A CLINICAL AND PATHOLOGICAL
STUDY OF GENITO-URINARY TUBERCULOSIS

by

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INTRODUCTION.
INTRODUCTION.

The subject of tuberculosis has been of genuine and absorbing interest to me during the past twenty-six years that I have been engaged in general practice, mainly in the North of Ireland. I propose, therefore, to base this Thesis on a discussion of the clinical and pathological aspects of tuberculosis as it occurs in the genito-urinary tract.

Cases of pulmonary tuberculosis in Northern Ireland have diminished greatly in number during the past ten years. There has been little decrease in the number of cases of bone and joint tuberculosis. The number of patients suffering from genito-urinary tuberculosis has, if anything, slightly decreased and I have been struck by the relatively large number of such cases amongst insured persons.
whom I have examined from time to time in my capacity as part-time Regional Medical Officer for the Ministry of Labour.

Ireland has a population that is mainly rural in character. There is no doubt that in the past tuberculosis has been activated by the primitive hygienic conditions, and by the prevalence of the disease in cattle. Many of the houses have earthen floors, no damp-proof courses in the walls, and very small windows designed with a view to permanent closure. Overcrowding under such conditions lowers resistance and drains vitality.

Fortunately the old conditions are changing. The Government, for the past ten years, have subsidised the building of houses, which have to be built to certain standards before a grant is allowed. It is an offence punishable by heavy fines to keep a cow that is tuberculous.
The people of Ireland, especially in the North, are gradually becoming more alive to the dangers of the disease. This is due not only to the fact that they are educated to a better standard than some years ago, but also to the fact that particular instruction with regard to the causation of tuberculosis is given by the various doctors practising in the rural districts.

The work which I have incorporated in this Thesis is based on personal observations both in life and after death, of a large number of cases of genito-urinary tuberculosis which I have seen during my years in medical practice. For the vastness of my subject I offer no apology. My intense interest in tuberculosis has been sufficient stimulus to enable me to carry out this work.
HISTORICAL SURVEY.
HISTORICAL SURVEY.

The progress which has been made in methods of diagnosis and treatment of genito-urinary tuberculosis since the beginning of this century has been spectacular. The early workers in this interesting sphere were seriously handicapped by lack of knowledge of the causative agent and of the modern methods of accurate diagnosis now at the beck and call of the present day seekers after the truth.

The first case of renal tuberculosis was recorded by Morgagni in 1767. Whilst doing
an autopsy he found the left kidney tuberculous and considered it due to spread from adjacent enlarged glands.

Howslip in 1823 described two cases of renal tuberculosis in a very accurate way, pointing out the frequency of bladder symptoms when the disease was originally situated in the kidney and giving no indication of its presence there.

Bozzini in 1805 invented an instrument to illuminate the bladder called the "light conductor". The instrument was condemned by the medical faculty on the grounds that it provided insufficient illumination. Then Fisher of Boston in 1824 invented the hollow metal tube with a reflected light. This was also condemned on the grounds of insufficient illumination. Desmoreaux in 1853 brought before the Academy of Medicine in Paris the first practical endoscope. In 1865 Cruise of Dublin made an
important improvement in the Desmoreaux instrument. His apparatus was provided with a speculum through which the light from a lamp was reflected on the part to be examined. Various improvements and modifications of this instrument were made, and Christopher Heath used it at the Lock Hospital, London, and introduced it to the medical profession in England in 1866.

Little change took place in this type of endoscope until the year 1879 which was the first year in which electricity was used as an illuminant. The Nitze-Leiter cystoscope of 1879 was hailed by the profession all over the world as a marked advance in diagnostic method. It had its disadvantages however. The water cooling system had to be very efficient, the platinum loop used for illumination fused easily, and the services of a competent electrician were continually found necessary to repair the delicate details of this complicated but
complete instrument.

The next advance was the investigation by Edison of the electric incandescent lamp in the year 1887. This point marks the introduction of the cystoscope as the reliable and almost fool-proof instrument that we have today. Once the principle of the cystoscope became established additions and improvements followed with great rapidity. In a few years it became a very efficient aid in diagnosis and treatment. Now any part of the bladder can be inspected, ureters catheterised, and operations of considerable magnitude can be performed by direct vision with little discomfort to the patient. James Brown of the John Hopkin's Hospital was the first to catheterise the ureters in a male subject through the cystoscope in 1893. This marked a further new development in the diagnosis of genito-urinary tuberculosis.
To the illustrious and epoch-making investigations of Robert Koch in 1882 we owe the discovery of the tubercle bacillus. This great advance lead to the tracking down of many other organisms which collected their human toll with rapid but unerring aim. Koch's discovery had a profound effect on the works of Behring and Kitasato thus bringing about the introduction of the science of immunology. Koch's discovery also was the first positive step in grouping and classifying the clinical condition caused by the tubercle bacillus, and the first effective step in the extermination of this ubiquitous fell scourge of the human race.

In 1883 Bates demonstrated tubercle bacilli in the urine from a case of renal tuberculosis, this being the first occasion on which the bacillus was detected in the urine of a patient suffering from genito-urinary tuberculosis.
THE ETIOLOGY OF GENITO-URINARY TUBERCULOSIS.
ETIOLOGY.

Tuberculosi of the genito-urinary tract is generally accepted as a disease of young adult life, occurring between the ages of fifteen and forty years. It is rare in children and in old people.

Tubercle bacilli enter by ingestion, inhalation or very rarely, by skin inoculation. From the tonsils and bronchi they are conveyed to the lymphatic tissue and glands of the small intestine, and thence via the blood-stream to various parts of
the body including the genito-urinary tract.

By the time a genito-urinary lesion is clinically evident, there is often no sign of tuberculosis in any other organ, although a past history of infection may often be obtained. Post-mortem examinations generally disclose the presence of healed or closed lesions in, for example, the lungs or mediastinal glands.

It is possible that in rare instances tubercle bacilli may pass through the kidney from distant lesions and appear in the urine without setting up a focus in the genito-urinary tract. Harris (1929) in a routine examination of the urine of children and adults suffering from bone and joint disease, and also from tuberculosis of the soft tissues, has pointed out the frequency with which tubercle bacilli can be repeatedly found in the urine.
despite an absence of symptoms referable to the urinary tract. In half his cases investigated by urological methods the bacilli appeared to have a renal origin. In some cases renal lesions subsequently developed; in others post-mortem lesions were found; in yet others no lesions were detectable and the presumption was that they had healed completely. The corollary to this is that the cases which progress to clinical genito-urinary tuberculosis are a small remnant of a large number in which relatively insignificant lesions have healed.

In general, it may be stated that the path of infection is by the blood-stream when the primary source is distant, but it may be by the lymphatics when the primary source is in the neighbourhood of the kidney.

Those who support the theory of
lymphatic origin of the infection emphasise the close connection between the lymphatics of the intestinal and urinary tracts. Brongersma (1908) found that in 60% of cases the primary lesion is in the lungs and pleura on the same side as the affected kidney, and suggests that the path of infection to the kidney is by the thoracic lymphatic glands and connections between them and the renal glands. He explains the unilateral infection of the urinary tract by this route.
THE PATHOLOGY OF

GENITO-URINARY

TUBERCULOSIS.
Renal tuberculosis is most probably never a primary condition, as the tubercle bacillus has no obvious means of gaining direct access to the kidney from without. The lesions to which it is usually secondary are tuberculosis of the lungs, lymphatic glands, and bones. In certain cases of Addison's disease, however, it may occasionally be impossible to demonstrate with certainty any definite active focus elsewhere in the body.

Tubercle bacilli may be passed out
from the blood into the urine by a healthy kidney. It is only when the bacilli are retained in the kidney that disease results. In over three hundred cases of pulmonary tuberculosis which came to autopsy at the Phipps Institute, Philadelphia, 82 per cent showed tubercle bacilli in the urine during life. At autopsy, however, in only 40 per cent were there miliary tubercles in the kidneys, and in only 1 per cent was the ulcerative form of tuberculosis present.

The disease may be of two very different types, the acute or miliary form and the chronic or ulcerative form.

**Acute Miliary Tuberculosis** of the kidney is the renal manifestation of general acute miliary tuberculosis. It may be compared with the
diffuse suppurative nephritis of pyaemia. It is always bilateral, and rapidly proves fatal, the patient commonly dying of tuberculous meningitis. The kidneys are slightly enlarged and studded with minute miliary tubercles.

Chronic Tuberculosis of the kidney may be compared with focal suppurative nephritis. It is always unilateral at the commencement, and may remain so for a remarkably long time. Eventually, however, the other kidney is liable to become involved. The very considerable variations in the statistics given in the literature may be explained by the fact that when the diagnosis made at post-mortem examination the condition will
probably be found to be bilateral, whereas a diagnosis made during life will frequently show only one kidney to be infected.

No adequate explanation has ever been given why the condition should at first be unilateral. The kidney cannot be an organ which is specially liable to tuberculous infection, owing to the comparative rarity of the condition. A large number of local predisposing causes have been suggested, but the arguments supporting no one of them can be regarded as satisfactory. It is distinctly remarkable that there appears to be no relationship between renal calculus and tuberculosis.

Route of Infection./
Route of Infection.

There are three possible routes by which the kidney might become infected:

(1) By the blood stream.
(2) By the lymph stream.
(3) By direct spread from neighbouring diseased organs such as the adrenal or the spine.

Whilst it is possible that in rare cases the second or the third of these routes may be that adopted by the tubercle bacillus, in the overwhelming majority of cases the infection is hematogenous in origin. Tuberculosis of the urinary tract always commences in the kidney. It never begins in the bladder, and then spreads up the ureter to the kidney.
Site of the Initial Lesion.

The primary renal lesion is nearly always in one of the poles, upper or lower, the intervening parenchyma displaying a remarkable degree of immunity. Tuberculous infection parallels in several respects infection by the bacillus coli. In both conditions the infecting agent passes through the kidney filter, settles down in the collecting portion, and then by a retrograde process passes up the lymphatics so as to infect the cortex. In both cases the urine is acid. The tendency towards destruction is, however, far greater in tuberculosis than in bacillus coli infection.

There are two sites in which the process is likely to originate:
(1) In the boundary zone at the base of a pyramid.

(2) In the apex of a papilla as it projects into the pelvis.

To have reached these positions the bacilli must either have traversed the glomerular capillaries and been carried downwards by the inter-tubular set of capillaries; or, what is more probable, they have been excreted into the capsular space, passed for some distance along the tubule, and there been arrested.

Subsequent Course.

A miliary tubercle
composed of endothelial cells surrounded by a zone
of lymphocytes is formed in one of the sites just
alluded to. The usual necrosis of tissue occurs,
and the focus may ulcerate into the pelvis, with
early infection of the ureter, and a resulting
tuberculous pyelitis.

At the same time a retrograde process
commences, the infection creeping up the lymphatics
along the line of the collecting tubules, and eventu-
ally infecting the cortex and even the capsule.
The process of the infection is evidenced by
yellowish-white streaks in the medulla and similar
rounded areas in the cortex. Owing to wide-
spread infection of the pelvis and calyces the
greater part of the kidney eventually becomes in-
volved in this retrograde process.
Gross Appearances.

The appearance of a tuberculous kidney varies greatly with the extent of the lesions and the degree to which destruction has taken place, so that no one description can be given which will be applicable to every case. We may say, however, that in the early stages small miliary tubercles or larger yellowish areas may be observed at one or other pole in the boundary zone, the papillae, or even in the cortex, so that when at operation the capsule is stripped from a small area of the surface the tell-tale tubercles may be detected, a point of diagnostic value. As involvement of the pelvis occurs at so early a date, the pelvic mucosa will be somewhat roughened and lacking in lustre, and the sides of the papillae may be ulcerated.
The later stages are characterised by progressive destruction of renal tissue with cavity formation. The kidney is enlarged, it may be to double the size, and on the surface are to be seen either distinct tubercles or larger bosses denoting cavity formation within. Where the surface is extensively involved there will be adhesions of the perirenal fat. The cut surface shows:

(1) Caseous yellow tuberculous masses, notably at the base of the pyramids, and at one or both poles.

(2) Cavities of varying size with rough, ragged walls, containing thick creamy pus without smell and sterile on culture.

(3) Yellowish lines of infection leading up to clusters of tubercles in the cortex.
(4) Extensive ulceration and dilatation of the pelvis with destruction of the papillae.

(5) Thickening and dilatation of the ureter, although in some cases there may be tuberculous stricture.

One part of the kidney may show early lesions and another part the most advanced lesions. In the late stages a condition of tuberculous pyonephrosis develops, and the kidney becomes converted into a multilocular sac, the various cavities opening into a greatly dilated pelvis, and the whole occupied by thick tuberculous pus. In many cases the condition is due to tuberculous stricture of the ureter; in some, however, the essential cause appears to be loss of tone of the musculature of the pelvis and ureter, the ureter being dilated along its entire length.
The final stage is one in which the kidney becomes a functionless mass of tuberculous tissue. The kidney may be enlarged from pyonephrosis, or may be shrunken and atrophic. The ureter is occluded. Lime salts are deposited in the caseous mass, and extensive calcification may occur.

Microscopically the picture depends entirely on the stage of the disease. The early lesion presents the characteristic appearance of a miliary tubercle. As the disease advances the unaffected part of the kidney may show a condition of toxic tuberculous nephritis, due to the action of the tuberculous toxins on the renal epithelium. In
the late stages amorphous areas of caseous material are surrounded by tissue which has become so fibrosed that all resemblance to kidney structure is lost.

Healing.

The question of whether tuberculosis of the kidney is capable of spontaneous cure is of great importance from the standpoint of treatment. Partial cure by fibrosis undoubtedly occurs. Outside the fibrosed area, however, it is sometimes possible to demonstrate small active lesions, and for this reason many clinicians claim that extirpation of the kidney is the method of
treatment par excellence.

At the same time it must be remembered that elsewhere in the body the surgical treatment of tuberculosis is becoming more and more conservative, and in the future the treatment of tuberculosis of the kidney may be less drastic than it is at present. It must always be borne in mind that the process is not primary in the kidney, that the kidney lesion is only a part of a generalised and systemic disease, and that at any time the remaining kidney may also become infected.

The Other Kidney.

Renal tuberculosis is at
first a unilateral condition. Sooner or later, however, it is most likely to become bilateral. Two different pathological conditions may occur in the other kidney:

(1) Toxic nephritis of tuberculous origin.
(2) Renal tuberculosis.

The toxic nephritis is due to absorption of toxins from the infected kidney. It manifests itself by the appearance of albumen and sometimes casts in the urine. When the primary source of infection is removed this nephritis speedily clears up.

Tuberculous infection may pass down the ureter from the first kidney, infect the bladder, and pass up the other ureter to infect the second kidney. Or the infection may be hematogenous
in origin. The tuberculous toxic nephritis just alluded to weakens the resistance of the kidney, and it succumbs to a blood borne infection. Which of these methods of infection is the commoner it is difficult to say.

The unilateral character of early tuberculosis is well exemplified in the case of a horse-shoe kidney. In this condition there may be widespread infection of one pelvis and yet the other may be quite free. Microscopic examination, however, will frequently reveal the presence of miliary tubercles in the cortex of the other half. These, apparently, are due to infection carried by the lymphatics in the capsule.
TUBERCULOSIS OF THE BLADDER AND URETER.

For practical purposes it may be said that tuberculosis of the bladder is never primary. It is always secondary, to tuberculosis of the kidney in the great majority of cases, to tuberculosis of the prostate, seminal vesicles, and epididymis in a few cases, and very rarely to tuberculosis of adjacent organs such as Fallopian tubes.

In renal cases the disease commences at the ureteric opening, in prostatic cases it begins at the neck of the bladder. As involvement of the ureteric opening occurs comparatively early in
renal tuberculosis, cystoscopic examination plays a most important part in the diagnosis of the condition. Only too often, however, by the time the patient presents himself for treatment the disease has spread to such an extent that the entire trigone is involved. The initial lesion is a tiny, white, translucent tubercle covered by epithelium and situated in the subepithelial connective tissue. Soon, however, ulceration of the overlying epithelium occurs. The tuberculous ulcer is characteristically round, with ragged, overhanging edges, and a grey, shaggy floor. Although occasionally covering a considerable area it is seldom deep, and perforation is almost unknown.

The infection is at first a pure tuberculous one, but sooner or later a mixed infection is sure to occur, and the condition becomes one of
tuberculous cystitis. Removal of the offending kidney will be followed, in the earlier cases, by recovery of the bladder lesions in the course of a few months. In the later and more advanced cases the duration may be for years or it may be forever.

In the ureter the infection passes down in the submucosa. In this layer there may be definite tubercles, or the formation of a diffuse, tuberculous granulation tissue, such as is found in the intestinal submucosa. Ulceration of the mucosa occurs, followed by infiltration and thickening of the muscular and serous coats. The upper and
lower thirds are usually much more involved than the middle third. Scar formation may give rise to stricture, but more often the ureter is converted into a rigid, thickened, and markedly dilated tube.
TUBERCULOSIS OF THE PROSTATE.

Owing to its anatomical situation the prostate may be infected with tuberculosis either from the urinary or from the genital tract. The subject will be considered in greater detail when genital tuberculosis is discussed. The disease process may commence in the prostate instead of spreading from the kidney or the epididymis.

Experimental work has shown that the initial lesion is found just under the epithelium lining one of the ducts. Miliary tubercles are
formed which increase in size, giving rise to caseous masses scattered throughout the gland. Although the early lesions are probably on the same side as the infected epididymis, by the time the disease is diagnosed clinically the greater part of the gland is involved, and firm nodular masses can be felt on both sides. Occasionally there is a diffuse tuberculous infiltration without nodule-formation. If caseation is extensive a tuberculous abscess is formed, which may burst either into the urethra or upon the surface. In the latter case a tuberculous fistula will result.

By the time that tuberculosis of the prostate can be diagnosed clinically both seminal vesicles will always be found to be involved.

Tuberculosis of the prostate resembles
enlargement of the prostate in that it gives rise to no distinctive symptoms per se. The symptoms of the patient are those of tuberculosis cystitis, or he may come complaining of an enlarged testicle.

Once tuberculosis has become established in the genito-urinary tract it seldom undergoes spontaneous cure, but when the kidney or the epididymis which is the source of the infection has been removed the disease in the prostate may become quiescent or may actually clear up completely.
TUBERCULOSIS OF THE GENITAL TRACT.

The site of origin of genital tuberculosis is still a matter for discussion. The general opinion is that the disease commences in the epididymis, but it has recently been demonstrated that in many cases, perhaps in most, the primary focus is in the seminal vesicles. From the seminal vesicles the globus minor of the epididymis is generally next attacked, and from the same source the prostate, the urethra, and the bladder may be involved later. The importance of removing the
seminal vesicles in any surgical treatment of genital tuberculosis is therefore self-evident.

One point too frequently forgotten must always be kept in mind. Genito-urinary tuberculosis is not an isolated manifestation of the disease. In the great majority of cases tuberculous lesions can be demonstrated in other organs, usually the lungs, and where these cannot be found it may confidently be assumed that they nevertheless exist. From the point of view of treatment, which is that of the patient and not merely of the affected organ, this conception is of paramount importance.

In whichever part of the seminal tract the disease may begin the mode of infection must be haematogenous, except in those cases where the bacilli
travel from the kidney down to the prostate.

Morbid Anatomy.

The lower pole of the epididymis is the first part involved. The disease may remain confined to that part, or it may gradually involve the whole of the epididymis, but particularly the upper pole. At first one and then several nodules are formed, so that the whole epididymis becomes considerably enlarged. In the early stages the nodules are very firm and hard, a characteristic which can readily be recognised on
palpation. Sooner or later, however, caseation, softening, and liquefaction occur. Tuberculosis in the genital tract seldom displays the natural tendency to healing which is so marked in the lungs. Even if the infection is not advancing it remains ready to spring into activity at the slightest provocation, and the patient once infected has the sword of Damocles continually suspended over his head. When softening occurs the skin of the scrotum may become adherent to the epididymis, with the formation of a tuberculous fistula. On the other hand softening may not occur, or may be delayed for several years.

Spread to Other Organs.

Tuberculosis does
not remain confined to the epididymis. Indeed it is the fact of its early spread to the other parts of the genital tract which makes the determination of the point of onset a matter of so much difficulty. When the patient is first seen by the clinician and still more frequently when the parts are examined at autopsy, the epididymis, the vas deferens, the prostate, and the seminal vesicles may all be involved. The path of spread of the infection is a matter of dispute. The obvious route is via the lumen of the vas, but it is more than possible that it is along the lymphatics in the wall of the vas that the bacilli travel. Finally, it must be remembered that if the blood stream has brought infection to the epididymis or seminal vesicles, the other genital organs may be similarly attacked.

The spermatic cord is thickened, and
hard masses may be felt here and there along its course. Similar nodules are present in the prostate and seminal vesicles. At a later stage these may caseate and soften.

The _tunica vaginalis_ is frequently involved, and its inner surface may be found to be studded with tubercles. A hydrocele is the natural consequence, and may have to be tapped before the outline of the testicle can be made out.

The testicle itself may escape for a considerable time, but it will not forever remain inviolate. The first part to be involved is the body of Highmore. The fact that the lymphatics of the testicle concentrate at this point suggest that the path of spread may be lymphatic rather than along the seminal tubules. The disease may
remain localised to this part of the testicle, or may spread diffusely throughout the organ.

The condition of the other epididymis must be considered in every case. It is a lamentable fact that involvement of the other side is apt to occur no matter what precautions are taken, no matter how radical the operative treatment. Once again the route of infection is a matter of discussion. It is improbable that the bacilli pass along the vas in the direction opposite to the normal stream. More likely the spread is by the lymphatics or by a fresh haematogenous infection.

Finally, the infection may spread to the urinary tract. From the prostate one or both kidneys may become involved. A more common occurrence, however, is the downward passage of the
bacilli from the kidney to the bladder, with spread to the prostate, vas, and epididymis. About 30 per cent of cases of renal tuberculosis in the male are associated with genital tuberculosis, but the spread is more likely to be downwards than upwards. Still more probable is an independent haematogenous infection of both kidneys and epididymis.
CLINICAL MANIFESTATIONS

AND DIAGNOSIS.
In the early stages of genito-urinary tuberculosis the symptoms may be so completely absent or trivial that the patient does not seek advice. In spite of advanced destruction of the kidney tissue, it may not be until the lower urinary passages, and more especially the bladder become involved that the presence of some serious disease is suspected.

Frequency of micturition is the commonest symptom, and is only rarely absent when patients apply for treatment. It sometimes begins
suddenly, but this is not usually the case. The frequency has a marked tendency to amelioration over long periods. It is present both night and day. It can occur when the only signs in the bladder are slight oedema of the affected ureteric orifice, so in the first instance must be due to irritation. Later, when the bladder has become scarred from extensive involvement, there is diminution of capacity, which leads to some degree of frequency after the disease is cured. The frequency is much less than when the disease is active, owing to the healing of the ulcers abolishing the irritation and the oedema of the bladder wall. Severe frequency may lead to incontinence from the patient not having sufficient time when the desire to micturate is felt.

Frequency is commonly, but not invariably
associated with some form of pain occurring with micturition; occasionally this may begin before the frequency. The commonest form of pain is scalding during micturition. Terminal pain at the end of the penis is quite common. Pain in the perineum or hypogastrium associated with micturition is not uncommon. Terminal pain radiating from the end of the penis up to the affected loin and down to the testicle of the affected side may occur. The hypogastric and perineal pain is really a very intense desire to micturate; it occurs before, during and after micturition, the latter pain being due to the contraction of the diseased bladder persisting after the bladder is emptied, as can be easily seen if an attempt is made to wash out the bladder of such a case.
About a third of patients when first seen either have or give a history of haematuria. In the majority of cases this is due to disease of the bladder and is associated with marked symptoms of this; in such cases the haematuria is often terminal. The haematuria of renal tuberculosis is occasionally renal, and severe renal haematuria is, at times, the first symptom of the disease, in which case years may elapse before other symptoms develop.

Pain in the lumbar, hypochondriac or iliac regions of the affected side is nearly as frequent as haematuria. The pain is most commonly behind the loin, and it may radiate to the external ring or down behind the buttock; less commonly it is immediately below the ribs in front and radiates
back to behind the loin. The pain has marked exacerbations and remissions, and in some cases is made worse by exercise. When severe it is associated with a varying degree of muscular rigidity of the abdomen and loin on the affected side. The pain is only rarely associated with vomiting. Pain in the iliac region by itself is much less common; when it occurs there is sometimes a history of previous lumbar pain. Occasionally lumbar pain may arise at the end of micturition, radiating upwards from the penis or hypogastrium. A typical renal colic occasionally arises from a tuberculous kidney from the passage down the ureter of a calcified concretion.

Patients occasionally complain of difficulty or retention of urine. This may be
due to tuberculosis of the prostate or to a calcareous concretion of the bladder. The symptom may be present in the absence of either of these conditions. In such cases, when a catheter is passed to relieve retention, it is found that only a small volume of urine is present in the bladder, the feeling of retention being produced by the disease of the bladder.

Patients with renal tuberculosis are seldom wasted until the last stages of the disease, and often not then.

Fever is generally absent, and when present should always arouse the suspicion of tuberculosis somewhere else. It does occasionally occur from renal tuberculosis if the kidney is becoming distended, or if the disease is advancing
with unusual rapidity.

Abdominal examination commonly shows suprapubic tenderness when there is well-marked disease of the bladder. As already stated, when lumbar pain is severe, abdominal and lumbar rigidity may be present. Palpable abnormalities in the kidney itself in renal tuberculosis are unusual. If the kidney is easily palpable it may feel harder than normal. Occasionally a palpable enlargement may be felt, but as the disease commonly runs its whole course without the kidney becoming enlarged at all, and as enlargement may occur in the upper half of the kidney to a very great extent without being palpable, this is very unusual.

Pus is constantly present in the urine, except in the very rare cases where closing of the
ureteric lumen, or the lumen of the branch of the renal pelvis draining the affected part of the kidney, has taken place after the disease has become quiescent. Except in very early cases the pus is present in sufficient quantity to form turbidity or even thickness of the urine when freshly passed; even in very early cases if repeated examinations are made by this method pus will generally be found before long; microscopic examination will show it in either case. The urine is strongly acid. The best chance of finding tubercle bacilli by direct examination is given by collecting the urine for twenty-four hours and examining the deposit of this after centrifugation. When examined carefully by the direct method a single examination in tuberculous cases is probably positive in 80 to 90 per cent. The
urine in renal tuberculosis usually contains no other organisms than tubercle bacilli, and therefore gives no growth when sown on agar. As already stated blood in the urine is common, the amount varying from time to time.

When any considerable degree of tuberculosis of the bladder is present a small amount (100 - 150 c.c.) of residual urine is commonly present; this may bear a very large proportion to the amount passed at each micturition, which it may considerably exceed. This occurs in other severe chronic inflammatory diseases of the bladder. It may be due to loss of elasticity of the bladder wall or to an unconscious means of preventing the pain resulting from completely emptying the bladder. The
fact that this pain is prevented by not emptying the bladder completely is readily seen if such a bladder is washed out; this, and the fact that failure to contract completely does not seem to occur in tuberculosis, renders the second explanation more probable.

Thickening of the ureter, which is a very common effect of tuberculosis, can often be felt in the anterior fornix on the affected side on vaginal examination. The same may occur on rectal examination in men, but it may be in this case very difficult to differentiate from a tuberculous vesicula seminalis.
Diagnosis.

The diagnosis of renal tuberculosis consists in establishing the existence of the disease, and subsequently in determining whether both kidneys are involved or only one, and if so which one.

Considerably over three-quarters of the cases apply for treatment on account of symptoms of cystitis with or without lumbar pain. Epididymitis and renal haematuria or symptoms directly due to a stone, such as renal colic, are the cause of seeking advice in most of the remaining cases. Very rarely patients apply for treatment on account of the development of a cold abscess, most commonly in the loin.

The most important examination for
diagnosis is that of the urine. If pus, sufficient in amount to produce turbidity, together with tubercle bacilli, are present in the urine, tuberculosis of the kidney or bladder, or both, must be present, except in the case of a man with an abscess of the prostate which has burst into the bladder or urethra. Therefore, if gross prostatic disease is excluded by rectal examination, such a combination always indicates the presence of renal tuberculosis, as primary tuberculosis of the bladder is either non-existent, or so rare as to be of no practical importance in diagnosis. This examination applied once, and properly carried out, will establish the existence of the disease in over three-quarters of the cases, but a negative finding is sufficiently frequent to make a negative result of very little diagnostic value. If
guinea-pigs are inoculated, negative results in positive cases are less frequent, but even in this case a negative result is not absolute.

Examination of the urine may show a considerable amount of pus in an acid urine with no tubercle bacilli or other organisms, either by direct examination or on culture. Apart from renal tuberculosis, this result occurs occasionally in renal stones, and occasionally in late stages of intractable gonorrheal posterior urethritis. If these two conditions are excluded, the presence of renal tuberculosis becomes very probable.

Renal tuberculosis may easily be confused with renal stones: firstly, the common combination of symptoms is not dissimilar, and secondly,
the patients do, in uncommon cases, actually have stones, which are really calcified tuberculous products, and which produce all the usual symptoms of stone. X-ray examination, therefore, is of considerable diagnostic importance. Renal tuberculosis gives an X-ray shadow when some part of the diseased kidney becomes calcified, the extreme case being when the kidney is destroyed and represented by a thin fibrous sac with incomplete septa filled with white mortary material. This condition gives a thin spongy shadow, which is lobulated, and the shape of the kidney or of a large part of it. This shadow can be simulated by stone when a large hydro-nephrosis containing very numerous, very minute stones, which settle into the lower loculi, is present. The shadow in the case of the hydronephrosis will be
in the lower part of the tumour, and in places will be much denser than that cast by calcified tuberculosis. When the area of calcification is smaller the shadow may be mistaken for that of a single stone, the fact that the shadow is not of uniform density and may be situated in some very unusual place for a single renal stone, such as the upper pole, should arouse suspicions as to its nature. A primary renal stone and renal tuberculosis occasionally co-exist.

When genital tuberculosis is present in a man renal tuberculosis may or may not co-exist. If the urine is clear, possibly containing a very few, small, heavy threads made of leucocytes, it may be assumed renal tuberculosis does not exist. If, on the other hand, the urine is turbid with pus,
renal tuberculosis is present, unless an abscess of the prostate is present, which must be found by rectal examination. If an abscess of the prostate is present, the diagnosis can usually be settled only by cystoscopy; this should not be done if the patient is unfit for a nephrectomy in any case, as it will require an anaesthetic and is not necessarily harmless in the circumstances.

When the existence of the disease is established it becomes necessary to know whether either of the kidneys is not diseased, and with one rare exception this can only be done by cystoscopy. The only combination of circumstances where the integrity of one kidney can be established without cystoscopy is when one kidney can be shown by X-rays to be completely destroyed and the urine is normal.
As cystoscopy is the most tedious of all the examinations, all other means should be taken to find conclusive evidence of disease of one or other kidney in order that time may not be wasted examining this side during the cystoscopy. It may occasionally happen that conclusive evidence of disease of both kidneys can be found without a cystoscopy, such as when X-ray shadows are present on both sides, or when there is marked deficiency of the total renal function shown by a marked rise in the blood urea, etc.; in such cases cystoscopy is unnecessary.

Lumbar pain, when severe, is good evidence of disease on its own side. Enlargement of the kidney has to be marked to be of any diagnostic value, as the unaffected kidney may feel enlarged and also rather tender. Tenderness of the diseased
kidney on pressure is often absent. If the kidney, without question, feels harder than normal it is a sign of disease. A palpable indurated thickening of the ureter on vaginal examination can be regarded as certain; the same cannot be said of rectal examination in a man because of error caused by the vesicula seminalis.

**Cystoscopic Examination.**

In the early stages of the disease no change is found by cystoscopy. In most cases very soon alterations
occur in the ureteric orifices of the affected side; the early change in this is that its edges become oedematous, and the oedema may spread to the bladder mucous membrane round it. Subsequent changes which take place in the ureteric orifice are that it may become ulcerated, it may become "holed", i.e. remain rigidly open at all times, or it may become retracted. Any of these changes may be combined. Except retraction, none of these changes is pathognomonic of tuberculosis, but each indicates the side of a diseased kidney. A retracted ureteric orifice occurs only in tuberculosis; the ureteric opening is displaced upwards, backwards and slightly outwards from its normal position, and generally lies in a pit, where two approaching ridges running from its old to its new position meet. A retracted ureteric opening appears to arise from shortening of
the whole ureter, together with its adhesions to the posterior abdominal wall. Besides changes in the ureteric opening, and efflux of pus or blood will indicate a diseased kidney, whether the orifice is changed or not.

Disease of the bladder outside the ureteric orifice commonly begins and is more extensive on the affected side, but it may involve the opposite side, so that the orifice is situated in a diseased part of the bladder and yet the opposite kidney is still uninvolved. The earliest change seen in the bladder is a circumscribed bright red area either in close proximity to the ureteric orifice of the affected side or on the anterior wall. These areas enlarge, become multiple and ulcerate, but except in very extensive cases quite normal bladder
mucous membrane will be seen between the diseased areas. Tubercles are sometimes seen near the edges of the ulcers or in the red areas. In late cases scarring produces sharp ridges of mucous membrane radiating from an ulcer. Patches of mucous membrane sometimes become markedly oedematous, and then have a superficial resemblance to growths.

By the end of the cystoscopic examination, therefore, it has become evident in the majority of cases that a particular kidney is certainly diseased. All that remains is to catheterise the opposite ureter and show that the urine has a good concentration of urea and is free from pus. A few leucocytes in the urine from the sound kidney are not uncommon, but if there are more than a few, and any doubt arises, nephrectomy should be postponed six weeks until the result of inoculating a guinea-
pig with the urine from the supposed sound kidney can be obtained. If obvious pus is present in the urine of the supposed sound kidney the disease is clearly bilateral. An indigo-carmine test applied to the sound side adds additional security.
TREATMENT.
GENERAL TREATMENT.

The treatment of genito-urinary tuberculosis neither begins nor ends with surgical intervention. The general treatment of the underlying disease is all-important. Resistance must be raised to the utmost by rest, fresh air, and good food. These are the essentials of sanatorium treatment.

The value of fresh air is inestimable. The patient should, as far as is possible, live continuously in the fresh air. Chill must naturally be prevented, and a patient should never complain of feeling cold.

Diet. The patient with tuberculosis should be put on a simple diet, rich in vitamins, and of adequate caloric value for the degree of exercise that he may be taking.

Zomotherapy in many cases has a beneficial effect. Personally, I have used it with great success.
With a satisfactory diet, patients, when appreciably below their normal weight, regain their loss often at the rate of several pounds per week. On reaching the average weight for their age and height, the caloric intake can often be reduced with advantage. The giving of fluids is important, diuresis certainly lessens the risk of infection to the opposite in cases of renal disease.

Drugs in the form of cod-liver oil, arsenic, iron, and so on, are all of secondary value. Urinary antiseptics too are usually a mere waste of time and money.

Rest and regulated exercise are both of paramount importance. It is difficult to formulate rules with regard to either the amount of rest that should be taken or the degree to which the patient may take exercise. Roughly speaking the extent of the lesion acts as a guide.
TUBERCULIN THERAPY.

Not long after Koch introduced tuberculin it was utilised in the treatment of genito-urinary tuberculosis. Since then clinical data have accumulated, differing widely in opinion. Emerging from the mass of statistics and the great amount of work that has been done, it may be stated that tuberculin is not a cure for genito-urinary tuberculosis but nevertheless it cannot be dismissed as a therapeutic agent of no value. There is considerable evidence that it aids in building up the general health of patients suffering from
this type of tuberculosis.

The reports of the results with tuberculin therapy may be logically divided into three groups. Those who find it of value, others who are uncertain, and those who believe that it has no practical value in treatment.

Tuberculin is given a place in treatment on the theory that it causes an inflammatory focal reaction at the site of the disease, and thus promotes fibrosis of the lesion. It is recognised that it is difficult to bring about unharmful focal lesions since the characteristics of the lesion vary so widely, particularly in renal tuberculosis. In the same kidney lesion tubercules may be present in all phases, and it is evident therefore, that the effect of a given amount of tuberculin on the various lesions must vary widely, (Wang 1930).
In prostatic tuberculosis, Lowsley and Duff (1930) maintain that Koch's Old Tuberculin in small doses, increased gradually at regular intervals, causes inflammatory reaction at the site of disease, and promotes fibrosis of the lesion. The clinical appearance of evidence of local or constitutional reaction is the guide to dosage, they state, the usual initial dose being 0.1 c.c. of a 1 in 10 million dilution, given subcutaneously.

The results of treatment with tuberculin are certainly difficult to evaluate, particularly when it is used as a part of a general regime. There are few if any standards for its administration in genito-urinary tuberculosis. The reactions produced differ widely. Dosage has therefore to vary with the exacerbations and remissions of the
disease, and the clinical manifestations are always the best guides. The giving of tuberculin requires some knowledge of its rationale, and it is necessary to exercise careful judgement in its application.

I have found that Morö's ointment, given by inunction, is a most convenient method of administrating tuberculin, and in my experience it is associated with results as encouraging as subcutaneous injection.
The scope of surgery in genito-urinary tuberculosis has given rise to much argument. The term "surgical tuberculosis" is to be deplored. To those who understand the genesis of tuberculosis and how it affects the human body, surgery can never be more than a useful adjunct to systemic treatment along the lines that have just been indicated. Eradication of the disease as we know it can never be effected by surgical intervention, but nevertheless
in certain well-chosen cases the surgeon can collaborate successfully.

Innumerable writers, most of them surgeons, have given is their results of the effects for example, of nephrectomy in renal tuberculosis. They claim that the immediate mortality from operation is small, and that it is undoubtedly lowered in proportion to the earlier date of diagnosis, and the greater care taken in investigating the function of the opposite kidney.

The patients who subsequently die of a spread of disease in the genito-urinary tract or elsewhere in the body, do so within one or two years. The majority survive for many years. It is claimed that those latter do not develop tuberculosis elsewhere, a statement, in our opinion, which must be
very difficult to substantiate.

Crabtree (1913) in reviewing seventy cases of unilateral nephrectomy had an operative mortality of 3.8 per cent, and a late mortality of 20 per cent, half the deaths occurring within two years, and the remainder within five years. The percentage of "cases" was sixty. In 35 per cent symptoms persisted. Judd (1923) reports, "complete restoration to health;" in a period of three years, in 53 per cent of his cases. Similar figures are given by Thomson-Walker (1927), 60 per cent, and Wildbolz (1913), 59 per cent.

It may be stated that conservation in surgical intervention has of recent years become more and more the attitude of clinicians, and it is becoming more widely recognised that only in carefully
selected cases, with gross disease, and where certain aggravating symptoms may be alleviated, can surgical interference be of universal benefit to the patient.
CASE No. 1.

M.C.F.  

History -  

Patient had a tuberculous testicle removed in 1922.  
No other previous history of illness.  
About 3 years ago the patient began to complain of frequency in passing urine.  
About every half-hour, sometimes more frequently at night.  
This improved temporarily with treatment but got worse again till about 2 months ago when there was some improvement.  
Patient has been occasionally passing pus and blood for the past 2 years.  
The blood came at the end of micturition.  
Pain before and during the actual passage of urine.
Examination -

Patient looks healthy and well nourished.

Teeth, tongue and pharynx - normal.

Heart, lungs, C.N.S. - N.A.D.

Cystoscopic examination - The capacity of the bladder 4 ozs. Mucous membrane appeared normal.

The right ureteric orifice was situated on the top of a small elevation evidently composed of tubercles. The left ureter was difficult to find as it was drawn up into a small thickened patch - the typical golf hole ureter. The field was obscured by large quantities of pus which came down each ureter. The ureters were catheterised.

Laboratory report - Right side Specific Gravity 1011. The left side 1010.

Tubercle bacilli more numerous on the left side.

Blood area 146 mgms per 100 c.c.
No operation was considered. The patient sent home where he is carrying out open air treatment, on a diet consisting mainly of eggs and milk. In addition he is having tuberculin.
CASE No. 2.

M. G. Aet 30 years.

History -

Glands in neck 10 years ago.

Recurrence 5 months ago; removal by operation.

Three months ago the patient began to have frequency and pain on micturition. After 10 days this difficulty cleared up but there was still some pain. For the past 2 weeks frequency and passing urine at hourly intervals during the day and about three times during the night. No loss of weight.

General health good.

The patient looks healthy and well nourished.

Teeth satisfactory. Tongue moist and clean.

Throat normal.
Heart, lungs, abdomen - N.A.D.

On 28. 6. 32. specimen of urine from left ureter showed large quantities of pus and cells, mainly monocytic. No organisms. Sterile on culture. Specific Gravity 1012. Right ureter showed a few blood cells. No organisms. Culture sterile.

Blood urea 37 mgms per 100 c.c.

Cystoscopic examination - The bladder held 4 ozs without discomfort. The right ureteric orifice looked normal; the left enlarged and showed thickening around the margins and several small ulcers. Ureters catheterised, marked diuresis on the left side. Nephrectomy of the left kidney, a drain inserted and the wound closed. The perinephritic tissues were healthy. The ureter was much thickened.
Pathological examination showed the presence of typical tubercles in the medulla.

On 27. 7. 32 the scar was well healed. No frequency.

This case was running an irregular temperature Maximum 99.8. This became sub-normal one week after the operation.
CASE No. 3.

E.S. Act 43 years.

History -
In April 1932 sudden severe pain in the right lumbar region with haematuria. There was no frequency of micturition. He vomited once.

On examination -
Temperature 99.
Heart, lungs and abdomen - normal.
Rectal examination - the prostate was normal.
The epididymis was normal.
The urine contained albumen, blood and pus.
Cystoscopic examination - 25. 4. 32.
Bladder holds 8 ozs. Bladder wall shows slight cystitis.
Right ureteric orifice - normal.
Left ureteric orifice – redness and inflammation.

One white spot suggestive of tubercle. The appearances are suggestive of an affection of the left kidney.

On 22. 4. 32 tubercle bacilli were found in the urine. The blood urea was 38 mgms per 100 c.c.

Uroselectan showed poor filling of both kidneys on X-ray. No diagnosis possible. This case was not considered sufficiently definite to warrant removal of the kidney.

It is now under medical care with tuberculin.
CASE No. 4.

J.S. Aet 23 years.

History -

Glands of neck removed in childhood.

Frequency of micturition since November 1931. This has become much more distressing and now occurs at very short intervals. He is much disturbed at night.

Haematuria - This has been marked on two occasions.

Pain is complained of in the region of the right kidney, marked on micturition.

Turbidity of the urine.

There is a history of pyrexia for an indefinite time past. This has touched 100 degrees and the fall has been accompanied by heavy sweating.
The patient states he has lost 2 stones in weight.
He has recently developed cough and spit.

Examination -

Lungs. - Dullness on percussion and crepitations on auscultation over the lower 2/3rds of the right lung. The left lung was normal.

Heart and abdomen - normal.

It was found impossible to pass a catheter or to cystoscope this patient owing to marked obstruction in the posterior urethra.

On rectal examination the prostate was found to be enlarged and nodular.

The right epididymis was found to be enlarged.

The sputum after repeated examination - no tubercle bacilli. There was a pure culture of microcococcus catarrhalis.
The blood urea was 32 mgms per 100 c.c.
X-ray examination of thorax showed dullness at the base of the right lung. Soft wooly infiltration in the right middle zone. There was little or no fluid. The heart was not displaced.
Uroseletan was given but the resulting X-ray did not reveal anything definite. There was evidence of calcified glands to the right side of the spine. There was no sign of calculus.

Progress of the case. - This case returned home after three months nursing home treatment. He is at present living on his farm in a shelter. General measures are being adopted and small doses of tuberculin are being administered. The patient, however, is slowly losing ground.
CASE No. 5.

A. B. Aet 38 years.

This patient had a severe haemoptysis in 1915. Since then he has suffered from pulmonary tuberculosis and has sought treatment in various parts of the world. He has been in Australia and has spent two years in Switzerland. The sputum is still positive to T.B.

The physical signs in the chest are those of a fibroid phthisis, with a large cavity in the upper lobe of the right lung.

The temperature was usually normal: pulse 50 to 60 regular: blood pressure 156/80.

The heart was normal.
In July 1932 albumen appeared in the urine with granular casts. The albumen rapidly increased in amount. No tubercle bacilli were found in the urine.

Blood urea was 314 mgms per 100 c.c.

He is now developing drowsiness and twitching and the amount of urine has fallen to two ounces in 24 hours. Uraemia is obviously impending.

I regard this case as belonging to the group described by Thomson Walker as tuberculous nephritis.
H.D. Act 12 years.

History -

Three years ago, when at school in England, he noticed that he had to pass water more frequently than usual. The urgency was so insistent that he consulted the school doctor. Urine examination showed blood and tubercle bacilli. He was then sent to Switzerland for two years, and on his return he came under my care.

Frequency and urgency not now so marked but still very troublesome. He had one attack of haematuria six months ago.

Cystoscopic examination not permitted.

Urine still positive T.B. No albumen nor casts found.
Temperature normal. Pulse rate 78 per minute, regular in time and force. No night sweats. Gaining in weight.

Treatment adopted. - Tuberculin in the form of 50% Moro's paste once weekly, rubbed in over sternum an area about the size of a five-shilling piece.

Is kept in a open shelter of approved style winter and summer. Allowed to play mild games and goes for short walks - one mile morning and afternoon.

Diet - Finely minced raw meat, meat juice, raw eggs and carbohydrate in limited amount. Milk given about 2 hours after meals.

This boy's appetite and general condition have greatly improved since he started on the finely minced raw meat.
CASE No. 7.

J.D.M. Aet 43 years.

History -

Patient who is a strong healthy man began about 8 years ago to be trouble with frequency of micturition. He had a slight haemorrhage before he consulted me.

Examination -

Heart and lungs, apparently healthy.

Both testicles and epididymes were normal in appearance and normal to touch. No sign of any thickening or enlargement.

The urine contained no albumen nor casts. No blood. A few pus cells present.
Dystoscopic examination - The bladder appeared normal but the opening of the right ureter was swollen and somewhat oedematous. The ureters were catheterised. Right side - Specific Gravity 1008. Left side - Specific Gravity 1014. Few pus cells from right specimen and tubercle bacilli after guinea pig inoculation.

It was decided to remove the right kidney; this was done 7½ years ago.

Patient enjoyed quite good health for about 7 years. He again visited me. Urine showed blood, tubercle bacilli and pus. The left kidney now obviously affected.

Both epididymes were involved. Prostate somewhat enlarged and nodular.

Temperature 98.6 evening. Pulse rate about 80.

Treatment adopted. - Tuberculin, zomotherapy, aerotherapy, and moderate amount of exercise.
CASE No. 8.

Male aet 50 years.

History -

This patient while a medical student, in Edinburgh, developed an attack of influenza in 1903, but recovered in the course of four weeks. He lived in a house where the landlady's daughter had consumption from which she shortly afterwards died.

All went well for about three months when suddenly he developed severe pain and swelling in left epididymis which ran an acute course. Removal of testicle was advised by Professor Annandale, and
thereafter patient went for a prolonged rest to his native country. Whilst there, a similar swelling started in the right epididymis. Sir Charles Ball of Dublin did not advise further operative interference. The epididymis became hard and fibrous in about six months, and has remained so ever since.

All went well for eleven years. Patient was vigorous, healthy and full of energy, but in 1915 he had a slight haematuria which subsided in the course of a couple of days. Urine was then sent away for guinea pig inoculation and tubercle bacilli were found. Haematuria recurred in 1916, more severe than before.

Patient got very dyspeptic, vomited frequently - generally at night. It is interesting to note that when a large protein meal was ingested no pain
followed. The most severe pain followed a light carbohydrate meal. Raw meat, eggs, underdone steak and chops never gave rise to any pain.

This see-saw sort of existence continued for at least two years, the patient carrying on a busy general practice. Things reached a climax when a twisting pain developed in the left costal margin and a severe stabbing pain in the left kidney. Mr Mitchell, Belfast, was consulted and diagnosed obstructive appendicitis with adhesions. He then operated with considerable success inasmuch as the patient was freed from the twisting pain, but there was persistent dragging pain in the abdomen and left kidney region for four years after the operation. This gradually cleared up then and except that the patient was very indiscreet in the way of exercise or standing about too much, he was able to live in
comparative comfort. The only remedy for the dragging and kidney pain appeared to be rest.

All went smoothly until about 1926 when another haematuria occurred. After this patient was debilitated and feeling very ill. He then visited Sir Robert Philip in Edinburgh who discovered that he was suffering from glycosuria, probably due to the septic condition of the kidney. He advised the use of Moro's paste and this has been continued ever since with marked benefit in general health and range of activity.

For six months after his visit to Sir Robert Philip the patient passed quantities of pus in the urine. When this clear up the glycosuria disappeared.

The patient would not allow cystoscopic examination to be carried out.
Urine examination in September 1932 showed blood, pus, and tubercle bacilli. The general systemic condition of the patient however is fairly good.
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SUMMARY AND CONCLUSIONS
SUMMARY AND CONCLUSIONS.

1. A brief note has been given of the earlier work on genito-urinary tuberculosis and its diagnosis by specialised methods.

2. The etiology and pathology of the disease has been discussed at some length. It is maintained that only by an accurate knowledge of the genesis of tuberculosis can we hope to be successful in our treatment.

3. The clinical manifestations described are based on personal observations made over a period of twenty-six years. Recent methods of diagnosis are mentioned.

4. A brief outline is given of treatment particularly in regard to the giving of tuberculin, and a plea is put forward for its more extensive use in the disease. The relationship of surgery to genito-urinary tuberculosis is discussed.
5. A number of case records are detailed, which are illustrative of the type of case met with in general practice.