GENITO-URINARY TUBERCULOSIS

A STUDY OF THE DISEASE IN ONE UNIT OVER A PERIOD OF 24 YEARS

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ONE of the earliest references to genito-urinary tuberculosis was made by Cohnheim in 1879[1], three years before Koch discovered the *Mycobacterium tuberculosis*. After post-mortem studies, he concluded that tuberculosis was a disease which spread from the kidney to the bladder via the ureter, that it could pass up the contra-lateral ureter and infect the other kidney and that it could affect the prostate and other genital organs. Moreover, he pointed out that it was able to affect areas of the genito-urinary tract whilst missing out others. Following the discovery of the *M. tuberculosis*, it was accepted that the organism could filter through the kidney without causing any symptoms, and 40 years elapsed before this view was challenged[2], [3]. In 1926 Medlar[4] published the results of his examination of over 100,000 serial sections of kidneys of 30 patients who had died of pulmonary tuberculosis. None of these patients showed any clinical evidence of renal tuberculosis, yet in the kidneys of 22 he found histological evidence of renal tuberculosis, mostly cortical lesions, and in all the cases in which both kidneys were examined the lesion was found to be bilateral. Later it was suggested[5] that such lesions should be termed metastatic rather than secondary, as in many cases the primary focus was not apparent. The term genito-urinary tuberculosis only began to be used as a clinical entity when Wildbolz[6] stressed that renal tuberculosis and tuberculous epididymitis were not diseases of isolated organs, but different examples of the same disease carried by the bloodstream. The same author[7] pointed out that two conditions must exist before renal tuberculosis could be diagnosed:

1. The urine should produce tuberculous lesions in guinea pigs.
2. Active or healed lesions must be present in renal tissue.

The acceptance of this concept has guided the principles of the modern treatment of genito-urinary tuberculosis.

Treatment

Before any patient was accepted for treatment, *M. tuberculosis* had to be isolated from their urine, either by culture or guinea-pig inoculation, hence the diagnosis of the condition and, more important, an

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assessment of success or failure of treatment depended on the efficiency of the methods used for the detection of this organism in urine, an organism which is notoriously difficult to isolate because it is excreted infrequently in small numbers. Investigations were carried out to assess the efficiency of various methods of isolating *M. tuberculosis* from urine. This is an extension of previous investigations[8]. Specimens of urine

TABLE I

EXAMINATION OF 10,029 SPECIMENS OF URINE BY CULTURE AND GUINEA PIG INOCULATION TEST

Total number of investigations — 10,029

Number positive by both culture and guinea pig inoculation	440 = 4.4%
Number positive by culture but negative by guinea pig inoculation	301 = 3.0%
Number positive by guinea pig inoculation but negative by culture	511 = 5.1%

Total number of positive: 1,252 = 12.5%

TABLE II

THE EXAMINATION OF 600 SPECIMENS OF URINE OF NEW PATIENTS BY CULTURE AND GUINEA PIG INOCULATION

Total number of specimens examined — 600

Number positive by both culture and guinea pig inoculation	126 = 21%
Number positive by culture but negative by guinea pig inoculation	37 = 6.1%
Number positive by guinea pig inoculation but negative by culture	62 = 10.3%

Total number of positive specimens — 37.4%

TABLE III

MICROSCOPIC EXAMINATION OF 164 URINARY DEPOSITS FROM WHICH M. TUBERCULOSIS WAS ISOLATED

		Total	*Urinary deposits*	
			Positive for M. tuberculosis	*Negative for M. tuberculosis*
Leucocytes:	Increased	75	47	28
	Normal	89	21	68

$p < 0.001$

passed before, during and after chemotherapy have been examined. Two investigations have been made:

1. A comparison of the efficiency of guinea-pig inoculation and artificial culture in the isolation of *M. tuberculosis* from urine:

(*a*) All specimens.

(*b*) Single specimens before onset of chemotherapy.

2. An assessment of the importance of pyuria in the diagnosis of renal tuberculosis.

The results are shown in Tables I, II and III.

Table I demonstrates, first, the small percentage of urines containing *M. tuberculosis,* and, secondly, that, whereas guinea-pig inoculations give a greater proportion of positive *M. tuberculosis* isolation than artificial culture, the latter method is essential if the highest number of positive results is to be obtained.

Table II shows the results of the investigation of 600 single specimens of urine of patients with known genito-urinary tuberculosis. It emphasizes once again the difficulty of isolating *M. tuberculosis* from urine and the necessity of using both culture and guinea-pig inoculation for the diagnosis of this condition. If, however, three consecutive early-morning specimens of urine are examined, the percentage is increased to 53.5%.

For many years it has been accepted that, unless there are an increased number of leucocytes in the urine, *M. tuberculosis* will not be found[9]. However, this concept can no longer be accepted. In Table III three early-morning specimens of urine were taken from 164 new patients over a period of two years immediately after admission and before chemotherapy was started, and the centrifuged deposits examined for leucocytes, the normal being taken as three in the male and six in the female per $\frac{1}{6}$ high power microscope field. The same specimens were specifically cultured and inoculated into guinea pigs. The results demonstrated that 12.8% had *M. tuberculosis* in the urine without any increase in the normal number of leucocytes, but that there is far greater likelihood of finding the organism in urine if leucocytes are present. The x^2 confirm the significance of this statement. Nevertheless, specimens of urine with or without pus cells should always be examined for *M. tuberculosis* if this disease is suspected clinically. However, reliance on the presence of pus cells in the urine as a screening test for genito-urinary tuberculosis should no longer be accepted.

The effective chemotherapy of tuberculosis started in 1952, after isoniazid was discovered. Before then, streptomycin, P.A.S. and thiosemicarbazones were available, but were given for too short a period of time, as the importance of prolonged continuous chemotherapy had not been realized. Furthermore, streptomycin and P.A.S. are toxic drugs and adverse reactions caused changes in drug regimes for many patients. Because it was thought that toxicity was directly responsible to the length of uninterrupted treatment, a rotating regime was evolved and was used for the period 1952–63[10]. This was changed in 1963 for triple continuous therapy, so that a direct comparison between two regimes of treatment used by one centre could be made.

In the period 1953–63, streptomyin, isoniazid and P.A.S., together with pyrazinamide, were used in combinations of two drugs, rotating every fortnight for six months, followed by isoniazid and P.A.S. for a further period of 18 months at home. For the period 1963–69, isoniazid, P.A.S. and streptomycin were used continuously for six months, followed by the same course of isoniazid and P.AS. at home. Although favouring intermittent therapy, as it seemed to overcome the main disadvantage of prolonged continuous therapy, i.e. the high incidence of side effects, it had fallen into general disfavour following the

Medical Research Council's report[11] which showed that isoniazid 100 mg. twice daily and streptomycin 1 G. twice weekly was of lower efficacy than other regimes, possibly due to the fact that the importance of the rapid inactivation of isoniazid in 50% of patients was not realized.

In 1965 a new drug, Rifampicin, was discovered, and was found to be highly active against *M. tuberculosis*[12]. It was of low toxicity, could be given orally and was bactericidal to the bacillus with an activity approximating that of isoniazid. Furthermore, there was no cross resistance between Rifampicin and other anti-tuberculous drugs. Extensive clinical trials were commenced with combinations of drugs, and it was discovered that Rifampicin with isoniazid and Rifampicin with ethambutol were the most effective combinations.

Prolonged treatment with a combination of isoniazid and Rifampicin has revealed an incidence of jaundice, which, although reversible, nevertheless necessitated caution and in some cases cessation of treatment. Furthermore, cases of thrombocytopaenia have been recently reported. All have had continuous therapy for three months, followed by an intermittent course.

These findings could lead to a re-appraisal of intermittent therapy, especially as it had been shown that twice weekly high dosage isoniazid together with streptomycin was a highly effective method of treating tuberculosis[13] and that with both ethambutol and Rifampicin, as the size of the dose was increased, increasing amounts were retained in the body tissues[14], thus quite a small increase in the dose size would produce a disproportionately large increase in serum concentration.

Rifampicin and isoniazid, alternating with Rifampicin and ethambutol, might make the treatment more acceptable to clinician and patient, and would give credence to the words of Hippocrates that healing is a matter of time, but it is sometimes also a matter of opportunity.

In this review 985 patients have been treated between 1946 and 1970. They have been classified into three groups recommended by Semb[15], who in 1953 pointed out the similarity between pulmonary and renal tuberculosis.

Fig. 1. 97.8% of these patients have been followed up until death or for a period of 10 years after the start of the treatment.

Fig. 2 shows the number of patients annually admitted, and reveals a gradual fall to year 1967, but since then there has been a slight increase in the numbers, and most of the more severe type. Males predominate in the rate of 2 : 1.

Results

The results of treatment were assessed by the conversion of urine in patients treated in hospital for six months, and the number of patients

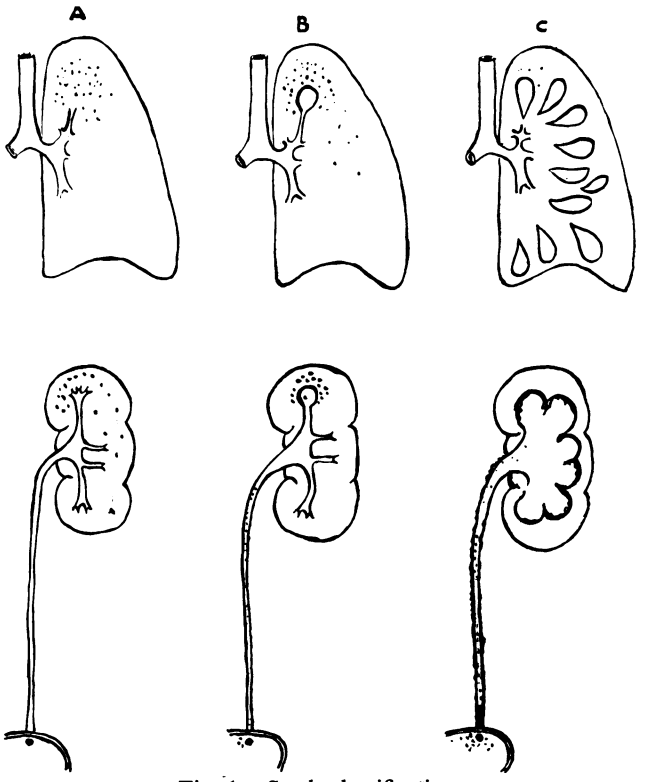

Fig. 1. Semb classification.

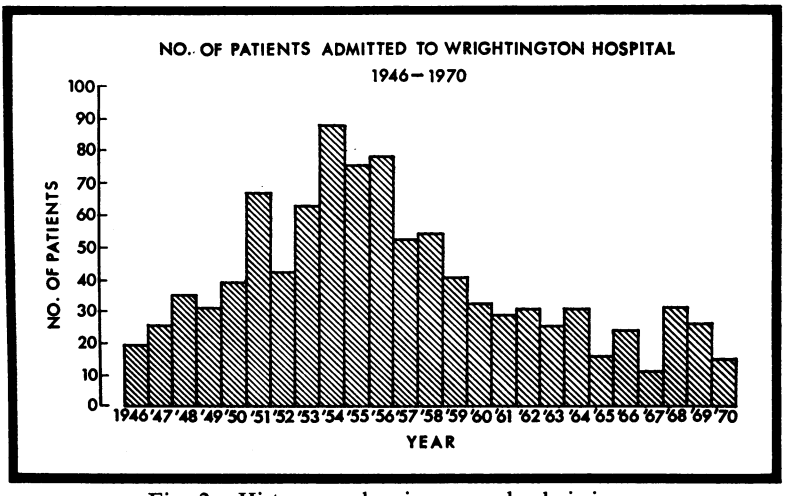

Fig. 2. Histogram showing annual admissions.

TABLE IV

DEATHS DUE TO GENITO-URINARY TUBERCULOSIS, INCLUDING NINE FOLLOWING SURGICAL PROCEDURES

Year	*No. of patients*	*Deaths*	*%*
1946–1952			
Pre-isoniazid era	262	20	7.9%
1953–1963			
Intermittent therapy	565	26	4.6%
1964–1970			
Continuous triple therapy	158	5	3.2%

whose urine reverted during the 10-year follow-up period after cessation of treatment. It should be emphasized that the disease should be considered quiescent, never cured.

Deaths. These are shown in Table IV. The five deaths in the later period were all post-operative and there have been no deaths due to tuberculosis since 1961.

Urinary conversion. The results are shown in Table V. It seems that there will be always a small number of patients whose urine remains positive, as a few harbour organisms which are either resistant to one or more antibiotics at the start of treatment, or else become resistant during the first period of chemotherapy. All the urines of these patients have been sterilized by variations in the combination of antibiotics.

Urinary reversion. The efficacy of treatment stands or falls by the number of patients whose urines revert after a full course of treatment. Reversion is defined as a patient whose urine becomes positive more than six months after a full course of anti-tuberculous treatment which includes chemotherapy and surgery as required. Table VI shows the number of reversions by years. Only five patients reverted after five years, therefore it is unnecessary to follow up patients for more than this length of time. The follow-up period of 10 years that was adopted has given the evidence that it is not necessary as a routine to review patients for more than five years. However, patients with bladder lesions and calcification should be followed up annually for an indefinite period, as there is a significantly increased number of bladder lesions in patients who reverted than those without. There appeared to be no obvious explanation of this fact, but patients who have had bladder lesions should be followed up with greater care than those with no bladder involvement. The number of patients whose urine reverted is small and, for an adequate analysis, cases from other centres will have to be collected. The analysis has been done

TABLE V

URINARY CONVERSION

1946–1952	92.1%
1953–1963	97.6%
1964–1970	98.1%

Percentage calculated after deduction of number of patients who died in hospital.

by the Woolf method, and it is an advantage that this method of analysis allows the utilization of data from different centres[16].

Uses of cortisone. Cortisone in the form of prednisolone, 10 mg. t.d.s. was given over a period of three years to patients with bladder lesions which were thought likely to produce bladder contracture, and those with obstruction at the lower end of the ureter leading to dilatation of the upper urinary tract. Over 50 patients were so treated. The course was continued for two months. In no case was there any evidence that it influenced the course of the disease, and in two a pulmonary lesion appeared to be reactivated despite concomitant adequate chemotherapy, and in two others a dormant peptic ulcer was reactivated, producing in one quite severe haematemesis. It is suggested that cortisone has no routine place in the treatment of genito-urinary tuberculosis but it can produce dramatic results in selected cases.

Hypertension

The first observations which associated the kidney with hypertension were made by Goldblatt[17], who showed that constriction of the renal artery of a dog, the other kidney having been removed, produced high blood pressure. Butler[18] was the first to report an improvement in

TABLE VI

NUMBER OF PATIENTS WHOSE URINES REVERTED

	< 1 *year*	1–2 *years*	2–3 *years*	3–4 *years*	4–5 *years*	> 5 *years*
Group 1	21	15	10	7	2	3
Group 2	10	7	4	2	—	2
Group 3	2	—	1	—	—	—

% calculated after deducting the number of patients who died in hospital.

hypertension by removing a diseased kidney, and Nesbit[19] was the first to report relief of hypertension by removal of a tuberculous kidney. There are many types of renal disease associated with hypertension, and all have one common histological picture, a reduction in the blood supply to either a part or the whole of the kidney. It might be thought as Black said, that 'tuberculosis is a neglected diagnostic possibility'[20]. This view is shared by Kaufman[21], who reported four cases of renal hypertension cured by nephrectomy, and Hsiung[22], who quoted 23 out of 30 cases of renal tuberculosis with hypertension cured by nephrectomy. The contrary opinion is stated by Schwartz[23], who quoted only one hypertensive patient with renal tuberculosis out of 20 improved with nephrectomy, and he had also acute ureteric obstruction, another possible cause of renal hypertension.

The records of 772 patients between 1952 and 1970 were examined. and those with a blood pressure of 150/90 on three separate occasions were accepted as cases of renal hypertension. A fall of 30 mm. Hg systolic was accepted as a reduction in hypertension. This period was chosen, as it was after 1952 that efficient chemotherapeutic treatment was introduced.

These were studied in three ways:

1. Effect of chemotherapy on hypertension.

2. Effect of partial nephrectomy and nephrectomy on hypertension.

3. Number of normotensive patients treated by nephrectomy or partial nephrectomy who developed hypertension during the follow-up period.

1. Effect of chemotherapy on hypertension. Of the 772 patients, 494 did not require surgery on the kidney. Only 39 showed evidence of hypertension. Four of these improved during the course of chemotherapy, but bed rest at the time was part of the treatment, so it was difficult to assess the true value of the drugs in the reduction of blood pressure.

2. Effect of nephrectomy and partial nephrectomy on hypertension. Two hundred and seventy-eight patients were treated by these surgical procedures. Fifty-two showed evidence of hypertension. Ten of these showed improvement, all with a blood pressure below 180/100. The six above 200/110 mm. Hg did not respond. Four of these 10 showed some deterioration during the 10-year follow-up period, but none returned to the original level.

3. Number of normotensive patients treated by nephrectomy or partial nephrectomy who developed hypertension during the follow-up period. Two hundred and twenty-six patients were examined annually. Twenty-one developed hypertension during this period, a little above the national average for the comparative age group.

These results suggest that renal tuberculosis is a rare cause of renal hypertension, an opinion shared by Smith[24], who stated that only 1/1,250 cases of hypertension are due to this condition.

Calcification in renal tuberculosis

Calcification is one of the major hazards of genito-urinary tuberculosis[25]. In this review no attempt has been made to differentiate between diffiuse calcification and a discrete stone, as there is no evidence to support a different pathogenesis. Occasionally, some of the precipitating factors for stone formation, e.g. recumbency, unlimited calcium intake, urinary infection, obstructive uropathy and hypercalciuria, were found, but there was no common denominator. From histological studies it seems likely that the calcification starts in healing tuberculous foci, and that the position of the focus determines whether the calcification spreads in the renal parenchyma or forms a calculus in the pelvi-calyceal system.

Calcification can be classified into two groups:

A. Calcification present at the time of the initial diagnosis:

(i) Small, irregular areas affecting one or more segments of the kidney.

(ii) Larger areas involving kidneys which retain some function:

(*a*) restricted to one pole.

(*b*) diffuse involvement.

(iii) Extensive calcification in a non-functioning kidney.

(iv) Calcification in a solitary kidney.

B. Renal calculi or calcification occurring during or after treatment.

The present study embraces the years 1952–1970, when 765 patients were under treatment. In this period 193 cases of calcification were seen, and the details are shown in Table VII. Most had calcified lesions on admission to hospital. No particular age group was affected and the sex incidence was almost identical.

TABLE VII

DETAILS OF CALCIFICATION

Total — 193

A.	Patients with calcification at the time of the initial diagnosis:			
	1. Small irregular areas affecting one or more segments of the kidney		23	
	2. Larger areas of calcification involving kidneys which retain some function:			
	(*a*) Restricted to one pole	18		
	(*b*) diffuse involvement	32	50	
	3. Large calcified non-functioning kidney		71	
	4. Calcification in a solitary kidney		17	
				161
B.	Patients developing calcification during or after treatment			32

Management. In the management of all cases of genito-urinary tuberculosis the aim is to retain as much functioning renal tissue as possible and to delay surgery until the maximum effect of chemotherapy has been achieved. Most of the small areas affecting one or more segments were treated initially without surgery, and the results are shown in Table VIII. The results are highly significant, as they reveal that one-half of the patients who had calcification at the start of treatment developed further renal damage. The 41 who were treated by partial nephrectomy after a period of chemotherapy were also studied. Only 14 showed evidence of active tuberculosis, but 27 showed pyelonephritic changes surrounding the calcification, and those near the cortex caused

TABLE VIII

PATIENTS WITH CALCIFICATION ON ADMISSION. NO SURGERY.

Number of patients — 73

	Increasing damage of renal structure assessed radiologically	*No change in renal structure*
Increase in size of calcification	27	8
No increase in size of calcification	9	29

$p < 0.001$

TABLE IX

ANALYSIS OF THE EFFECT OF CALCIFICATION ON 17 PATIENTS WITH A SINGLE KIDNEY

No effect	5
Urinary reversion	1
Increase in size	9
Death	6
Formation of staghorn calculus	2

atrophy and scarring of a local area of cortex. Of the 71 patients requiring nephrectomy, 40% showed evidence of active tuberculosis on histological examination. The analysis of the progress of patients with calcification in a solitary kidney is shown in Table IX. The disease was often extensive and in 50% the increase in size was progressive, eventually causing renal failure.

Those cases in which the calcification occurred after treatment were managed as uncomplicated renal calculi. The incidence in this series was over 4% as against the national average of 0.03 so the tuberculous infection must play some part, but did not alter the normal pattern of the condition. Hypercalciuria was only seen in 5% of cases. Small calculi were treated conservatively and many have been followed up for more than 10 years without any change, but larger calculi should be removed, and the patients followed up annually.

These studies show that calcification is a severe complication of renal tuberculosis, and where indicated should be removed.

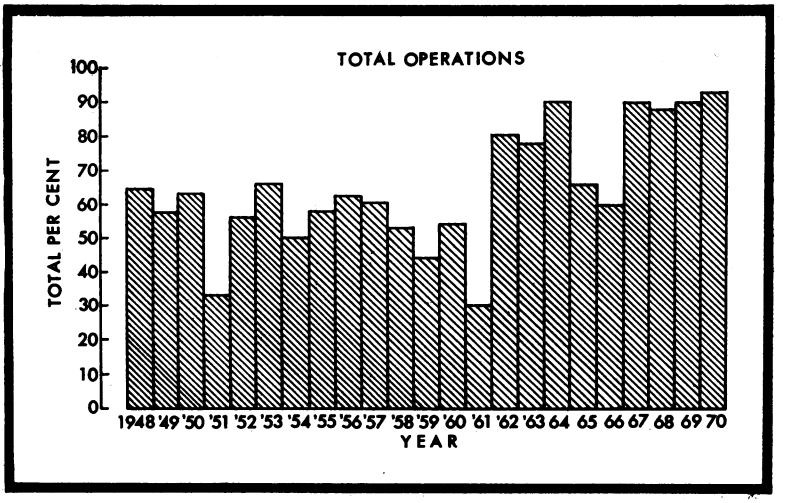

Fig. 3. Histogram showing percentage of patients requiring surgical treatment, percentage being the ratio of patients requiring surgical treatment over the number of those admitted.

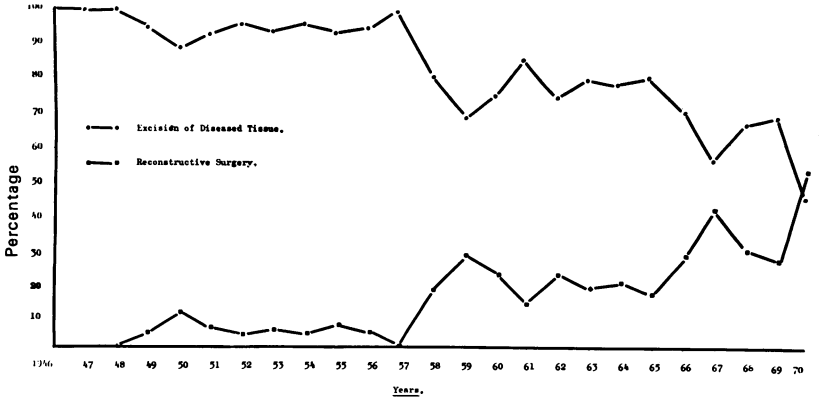

Fig. 4. The changing incidence of excision of diseased tissue and reconstructive surgery.

SURGERY

Surgery has still a vital rôle to play in the treatment of genito-urinary tuberculosis, and in recent years there are very few cases which have not required some form of treatment. Figure 3 shows the percentage of patients requiring such treatment. In 1970 14 out of 15 patients treated required surgical treatment. Surgery should be carried out during a period of chemotherapy and not less than three months after the start of antibiotic treatment. It can be divided into two types:

1. Excision of diseased tissue, e.g. nephrectomy, partial nephrectomy and epididymectomy.

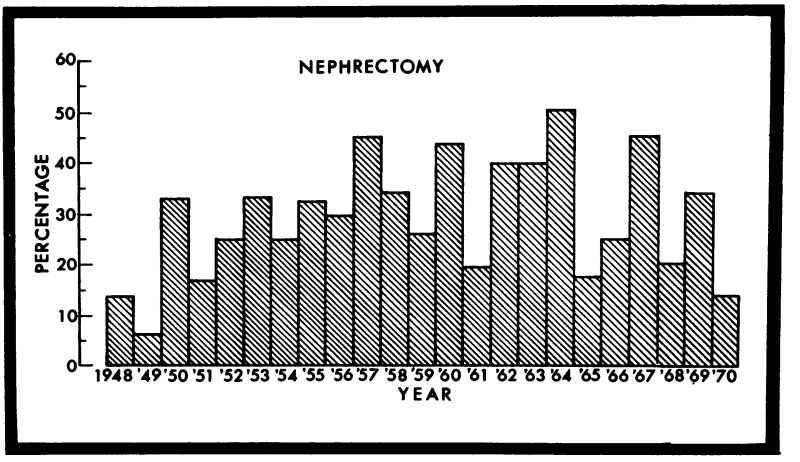

Fig. 5. Histogram showing the percentage of nephrectomies, the percentage being the ratio of the number of nephrectomies carried out over the number of patients admitted.

2. Reconstructive surgery, e.g. enterocystoplasty, ureterocolic anastomosis, pyeloplasty for strictures at the pelvi-ureteric junction and reimplantation of the ureter into the bladder for strictures at the lower end of the ureter.

Figure 4 shows the changing incidence of excision of diseased tissue and reconstructive surgery, demonstrating the increasing number of cases requiring reconstruction, and that in 1970 for the first time more patients required reconstruction than excision of diseased tissue.

Excision of diseased tissue

Nephrectomy. Two hundred and sixty-five nephrectomies have been carried out in the period under review, all for grossly diseased organs, and Figure 5 shows the number of nephrectomies carried out year by year. Nephrectomy will always be required, as there will be patients whose kidneys are irreparably damaged before treatment is started. Nevertheless, it was stated[26] that nephrectomy is never necessary provided adequate chemotherapy has been given. Evidence is presented in support of the contrary view.

1. Eight patients have been admitted to the unit with an active renal lesion, having received two years chemotherapy for a pulmonary lesion within five years prior to admission for treatment of the genito-urinary disease.

2. Four patients with a non-functioning kidney who had received two years chemotherapy presented between one and five years later with a sinus in the loin, the discharge from which contained viable sensitive organisms.

3. Over a period of three years the kidneys of all patients undergoing nephrectomy, who had intensive chemotherapy for at least three months, were cultured for *M. tuberculosis*. In 28% viable sensitive organisms were isolated.

Partial nephrectomy

Until 1952 the standard treatment for advanced renal tuberculous lesions was nephrectomy. This radical treatment, although probably the best available at the time, had a five-year mortality rate of 20%. Furthermore, almost 14% developed discharging wounds, some even leading to amyloid disease. With the introduction of effective chemotherapy it seemed logical to apply a more selective principle in the surgical treatment of renal tuberculous lesions. Clearly such a principle depended on the extent of the diseased renal tissue, but localized lesions in the middle, upper and lower poles could be excised. Semb[15] was the first person to point out the importance of this method of treatment, and ultimately reported on 262 cases[27]. He was an enthusiastic advocate, but others tended towards a more conservative regime, and relegated surgery to a few definite indications[28]. Certainly it should not be considered until a prolonged course of chemotherapy has been administered, and no case without calcification has been treated by partial nephrectomy in the last 10 years.

The present series is an examination of 92 operations carried out between 1952 and 1970. Figure 6 shows the number of operations carried out each year. Forty-five resections were in the lower pole, 40 in the upper pole, three both upper and lower poles and in four a middle segment was excised. The size of the cavity bore no relationship to the presence or absence of *M. tuberculosis* in the urine. Three secondary nephrectomies have been required, two for persistent haematuria and one for an uncontrollable tuberculous infection. In this last case it was found that either the previous resection was not extensive enough, or else a parenchymatous lesion close to the line of resection had progressed. Furthermore, the organism was resistant to both isoniazid and streptomycin. All the resected specimens were examined histologically

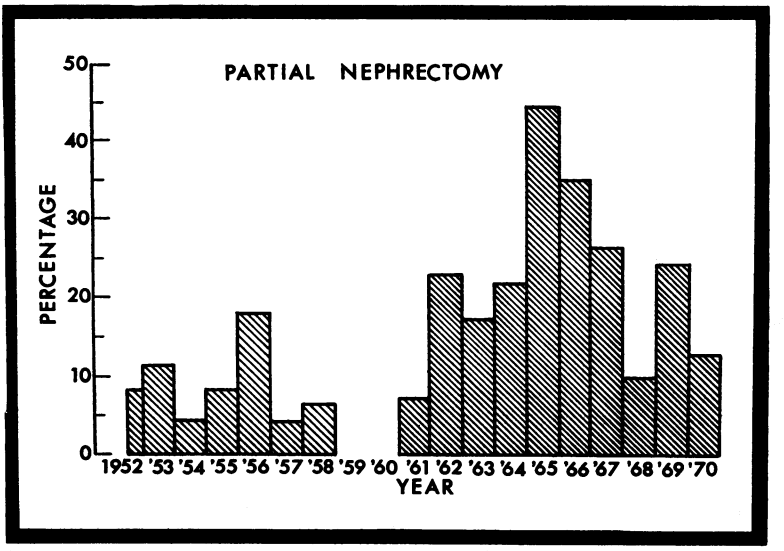

Fig. 6. Histogram showing the percentage of partial nephrectomies, the percentage being the ratio of the number of partial nephrectomies carried out over the number of patients admitted.

and were divided into active, moderately active and quiescent. Forty per cent were found to be active, and a third of these contained calcification. Partial nephrectomy is recommended for the following reasons:

1. Lesion in a localized area of the kidney which has failed to respond to chemotherapy.

2. Lesion in one or more poles containing calcified material which is increasing in size.

3. Calcified lesions affecting one-half of a duplex kidney.

Technique. Only the artery is clamped and this should not be occluded for more than 30–40 minutes without release. The sinus of the kidney

is dissected free and it is only on rare occasions that this is impossible. Next the capsule is reflected back from the tip of the pole to the site of the renal incision, and the diseased area resected in a 'V' fashion. The capsule is then turned into the bare area which is closed with a few interrupted atraumatic sutures (Fig. 7).

Tuberculous epididymitis

The incidence of tuberculous epididymitis is decreasing, and in the last six years only 10 cases have been seen. All these have been combined with some other tuberculous lesion in the urinary tract, whereas in the previous decade 20% of genital tuberculoses were seen

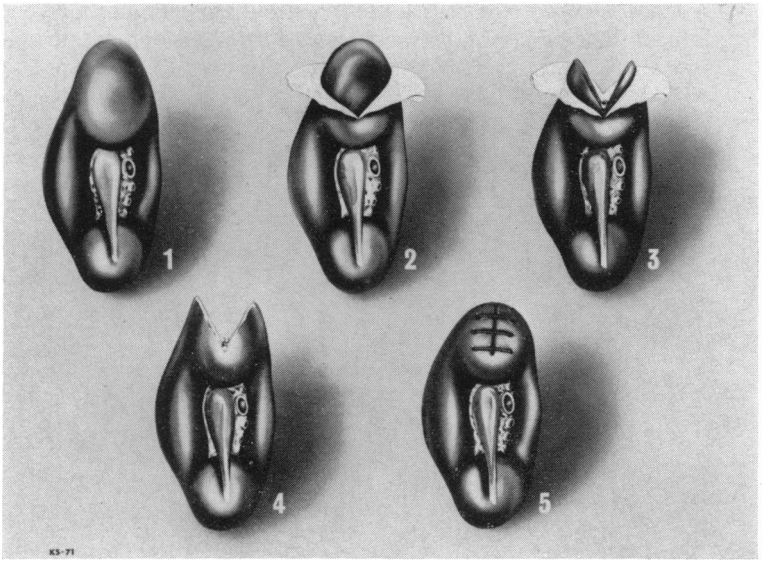

Fig. 7. Technique of partial nephrectomy.

without an overt renal lesion. In discussing tuberculous epididymitis two aspects have been considered:

1. The route of infection to the epididymis.
2. Management.

Route of infection

Via lumen of vas. This theory is widely held and envisages that there is coincidental renal tuberculosis. It is not denied that retrovasal migration does occur, e.g. the acute epididymitis after prostatectomy, the presence of dye in the ejaculatory ducts after urethrography[29] and urinary fistulae through the stump of the vas[30], but nearly all these examples occur in the presence of some abnormality of the posterior urethra, but, as has been pointed out, *M. tuberculosis* are difficult to

isolate from urine, are present in small numbers, intermittently excreted and therefore the chances of organisms migrating up the vas must be remote. It is suggested that tuberculous epididymitis is rarely caused by organisms passing up the vas.

Via the bloodstream. Renal tuberculosis is caused by metastatic focus which is blood-borne, spread from a primary. This implies that at some time *M. tuberculosis* has been circulating in the bloodstream, and has been carried to all parts of the body. Organs such as muscle, skin and testes are relatively immune, and therefore rarely produce a lesion. Others such as bone and joints and epididymes are susceptible and are therefore frequently invaded. This brings tuberculous epididymitis into line with renal tuberculosis. Furthermore, the disease commonly occurs in the globus minor which has been shown to have a much greater blood

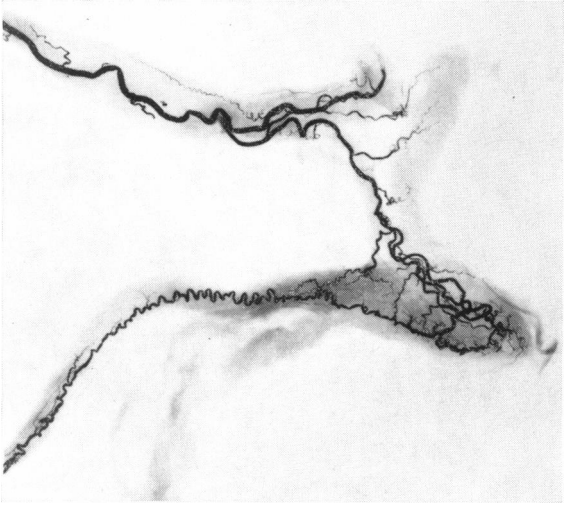

Fig. 8. Blood supply to the epididymis.

supply when compared with other parts of the epididymis[31] (Fig. 8). This evidence suggests that the source of the infection in most cases of tuberculous epididymitis is blood-borne.

Management

Within recent years the incidence of epididymitis has decreased but the cases that have been seen are more severe, with abscess formation. and rarely have responded to chemotherapy. Figure 9 shows this incidence, and demonstrates the diminishing numbers. In the years 1955–62 there was a trend towards a more conservative management, but the results were unsatisfactory, and since then a more radical approach has been adopted—an approach which has been vindicated by the findings on histological examination as, in two of the cases diagnosed as

tuberculous epididymitis, seminoma were found[32]. Both were dead within six months.

Trauma

In many papers discussing tuberculosis of the male genital tract, trauma is scarcely mentioned[33, 34, 35]. In only one paper was the rôle of trauma discussed at any length[36]. It was pointed out that the importance of trauma was to cause an activation of a quiescent tuberculous focus not previously recognized. In a previous review of tuberculous epididymitis[37] only six cases of trauma were encountered. Since, there have been four more, one developing an infection 30 years after the primary lesion.

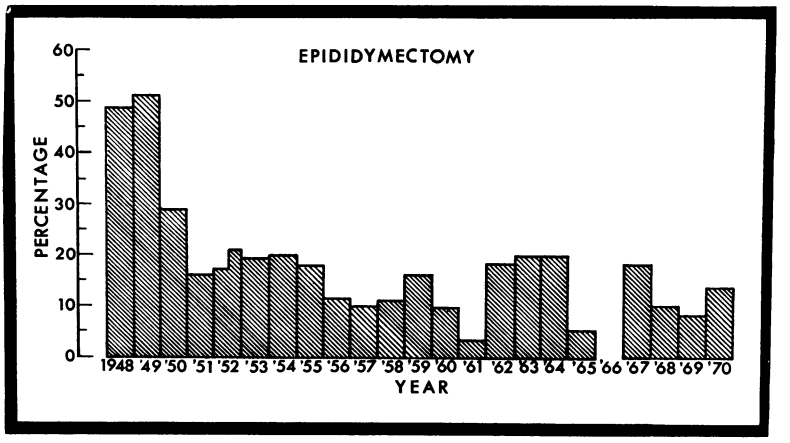

Fig. 9. Histogram showing the percentage of patients requiring epididymectomy, the percentage being the number of patients requiring epididymectomy over the number of patients admitted.

Reconstructive surgery

This has been reviewed under three headings:

1. Diversion of the urine.
2. Strictures of the ureter.
3. Methods of increasing bladder capacity.

1. Diversion of the urine. In this series such treatment was carried out prior to 1959. After this it was superseded by enterocystoplasty. Twenty-three operations were performed, all for patients with a small fibrotic bladder producing intractable frequency of micturition both day and night. The results were poor and the longest surviving patient was 12 years. Two patients had the segment of bowel containing the ureters re-anastomosed to the healed bladder five years after the original ureterocolic anastomosis. In both these patients the result was excellent, especially the way the bladder recommenced function after the long

quiescent period. Frequency of micturition was stabilized at two-hourly during the day, and twice at night. There is very little place for diversion of the urine in the present-day treatment of genito-urinary tuberculosis.

2. Strictures of the ureter. These were situated either at the pelvi-ureteric or ureterovesical junction (Table X). None were seen affecting the middle third of the ureter.

TABLE X

STRICTURES OF URETER

Total — 41

Pelvi-ureteric junction		5
Vesico-ureteric junction:		
Direct anastomosis		9
Boari technique:		
Direct anastomosis	16	
Tunnelling procedure	11	27

Five were at the pelvi-ureteric junction, a small number, but most strictures at this level produce irreparable damage to the kidney before the patients first attend for treatment. All were treated by Anderson Hynes pyeloplasty, with four successes and one failure, the failure being an error of judgement, as the ureter was thickened and fibrotic, and produced a urinary fistula, which persisted, necessitating a secondary nephrectomy. Two of the other four leaked urine for eight and 14 days respectively, but the ultimate result was satisfactory.

Twenty-seven were at the ureterovesical junction. The complications of this procedure are shown in Table XI. They were due to:

(*a*) technical inadequacies which have been eliminated in later procedures,

(*b*) the poor state of the tissues which were being treated.

Many of the ureters were fibrotic, thickened and adherent, so that their blood supply was impaired, probably the key factor in the urinary leaks. However, all leaks except one dried up within two weeks. All the direct implants showed reflux, and one has since been converted to a Boari with tunnelling technique. Six of the nine patients who had a direct implant require periodic courses of chemotherapy for a recurrent renal infection.

TABLE XI

COMPLICATIONS OF TREATMENT OF STRICTURES AT THE URETERO-VESICAL JUNCTION

	Direct implant	*Boari*	
		Direct anastomosis	*Tunnelling technique*
Urinary leak	3	4	2
Nephrectomy for persistent urinary leak	1	1	—
Ruptured deep epigastric artery	—	1	—
Stenosis at site of anastomosis	1	1	—
Renal failure	—	1	—

The Boari technique using the tunnelling modification is satisfactory, and only one patient showed evidence of ureteric reflux. Provided the ureter is not thickened and fibrotic the method can easily be performed. Even if the ureter is dilated it can be fashioned to produce a ureter of a size appropriate for this technique.

Figure 10 shows the technique of the tunnelling procedure. It is helpful in marking out the flap if the bladder is filled with fluid through an

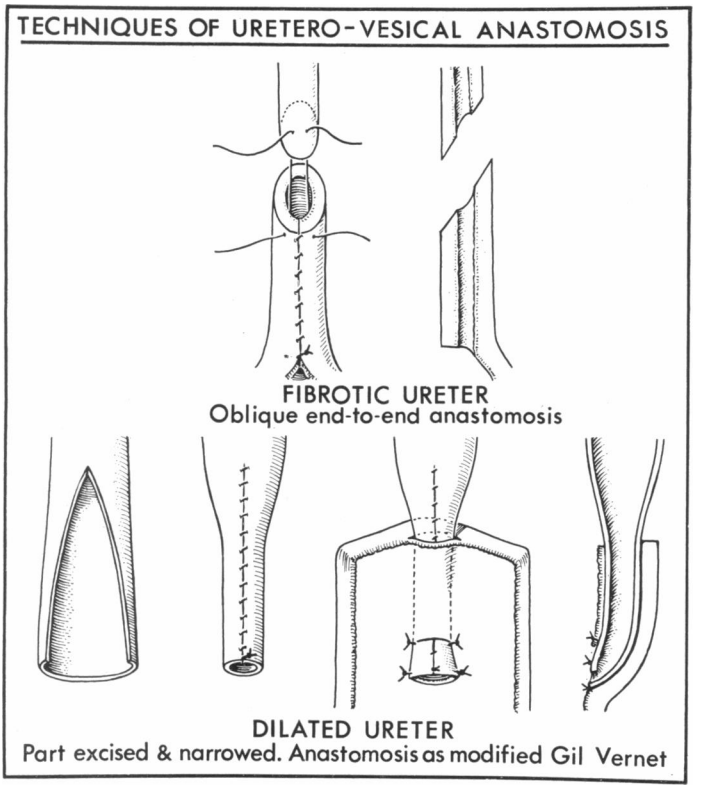

Fig. 10. Technique of Boari tunnelling procedure.

indwelling catheter inserted at the start of the operation just before the flap is cut. Up to 14 cm. of ureter have been removed.

The psoas hitch has not been used in any of the cases, but this is an eminently satisfactory alternative for most cases of reimplantation of the ureter into the bladder.

3. Methods of increasing bladder capacity. Enterocystoplasty has been carried out in 44 patients, and Table XII shows details. The ileocystoplasty was carried out by the side to fundus technique. The result

was not satisfactory, so the colocystoplasty was then employed, using the method suggested by Yeates as applied to the colon[38]. Fourteen patients were treated. The functional results were satisfactory, but urinary infection was frequent and the patients complained of always passing large quantities of mucus. Furthermore, occasional technical difficulties arose because of the short pelvic mesocolon. It was decided, therefore, to use the caecum as a bladder substitute and this procedure has been adopted in the last 29 cases. Table XIII shows the complications of both procedures. There is no significant difference from this aspect. The two cases of incontinence in the caecocystoplasty were errors of

TABLE XII

ENTEROCYSTOPLASTY

Total — 44

Ileocystoplasty	... ...	1
Colocystoplasty	... ...	13
Caecocystoplasty	... ...	23
Caecocystoplasty with transplant of ureters into ileum	... ...	7
		30

TABLE XIII

COMPLICATIONS OF COLO- AND CAECOCYSTOPLASTY

	Colocystoplasty — 14	*Caecocystoplasty* — 29
Urinary leak	4	5
Incontinence	1	2
Haematuria	—	2
Retention of urine	—	1

TABLE XIV

RESULTS OF COLO- AND CAECOCYSTOPLASTY

	Colocystoplasty — 14	*Caecocystoplasty* — 29
Death	1	2
Continence	13	27
Residual urine	Average 75 ml. Highest 120 ml. Lowest 30 ml.	Average 60 ml. Highest 120 ml. Lowest 20 ml.
Infection	9 (6 *B. coli*, 2 *B. proteus*, 1 *B. pyocyaneus*)	6 All *B. coli*
Frequency	D ½ – 2 hourly Night: 2–3 times	D 2-hourly Night — twice
Mucus	13	7

judgement. One was a young man with a very low I.Q. and was partially incontinent before he contracted tuberculosis. The other was a lady with advanced Parkinson's disease. The results of the two procedures are shown in Table XIV. There is marginal improvement with regard to residual urine infection, frequency and mucus, but it would take a much larger series of cases for a statistical analysis to be made. Infection responded to long-term chemotherapy repeated whenever the infection recurred. In all the cases of infection in the caecocystoplasty the infection was symptomless. Careful monitoring of the serum biochemistry has been carried out, and in no case has any significant alteration been seen.

Bladder neck resection is carried out in the males where there is evidence of bladder neck obstruction.

An advantage of the caecocystoplasty is seen when there is reflux up one or both ureters, as the ureters can be anastomosed to the segment of ileum attached to the caecum, using a nipple technique for the uretero-ileal anastomosis. Reflux can be avoided through the combined preventative action of the nipple and the ileocaecal valve. This technique has been carried out in seven cases, three with a single ureter and four with double. Six showed no evidence of reflux up to one year, and the renal function remains excellent. One showed a small amount of dye entering one ureter. This was due to a poor ileocaecal valve mechanism, as the whole of the ileal segment was filled with urine during micturition. When this operation is being performed the ureters should be anastomosed to the ileum before the caecum is anastomosed to the bladder, otherwise the technical difficulties could be insuperable.

Conclusions

1. Both guinea-pig inoculation and artificial culture media must be used, otherwise a significant number of urines containing *M. tuberculosis* will be missed.

2. Pyuria is absent in 12% of urine specimens from which *M. tuberculosis* are grown.

3. Chemotherapeutic treatment is still prolonged therapy with a combination of drugs, but Rifampicin may open up a new era. Cortisone has very little place in the treatment of this condition.

4. Tuberculosis is rarely a factor in renal hypertension.

5. Renal calcification is a serious complication of genito-urinary tuberculosis.

6. Surgery has still an important rôle to play in the treatment, but reconstructive surgery is assuming a more important rôle than excision of diseased tissue.

REFERENCES

1. Cohnheim, J. (1879) *Die Tuberkulose von Standpunkte der Infectionslehre.* Leipzig.
2. Medlar, E. M., and Sasano, K. T. (1924) *Amer. Rev. Tuberc.* **10**, 370–391.
3. Harris, R. I. (1928–29) *Brit. J. Surg.* **16**, 464–484.
4. Medlar, E. M. (1926) *Amer. J. Path.* **2**, 401–415.
5. Medlar, E. M., Spain, D. M., and Holliday, R. W. (1949) *J. Urol.* **61**, 1078–1088.
6. Wildbolz, H. (1937) *Schwerz. Med. Wschr.* **67**, 1125–1129.
7. Wildbolz, H. (1939) *Trans. Int. Soc. Urol.* 267–320.
8. St. Hill, C. A., Gow, J. G. (1966) *Brit. J. Urol.* **38**, 163–170.
9. Ljunggren, E. (1957) *Brit. J. Urol.* **29**, 263.
10. Gow, J. G. (1963) *Lancet*, **2**, 261–265.
11. Medical Research Council (1955) *Brit. Med. J.* **1**, 435.
12. Fursez, S., Arioli, V., and Pallanza, R. (1965) *Antimicrobial Agents and Chemotherapy*, **5**, 770–777.
13. Menon, N. K. (1968) *Tubercle* (Lond.) **49**, March Suppl. 76.
14. Dickinson, Jean M. (1969) *Scand. J. Resp. Dis.* Suppl. **69**, 91–98.
15. Semb, C. (1953) *J. Oslo Cy. Hosp.* **3**, 45–114.
16. Woolf, B. (1955) *Ann. Hum. Genet.* **19**, 251–253.
17. Goldblatt, H., Lynch, J., Hanzal, R. F., and Summerville, W. U. (1934) *J. Exper. Med.* **59**, 347.
18. Butler, A. M. (1937) *J. Clin. Invest.* **16**, 889.
19. Nesbit, R. M., and Ratliff, R. K. (1940) *J. Urol.* **43**, 427–431.
20. Black, D. A. K. (1962) *Renal Disease.* **485**. Blackwell Scientific Publications, Oxford.
21. Kauffman, J. J., and Goodwin, W. E. (1965) *Amer. J. of Med.* **38**, 337–344.
22. Hsiung, J. C., Miao, T. C., and Ch'en, C. C. (1965) *Chinese Med. J.* **84**, 327–332.
23. Schwartz, D. T., and Lattimer, J. K. (1967) *J. Urol.* **98**, 651–652.
24. Smith, H. W. (1956) *Journal of Urology*, **76**, 685–701.
25. Gow, J. G. (1965) *Brit. J. Surg.* **52**, 283–288.

26. BLOOM, S., WECHSLER, H., and LATTIMER, J. K. (1970) *J. Urol.* **104**, 654–657.
27. SEMB, C. (1956) *Ann. Roy. Coll. Surg. Eng.* **19**, 137–155.
28. GOW, J. G. (1959) *Lancet*, **2**, 759–761.
29. PERVES, J., and DUBERGEY, H. (1939) *J. Urol. Med. Chir.* **48**, 97
30. AINSWORTH-DAVIES, J. G. (1944) *Brit. J. Urol.* **16**, 55.
31. MACMILLAN, E. W. (1954) *Brit. J. Urol.* **26**, 60–70.
32. GOW, J. G. (1963) *Urologia*, **30**, V.
33. VEENEMA, R. J., and LATTIMER, J. K. (1957) *J. Urol.* **78**, 65–77.
34. MENVILLE, J. G. (1938) *J. Urol.* **40**, 66–72.
35. ROSS, J. COSBIE (1948) *Brit. J. Urol.* **20**, 173.
36. KRETSCHNER, H. L. (1928) *Surg. Gynae. and Obstet.* **47**, 652.
37. ROSS, J. C., GOW, J. G., and ST. HILL, C. A. (1961) *Brit. J. Surg.* **48**, **212**, 663–666.
38. YEATES, W. K. (1956) *Brit. J. Urol.* **28**, 410–413.

POINTS OF VIEW—SURGEON TRAINING IN THE U.S.A.

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IN THE LIGHT of letters from the United States, all of which were couched in the kindest terms, some false impressions were revealed in my observations on Surgical Training in the United States of America which were published in the *Annals of the Royal College of Surgeons* in November last, which I am most anxious to correct. After considerable thought I have come to the conclusion that this can best be done by quoting from the letters themselves rather than by an attempt to revise the original article. For example, referring to my comments on the theory and practice of surgery, Dr. Owen Wangensteen writes: 'There is still, I believe, throughout university communities in our country an abiding interest in research, a most significant device in the advancement of surgery.' He then goes on to note that the long Viet Nam conflict and other domestic problems have crippled Federal support to research grants in many institutions, but he adds: '. . . even so, I believe you are quite right in suggesting that the men you visited in 1970 exhibited a greater interest in clinical surgery perhaps than they did in 1951.'

Dr. Englebert Dunphy writes: 'In the discussion of the examinations you have confused the type of program with the examination. A type one program involves four years of residency training. Type two involves three years. The examination, both Part 1 and Part 2, are taken by men graduating from both types of programs. The type two, namely the three year program, is to be phased out by 1972. The changes in the examinations is that the Part 1 examination will be a comprehensive examination for all surgical specialists. The Part 2 examination will be given after the man completes the training in his own particular field of Urology, General Surgery, etc.'

Dr. George Stevenson, the Assistant Director of the American College of Surgeons, writes: 'If you were to expand this paper, on another occasion, I would suggest that it might be well to emphasize the fact that the American Board of Surgery is related only to general surgeons, and that there are nine other Boards for the recognized surgical