a single capsular loop. Normally, each loop is covered externally by a layer of epithelium and lined by endothelial cells. The epithelial cells first hypertrophy, then undergo hyperplasia, so as to fill the space between the loops. Later, these cells usually degenerate and are cast off into the capsular space. After this, the endothelial cells swell and proliferate to such a degree that the lumen is narrowed, and later, become further narrowed by the formation of intracapillary hyaline material. The capsular space may be seen to contain an exudate composed of albumin, fibrin, leucocytes and red blood cells. The condition of the tubules varies with the corresponding glomerular damage. In anaemic glomeruli, tubular degenerative changes are slight. The epithelial cells show cloudy swelling, perhaps fatty degeneration, and occasionally the tubules contain epithelial cells. Other tubules may be normal. Far advanced tubular damage is rarely seen in acute nephritis and if present is believed to indicate an older process.

Signs, Symptoms and Laboratory Findings

Nephritis produces changes which can be demonstrated by examination of the eye grounds, blood pressure, heart, blood and urine.

Acute nephritis may onset insidiously, or in other cases, the patient may not feel particularly ill but present only a swollen face or swollen eyelids. However, in children it is common for acute nephritis to begin suddenly. Here it is associated with general malaise, slight fever, headache, nausea, vomiting, lumbar pain and perhaps convulsions.

If the pathology is interpreted in terms of function, it is conceivable that eye ground changes are uncommon; the blood pressure may show a moderate rise (20 to 30 mm. Hg.) ; the heart shows no changes; while the blood chemistry and kidney function tests are unaltered. At this early stage, then, the most important changes are seen in the urine. The damaged glomeruli necessarily excrete a decreased amount of urine, of a high specific gravity, smoky or reddish in appearance, and containing albumin, erythrocytes and leucocytes. Depending upon the amount of tubular damage, there may or may not be found hyaline and granular casts.

FOCAL NEPHRITIS

Some authorities have described an acute focal nephritis which differs from the diffuse type in that it is localized to a part of the kidney, and early, does not show any clinical changes except in the urine. In contrast with acute nephritis it occurs at the height of the infection, with actual bacterial invasion of the kidney. This condition

is often discovered on routine urine analyses. Usually, there is a gradual spread, resulting in a terminal picture of renal failure.

In the gross, the kidney shows small haemorrhages on the surface and microscopically, these haemorrhages are into the capsular spaces. Frequently, also, accumulations of leucocytes are seen in the glomerular tufts.

SUB-ACUTE NEPHRITIS *(Chronic Parenchymatous Nephritis)*

Pathology

Classically, the kidney in sub-acute nephritis is described by the pathologist as the "large white kidney," a term also used for the kidney in nephrosis. The organ is pale and usually enlarged, but this enlargement may be slight or absent, so that the gross appearance can be very misleading in this as well as in other stages of nephritis.

The normal architecture of the kidney in sub-acute nephritis is markedly altered. The glomeruli are considerably enlarged and ischaemic, some are fibrosed with their tubules degenerated, and there are many round cells in the increased interstitial tissue. Essentially the lesions are a continuation of the first (acute) stage of nephritis, but with a greater preponderance of degenerative changes. In some of the glomeruli, progressive hyalinization plus the preceding endotheliosis leaves the glomeruli as hyaline masses. In others, the capsular epithelium proliferates into folds, forming epithelial crescents. In still other glomeruli, the capsular space is obliterated by the fusion of the capsular and vascular epithelium. The capsular fluid contains red blood cells, desquamated cells, albumin and fibrin.

Most of the tubules present a picture of degeneration, the amount depending upon the damage in the corresponding glomeruli. The epithelial cells show cloudy swelling, fatty degeneration or even necrosis. This degeneration leads to desquamation and the formation of casts in the tubules. Special stains reveal large amounts of lipoid material in the cells, which may be secondary to the ischaemia.

The interstitial tissue shows replacement fibrosis and accumulations of round cells. As a rule, the blood vessels in this stage show few changes, but evidences of the on-coming third stage, such as distinct intimal thickenings, may be found.

Signs, Symptoms and Laboratory Findings

The onset of sub-acute nephritis is usually gradual and may or may not follow a clinically recognizable acute stage. However, this is the stage of oedema, which may be extreme, resulting in effusion into the pleural, pericardial and peritoneal cavities, or even anasarca. Malaise and pallor may be marked. Early, the fundi may show no changes, but later an "albuminuric retinitis" is seen. The blood pressure usually is

normal at the onset, but as the disease progresses a moderate hypertension may develop. The degree of cardiac hypertrophy depends on the duration of the hypertension. The blood begins to show changes such as high cholesteral, reversal of the albumin-globulin ratio, and perhaps an increase in the non-protein nitrogenous substances, as well as a secondary anaemia. In spite of the renal damage, the kidney function tests are often normal. The urine still shows very characteristic changes; the oliguria persists, the specific gravity is high, and the albuminuria is marked with the presence of numerous casts (hyaline, granular, cellular and fatty), fewer erythrocytes and a moderate number of leucocytes.

CHRONIC NEPHRITIS

Pathology

The kidney in chronic nephritis is called the "small white kidney" or by others, the granular contracted kidney. Here the capsule is adherent with small retention systs on the surface; the pelvis may be dilated; and the walls of the arteries obviously thickened.

In chronic nephritis there is an almost complete loss of renal structure by scarring, and replacement fibrosis of the parenchyma. The blood vessels show diffuse hyperplastic sclerosis and endarteritis obliterans.

Signs, Symptoms and Laboratory Findings

This is the end stage of nephritis. Even prior to renal failure, the body shows widespread damage. This condition usually has an insidious onset and is marked by increasing debility, pallor, loss of weight and strength, digestive disturbances and headaches. As a rule, the fundi show haemorrhages and exudates, choking of the discs, and the arteriolar sclerosis which is associated with the moderate hypertension. The heart is hypertrophied and signs of cardiac failure may develop. Early in the course of the disease, the blood picture is essentially normal, but eventually the non-protein nitrogenous substances increase, and a progressive anaemia ensues. The dilution and concentration kidney function tests, as well as the urea clearance tests, are impaired early. The urine reflects the function of the badly damaged kidneys. The loss of concentrating power results in a low, fixed specific gravity of the urine and polyuria. The amount of urine excreted at night is equal or may even exceed that during the day. The cellular and other elements are decreased. There is a slight albuminuria, with casts, especially hyaline, also a few erythrocytes and leucocytes.

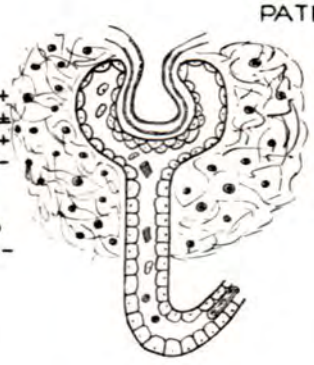
NEPHROSIS

Nephrosis as an entity is debatable. Some pathologists label as nephrosis only those degenerative conditions which give rise to oedema, massive albuminuria, hypercholesterolaemia, reversal of the albumin-

ACUTE NEPHRITIS

CLINICAL RESUMÉ

SUDDEN ONSET
GENERAL MALAISE
OEDEMA (in loose tissues) +
EYE GROUNDS ±
BLOOD PRESSURE +
HEART –
URINE HAEMATURIA
ALBUMINURIA
SP. GR. ELEVATED
BLOOD –



PATH.- PROLIFERATIVE

SWOLLEN GLOMERULI
ENDOTHELIOSIS
INTRACAPILLARY HYALINE MATERIAL
ISCHAEMIA OF CORRESP'G LOOPS
P.M.N'S INCREASED
EXUDATE

SUB-ACUTE NEPHRITIS

DEGENERATIVE

OEDEMA ++
EYE GROUNDS +
BLOOD PRESSURE ±
HEART ±
URINE
ALBUMINURIA
CASTS
OLIGURIA
SP.GR. ELEVATED
HAEMATURIA ±
BLOOD
N.P.N. RISING
HIGH CHOLESTEROL
ANAEMIA
REVERSAL OF ALB.-GLOB. RATIO

CONTINUATION OF 1st STAGE
SOME GLOMERULI OCCLUDED, OTHERS ONLY PARTIALLY
EPITHELIAL CRESCENTS
TUBULES
CLOUDY SWELLING
FATTY DEGEN.
CASTS
INCREASED INTERSTITIAL TISSUE (REPLACE'T FIBROSIS)
BL. VESSEL CHANGES

CHRONIC NEPHRITIS

SCARRING

HYPERTENSION
EYE GROUNDS +
HEART CHANGES +
URINE
SP.GR.- LOW + FIXED
ALBUMINURIA } SLIGHT
CASTS }
BLOOD CHANGES
NITROGEN RETENTION
SECONDARY ANAEMIA

FEW SCATTERED HYPERTROPHIC GLOMERULI
MANY HYALINIZED GLOMERULI
TUBULES
MARKED ATROPHY
FEW DILATED (HYPERTROPHIC)
INTERSTITIAL TISSUE
MARKED INCREASE
(REPLACE'T FIBROSIS)
ROUND CELL INFILTRATION
ARTERIES
ENDARTERITIS OBLITERANS
ARTERIOLARSCLEROSIS

globulin ratio, persistently normal renal function tests and low basal metabolism rate but which do not produce haematuria, retinitis or hypertension. Nephrosis is primarily a result of tubular degeneration, but in special types of nephrosis pathology is not thus restricted, but also involves the glomeruli. It may be that, fundamentally, true nephrosis is a hyperpermeability of the glomerular capillaries, leading to a secondary tubular degeneration. Boyd[3] considers that lipoid nephrosis is the only true type. Platt[8] gives the impression that lipoid nephrosis and sub-acute nephritis should not be looked upon as entities, but rather, that nephrosis is a variety of sub-acute nephritis, in which some of the findings are minimal or absent. He substantiates this view by saying that both conditions commonly arise in connection with some septic process. Certain English authors regard lipoid nephrosis and chronic nephritis as synonomous terms. Clinically, on the other hand, cases do fall into line with our above-mentioned definition and as the nephrotic kidneys are seldom seen at necropsy, and when they are, the condition is a long-standing one and undoubtedly shows superimposed infection or other terminal changes. Lipoid nephrosis is a condition which the pathologists have not been able to include among the inflammatory changes. Whether lipoid nephrosis is accepted as an entity or not appears to depend upon whether a clinical or pathological viewpoint is held. In this paper, lipoid nephrosis will be considered as an entity and the other types of nephrosis discussed separately.

LIPOID NEPHROSIS

Pathology

The kidney is large and pale and has been described as the "large white or myelin kidney." On section, the cortex is swollen and pale, and yellow spots or streaks are often discernible.

Lipoid nephrosis presents a microscopic picture which is essentially a tubular degeneration. The convoluted tubules are dilated and the greatly swollen epithelium contains large numbers of droplets of neutral fat. In addition to this fatty degeneration, numerous other droplets are seen on looking at frozen sections through Nicol's prisms. These are composed of myelin, an ester of cholesterol. Since this material is filtered through the glomeruli and absorbed by the tubules, lipoid nephrosis may be regarded as a lipoid infiltration. According to our definition, the glomeruli are not affected in lipoid nephrosis, but Bell[1] states that special staining technique does show definite lesions in every case. These consist of a proliferation of the capillary endothelium of the tuft and a thickening of the basement membrane. This proliferation produces only a partial capillary obstruction. This is important, in that it differentiates this condition from glomerulonephritis and also explains the absence of hypertension in lipoid nephrosis.

THE KIDNEY OF THE TOXAEMIAS OF PREGNANCY

The etiology of kidney lesions in pregnancy is still not understood, but several theories have been suggested. Formerly, the hypothetical toxin which was supposed to cause the damage of the other organs was blamed, but there appeared to be little to support this idea. It seems probable that vascular lesions are of most importance. Elwyn[5] suggests that the advancement of pregnancy may cause an increased irritabilty of the neuro-muscular apparatus of the blood vessels, resulting in vascular spasm and hypertension. Volhard, as quoted by Boyd, states that afferent glomerular arteriolar spasm, of undecided etiology is the underlying pathology. This leads to a degeneration of the glomerular capillaries, resulting in albuminuria. The tubular degeneration is probably of secondary consequence.

The kidney appears to be attacked in its entirety. The glomerular loops are swollen and thickened, due to marked hypertrophy of the capillary basement membrane, without nuclear proliferation. The afferent arterioles frequently show changes similar to those of essential hypertension. The tubules present a picture more like that of nephrosis than of nephritis. The epithelium of the proximal convoluted tubules shows marked albuminous and fatty degenerations. These cells may contain cholestrol esters.

This condition occurs during the last trimester of pregnancy and onsets gradually with headache, slight malaise and oedema of the legs. As these cases progress towards eclampsia, vomiting, epigastric distress, failing or even loss of vision, oliguria and increased oedema supervene. The eye grounds may show an "albuminuric retinitis"; a moderately severe hypertension develops, but the heart shows no hypertrophic changes unless the condition is of long duration. Blood urea and total non-protein nitrogenous substances show little or no elevation. The urine, which is diminished in amount, has a high specific gravity and contains much albumin as well as granular and hyaline casts. All of these symptoms usually disappear with the termination of pregnancy, but in some cases a picture of chronic nephritis may persist.

CORROSIVE SUBLIMATE POISONING *(Mercurial Nephrosis)*

Clinically, corrosive sublimate is the most important poison to the kidneys, particularly since it is prone to cause death from anuria. The pathological picture in these swollen pale kidneys is one of acute nephrosis. The glomeruli are unaffected, but the tubules, especially the convoluted ones, show extensive areas of necrosis. The necrotic cells are sloughed off and may block the tubules, causing anuria. "Acute calcification," by the deposition of lime salts in these necrotic cells, frequently occurs within a week. If the kidneys can eliminate the cellular debris, the tubular epithelial cells will regenerate and recovery be complete.

The clinical picture is one of increasing oliguria and perhaps anuria for one to two days; a rising non-protein nitrogen and blood pressure, especially during the stage of anuria; but no oedema. During the stage of epithelial regeneration, the patients will excrete a urine of low specific gravity and containing albumin. Clinically, they may become negative or show a picture of chronic nephritis.

AMYLOID NEPHROSIS

Amyloid disease usually follows chronic infections such as suppurative, tubercular or syphilitic lesions but there are some idiopathic cases. Clinical and pathological evidence is conflicting since the disease has features characteristic of nephrosis, nephrosclerosis and even nephritis. Clinically, it does resemble a nephrosis by the oedema and albuminura, but it differs in that there is the added picture of hypertension and rising non-protein nitrogen. Pathologically, it differs from nephrosis in that the lesions are primarily in the glomeruli and not in the tubules. An adequate explanation of amyloid disease is still lacking. These chronic processes may excite in some way the production of a compound composed of albumin and chondroitin-sulphuric acid.

The kidney is large and firm, closely resembling the large white kidneys of lipoid nephrosis and sub-acute nephritis. In amyloid disease, however, the organ is usually much firmer and with a consistency of india-rubber.

Amyloid disease is essentially a connective tissue change and the amyloid is deposited in the kidney in three special sites, namely, the glomeruli, arterioles and around the collecting tubules. In the glomerular loops, the amyloid substance is deposited between the basement membrane and the endothelium, thereby diminishing the circulation, and in time the glomerulus is converted into a hyaline mass. Occurring concommitantly and as a result of the above changes, the tubules degenerate and the epithelium is seen to contain fatty and hyaline droplets and perhaps cholesterol esters. Later, these tubules atrophy and are replaced by fibrosis and amyloid material. The atrophy of the kidney is further aggravated by the deposition of amyloid in the arteries.

The kidney dysfunction occurs insidiously and is masked by the accompanying disease. Early oedema may be the only sign, but later, ascites may also develop. Hypertension may be an early and marked sign, and if present, the fundi and heart will show changes referable to the degree and duration of the increased blood pressure. The blood shows hypercholesterolaemia, decrease in plasma proteins, secondary anaemia, and an increase of the non-protein nitrogen. Renal function tests may yield normal results, but the Congo Red test is diagnostic and indicates the degree of amyloidosis. The urine has a high specific gravity, massive albuminuria and epithelial and "colloid" casts.

OTHER FORMS OF NEPHROSIS

At times, nephrosis results from excessive metabolic poisons such as blood pigment, bile acids, uric acid and glycogen. In this type, deposits occur in the cells of the loops of Henle or convoluted tubules. Such nephroses are usually transitory and clear up when the underlying cause is irradicated.

NEPHROSCLEROSIS

Nephrosclerotic kidney disease includes the renal pictures associated with benign and malignant hypertension as well as that of old age or, in other words, arteriosclerosis. The etiology of arteriosclerosis seems to be closely bound up with the process of aging, about which very little is known. The cause of benign or essential hypertension is debatable. It seems to be associated with emotional strain, endocrine and neurological changes and renal ischaemia. Probably one or more of these factors are implicated in each case. Malignant hypertension shows necrotic changes of unknown origin in the arteries.

THE ARTERIOSCLEROTIC KIDNEY

Pathology

The arteriosclerotic kidney is often contracted with scars, suggesting old infarcts. The renal artery shows marked atheromatous changes and in severe cases the sclerotic interlobar arteries are prominent.

The gross appearance suggests the microscopic findings. Because of the fan-shaped arterial distribution, triangular areas of hyalinized glomeruli and fibrosed tubules are observed. Intervening areas appear to be essentially normal. In some of the triangular zones, the proximal convoluted tubules may be unchanged or hypertrophied because of a newly formed blood supply to these tubules. These hypertrophied tubules are believed to function like glomeruli.

ESSENTIAL HYPERTENSION

Pathology

The kidney of essential hypertension is classically described as the "primary contracted kidney," but the gross appearance depends upon the duration and severity of the vascular lesions. Early, except for some congestion, the kidneys appear normal. Later, they become small, firm, granular, shrunken, and small cysts may form on the surface. In the gross it is usually impossible to distinguish this type from the secondarily contracted kidney of chronic nephritis.

In essential hypertension, the microscopic picture is probably more varied than in any other renal disease. Wedge-shaped areas of atrophy are seen as in the senile arteriosclerotic kidney, while the intervening glomeruli and tubules are either normal or they may become hypertrophied, as a compensatory mechanism.

In the glomeruli, the capillary basement membrane is thickened and wrinkled, the capsule shows a fibrous thickening, and these changes occurring together result in a complete obliteration of the capsular space. Other glomeruli, due to relative ischaemia, are converted into hyaline masses with fibrous replacement of the corresponding tubules. The unaffected tubules may undergo a compensatory hypertrophy or dilatation or appear normal.

It is in the arterioles that pathognomonic changes are found. The afferent arterioles present a picture of uniform hyaline thickening of the sub-endothelial layer of the intima. This thickening is greatest at the arterio-glomerular junction, and prior to the hyaline deposition, a "lake-like" dilatation of the arteriole at this area is seen. Later, fat is deposited in the intima. The interlobar arteries later show diffuse hyperplastic sclerosis.

MALIGNANT HYPERTENSION

Pathology

The gross appearance of the kidney in malignant hypertension resembles the "flea-bitten" kidney of focal embolic glomerular nephritis. Frequently the two kidneys differ in size; they may be granular and contracted, or normal in size and smooth. The presence of a "flea-bitten" kidney in a known case of essential hypertension is practically pathognomonic of malignant hypertension.

The microscopic picture is one of two added aretriolar lesions, either a necrosis of the arteriolar wall, or an endarteritis obliterans, and both may be superimposed on the changes resulting from benign nephrosclerosis. The arteriolar necrosis is represented as patchy areas of necrosis, aneurysmal dilatations and irregular outline of the vessel walls. The nuclei of the endothelial cells disintegrate and disappear with a resulting destruction of the intima and media. This may be extensive enough to result in haemorrhages into the surrounding tissues. Thromboses are prone to develop in these necrotic walls, often obliterating the entire vessel. The small arteries and arterioles exhibit endarteritis obliterans. In other words, the picture may be described as the glomerular lesions of benign nephrosclerosis to which patchy areas of necrosis are added, as well as the epithelial proliferation seen typically in acute nephritis. The tubular changes are predominately degenerative rather than atrophic, since the vascular pathological lesions occur rapidly.

The clinical pictures and differential diagnosis of the types of nephrosclerosis may be summarized as follows:

ARTERIOSCLEROTIC KIDNEY DISEASE

Insidious onset in old age.

Eye grounds show arteriosclerotic vascular changes.

Blood pressure is slightly increased, with relatively low diastolic pressure.

Heart is usually normal or atrophic.

Blood chemistry may reveal nothing except a rising N.P.N. as a terminal feature.

Urine shows (1) low, fixed specific gravity,
(2) occasional casts,
(3) slight albuminuria.

Death is usually due to cerebral thrombosis or thrombosis elsewhere, rarely by renal failure.

BENIGN NEPHROSCLEROSIS *(Benign Hypertension)*

Gradual onset in middle age.

Eye grounds show retinal arteriolar sclerosis, increased tortuosity and light reflex, arterio-venous compression, irregular narrowing of lumen, haemorrhages and exudates. These usually occur in the above sequence and indicate the severity of the condition.

Blood pressure is always elevated and the diastolic pressure is of greater value in prognosis.

Heart is enlarged to grade ii, on grade iv basis.

Cerebral haemorrhages occur in about 25 to 33 per cent of cases.

Blood picture is negative except with renal failure.

Urine shows (1) lowering of specific gravity,
(2) gradual loss of concentration,
(3) increasing number of hyaline casts,
(4) trace of albumin.

Death is usually due to heart failure in 60 per cent of cases, cerebral haemorrhage in 25 per cent of cases, and renal failure in 15 per cent of the cases.

MALIGNANT HYPERTENSION

There are two types:

(1) Superimposed or the terminal phase of benign nephrosclerosis.

(2) Primary acute fulminating type occurring in the young.

(In the latter type, onset is sudden, with development of cachexia, while in the former type the onset is gradual.)

Eye grounds show a severe retinal arteriolar sclerosis with choked discs.

Blood pressure is elevated with a diastolic pressure above 125 mm. of Hg.

Heart is enlarged to grade iii.

Blood changes: Urea, creatinine, N.P.N. all elevated; secondary anaemia.

NEPHROSIS

CLINICAL RESUMÉ

OEDEMA
EYE GROUNDS
B.P. + HEART - NORMAL
LOW B.M.R.
URINE
- ALBUMINURIA
- CASTS
- LIPOID BODIES

BLOOD
- PROGRESSIVE ANAEMIA
- SALT RETENTION
- PLASMA PROTEINS DECREASED & REVERSAL OF ALB.-GLOB. RATIO
- HYPERCHOLESTEROLAEMIA

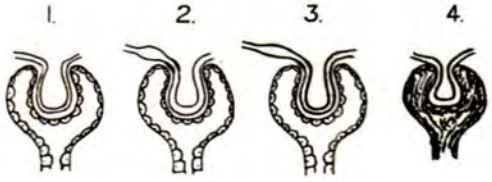
PATHOLOGY

GLOMERULI
- CAP. ENDOTHELIAL PROLIFERATION
- THICKENED BASEMENT MEMBRANE

TUBULES
- DILATED
- SWOLLEN EPITHELIUM
- FATTY DEGEN.
- MYELIN DEPOSITS IN CELLS

NEPHROSCLEROSIS

BENIGN



1. ARTERIOLAR SPASM
2. LAKE-LIKE DILATATION OF ARTERIOLE
3. CAPILLARY BASEMENT MEMBRANE THICKENED + WRINKLED
   THICKENING OF INTIMA
   ARTERIOLAR SCLEROSIS
4. CONTINUATION OF (3)
   OBLITERATION OF CAPSULAR SPACE

   GLOMERULI
   - SOME FIBROSED, OTHERS SHOW COMPENSATORY HYPERTROPHY

   TUBULES
   - VARY & GLOMERULAR CHANGES

MALIGNANT

MODES OF ONSET
1. TERMINAL PHASE OF BENIGN
2. ACUTE FULMINATING

EYE GROUNDS +++
BLOOD PRESSURE +++
HEART ++
URINE
- ALBUMINURIA
- CASTS
- R.B.C.

BLOOD
- RISING N.P.N. (URAEMIA COMMON)
- SECONDARY ANAEMIA

FAR ADVANCED LESIONS OF BENIGN
+
1. ARTERIOLONECROSIS
2. ENDARTERITIS OBLITERANS

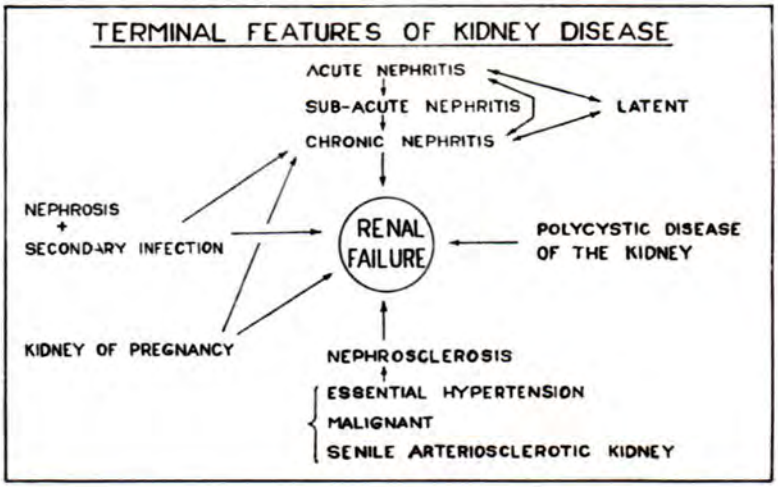
Urine shows (1) concentrating power last,
(2) specific gravity below 1010,
(3) albuminuria and casts marked,
(4) blood and pus present.

Death is usually due to uraemia or cerebral haemorrhage.

RENAL FAILURE *(Renal Insufficiency + Uraemia)*

Renal insufficiency is the end stage of all kidney conditions. There are two forms, which may occur separately or together: it may be an inability to eliminate water and sodium chloride, manifested by oedema, or it may be an inablity to eliminate the nitrogenous waste products of the blood, which is manifested by uraemia. Renal insufficiency is characterized by albuminuria, polyuria, low fixed specific gravity of the urine, and later by the development of oedema or uraemia; in other words, the picture of chronic nephritis. Although seen typically here, the syndrome of renal insufficiency is not a specific one, since it occurs wherever there is sufficient destruction of renal tissue. The inability of the tubules to absorb is manifested in oedema, resulting from a gradually increasing loss of albumin with a subsequent reversal of the albumin-globulin ratio and also a retention of salt in the blood. There is no increased accumulation of waste nitrogenous products in the blood unless there has been a previous nephritis. Uraemia is the final manifestation of renal failure to secrete the main waste products, of which the non-protein nitrogenous substances are the most important. However, clinical evidence shows that the concentration of the blood N.P.N. varies at the onset of convulsions. The causative agent of convulsions in uraemia is not known.

The accompanying diagram shows graphically the various phases through which the common kidney diseases progress before renal failure occurs.



SUMMARY

In this paper the author has attempted to correlate the clinical and pathological classifications of kidney diseases, and to interpret the clinical changes on a physiological-pathological basis. The diseases discussed have been classified under three groups, namely: nephritis, nephrosis and nephrosclerosis. No new work is presented here.

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Leonardo Da Vinci: Anatomist*

By JOHN G. STAPLETON, B.A., '41

IT is frequently stated that this is an age of specialization in which those who would make great contributions in literature, science, or art, must confine their endeavours to a very narrow field in order to excel. It would be equally true to say that the period of the Renaissance was an age of versatility. The great variety of realms of knowledge which felt the influence of single individuals in those days constantly amazes us who have been reared in the modern atmosphere of specialism. Probably no one person left such an imprint on so many widely diversified fields as did that great Florentine, the leader of the Golden Age of the Renaissance, Leonardo Da Vinci. His lifetime stretches over that great period centering around the close of the fifteenth century, when the increasing birth pangs of two centuries had given to the world that rebirth of knowledge and intelligent speculation without which it had languished in darkness for many centuries. In such a world, there was almost no limit to what could be achieved by one who was wise enough and fearless enough to reject the well-tracked roadsteads of tradition and chart a new course into unknown seas. Leonardo Da Vinci was such a man. His versatility was so amazing that there is surely nothing like it in the history of science. His name is a household word as a great artist, the originator of the Virgin of the Rocks, the inspired creator of the Mona Lisa, the man whose spirit conceived the masterpiece that is the Last Supper. His contributions to the new technique of the use of contrasting light and shade and his accurate perspective, an outgrowth of his passion for mathematics, have stamped his name indelibly upon the pages of the history of art. But his prolific genius was turned as well to so many other fields that Osler refers to him as a great "universal genius," the intellectual equal of Aristotle. As an architect, he was the builder of bridges, canals, and castles which were the wonder of his time. His studies of the flight of birds gave him knowledge of almost all the principles of heavier-than-air flight—centuries before Lilienthal and Langley. In addition, he was a chemist, a sculptor, a musician, an engineer and a natural philosopher. And finally, he is eminently deserving of his place in the history of medicine because of his work as an anatomist—an anatomist who had the rare privilege of being his own illustrator; who was able to show with a few strokes of his facile pen and brush what all the verbosity of his scholastic contemporaries could never have made intelligible, even had they understood it. As an anatomist, therefore, we accord to Leonardo a place in the front rank of the immortal pioneers of medicine.

The life of Leonardo Da Vinci is so interesting in all its aspects that it is only with difficulty that we can confine ourselves to the study of any one phase of it. He was born in the little village of Vinci, near

* Read at the December Meeting of the Osler Society.

Florence, in 1452, the illegitimate love-child of a well-to-do Florentine notary and a humble village barmaid. His father openly acknowledged the boy and brought him up in his own house, providing him with a fairly liberal education until, at the age of 18, he entered the studio of Verrocchio to study painting. Seven years later he commenced his own artistic career under the patronage of Lorenzo di Medici, a career which is marked by repeated migrations from one patron to another and by an almost incredible variety of activity in many fields. He died at Amboise in France in 1519, having spent the last four years of his life in the service of Francis I of France.

In order to understand Leonardo's place as an anatomist we must, of course, consider the world in which he lived and the knowledge which was then available. He lived his life at a time when the vitalizing force of the Renaissance, which began about 1300, still had only partly severed the shackles of blind adherence to the past. Science lagged behind art in this great movement of emancipation, and anatomy particularly was still dominated largely by the Galenic tradition. With the death of Galen at the end of the second century the study of anatomy had entered upon its dark days, and for almost thirteen centuries men learned nothing new of the structure of the human body. The so-called Dark Ages were the days of dogmatism. Faith and not reason was upheld as the basis of all knowledge of both the natural and supernatural worlds and, of the two, men were more concerned with the supernatural. There existed no stimulus to personal observation or to experiment, and science languished and anatomy became conventionalized largely according to the Galenic tradition, which possessed a finality and unassailability which precluded any question. Had it not been for the appreciation by the Arabs of what was best in the philosophy and science of the Greeks even Galen would have been lost. But they were indefatiguable translators who saved for the world much that would have perished in the medieval darkness. Although they contributed little, they were the keepers of the light throughout the Dark Ages when the Greek language of Galen, Aristotle and Hippocrates had become practically extinct. Arabistic versions of the masters were the sources of most of the anatomical knowledge of Leonardo's time, and were the basis of the two anatomies which Leonardo probably used, those of Mondino and Avicenna. Mondino's "Anathomia" undoubtedly served as an introduction to Leonardo's anatomical studies and as a source of most of his nomenclature. He probably was familiar also with the "Canon" of the Arabian Avicenna, which was very popular in the schools of medicine of the time, and went through many editions during Leonardo's lifetime. Probably Leonardo's indebtedness to his predecessors was limited almost entirely to what he found in these two books, with the possibility that he may have occasionally referred to Pliny, Celsus, Galen, Aristotle, and Hippocrates, probably in Arabistic translations.

Nevertheless, in the emancipated atmosphere of the Renaissance some practical progress was being made. Dissection of cadavers was frequently performed in Italy in the 14th and 15th centuries, and the practical study of anatomy was the rule at Padua, Bologna and Pisa. Hence Leonardo commenced his anatomical studies in a world where actual dissection was a well-recognized feature of medical instruction. Nevertheless little that was actually new was being added to anatomical knowledge due to the speed and superficiality of the dissection, inability to preserve the material, and reliance throughout upon the written text. Dissection was a privilege that was granted to the guild of artists as well as to the physicians and surgeons, and Leonardo himself performed many autopsies at the Ospedale Santa Maria Nuova at Florence.

We cannot say that Leonardo erased all errors and founded modern anatomy single-handed. To Vesalius, a generation later, goes the distinction of being the founder of modern anatomy. But, as McMurrich says, "Leonardo was his forerunner—a St. John crying in the wilderness." Certainly we can agree with William Hunter that he was "the greatest anatomist of his epoch," and no one can deny that in scientific accuracy many of his drawings eclipse those of Vesalius. Leonardo was singularly fortunate in that his knowledge of Latin and Greek was meagre and insufficient for abundant reading. Thus he was spared the dead weight of that dialectical and empty learning which had accumulated for centuries, and which by its authority made originality well nigh impossible.

The revolution accomplished by Leonardo was brought about chiefly by his use of accurate illustration. No lecturer, or essayist, he left little that could be called a written text, but he laid great emphasis upon the use of illustration as a didactic method. Thus it comes about that Leonardo's anatomical manuscripts are largely collections of drawings, accompanied by a few secondarily added notes suggesting further observation or illustration. Some of the drawings are very elaborate, others are very simple. Many are sufficiently finished to require no further additions; others are incomplete outline sketches, and still others are merely diagrams. He filled many pages with drawings that are merely crude suggestions — notes on problems requiring further study. Professor McMurrich has classified the more finished drawings into three main types:

(1) Representations of Leonardo's personal observations, without modification due to preconceived notions. He made many such drawings, some of them suitable for illustrating a modern textbook.

(2) Representations of structures which he had not himself sufficiently investigated; in these he was influenced by the dictates of tradition, a serious enough mistake but one which is daily repeated in all modern anatomy and histology classes.

(3) Representations of human structures on the assumption that they were the same as those of animals, a Galenic error; for Galen's

work was founded largely upon dissection of monkeys, a fact that was lost sight of in the medieval translations by which he was largely known. So we find Leonardo representing a canine hyoid bone in the human throat and the cotyledons of the cow's placenta in the human.

Leonardo's inaccuracies are inevitable in the work of a pioneer, and even with them he was representing human structures with an exactness and a skill never seen before. We have only to compare Leonardo's figures with other contemporary ones to realize how important was his revolution in anatomical illustration in establishing the foundations of modern anatomy. Not only did he illustrate better but his whole approach to the subject was different, emphasizing nature as a model, and turning away from highly conventionalized diagrams based largely upon hoary anatomical traditions. The difference is striking when one takes an example. In the 1491 edition of Ketham's "Fasciculus Medicinae," there appears the earliest printed figure of the internal organs, an extremely diagrammatic drawing of a female figure in a highly conventionalized posture. It was obviously drawn by someone quite unfamiliar with anatomy, for not one of the organs shows its true form or relations. Leonardo's contemporary figure of the female situs viscerum is quite different. Though it embodies many inaccuracies, too, it does attempt to portray correctly the form and relations of the organs, a profoundly significant difference because it shows evidence of a modern scientific approach to the problem of illustration—a change similar to what had already taken place in painting, where the conventionalism of Byzantine art had been superseded by the return to nature for a model inaugurated by Cimabue and Giotto. Leonardo was a pioneer here in a field that was later successfully exploited by Vesalius and Eustachius, and his work offers a profound contrast to the artistic crudity of that of his contemporaries. In his works, he combines also the appreciation of form and the accurate draughtmanship of a great artist. Thus a new period in the history of anatomy was inaugurated—a period when the anatomist and the artist were to collaborate in obtaining and recording advancing knowledge. Leonardo was unique in the breadth of his curiosity in a world where all curiosity was discouraged. His insatiable desire to know nature in all her aspects led him far beyond the confines of mere artistic anatomy which bound his fellow-artists, like Michelangelo and Raphael. With his enormously versatile mind he endeavoured increasingly to be simultaneously active in several subjects quite foreign to his art. His interest in form was in no way secondary to a profound curiosity about function. He was especially interested in the problems presented by the actions of muscles and the flow of blood through the heart. In fact, he seems to have planned all his life a monumental textbook which was to bear his name and was to contain the results of his investigations of the structure and function of the human body, combining physiology with anatomy as he always did. All his life he made illustrations and scraps of notes which were

intended for his magnum opus. But his conception was so vast that it was far beyond his ability to execute the work. The book, if completed, would have been an encyclopedia of man, a compendium of all knowledge about his form, function, growth and action from conception to the grave. As in all things, Leonardo's interest lay not in planning or in executing small things—his dreams were panoramic and his visions vast, which is the reason that he left so little finished work. Occupied as he so often was with the plans and preliminaries of a sublime conception, he was so critical of his own efforts that his actual production was quite small. His lifelong anatomical work, nevertheless, is quite extensive, and we can only refer to some of it. He left at least 750 separate anatomical sketches. He was greatly interested in the heart, its mechanism and structure; he recognized four chambers, instead of two like his contemporaries, and showed correctly the columnae carnae, papillary muscles, and moderator band. He traced the left vagus nerve to the heart and speculated about its relation to the heart beat. He says of the heart: "the heart in itself is not the beginning of life, but it is a vessel made of muscle, vivified and nourished by the artery and vein, as are the other muscles." This is interesting, for Galen considered the heart a tissue sui generis, not a muscle. He did fall, however, into the pitfall of Galenic tradition regarding the circulation of the blood, and even figured the pores in the heart's septum referred to by Galen, thus proving himself human and fallible, as even modern scientists are human and fallible. Probably some of his best illustrations are those of the limb muscles, many of which embody accuracy and detail which would satisfy even modern standards. He showed accurately the muscle insertions on the greater tuberosity of the humerus, and the curious twist of the Pectoralis major tendon at its insertion, as well as indicating a remarkably good understanding of their function. He pronounced the very modern view that the biceps is first a supinator and then a flexor, and seems to have grasped the principle of the synergistic action of muscles embodied in Sherrington's modern theory of reciprocal innervation. In connection with his studies of the lower limb he suggests the idea of studying anatomy by means of serial sections, and shows surface drawings of some of the sections—a method of study now used in many teaching centres. He is remarkable also for his cross-sections of the brain, and as being the first to make casts of its ventricles, as well as for his probable injections of the blood vessels, his unique and accurate delineation of the position of the fetus in utero, and for his investigations of the hydrodynamics of the blood current. These examples are but a few from a list which includes studies of the skeleton, the blood vessels, the digestive, respiratory, excretory and reproductive organs, the nervous system and sense organs, as well as excursions into embryology and comparative anatomy.

Accustomed as we are to all the inheritance with which the activities of four or five hundred years of able and inquiring minds

have provided us, we can scarcely appreciate the difficulty encountered by one like Leonardo, who broke away from tradition and blazed a new trail into the wilderness of ignorance and superstition in which his contemporaries dwelt. In this modern day, the debunking of traditionally great figures is a favorite indoor sport, and there are few indeed who can escape the pitiless exposure of him who seeks out only weakness. This is no less true of Leonardo than of any other great figure, ancient or modern. Despite his extraordinary ability, and the degree to which he was able to divorce himself from the errors of his contemporaries, he still belongs like any man to his environment, which was 15th century Italy. So we find even in his manuscripts some representations of tradition handed down from earlier times, especially from Galen. Undoubtedly he found much worthy of imitation in the works of antiquity, although he had nothing but condemnation for those who followed them servilely. His desire was to prove by observation the teachings of his predecessors, but when observation and tradition were at variance he promptly accepted the results of observation. His place in the history of anatomy is assured largely by the fact that he was the first to strive for accurate representation in anatomical illustration, the first to discard blind adherence to tradition and to recognize the educational and scientific value of accurate illustration. What might have happened if Leonardo's drawings had been made available at once to the scientific world is a fruitful field for speculation. They were probably known to a fairly extensive circle of friends and acquaintances and must have opened the eyes of many contemporary physicians to the possibilities for improvement. In any case, improvement began to show itself shortly after his death. These later drawings, mostly inferior to Leonardo's, belong to the new era which he inaugurated, for they are attempts to show the human body as it really is, freed from the sterile conventionalism of the past.

So we find in Leonardo the originator of a new productive and revitalizing approach to anatomy—an approach which pitilessly exposed the weakness and inadequacy of the past, and provided the impulse which carried it from an inauspicious and superstition-bound beginning to the level of the exact but still advancing science which it is today.

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Hunner's Ulcer

By HARRY H. HERSHMAN, B.A., '41

HUNNER'S ULCER is an inflammation of the urinary bladder of uncertain ætiology. It is characterized anatomically by a chronic inflammatory process predominantly of the submucosa but extending into the mural structures, and clinically by an insidious onset with long-standing symptoms of pain and frequency of urination usually of increasing severity.

First described by Skene in 1887 under the name of interstitial cystitis, the condition has been known since by a multitude of names, for example: submucous cystitis, Hunner's ulcer, elusive ulcer, cystitis parenchymatosa and congested cystitis. In the American literature and parlance, the disease is referred to generally as Hunner's ulcer. Hunner accurately described the condition in 1914, and in spite of the voluminous literature subsequently, little has been added to Hunner's original observations.

ÆTIOLOGY

The ætiology of the elusive ulcer is unknown. Bumpus and Hinnan ascribe the cause to a specific bacterium (streptococcus), blood-borne, presumably, from some focus of infection. There is, however, no evidence that removal of these alleged foci have any effect on the progress of the lesion. Rosenow believes that he has produced an elusive ulcer in rabbits by simple intravenous injections of streptococci obtained from a dental abscess in a patient suffering from Hunner's ulcer.

The condition occurs most commonly in females during the child-bearing period. Some writers are of the opinion that the co-incidental infection of the kidneys and ureteral stricture may in part be responsible for it. As a matter of interest, chronic bilateral pyclonephritis in the adult is much more common in the male than in the female.

PATHOLOGY

The ulcer is usually found in the vertex or free portion of the bladder, as contracted with the location of the Fenwick ulcer on the base or fixed portion. The actual abrasions of the mucosa may be single or multiple. These ulcers are very small, varying in diameter from two to five millimeters. They appear to be very superficial and are exceedingly sensitive to the touch.

The histological sections of specimens removed at operation or necropsy have a fairly uniform appearance in all cases. The picture is that of an inflammatory process with typical granulation tissue involving all the coats of the bladder. The mucosa shows a preponder-

ance of connective tissue with infiltration of small, round cells and leucocytes. Except where the mucosa has been broken by overdistention, its surface is more or less covered with low cuboidal epithelium. The margin of the lesion bordering on the normal mucosa frequently is jagged with marked perivascular infiltration suggesting tubercles. Some areas show fibrosis; others appear to be quite vascular and in certain sections there is present an unusual distribution of nervous tissue. A thick basement membrane is a prominent feature. The muscularis is hypertrophied and shows enlarged lymph spaces filled with small, round cells.

Occasionally, the inflammatory process may extend beyond the confines of the bladder wall and involve the paravesical tissues and adjacent peritoneum. In such cases, adhesions are formed between these structures and the bladder wall.

SYMPTOMS

The chief symptom associated with ulceration of the bladder is constant discomfort in the region of the bladder often with definite pains in the suprapubic area, aggravated by jarring or overdistention of the bladder. Along with the pain, the other symptoms of cystitis occur in varying degree, namely, frequency during the day and night, and strangury, burning and smarting. The pain is often of the most extreme grade, the patient complaining of a jabbing or stabbing knife-like pain or of a sensation of a sharp stick being driven into the bladder wall. The symptoms are apt to become aggravated, or exacerbations precipitated by acute infections, fatigue, constipation and long automobile rides, may occur.

The urine is always sterile on ordinary culture media. There are a few leucocytes or erythocytes, or both. That such a widespread destructive process can occur in the bladder wall without giving rise to objective urinary findings is probably responsible for the condition being so frequently overlooked.

The disease is progressive and the urinary frequency increases as well as the pain on urination.

Because of the severity of the symptoms and the long-standing course of the disease, some patients become suspicious that they are suffering from a cancer. Only three cases of malignant change in an elusive cancer have been reported.

DIAGNOSIS

The diagnosis is made by the presumptive evidence as suggested by the history and urine analysis, together with the cystoscopic findings. Because of the reduced bladder capacity often amounting to but two or three ounces, the characteristic appearance of the lesion cannot always be recognized. On cystoscopic examination, the normal mucosa

tends to remain intact when the bladder is not distended beyond the normal capacity for the patient. The classical appearance of the lesion is that of a salmon-red area which may be single or multiple and scattered. Occasionally, there may be small punctate hæmorrhages on the mucosa. If, under anæsthesia, the bladder be subjected to over-distension, bleeding fissures and striations appear at the site of the lesions. The original lesion, being replaced by fibrous tissue, is inelastic and attached to the overlying mucosa and when stretched, ruptures. The cracking and bleeding upon over-distension of the bladder is the most characteristic finding. In fact, this accounts for the occasional red blood cells discovered in the urine. The touching of the ulcer with a catheter, fulgurating wire or probe immediately produces bleeding and the characteristic sharp pain experienced by the patient when the bladder is distended. As a rule, the patients are so intolerant to instrumentation that local anæsthesia does not suffice and sacral or general anæsthesia is required for a satisfactory examination.

TREATMENT

There is no standard treatment for elusive ulcer. The forms of treatment that have been employed are legion, ranging from the original recommendation by Hunner of wide resection of the ulcer-bearing area to the recent suggestions of Alexander and Christie of injecting absolute alcohol into the ulcer and the use of emetine by Howard. The multiplicity of therapeutic measures and the rarity of positive results condemn the specific therapy for interstitial cystitis.

The treatment of this vague disorder is purely symptomatic. The eradication of any possible septic focus is advised empirically as a part of the general treatment.

Radical treatment consists of segmental resection. Hunner wrote, "I believe it safe to say that no form of treatment will suffice in these cases except complete excision of the inflamed area." But there was a marked tendency for recurrence, after which the symptoms were more aggravated. The unsatisfactory results have placed the procedure in disrepute.

As pain is the most distressing feature of the disease, attempts have been made to minimize this by interrupting the sensory pathways of pain perception. This has been accomplished to some extent by resection of the presacral or hypogastric plexus of nerves. It is worthy of trial in the more severe cases of interstitial cystitis. Learmouth and Quinby claim much success with this treatment for intractable bladder lesions which resist other forms of therapy.

One other radical surgical procedure has been utilized in affording the patients relief, namely, bilateral transplantation of the ureters to the sigmoid. The satisfactory results reported by Counsellor are overshadowed by an operative mortality of 23 per cent.

Topical applications of strong solutions of silver nitrate have not been satisfactory. Intravesical injections of bacteriophage and deep roentgenray therapy were unsuccessful. In a preliminary report by Folsom and O'Brien, absolute alcohol, two to six c.c., was injected about and directly into the ulcer and as a result some temporary relief was obtained.

Recently, Fister reported marked improvement in the symptoms in a patient with an elusive ulcer following the intravenous injection of gold sodium thiosulphate and the intramuscular injection of bismuth. This treatment is based on the apparent similarity in the histopathology of elusive ulcer and lupus erythematosis.

The present tendency is toward conservative treatment, namely, transuretheral electro-coagulation, over-distention of the bladder, or a combination of these two procedures. With the patient under an anæsthetic, particularly spinal or sacral combined with sacral block, the bladder is distended with fluid to a capacity of 250 to 350 c.c. The majority of the patients show an immediate relief from symptoms. Complete cures do not generally follow. Relapses are common and repeated electro-coagulations may be contraindicated. Following repeated electro-coagulations, the bladder wall becomes thin and cicatricial, almost like tissue paper. Several cases of fatal peritonitis have developed under such circumstances.

Using the various methods of treatment enumerated above, Kretschmer claims 20.4 per cent of cures.

SUMMARY

1. Hunner's ulcer is a chronic inflammation of the urinary bladder of uncertain ætiology, but presumably of bacterial origin.

2. If a case history reveals frequent and painful urination that has been present for many years, one or more operations without relief or repeated courses of ureteral dilatation for stricture without cessation of bladder distress or bladder irrigations and installations without relief, a tentative diagnosis of elusive ulcer should be made, especially if the urine is clear to the naked eye.

3. On systoscopic examination, the cracking and bleeding upon over-distention of the bladder is the most characteristic finding of the lesion.

4. Treatment is purely symptomatic. The present tendency is toward conservative treatment, namely, transuretheral electro-coagulation, over-distension of the bladder, or a combination of such methods.

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A Case Presented at a Clinico-Pathological Conference

By JEROME A. CHIKOVSKY, B.A.

A WHITE male, aged 75, was admitted to Victoria Hospital, London, Ontario, in February, 1940, complaining of an enlargement of the abdomen.

The patient had been well until six years previously, when he developed "colitis", characterized by increasing constipation, frequency of micturition, and a loss of 40 pounds weight. It was discovered at that time that he had diabetes, which condition subsequently had been controlled by diet only.

He had been a building contractor and a farmer until four years before admission, when he retired. The present illness began with dyspnea on exertion for one year, but not so marked lately. The abdomen began to distend rapidly on December 25, 1939, and three days previously swelling of the feet was noticed.

The patient's mother died at the age of 51 with cancer of the breast. His first wife died at 50 of pulmonary tuberculosis. The second wife and three sons were alive and well. Functional enquiry was negative except for increasing weakness.

Physical examination showed the patient to be markedly emaciated and dehydrated, lying flat in bed without dyspnea. There were several discrete, firm lymph glands, the size of a split-pea, in the right supraclavicular fossa and one in the left; also several small, palpable glands in both groins. The lung fields were clear except for a few moist rales at both bases. The heart rhythm was irregular, due to frequent extrasystoles, and the heart was enlarged to the left. The abdomen was markedly distended, the skin over it being dry and scaly. There were prominent superficial veins over both flanks, but no caput medusae.

A fluid wave and shifting dullness were elicited. The liver was not palpable but it was dull to percussion two and one-half cm. below the costal margin. The spleen was palpable. There was moderate oedema of both ankles. Rectal examination revealed small external and internal haemorrhoids. The temperature, pulse and respirations were normal. The blood pressure was 140/90 mm. Hg.

The specific gravity of the urine averaged 1.026, and there were no abnormal urinary findings at any time. The erythrocytes numbered 4,500,000 per cu. mm. The hemoglobin was 76 per cent and the colour index 0.84. The white cells numbered 9,800 per cu. mm. with 76 per cent of polymorphonuclears. The blood sugar was 156, the non-protein

nitrogen 36.7, and the creatinine 1.3 mgm. per 100 cc. of blood. The total blood proteins were 5.2 per cent, consisting of albumin 2.5 per cent, globuiin 2.3 per cent, fibrinogen 0.4 per cent. (Albumin-globulin ratio 1.1.) The stools were negative for occult blood. The Wasserman and Kahn tests were negative. Gastric analysis showed the total acidity to be 80 units at 1 hour and 70 units at two hours. The free HCL was 55 and 35 units at the same periods without histamine. There was a delayed direct qualitative Van den Bergh reaction. The serum bilirubin was 0.6 mgm. per 100 cc. The Icteric index was 10.9. The Venous pressure was nine cm. H_2O and the circulation time was 25 seconds by the arm to tongue method with calcium gluconate. A barium enema was given but it could not be retained. An electrocardiagram showed left axis deviation and findings indicative of myocardial damage with probable coronary disease.

Abdominal paracentesis on the day following the patient's entry to the hospital produced 1100 cc. of clear, dark, straw-coloured fluid with a specific gravity of 1.008, albumin 1.5 per cent, no mucin, occasional pus cells, many epithelial cells and a few red blood cells. The white blood cells were all lymphocytes. After removal of the fluid, the spleen was palpable three fingers breadth below the costal margin, and the smooth, firm edge of the liver was felt well under the right costal margin. The heart border was still displaced to the left so that the enlargement was not merely an apparent one due to the accumulation of the ascitic fluid.

Pathological examination of the fluid revealed the presence of carcinoma cells, consequently, during the month of March deep X-ray therapy was applied to the abdomen in the hope of retarding the recurrence of the ascites. However, on further examination of other specimens of the fluid, it was decided that there were no malignant cells but only desquamated serosal cells. Further treatment consisted of repeated paracentesis and mercurial diuretics and the patient was discharged on March 19 with his condition unimproved.

He was readmitted one month later complaining of abdominal distention and pain. Nine thousand cc. of fluid were removed from the abdomen and the patient was discharged in a few days upon his own request.

The third admission was as an emergency one, on May 19, 1940. The patient had fallen while at work in his yard and complained of weakness, shortness of breath and abdominal discomfort. He had been coming every five days for abdominal paracentesis with about 11 litres of fluid being removed each time. The emaciation, weakness and dependent oedema had increased; otherwise his condition had not changed. The liver's edge was palpable, still soft and sharp. The spleen was moderately enlarged, and there were no other masses.

On May 21, the non-protein nitrogen of the blood was 49.1 mgm. per cent, the sedimentation rate 59 mm. in one hour, and the total plasma proteins 5.1 per cent. Abdominal tap, on May 22, produced 10 litres of fluid with a specific gravity of 1.010, but otherwise not different from previous samples.

The course of the illness was steadily downward. The patient complained bitterly of precordial pain. He became mentally confused on several occasions. Terminal decubitus ulcers developed, and became secondarily infected. The temperature, pulse and respirations increased and the patient died on July 13, 1940.

DIFFERENTIAL DIAGNOSIS

The more important causes of massive ascites may be roughly classified into:

1. Diseases of the Peritoneum.
    1. Tuberculosis.
    2. Carcinomatosis.
2. Diseases of the Liver.
    1. Cirrhosis.
    2. Carcinoma.
    3. Sarcoma.
    4. Syphilis.
    5. Hydatid disease.
3. Diseases of the Heart resulting in right-sided heart failure.
    1. Rheumatic endocarditis.
    2. Adherent pericardium.
    3. Chronic pulmonary disease.
    4. Hypertensive Heart Disease (secondary to left-sided heart failure).
4. Diseases of the Kidney.
    1. Subacute glomerulo-nephritis.
    2. True nephrosis.
    3. Chronic glomerulo-nephritis resulting in heart failure.
5. Obstruction of the Portal Vein (due to causes outside the liver).
    1. Thrombosis.
    2. Enlarged portal lymphatic glands.
        (a) Primary — Hodgkin's disease, lymphosarcoma, lymphatic leukaemia.
        (b) Secondary — Carcinoma.
    3. Pressure by tumours of adjacent organs as:
        Liver.
        Pancreas.
        Stomach.

6. Obstruction of the Inferior Vena Cava above the hepatic veins.
    1. Mediastinal new-growth.
    2. Thrombosis.
7. Obstruction of the Thoracic Duct (chylous ascites).
8. Multiple Serositis (Pick's or Concato's disease).
9. Banti's Disease.

In the interpretation of any case of ascites, the history is of the utmost importance. We know definitely in this case that the ascites preceded the oedema of the legs, which tends to rule out heart disease, or obstruction of the inferior vena cava. The lack of any general anasarca is good evidence that kidney disease with its diminution of the serum proteins is not at fault. The appearance of the ascites first, its massiveness wholly out of proportion to oedema elsewhere, point to some form of portal obstruction, peritonitis or liver disease.

Tuberculous peritonitis is excluded by the repeated negative cultures and inoculations of guinea pigs with the ascitic fluid, and the absence of any demonstrable tuberculous lesion elsewhere in the body. Diffuse carcinomatosis is usually secondary and the ascitic fluid, ordinarily, would be grossly haemorrhagic and might contain cancer cells.

Heart disease is not likely to be the cause of such marked ascites without giving physical signs directly referable to the heart such as enlargement, high venous pressure and pulmonary engorgement.

Kidney disease is ruled out by the many negative urine samples of varying specific gravity.

Thrombosis of the portal vein very rarely occurs except in association with portal cirrhosis of the liver. Enlarged portal lymphatic glands usually obstruct the bile ducts at the same time they block the portal vein, and so an increasing depth of jaundice accompanies the ascites. Also, there would be usually a generalized lymphadenopathy, or a typical blood picture might point to leukaemia.

Tumours of adjacent organs which might obstruct the portal vein by mechanical pressure would probably first present other symptoms which would suggest the diagnosis.

Chylous ascites is quickly ruled out by an examination of the fluid. Multiple serositis is not likely to present such marked effusions into one serous sac without clinical evidence of similar effusions into the others. Banti's disease may give massive ascites in the terminal stages but the enlargement of the spleen would be more pronounced; there would be a greater degree of anaemia, a leukopaenia, and probably gastric haemorrhages to complete the syndrome.

Disease of the liver is fairly definitely indicated in this case. The

slight jaundice, the moderate splenomegaly and the massive recurring ascites indicate portal or atrophic cirrhosis. Other factors in favour of this diagnosis are the transudate-like character of the ascitic fluid, the dilated abdominal veins, the haemorrhoids, the history of constipation and the progressive cachexia. Syphilis of the liver is easily ruled out; hydatid disease and sarcoma are rare.

The co-existence of primary carcinoma of the liver is not an unusual occurrence. Ewing and Winternitz[1,6] regard cirrhosis as predisposing to carcinoma. Rolleston[4] believes that the liver cells have acquired the habit of proliferation, at first as a compensatory hyperplasia, which becomes so excessive as to constitute carcinoma. On the other hand, there is the theory that the invasion by carcinoma gives rise to the cirrhosis. In this case there is no hint of an aetiological factor for a primary cirrhosis in the history, which might be in favor of the carcinoma preceding and bringing on the cirrhosis.

However, the carcinoma was so slight, and the cirrhosis so far advanced that there could be no doubt that the latter had produced the clinical signs long before the former came into being.

The clinical diagnosis was: Atrophic cirrhosis of the liver.
Post-mortem examination was performed by Dr. J. H. Fisher.

The pathological diagnosis:

1. Portal cirrhosis
   - Ascites
   - Splenomegaly
   - Oesophageal varices
   - Oedema of feet.
2. Primary carcinoma of liver (liver cell type).
3. Bilateral lobular pneumonia.
4. Large chronic gastric ulcer (benign).
5. Secondary anaemia.
6. Decubitus ulcers.

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Abstracts

ESSENTIAL UNSATURATED FATTY ACIDS IN THE RELIEF OF THE COMMON COLD

By ELDON M. BOYD and W. FORD CONNELL

C.M.A.J., 43:365, 1940

A definite psychosomatic element has been revealed in this disease by the investigations of these authors. There are great difficulties inherent in assessing the value of any drug or agent in treatment of common cold. Their investigations have failed to reveal any therapeutic value of linseed oil concentrate. As part of the experiments placebo perles of liquid paraffin were given to a group of patients and results in reducing colds were quite as good as those using perles of a linseed oil concentrate. Many other commercial drug products sold for this condition are also well-nigh worthless.

CARCINOMA OF THE STOMACH

By R. H. ABRAHAMSON and J. W. HINTON

S. G. & O., 71:135, 1940

Of 1,040,784 admissions to Bellevue Hospital, New York, from 1919 to 1938 inclusive, 444 cases were gastric carcinoma. The authors review these cases to emphasize the inadequacy of present methods for early diagnosis. Two-thirds of these cases were inoperable on admission. Of the 148 operable cases, resectability was possible in only 24 cases and cures in only 10 or 2.25% of the total cases. Knowledge of the present dark outlook of the patient with carcinoma of the stomach should be disseminated among all practitioners and students of medicine. Hope for lengthening the life span of gastric carcinoma patients lies in the development of a means of diagnosis which will permit resection. Until more specific tests are developed each patient should have the benefit of such methods as are at our disposal. Faithful use of X-ray, the gastroscope and the gastric test meal is the best confirmation of diagnosis and may help to disclose early carcinomatous lesions.

—R. B. PALMER, '41.

IS PHYSICAL EXAMINATION OF THE LUNGS WORTHWHILE?

By J. D. ADAMSON

C.M.A.J., 43:345, 1940

A "modern position" of physical examination of the lungs is presented:

1. Physical examination can be reduced to a few simple procedures. Any significant alteration from the normal is not subtle.
2. X-ray has long since superseded it in the diagnosis of curable tuberculosis.
3. It gives earlier and more reliable information in chronic sepsis and emphysema.
4. It is frequently best in acute pulmonary and pleural disease.
5. It is a worthwhile art and should be thoroughly taught in medical schools.

A NEW CONCEPTION OF PARATHYROID FUNCTION AND THE CLINICAL APPLICATION

By A. J. HELFET

Brit. J. Surgery, 27:657, 1940

Helfet bases his work on the theory that the secretion of the parathyroid, parathormone, is primarily concerned with control of the blood inorganic phosphate level. Accumulation of blood phosphate causes increased production of parathormone, which keeps the phosphate level down by drawing calcium from the bones and by increasing kidney excretion of phosphate. Thus, in removal of the parathyroid, the blood phosphate rises. Helfet suggests that this theory is capable of explaining both the

clinical and laboratory findings in hypo- and hyperparathyroidism.

The role of hyperparathyroidism in generalized fibrocystic disease, in rheumatic arthritis and osteitis deformans is discussed. A classification of hyperparathyroidism is given.

The author's treatment of hyperparathyroidism is to reduce the intake of phosphorous by using aluminium acetate which forms insoluble phosphorous. By reducing phosphorous intake, the stimulus to overproduction of parathormone is removed.

A scheme of dosage and fourteen case reports are presented.

—R. PARKER, '41.

THE RELIABILITY OF THE LEUCOCYTE COUNT IN THE DIAGNOSIS OF APPENDICITIS

By ALLAN S. JOHNSON

N.E.J. Med., 223:10, 1940

This is a study of the significance of the total and differential white cell counts in a series of 221 cases of suspected appendicitis (operated upon) and 50 control subjects without inflammatory lesions. For purposes of this study 10,000 was considered the upper limit of normal white count, and 70% the criterion of polymorphonuclear leucytosis.

When the white cell count was 10,000 or more, 62% of those suspected of appendiceal disease had appendices which actually needed removal.

When the white cell count was 15,000 or more, 81% of the suspects actually had appendices which needed removal.

It must be noted, though, that 29% of the suspected cases, with counts below 10,000, also had acutely diseased appendices, and 10% had necrotic or ruptured appendices.

Also included in the paper is a study of the oral temperature from which the author concludes that the thermometer is of little help in the diagnosis of appendicitis.

The author concludes with a plea not to trust blindly to the white count as conclusive evidence either for or against appendicitis.

—J. G. STAPLETON, '41.

ESTIMATION OF BLEEDING TENDENCY IN JAUNDICE

By L. K. FERGUSON, D. G. CALDER and J. G. REINHOLD

S. G. and Obs.

S. G. and Obs., 71:603, 1940

Three methods of comparing the bleeding tendency in patients with jaundice or biliary fistula are compared. Quick's method for determining blood prothrombin percentage requires equipment and personnel not always easy to obtain. Two other tests are dealt with which are "bedside" and compare favourably with Quick's method. These two tests give adequate information regarding the bleeding tendency in such conditions as mentioned.

NEUROGENIC DYSFUNCTION OF THE BLADDER DUE TO SPINAL ANESTHESIA

By E. L. PEIRSON and C. F. TWOMEY

N.E.J. Med., 223:5, 1940

A number of articles on nerve damage following spinal anesthesia have appeared. The authors of this paper emphasize that nerve damage is probably commoner than is thought, and discuss paralysis of the bladder as one of the most serious types. The literature on the subject is reviewed and tends to support the thesis of Critchley that "a very significant number of nervous sequelae are completely overlooked by both the surgeon and the anesthetist." Cases of bladder dysfunction are known to have persisted over a period of months or years, and not infrequently death eventually occurred as a result of infection of the urinary tract.

The authors present an interesting case report of a man 60 years of age who had complete retention of urine for 10 weeks following an appendectomy performed under spinal anesthesia. Complete study of the case failed to show any other reason for the retention.

This patient was treated by presacral nerve resection. Retention was relieved and he has remained well.

—J. G. STAPLETON, '41.

INSTANTANEOUS "PHYSIOLOGIC" DEATH

By SOMA WEISS

N.E.J. Med., 223:20, 1940

One of the most perplexing problems facing both physicians and pathologists is the explanation of instantaneous death. The term "sudden death" is frequently used to describe the unexpected occurrence of death within a space of several minutes or even hours after the onset of alarming symptoms.

The underlying diseases and mechanisms of sudden death are multiple; coronary disease, in particular, and other types of heart disease; cerebrovascular accidents; pulmonary embolism; dissecting aneurysm of the aorta; internal hæmorrhage; obstruction of the trachea, and various types of poisons, are mainly responsible. But in the sub-group in which death occurs without warning symptoms and practically instantaneously, one finds that in the majority of cases acute structural lesions in vital organs are often meagre or absent. The lesions offered as an explanation of death are usually chronic. Post-mortem examination, as a rule, fails to reveal proof of, or even evidence for, the cause of death.

The mechanism of instantaneous death has been little studied, but the observations of the author indicate that it is usually cardiac in origin, and that its occurrence depends on an underlying physiologic mechanism. There is a close similarity and interrelation between the mechanism of instantaneous death and that of syncope; frequently instantaneous death is merely fatal syncope.

The difference between syncope in health and in certain diseased states lies in the ability of the patient to re-establish normal equilibrium. Whereas, in normal subjects, owing to the action of the numerous emergency functions and to the reserve capacity of the organs involved, a return to the normal cardiovascular equilibrium is accomplished with relative ease and promptness; in diseased persons, because of damaged systems or organs, a return to the normal level is more difficult, usually slower, and may not even occur. In the presence of ischemic myocardium and hyperactive reflexes, fright or other emotional stress may induce cardiac arrhythmia, syncope and death. Asystole of various types and ventricular fibrillation are the usual causes.

SYPHILITIC AORTITIS AS A CAUSE OF SUDDEN DEATH

By TIMOTHY LEARY

N.E.J. Med., 223:20, 1940

The author states that there has been a remarkable change in the character of syphilis in the last few decades. Dramatic types of skin lesions, not unusual a generation ago, have become rarities, and the more extensive skin lesions met with today in luetic individuals are in the form of dermatitis exfoliativa due to treatment and not to the disease. The important forms of the disease seen by the pathologist today are those which centre in the cardiovascular and the central nervous system.

In the discussion of syphilitic aortitis as the cause of sudden death three possibilities are considered.

1. In the early stages of the disease, the excessive growth of fibroblastic tissue which thickens the intima and tends to narrow and occlude the portions of the coronary arteries lying within the aortic wall may be so marked as to cause occlusion of the ostia and result in sudden death of the coronary type.

2. In the later stages of the disease, in addition to the widening of the commissures and the rolling of the cusps, the association of atherosclerosis with late syphilitic aortitis tends to be followed by calcification and diffuse dilatation of the aorta including the ring. Dilatation of the ring produces aortic insufficiency which may be followed by sudden death of coronary type, but usually leads to late progressive cardiac decompensation.

3. The third cause of sudden death discussed is death due to rupture of an aneurysm, as in the production of local dissecting aneurysms in the lower ascending aorta, with rupture into the pericardium.

Editorial

SELECTION OF AN INTERNSHIP

By L. D. RUTTLE

PROBABLY the most important decision that a student makes during his undergraduate years is his choice of an internship. It is the foundation upon which he builds his entire post-graduate career. Unfortunately, in the past, the student has had little or no organized assistance in this choice of internship. Hence, year after year, the same unsatisfactory hit-and-miss methods were repeated.

To help solve some of these vexing problems, the Canadian Internship Board came into being. The plan put forth by the Board has been given the acid test during the last two years and has been found to be much better than the former arrangement. However, criticism and suggestions for the future are welcomed at all times.

By the plan, the C. I. B. submits to all hospitals a list of the students who are applying through them. That the plan has been successful can be seen by the fact that the Board placed 93 per cent of its applicants last fall. The number of broken contracts and "jumping from one hospital to another" has been decreased. Eighty per cent of the students received the appointment which was their first choice. Under the old method only 55 per cent received an appointment at their first choice hospital. Furthermore, all students were placed before Christmas. The Board has only one criticism to make to the students and that is that some failed to apply *personally* to *all* of the hospitals which they had listed on their forms.

This system has worked out well for the hospitals, too. There were 246 positions to be filled and the Board had only 203 students with which to fill them. Therefore, some hospitals did not get their full quota. In several cases, the hospital was at fault because of failure to send in complete lists and to indicate which were preferred and which were alternate choices.

The most serious problem that the Board faced this year was that several hospitals announced their appointments and required those who were appointed to sign contracts before the allocations were made by the C. I. B. Nothing is gained by this practice since, if a student later receives an appointment through the C. I. B. which he prefers he usually breaks his contract. He should have few scruples about doing so since that hospital is breaking the rules of the Board.

From this short summary of the *C. I. B. report for 1940-1941, it is evident that it is to the student's advantage to know the working of the Board and co-operate with it to the fullest possible extent.

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*There is a complete copy of the report for 1940-1941 in the U.W.O. Medical Library.

INJURIES OF SKULL, BRAIN AND SPINAL CORD: NEURO-PSYCHIATRIC, SURGICAL, AND MEDICO-LEGAL ASPECTS

Edited by SAMUEL BROCK, M.D.

632 pp.; 63 illustrations; William Wood & Co., 1940; $7.00 (in U.S.A.)

Because of their frequency, injuries of the skull, brain and cord have assumed a very important place in the practice of neurology, psychiatry and neuro-surgery. The consequences of such trauma also claim a great deal of attention on the part of the officials of insurance companies, referees of compensation commissions, lawyers and judges. For this reason a volume describing in detail all of the immediate and remote effects of physical injury to the skull, brain and spinal cord, together with their treatment, should be of great interest to the undergraduate.

A series of comprehensive monographs on neurology, psychiatry, neuro-surgery, radiology and forensic medicine has been included in this volume in as far as these subjects relate to injuries of the skull, brain and spinal cord.

"Trauma" is used in its every-day meaning; *i.e.*, "a physical force applied to the head or spine, directly or indirectly, transmitting its effects to the brain, and the spinal cord and their coverings". The secondary results of injuries are touched upon with respect to fat and air embolism. Electrocution and caisson disease are included.

The reader will find that some of the subjects are considered in several places by different authors, but to have eliminated all overlap would have impaired the unity of individual chapters and would have weakened at times the point of view expressed by the various contributors.

—CHARLES E. BODKIN, '42.

TEXTBOOK OF SURGERY

Edited by JOHN HOMANS, M.D.

5th Edition, 1,272 pp., illustrated. Published by Charles C. Thomas, Springfield, Ill., 1940; $8.00 (in U.S.A.)

Since 1931, this rather unique textbook has passed through five editions—sufficient evidence of its popularity.

The first few chapters, dealing with surgical fundamentals, will repay the student's careful study; while the later more detailed sections treat adequately their particular subjects. The study of wounds is dispersed throughout the book, instead of being gathered into one chapter where it would be much more accessible. Chapters on anesthesia and amputations have been added since the appearance of the first edition; otherwise, the text retains its original arrangement. Fifty odd pages of revised material have replaced items now outdated.

A feature which should be of interest to "overburdened" students and "overactive" practitioners is the historical introduction to each chapter. These sketches reflect the enthusiasm with which Dr. Homans and his collaborators have endeavoured to trace medicine to its fetish roots. By the historical approach to a study, one obtains a mental perspective without which Williamsburg might as easily be considered the place of origin of toe-twisting as Lindbergh the father of aviation.

The excellent bibliographical and subject indices add immeasurably to the value of this book as a reference work.

—JOHN LINDSAY, '43.

THE ABDOMINAL INJURIES OF WARFARE

By GORDON GORDON-TAYLOR, O.B.E., F.R.C.S.

87 pp.; 68 illustrations; John Wright & Sons Ltd., Bristol, 1939; 10s. 6d.

This little book, which can easily be read in a night, represents the basis of two lectures delivered at the British Post-Graduate Medical School in March, 1939. The foundation of the volume constitutes a series of excellent pen-and-ink and coloured illustrations of specimens from the war office collection in the Museum of the Royal College of Surgeons of England. The text, the author modestly claims, serves merely as a companion-guide to the illustrations. However, anyone who might read it will surely agree that Gordon-Taylor's style is lively and entertaining without detracting in the least from the value of the text; it is typically English, reminiscent of Hamilton Bailey, and awakens in one the vague regret that most of our North American surgeons seem to consider the presentation of facts unembellished by literary niceties sufficient. Case histories accompany many of the illustrations and often awake, as one reads, both awe and wonder that this delicate human body can, at times, stand so much punishment and recover.

A survey of the book also provides one with a short but complete history of the technical triumphs in the surgery of the first great war.

Since total war has brought horror and pain once more to soldier and civilian alike, and since we have now ceased to ask,

"Why a maddened people rush to arms
And rob the world of peace."

we might well, each of us, look this book over and give it a few minutes' thought. We shall probably be glad of its teaching.

—ALAN S. DOUGLAS, '42.