A British Medical Association Lecture ON UROGENITAL TUBERCULOSIS.

GIVEN TO THE SOUTH-WESTERN BRANCH

BY

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THERE was a time when urogenital tuberculosis was regarded as a more or less hopeless disease, and the unfortunate sufferer was condemned to a life of misery interspersed with courses of tuberculin, cod-liver oil, and sanatorium treatment, until, worn out by painful and frequent micturition, and wasted to a shadow by toxæmia, death put an end to his sufferings.

Within recent years research in the laboratory and in the *post-mortem* room has revealed the true pathology of the disease, and this knowledge, combined with the perfection of the cystoscope, has enabled the clinician to make an accurate and often an early diagnosis, and by means of active surgical treatment to give the patient a good prospect of complete recovery. But even now the ultimate mortality is still too high, and the reason is not far to seek. Successful treatment in this, as in all other diseases, depends on an early diagnosis, but unfortunately very few cases reach the surgeon before the signs and symptoms are unmistakable. Therefore, at the outset I want to emphasize the importance of a thorough clinical and bacteriological examination in all urinary cases, particularly those with intermittent bladder irritation and so-called cystitis. The following is a case in point.

A stout, healthy looking woman, aged 49, dislocated her shoulder; a week later micturition suddenly became frequent and painful. This was diagnosed as cystitis, treated with an alkaline mixture, and soon subsided. A recurrence four weeks later led her to seek further advice. Cystoscopy revealed slight congestion around the right ureteric orifice. Both ureters were catheterized. Urine from the left was normal; that from the right contained a few pus cells and a number of tubercle bacilli. The right kidney was removed and found to contain a small tuberculous focus in its lower pole. Six years later she was in perfect health and the urine was normal.

In discussing urogenital tuberculosis it is most convenient to commence with the kidney, for disease of this organ forms a large proportion of the cases of urinary tuberculosis; also a lesion in the kidney is very commonly the primary focus from which the lower urinary and the genital tracts are directly infected.

RENAL TUBERCULOSIS.

It is now well known that tubercle bacilli reach the kidney from some other focus in the body, either a quiescent and obscure lesion such as a tuberculous mediastinal gland or an active and obvious source such as pulmonary, articular, or osseous tuberculosis. These two sources of infection, generally speaking, give rise to two distinct clinical and pathological types of renal tuberculosis, called surgical and medical respectively. Though this is the usual rule, statistics show that in about 25 per cent. of cases the surgical form is secondary to active pulmonary and pleural tuberculosis.

Surgical or chronic renal tuberculosis was once thought to be primarily unilateral, but animal inoculation has now shown that a few early cases are bilateral. It is characterized by caseation, ulceration, and slow but progressive destruction. The urine contains tubercle bacilli, pus cells, caseous material, fragments of renal tissue, and a small trace of albumin of pyogenic origin.

The *medical* form is bilateral, the renal parenchyma is studded with miliary tubercles and the urine is clear, containing a few bacilli, many hyaline and granular casts, and a large trace of albumin. It is accompanied by typical physical signs of chronic nephritis and often ends in uræmia. Therefore the presence of tubercle bacilli in the urine does not necessarily imply that surgical treatment is required, but it does call for a careful and exhaustive examination. A kidney urine containing tubercle bacilli and pus cells demands immediate surgical treatment.

It is difficult to account for the two widely different pathological types, but probably the difference is only one of degree. In the miliary or medical form bacilli and toxins from an active lesion such as pulmonary tuberculosis are constantly passing through the kidney. Many of the bacilli are undoubtedly excreted in the urine, but eventually their escape is prevented by endarteritis and chronic nephritis; they therefore lodge in the glomerular zone and give rise to multiple tubercles. At this stage death takes place before caseation and liquefaction can occur. In the surgical form the kidney receives from a quiescent focus an occasional tuberculous embolus which becomes isolated by a barrier of protective inflammation. This is followed by the formation of a tuberculous abscess which either increases at the expense of the surrounding parenchyma or discharges into the renal pelvis.

CHRONIC RENAL TUBERCULOSIS.

As the miliary form is part of a general disease, and so not amenable to surgical treatment, my future remarks will apply to chronic or surgical renal tuberculosis.

Paths of Infection.

Tubercle bacilli reach the kidney either by the blood stream or by the lymphatics, and of these two routes the hæmatogenous is undoubtedly the commoner. The exact site of the initial lesion in the kidney is disputed. The peculiar arrangement of the intrarenal arteries favours a cortical lesion, but clinically, though many tubercles are seen in the cortex, the grossest lesion is nearly always at the base of a papilla.

The lymphatics are considered by some to be the means of infection in those cases which are secondary to quiescent pleural and pulmonary tuberculosis. The bacilli are thought to be carried in the lymphatics passing through the diaphragm by a retrograde current set up by pleural adhesions. Though primary lymphatic infection is doubtful, secondary infection may be conveyed from a diseased to a healthy kidney by the communicating channels between the two renal plexuses, or from an ulcerated bladder by the peri-ureteral lymphatics.

Ascending infection up the lumen of the ureter does not occur. Animal experiments by English and Continental workers have proved (1) that pyogenic and other organisms placed in the bladder cannot pass the normal uretero-vesical valve, and (2) that when pyogenic organisms are injected into the ureter and pelvis the renal papillæ prevent their entry to the kidney.

An ascending infection can only occur when the mucous membrane of the bladder or ureter is damaged, and it then travels in the wall of the ureter or in the peri-ureteral lymphatics. In all probability ascending tuberculous infection seldom occurs, for even when all the essential factors are present in the form of advanced genital and vesical tuberculosis the blood stream as a vehicle can never be discounted.

The pathological anatomy of the disease is so intimately connected with its clinical aspect that a brief description is essential for a proper understanding of the symptoms and treatment. Three types are recognized.

I. *Primary closed parenchymatous tuberculous nephritis* consists of one or more encapsuled foci in the parenchyma which progress to caseation and abscess formation and ultimately destroy the whole kidney. This is accompanied by an interstitial inflammation of the pelvis and calyces, ending in the formation of fibro-fatty tissue which fills the pelvis and excludes a portion or the whole of the kidney and its tuberculous products from the urinary tract. This process is called spontaneous cure, or auto-nephrectomy. The term is misleading, for it suggests that the disease is arrested or removed, but this is not the case. The bag of fibro-fatty caseous material never disappears; it may remain inert for years, but sooner or later the disease either invades the perinephric tissues or is disseminated to other parts of the body through the blood stream. The clinical signs of this condition are characteristic. In its early stage the urine of the infected kidney is increased in quantity, its specific gravity is lowered, and it contains a trace of albumin and a few pus cells. It is sterile and seldom contains tubercle bacilli. The renal function is

defective when compared with that of the opposite side. As destruction and exclusion progress the secretion of urine diminishes and finally ceases. The patient complains of frequency and vesical irritation, which is often diagnosed as mild cystitis and treated medicinally.

II. *Primary open tuberculous pyelonephritis* is characterized by ulceration of the pelvis combined with progressive destruction and ulceration of the papillae and parenchyma. There may be deeper lesions and miliary tubercles in other parts of the kidney, but the condition is essentially a tuberculous pyonephrosis. The changes spread down the ureter, its mucous membrane becomes ulcerated and its walls thickened. Its vesical orifice is wide open and dragged out, and the surrounding mucosa is ulcerated and sometimes studded with tubercles. The clinical picture is one of painful frequency (pollakiuria) with pyuria and occasional haematuria. The urine from the affected side contains pus, caseous material, renal elements, blood cells, and tubercle bacilli in large numbers. Often the efflux from the ureteric orifice resembles tooth-paste emerging from a collapsible tube.

III. The third type consists of a combination of the open and closed varieties. Its clinical features are similar to those of the second type.

Clinical Signs.

The clinical signs seldom indicate the extent of the lesion. The affected kidney may be enlarged, irregular, and tender, but enlargement is unusual. The ureter is often thickened, and can be readily felt as a firm, round, tender cord on bimanual examination—rectal in the male and vaginal in the female. It is unfortunate that thickening of the ureter should have come to be regarded as a diagnostic sign of renal tuberculosis, for it indicates long-standing disease. It is to be hoped that in the near future an accurate diagnosis of the renal lesion will be made long before the ureter has become thickened. The opposite kidney is very often enlarged and tender from compensatory hypertrophy and toxic nephritis. In this context it is interesting to note that the urine from the healthy kidney often contains albumin, which disappears after removal of the diseased kidney.

Cystoscopic examination may show the bladder to be inflamed and ulcerated, or the trigone alone may be congested. The appearance of the ureteric orifice is often the only visible sign of the hidden focus in the kidney. It may be congested or oedematous, or surrounded by minute bullae. There may be tubercles on its lips, or it may be surrounded by a ragged ulcer with tubercles on its margins. In long-standing cases the orifice is wide open, rigid, and ulcerated, and when there is much thickening and fibrous contraction of the ureter the bladder is dragged out, forming a small hollow cone with the ureteric orifice at its apex. The efflux may be forcible and clear, or turbid, or even putty-like, when it appears like a worm-cast, which curls up as it is ejected and falls to the bottom of the bladder. When the orifice is open and rigid, turbid urine trickles out in a slow, continuous stream, or a ragged mass of caseous material is slowly forced out. Absence of efflux indicates obstruction of the ureter by caseous material or complete exclusion of the kidney.

Symptoms.

The symptoms are essentially those of cystitis—pain, frequency, pyuria, and occasionally haematuria. They commence gradually and increase steadily. Sometimes the onset is sudden. This is due to the escape of tuberculous products from a focus in the kidney which has suddenly ruptured into the pelvis, giving rise to bladder irritation and frequent passage of turbid acid urine. In a few days the cavity in the kidney closes, the symptoms subside, and both patient and practitioner are lulled into a false sense of security. After an interval, during which the cavity refills, the process is repeated and the symptoms recur. These intermissions may last for months or years, the intervals of freedom becoming progressively shorter, until finally the discharge from the kidney becomes continuous and the lower urinary tract grossly diseased.

Pain.—The kidney may ache, but it is seldom painful. In rare instances there are definite attacks of colic, due to

the passage of caseous material or blood-clot. In some cases pain is felt only in the healthy kidney, when it is probably reflex, for it disappears after removal of the diseased organ. The pain of cystitis is felt at the neck of the bladder, along the urethra, and in the perineum.

Frequency is at first due to trigonal irritation and later to cystitis. In the beginning it is diurnal only, soon it becomes nocturnal as well, and finally merges into total incontinence. Nocturnal frequency is one of the most constant and important symptoms of the disease.

The changes in the urine have already been described, but there are certain features which require further consideration. Polyuria, due to a defective renal function, is present from the commencement, and is replaced by oliguria as the kidney is destroyed. Albuminuria can be accounted for by pus and blood from the diseased kidney and by toxic nephritis on the opposite side. In the latter case it is often associated with hyaline and granular casts. Haematuria is a variable symptom. A profuse haemorrhage may be the first indication of the disease; as destruction progresses there may be slight haemorrhages at long intervals, or there may only be an occasional microscopic trace of blood.

The presence of tubercle bacilli in the urine is, of course, the only definite diagnostic sign. When found in a clear urine they are either being excreted by a normal kidney or by one containing miliary tubercles, but when a urine obtained by catheterization of the ureter contains both tubercle bacilli and pus cells there can be no doubt that the kidney is the seat of a surgical lesion.

When a secondary infection is present colon bacilli and other pyogenic organisms may be found in the urine, and may mask the true nature of the disease. The smegma bacillus should not cause an error in diagnosis, for it can now be identified by careful staining. Its presence is due to faulty collection of the specimen.

Diagnosis.

Bladder irritation, nocturnal frequency, and the passage of hazy or purulent acid urine in a subject between the ages of 20 and 40 are strongly suggestive of tubercle.

The abdomen, lungs, lymphatic glands, and in the male the epididymes, prostate, and seminal vesicles, must be carefully examined. Tuberculous deposits in the genital tract are not necessarily primary infections; they may quite well be secondary to renal tuberculosis. Specimens of bladder urine must be examined both bacteriologically and microscopically by a competent pathologist. If tubercle bacilli are not found, even after repeated examinations, the diagnosis is still in doubt.

The next step should be a cystoscopic examination. Prior to this a dose of urea (for Maclean's urea concentration test) or an injection of indigo-carmin should be administered to enable the surgeon to ascertain the function of the kidneys. The appearance of the bladder and ureteric orifices is carefully noted. If the two orifices are similar in appearance both are catheterized and the urines collected. The renal function is estimated from a portion of these specimens, and the remainder is examined for tubercle bacilli, pus cells, etc. In doubtful cases guinea-pigs are also inoculated. If one ureteric orifice is obviously tuberculous the opposite one alone is catheterized; should its urine contain the slightest trace of pus a guinea-pig should be inoculated and the result awaited before the kidney can be declared healthy.

Treatment.

As surgical renal tuberculosis is primarily unilateral, and as spontaneous cure is unknown, the treatment can be summed up in two words—early nephrectomy. Before an operation is performed the function and condition of the opposite kidney must be carefully investigated. The diseased kidney and any adherent fat should be removed by the lumbar route. It should never be incised to verify a diagnosis which should have been certain before the operation, otherwise the surrounding tissues will be infected and the wound take months to heal.

In bilateral cases where the disease of one kidney is slight the grossly infected one should be removed, for nephrectomy relieves the bladder symptoms and may benefit the remaining kidney by increasing its congestion.

Nephrectomy is contraindicated (1) when the function of the opposite kidney is defective; (2) in advanced bilateral tuberculosis; (3) in slight bilateral tuberculosis; (4) when the disease is secondary to pulmonary or other gross tuberculous lesions.

Treatment of the Ureter.

When the ureter appears healthy it may be ligatured in two places, injected with pure phenol, and divided a few inches below the kidney. When it is thickened or much enlarged and dilated total ureterectomy is advisable, for high division may be followed by a tuberculous sinus or a urinary fistula. The operation I perform in these cases is nephro-ureterectomy through a lumbar and an inguinal incision, the ureter being divided at the base of the bladder and removed, still attached to the kidney, through the lumbar wound.

In most cases the wounds heal per primam, but occasionally in debilitated subjects, and particularly if the perinephric tissues are infected, there may not be the least attempt at healing, and after removal of the stitches the whole wound gapes widely open. This has happened to three of my cases, and in each the wound took more than a year to heal.

Results.

The operative mortality is about 2 per cent. Out of 100 cases which have survived the operation 60 will be cured and 40 will have persistent urinary trouble. Of these latter, 30 will die in from three to five years of tubercle of the opposite kidney or elsewhere in the body, and 10 will be left with either frequency alone or frequency and pyuria.

TUBERCULOSIS OF THE BLADDER.

Vesical tuberculosis probably never occurs as a primary lesion, but is always secondary either to renal tuberculosis, the infection reaching the bladder in the urine or from the walls of the ureter; to genital tuberculosis via the seminal vesicle and prostate; or, in rare instances, to tuberculous salpingitis by direct extension from an adherent Fallopian tube.

The infection tends to spread in a downward direction to the urethra and prostate and thence to the seminal vesicle and epididymis. When the walls of the bladder are ulcerated the infection may ascend to the kidney by the peri-ureteral lymphatics.

The tuberculous process, when secondary to renal disease, commences around the ureteric orifice, and on the base of the bladder when secondary to genital tuberculosis. Tubercles form in the submucous tissue, giving rise to raised hyperaemic patches in the mucous membrane, which tend to form superficial or deep ulcers, with a ragged shaggy base. As the ulcers deepen the muscular wall of the bladder is invaded, causing contraction and great diminution in the capacity of the viscus, and thus accounting for the distressing frequency and incontinence so typical of vesical lesions.

A diagnosis cannot always be made by examination of the urine, for in many cases tubercle bacilli cannot be found. In these cases tuberculous nodules in the genital tract or a thickened ureter, combined with the cystoscopic appearances, will solve the problem.

Treatment.

The immediate treatment is removal of the primary focus—either the kidney or the affected portion of the genital tract. When both renal and limited genital tuberculosis coexist the kidney and ureter must be removed first, and the genital tract operated on at a later date. Provided the patient is otherwise healthy the results of such radical treatment are often remarkably good. After removal of the primary focus the bladder will often recover without further treatment, but when the cystitis does not subside both general and local treatment are necessary.

General treatment consists of rest in bed, bladder sedatives, and tuberculin.

Local Treatment.—Instillation of many different chemicals has been advocated and employed, but they seldom have much effect, and their introduction into the bladder may

lead to septic infection. In my experience surgical diathermy is the best form of local treatment, but if this cannot be obtained relief is sometimes given by instillation of either gomenol oil 20 per cent., or of iodoform (1 part) and guaiacol (5 parts) in sterile olive oil (100 parts). Diathermy is performed at intervals of three weeks with a catheterizing cystoscope, and the granulation tissue or ulcer is gently fulgurated. Fulguration relieves pain, diminishes frequency, and assists healing. Suprapubic cystostomy and drainage is not advisable, for it is difficult to perform on a contracted bladder, gives little relief, and is followed by tuberculous infection of the wound.

GENITAL TUBERCULOSIS.

Genital tuberculosis usually occurs between the ages of 20 and 45. The infection may be primarily haemic, when it can commence in any part of the genital tract; or it may be secondary to urinary tuberculosis, when it spreads by way of the prostate to the seminal vesicles and epididymes.

Clinical and experimental evidence shows that the primary focus is generally in the prostate or seminal vesicle, and statistics favour the seminal vesicle. Young of Baltimore, in a recent article, states that the seminal vesicle is affected in 83 per cent. of cases of genital tuberculosis, and that in a large majority of these the vesicle was the seat of the primary focus. The work of Macfarlane Walker and others points to the same conclusions.

With certain peculiarities the pathological changes are similar to those of tubercle elsewhere, and, as in the kidney, the foci may be closed or open. The prostate is generally more often infected from the urine or from the seminal vesicle than from the blood. Cold abscesses form and discharge into the urethra or rupture into the base of the bladder at the side of the trigone, leaving a ragged cavity which never heals. The lower end of the seminal vesicle is more often affected than the upper, but the whole tube may contain caseous foci and tuberculous "pus."

The character and sites of the tuberculous deposits in the epididymis and vas are of great interest, and point strongly to the infection of the epididymis being secondary to a primary lesion of the seminal vesicle or prostate. The earliest and also the oldest foci are found in the globus minor, whence extension takes place along the convoluted tubes to the globus major and finally to the body of the testis. Primary deposits in the globus major are comparatively uncommon, and are probably correctly regarded as haemic infections.

Thickening of the vas is most marked at its two extremities, the intermediate portion being often comparatively healthy. This intermediate portion often feels thickened, but the increase in size is due to surrounding inflammatory fibrosis and not to changes in its walls or lumen. In advanced cases deposits of caseous material are found throughout its length. Macfarlane Walker in his Hunterian Lecture (*Lancet*, February, 1913) showed that the thickening of the prostatic end of the vas was due to tuberculous infiltration of the perideferential lymphatics, which gradually diminished as the epididymis was approached, and that thickening of the testicular end was due to tuberculous deposits in its mucosa. He therefore considered that the clinical fact of the earliest lesions of the epididymis being in the globus minor was explained by extension of infection from a primary focus in the seminal vesicle or prostate by way of the perideferential lymphatics and a return in the lumen of the vas.

The conclusions arrived at by many clinicians that genital tuberculosis is secondary to a primary focus in the seminal vesicles are borne out by the results of simple castration, for 50 per cent. of such cases develop tuberculous epididymitis in the opposite side within a year or more.

The disease commences insidiously and progresses slowly. Though complete arrest is uncommon, there may be long periods of quiescence ending in increased activity and rapid extension. The prognosis is bad, owing to the frequency of relapses, the constant danger of extension to the bladder, and of dissemination to other parts of the body.

Treatment.

In advanced cases medical and general treatment is indicated, as surgery offers little prospect of cure, but when the disease is limited to the epididymis and seminal vesicle radical operation is indicated. It should consist of removal *en bloc* of the epididymis (or epididymis and testis), the vas deferens, the seminal vesicle, and when necessary a portion or the whole of the prostate. The operation can be accomplished through an inguinal incision by Loughnane's method, but if the prostate is involved Young's combined inguino-perineal method is necessary. Should the disease be secondary to unilateral renal tuberculosis the affected kidney must be removed first and the operation on the genital tract postponed to a later date.

In all cases tuberculin is of great value, but only after operation. In my experience it has not the least effect on well established urogenital tuberculosis, and should never take the place of surgery if removal of the disease is possible.

An Address

ON

**THE CONSTITUTIONAL FACTOR
IN DISEASE.**

DELIVERED BEFORE THE ULSTER MEDICAL SOCIETY,

BY

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(Concluded from p. 827.)

BLOOD PRESSURE AND CONSTITUTION.

DISCUSSIONS on abnormal blood pressure have in the past suffered from our ignorance as to what constitutes the normal. Stocks²¹ has recently investigated the blood pressure of 1,323 English boys, and the extensive investigations of Alvarez²² and his colleagues on 6,000 male and 8,934 female first-year students at the University of California have provided reliable figures for young adults. The statistics published by Symonds²³ of 141,840 applicants for insurance in an American company give additional data for all ages above 15, the smallest group (over 60) being no fewer than 1,889. In children there is a steady rise in systolic pressure from an average of 85 mm. at 5 to 101 mm. at 10 and 123 mm. at 15. Contrary to what might be expected, there is no rise with advancing years from 17 to 40, the average systolic pressure being 125 mm. in men and 115 mm. in women. After 40 there is a slow rise, the maximum average being 135 mm. for 60 and over, but there is no longer any difference between the sexes.

In healthy individuals of the same age, sex, and race considerable variations occur. Thus Alvarez and his associates found that among the students they examined 2.77 per cent. men and 11.74 per cent. women had a systolic blood pressure less than 105 mm., and 20.81 per cent. men and 3.75 per cent. women had a blood pressure over 140 mm. It is, of course, impossible to draw exact lines above and below which the blood pressure should be regarded as abnormal. For convenience I would suggest that hyperpiesia is present when the blood pressure is more than 25 mm. above the average normal for an individual's age and sex. This would give a limit of 150 mm. in men and 140 mm. in women up to the age of 40, and 160 mm. for both sexes at 60. According to this standard, 8.15 per cent. men and 3.75 per cent. women among the 15,000 healthy students examined by Alvarez had hyperpiesia.

No such sliding scale is required for hypopiesia. American writers generally take 110 mm. as the minimal normal figure, but Alvarez's investigations clearly show that this is too high. A preferable limit would be 105 mm. for men and 100 mm. for women. According to this definition it was present in 2.77 per cent. of the 6,000 men and 3.50 per cent. of the 8,934 women among Alvarez's first-year students.

There is no doubt that the tendency to hyperpiesia is hereditary. Long before the invention of the sphygmomanometer the familial occurrence of high blood pressure,

apart from disease of the kidneys, was recognized, and it was well known that many members of successive generations of a family might die from such results of high pressure as cerebral haemorrhage and cardiac failure, associated with arterio-sclerosis and hypertrophy of the heart. The tendency to hypopiesia is probably inherited with equal frequency. The actual cause of the high or low blood pressure is quite unknown, though it is probably, at any rate in part, due to variations in the activity of such internally secreting organs as the suprarenal, the pituitary, and the thyroid glands.

I believe that hyperpiesia and hypopiesia should be regarded as constitutional conditions or diatheses, being simply extreme degrees of variation from the average normal. They are not diseases and need never give rise to symptoms, but an individual with either condition is apt to react in a special way to toxæmias and to mental and physical fatigue.

The diffuse hyperplastic sclerosis of the arteries, described by Evans as a constant pathological finding in hyperpiesia, is, I am convinced, a result, like the cardiac hypertrophy, of the constitutional high blood pressure, and not its cause. Clinically hyperpiesia may be associated with apparently perfectly healthy blood vessels, especially in young subjects before the pressure has become excessive.

(a) Hyperpiesia.

So much has been written on the subject of hyperpiesia since the classical analysis of the condition by Sir Clifford Allbutt that I have little to add. I think, however, that too much time has been expended in looking for some toxic cause, when in reality true hyperpiesia, as distinct from the high blood pressure secondary to chronic renal disease, is not a malady but a diathesis—a constitutional inherited condition. So far as I know, Dawson and Ryle⁴ are the only recent writers who have laid much stress on this aspect of the subject. Ryle has pointed out that the stout, healthy looking, John Bull type of individual is generally the subject of hyperpiesia, which is not acquired but is inborn. Among his patients with hyperpiesia 62 per cent. were of this type, 18 per cent. of average build, 4 per cent. "lean and nervous," and 16 per cent. of poor physique. The question might arise whether the exceptions were really suffering from primary hyperpiesia; in the lean and nervous individual I believe that a high blood pressure is generally secondary to the mental state. I found during the war, in cases of functional hyperthyroidism associated with a high blood pressure, that simple hypnosis often resulted in an immediate fall, amounting even to 45 mm., when the state of anxiety in which the patient lived was temporarily removed. A boy of 16, recently under my care, had an acute attack of nephritis from which he recovered completely, except that the blood pressure remained constantly in the neighbourhood of 156 mm.; as his urine was normal, the blood urea not raised, and the urea concentration test gave a normal result when he came under my observation six weeks after the onset, I allowed him to get up, and told him that his prolonged stay in bed had at last cured him. The next day his blood pressure had fallen to 120 mm., and it remained at this normal level, the previous rise having apparently been due to continuous anxiety, although he always appeared cheerful enough, but bored at having to stay in bed.

The typical hyperpietic has certainly a hypersthenic habitus; he is well built, with broad shoulders and a short, deep thorax with wide costal angle; he has a ruddy complexion, and may be actually plethoric; he is often athletic, unless overindulgence in food and drink has led to a crippling degree of obesity.

Hyperpiesia is often an accidental discovery in an apparently healthy man of exceptional mental and physical vigour. But sooner or later symptoms appear, generally as a result of mental or physical overwork, prolonged worry, excess of food and drink and perhaps of tobacco, or an acute or chronic infection. In women symptoms may develop at the menopause.

The symptoms are generally not due to the high blood pressure itself, but to its effects on the blood vessels or heart. Thus arterio-sclerosis impairs the blood supply to various organs, such as the brain and kidneys, which become