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Testicular obstruction: clinicopathological studies

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Genital tract reconstruction has been attempted in subfertile men with obstructive azoospermia (370 patients) or unilateral testicular obstruction (80 patients), and in vasectomised men undergoing reversal for the first (130 patients) or subsequent (32 patients) time. Histopathological changes in the obstructed testes and epididymes, and immunological responses to the sequestered spermatozoa have been studied to gain insight into possible causes of failure of surgical treatment. The results of surgery have been assessed by follow-up sperm counts and occurrence of pregnancies in the female partners. The best results were obtained with vasectomy reversal (patency 90%, pregnancy 45%), even after failed previous attempts (patency 87%, pregnancy 37%). Epididymovasostomy gave good results with postinfective caudal blocks (patency 52%, pregnancy 38%), while postinfective vasal blocks were better corrected by total anatomical reconstruction (patency 73%, pregnancy 27%) than by transvasovasostomy (patency 9%, no pregnancies). Poor results were obtained with capital blocks (patency 12%, pregnancy 3%), in which substantial lipid accumulation was demonstrated in the ductuli efferentes; three-quarters of these patients had sinusitis, bronchitis or bronchiectasis (Young's syndrome). There is circumstantial evidence to suggest that this syndrome may be a late complication of mercury intoxication in childhood.

After successful reconstruction, fertility was relatively reduced in those men who had antibodies to spermatozoa, particularly amongst the postinfective cases. Similarly, impaired fertility was found in men with unilateral testicular obstruction and antibodies to spermatozoa. Mononuclear cell infiltration of seminiferous tubules and rete testis was noted occasionally, supporting a diagnosis of autoimmune orchitis; although rare, this was an important observation as the sperm output became normal with adjuvant prednisolone therapy.

The anatomy of the human vas deferens was demonstrated by John Hunter in 1752 by injection of mercury *(1)*. In a comprehensive treatise on the comparative anatomy and function of the seminal vesicles *(2)* Hunter described a subject with absence of one vas and an incomplete epididymis on the other side; he noted that the testicles were sound and found that the epididymes contained semen. The fine internal structure of the epididymis was studied and illustrated by Hunter's pupil, Astley Cooper *(3)*, who confirmed that the testicle continued to produce semen after experimental vasoligation. The operation of epididymovasostomy for correction of testicular obstruction due to previous epididymitis was developed and first successfully carried out by Martin in 1901 *(4)*, who aptly described it as "a means of switching out the seat of obstruction by a short circuit". Vasography to establish vasal patency was first described by Belfield in 1913 *(5)*. Tubule-to-tubule epididymovasostomy was developed by Lespinasse and published in

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1918 *(6)*. In the first large series of operations for male sterility with azoospermia, Hagner, in 1936 *(7)*, reported 21 cures (63.6%) in 33 favourable cases, all of whom had a history of bilateral epididymitis; however, in another 30 cases, cure was considered impossible due to either occlusion of the vas deferens or absence of spermatozoa in the epididymis.

The importance of testicular biopsy in distinguishing azoospermia due to deficient spermatogenesis from that due to obstruction of the ductal system was stressed by Michelson in 1947 *(8)*. Thereafter, details of the findings in 12 patients with absent vasa were reported *(9)*. In 1952, Bayle (10) recognised that gonorrhoea was the most common cause of correctable epididymal obstruction, and pointed out that the vas may also be blocked in up to 25% of these cases. Nevertheless, 43 of 84 azoospermic cases were cured by epididymovasostomy, and 20 couples successfully produced pregnancies. These results were in stark contrast to those reported in 1955 by Hanley *(11)* in a previous Hunterian lecture: amongst 148 azoospermic men, only 15 had a history of previous inflammation; of nine who had corrective surgery, six were cured and four produced pregnancies. From 83 other epididymovasostomies, in which sperms were solely detectable in the *heads* of the epididymes, only four subsequently showed an occasional sperm in the ejaculate with but one pregnancy resulting. Comparing these results, Hanley and Hodges *(12)* proposed that there must be a fundamental difference in the anatomy and physiology of the postinfective and other groups, rather than any surgical or technical fault leading to failure of treatment.

A new aspect was identified by Young in 1970 *(13)*. Working in Liverpool, it was observed that of 52 men with obstructive azoospermia seen between 1968 and 1970, 28 (54%) had lung disease; 18 with bronchitis since childhood, seven with bronchiectasis and three had had lobectomies—an association now known as Young's syndrome. Shortly thereafter, in France, Guillon and Chuberre *(14)* confirmed that bronchiectasis was frequently associated with obstructive azoospermia, and observed that in these cases the obstruction was usually in the heads of the epididymes. Interestingly, this association was more commonly seen in patients from Brittany than among those in Paris. Although large series of cases with Young's syndrome have been described since then from England *(15–17)*, Australia *(18)* and France *(19)*, this condition seems to be very rare in the USA, with only two definite case reports in the literature *(20)*.

The increasing popularity of vasectomy as a method of contraception has produced numerous examples of acquired testicular obstruction. Since some of these men subsequently request vasectomy reversal, the effects of the blockage on testicular and epididymal function can be studied and related to subsequent fertility. Collected reviews have shown that spermatozoa return to the ejaculate in about 80% of cases, and 40–50% of female partners eventually become pregnant *(21–23)*. Meticulously careful vasovasostomies with microsurgical techniques were shown by Silber *(24)* to produce improved pregnancy rates of up to 71%. However, the results were less favourable if more than 10 years had elapsed between the vasectomy and the reversal procedure *(25)*. One cause of failure was rupture of epididymal tubules due to back-pressure, leading to granuloma formation and failure of flow after reconnecting the vasa—a problem that could be overcome by epididymovasostomy *(26)*.

Antibodies against spermatozoa developed in 60–80% of men after vasectomy *(27,28)*. These could be detected by serum sperm-agglutinating and sperm-immobilising tests *(29,30)*. Although these antibodies appeared to be harmless to the individual *(31)*, there was controversy about their possible effect on fertility following vasectomy reversal *(32,33)*. Similarly, antisperm antibodies could be demonstrated after experimental *unilateral* vasoligation *(34)*, but the effects of such antibodies on the spermatozoa emerging from the contralateral unobstructed testicle were unclear *(35)*. Indeed, the clinical relevance of the immunological sequelae of testicular obstruction remained to be defined.

There was little point in exploring azoospermic men with small testes and elevated serum FSH levels, since it was shown that spermatogenesis was always inadequate *(36)*. As a result, surgery has only been recommended for azoospermia with normal or near normal serum FSH levels and at least one normal size testis; possible unilateral testicular obstruction, usually with antisperm antibodies; and for vasectomy reversal.

Patients and methods

Experience gained with 450 subfertile males with testicular obstruction (370 bilateral, 80 unilateral) and 162 men requesting vasectomy reversal (130 first attempt, 32 after a failed previous attempt) is analysed to study the pathological and immunological problems associated with the surgery of this condition. Antisperm antibodies were measured throughout by the tray agglutination test (TAT) *(37)* or gelatin agglutination test (GAT) *(29)*.

Obstructive azoospermia

In 370 men with azoospermia and normal serum FSH levels, the scrotum was explored through a midline incision under general anaesthetic. Both testes were exposed, and the epididymes were examined with 2× magnifying loupes. If no distended tubules were seen, testicular biopsies were taken and the scrotum was closed. If distended tubules were seen, the extent of the dilatation was defined as either capital (confined to the head) or caudal (extending down towards the tail) (initially these changes were photographed and a record kept for subsequent comparative analysis). The vas was then picked up with Allis forceps, and a 0.5 cm linear incision made opposite the lowest part of the epididymis exhibiting tubular distension. A nylon probe was inserted first to define the lumen, and then a 2G Portex®

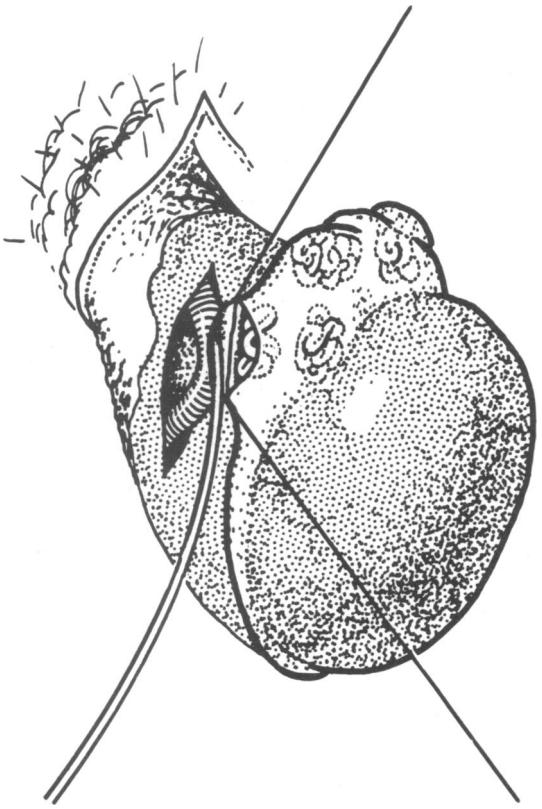


Figure 1. Side-to-side epididymovasostomy using 6.0 prolene; the vasogram cannula is removed as the anastomosis is completed. (By courtesy of the *British Journal of Urology (15).*)

intravenous cannula through which 5 ml 25% Hypaque® was injected before taking the vasogram X-ray. If the vas was clear, epididymovasostomy was performed by side-to-side anastomosis using continuous 6.0 prolene; the vasogram cannula was removed after completion of the upper end of the anastomosis (Fig. 1). If a vasal block was found, vasovasostomy was performed as described below, usually through a separate groin incision. If ejaculatory duct obstruction was demonstrated, cystourethroscopy was carried out to examine the posterior urethra. The scrotum was closed with 3.0 chromic catgut to the dartos layer and subcuticular nylon or Vicryl® to skin. Cotton wool was applied to the scrotum and a Litesome® support was worn for 5–7 days.

Unilateral testicular obstruction

A total of 80 subfertile men underwent scrotal exploration for unilateral testicular obstruction using the technique and methods of repair described above. In 10 men with irreparably blocked testes and high titres of antisperm antibodies, the obstructed testis was removed and replaced with a prosthesis.

Vasectomy reversal

Bilateral microsurgical vasovasostomies were performed in 130 cases, usually through bilateral oblique scrotal incisions, by an overlapped side-to-side technique using 6.0 prolene (Fig. 2). Failed vasectomy reversal was

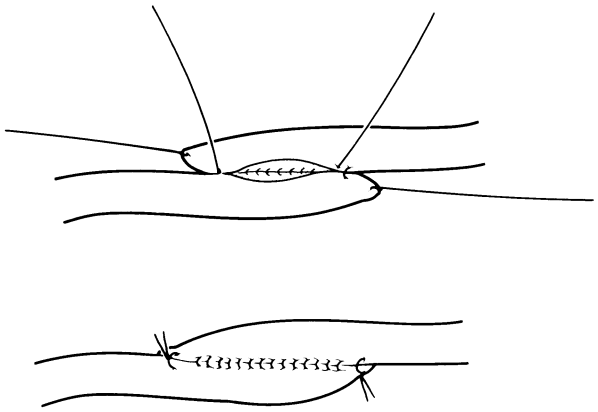


Figure 2. Side-to-side vasovasostomy using 6.0 prolene. (By courtesy of the *British Journal of Urology (46).*)

studied in 32 men by re-exploration of the scrotum. Among 23 azoospermic men, microsurgical revision of the vasovasostomies or epididymovasostomies were carried out as indicated by the operative findings; unilateral obstruction was found and corrected in nine oligozoospermic men.

In all cases, follow-up sperm counts were taken whenever possible at 3, 6 and 12 months. Enquiry at clinic visits as to production of pregnancy was supplemented by a postal survey.

Results

Obstructive azoospermia

The operative findings in 370 cases are shown in Fig. 3. Each group will be considered in turn, starting at the testicle and working towards the ejaculatory duct.

Empty epididymes (49 cases; 13.2%)

Impaired spermatogenesis was found in the vast majority of these cases—maturation arrest in 31 and sertoli cell only syndrome in 14. Five men with maturation arrest were treated with Pergonal® 2 ampoules 3 times a week for 3 months—one responded, the sperm count became normal and his wife became pregnant.

Four men had normal spermatogenesis, with very high serum TAT titres (≥512). One of these biopsies showed

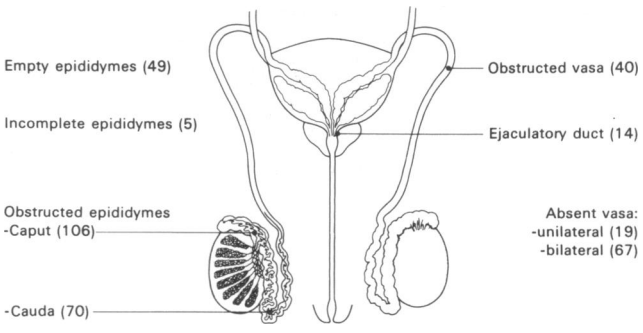


Figure 3. Sites of obstruction in 370 azoospermic males.

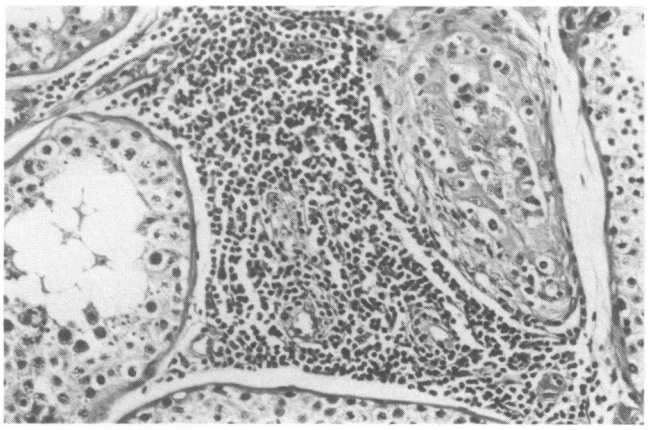


Figure 4. Testicular biopsy with mononuclear cell infiltrate supporting a diagnosis of autoimmune orchitis (H + E × 200).

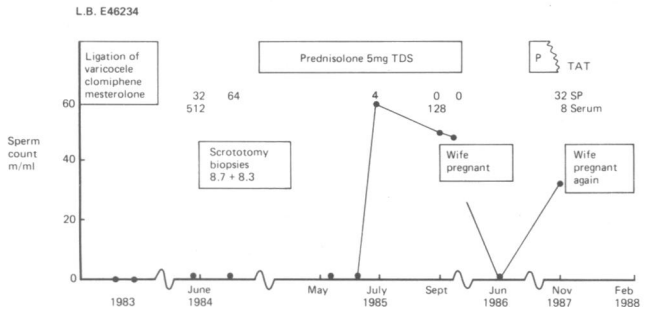


Figure 5. Serial sperm counts and TAT titres in patient with empty epididymes, and good spermatogenesis (Johnsen counts indicated) but no demonstrable mononuclear cell infiltrate on biopsy. Note response to prednisolone supporting a presumptive diagnosis of autoimmune orchitis.

focal mononuclear cell infiltration of seminiferous tubules supporting a diagnosis of autoimmune orchitis (Fig. 4). All of these men responded to oral prednisolone, 5 mg three times a day for 6 months, with normalisation of sperm counts. One successfully produced two pregnancies (Fig. 5).

Incomplete epididymes (5 cases; 1.4%)

In four cases, part or all of the epididymes had been excised surgically as treatment for epididymal cysts or chronic epididymitis; one was successfully repaired but no pregnancy resulted. One case showed congenital lack of continuity between head and midbody of the epididymis on both sides; repair was unsuccessful.

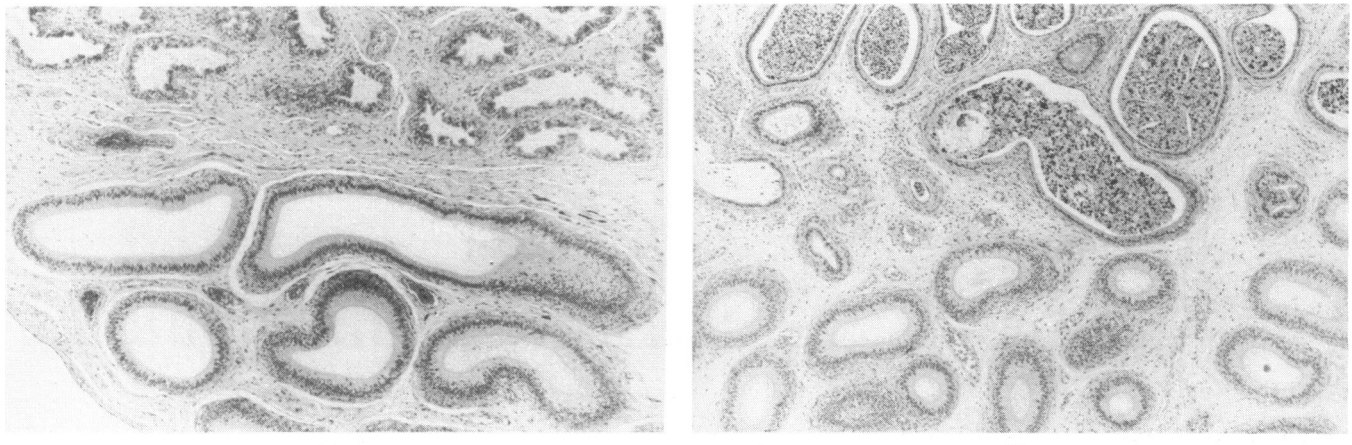
Caput epididymis (106 cases; 28.6%)

Histological examination of the transition zone between distended and empty tubules revealed that this coincided with the change from ductuli efferentes, lined by ciliated columnar epithelium, to ductus epididymis, lined by stratified columnar epithelium with microvilli (Fig. 6). The ductuli efferentes were distended with inspissated spermatozoa and lipid; fat stains (oil-red-O and Sudan black) were strongly positive for neutral lipid in the epithelium, interstitium and tubular lumina (Fig. 7).

Electron microscopy showed that the cilia were ultrastructually normal *(15)*, and cilia beat frequency was normal in these patients *(38)*; however, mucociliary clearance was significantly impaired *(39)*. A total of 82 patients (77.4%) had chronic sinusitis, bronchitis or bronchiectasis (Young's syndrome), whereas only 9 (8.5%) had a past history of genital infection. Twelve (11.3%) said they had produced children previously.

After epididymovasostomies, 90 (85%) were followed for 6 months or more: 11 (12%) developed sperm counts of 10 m/ml or more and three produced pregnancies. Only one patient out of 46 recovered a normal sperm count with surgery alone, compared with 10 of 44 given carbocisteine 375 mg three times a day for 6–12 months after surgery ($\chi^2 = 8.85$; $P < 0.01$).

Ten patients with Young's syndrome had a definite history of Pink Disease (mercury intoxication) in infancy. Since this condition was virtually eliminated in 1955 with the withdrawal of calomel from teething powders and proprietary worm medications *(40)* (Fig. 8*a*), the dates of birth of all patients with Young's syndrome are charted in Fig. 8*b*; it may be seen that only



(*a*) (*b*)

Figure 6. Junction of ductuli efferentes and ductus epididymis. (*a*) Normal. (*b*) Patient with Young's syndrome and capital block—note the ductuli efferentes containing masses of degenerate spermatozoa (H + E × 35).

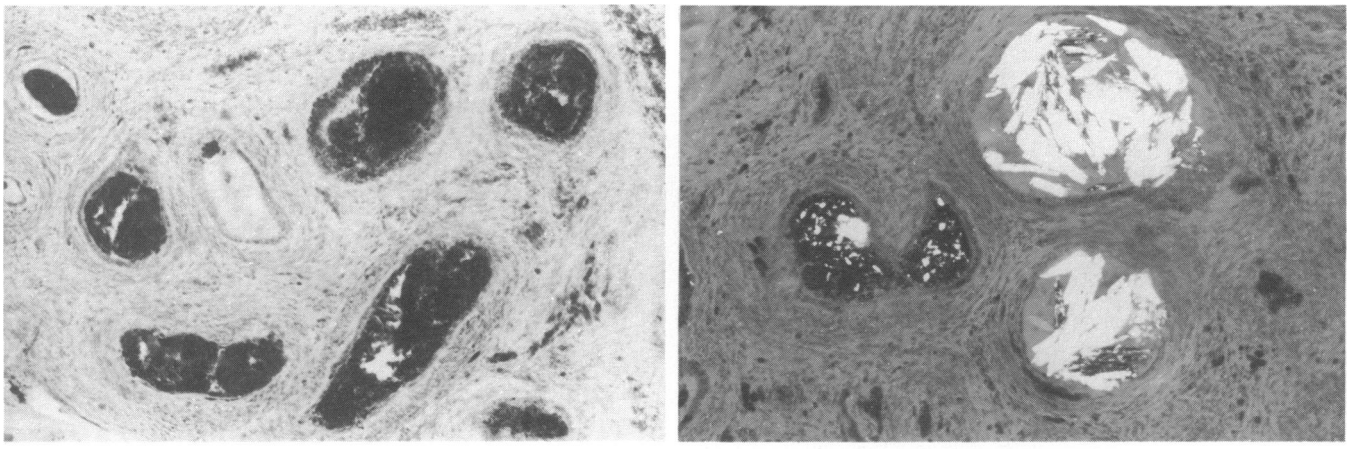


Figure 7. Frozen sections of caput epididymis from patient with Young's syndrome. *(a)* Lipid-rich debris in lumens and some epithelial cells. *(b)* Cholesterol crystals demonstrated by crossed polarised light (Sudan Black × 65).

one such patient was born after this year, whereas there was no change in frequency of those with caudal blocks.

Cauda epididymis (70 cases; 18.9%)

Histological examination revealed that these blocks occurred in the ductus epididymis; fat stains were negative.

A history of epididymitis, urethritis or smallpox was given by 38 men (54.3%), whereas only one had chronic bronchitis (different from those with capital blocks ($\chi^2 = 87.2$, $P < 0.001$)). Seven (10%) said they were previously fertile.

After epididymovasostomies, 26 (43%) of 60 patients followed for 6 months or more regained normal sperm counts, and 18 (30%) produced pregnancies. Of the failures, 10 had repeat epididymovasostomies with five successes; the final patency rate is therefore 52%, the pregnancy rate 38%. These sperm count results are significantly better than those obtained with capital blocks ($\chi^2 = 27.78$; $P < 0.001$).

Absent vasa

Bilateral absence of the vasa was found in 67 cases (18.1%)—no treatment was attempted.

There were 19 cases (5.1%) with unilateral absence of the vas. The contralateral testis was not connected proximally in seven, the testis was atrophic in three, but nine were suitable for reconstruction; three regained normal sperm counts but no pregnancies resulted.

Vasal blocks (40 cases; 10.8%)

Vasal blocks were found most commonly in the groin (15), but also occurred in the abdomen (8), in the scrotum (3) and in multiple sites (14). On the opposite side, caudal epididymal (15) or vasal (13) blocks were

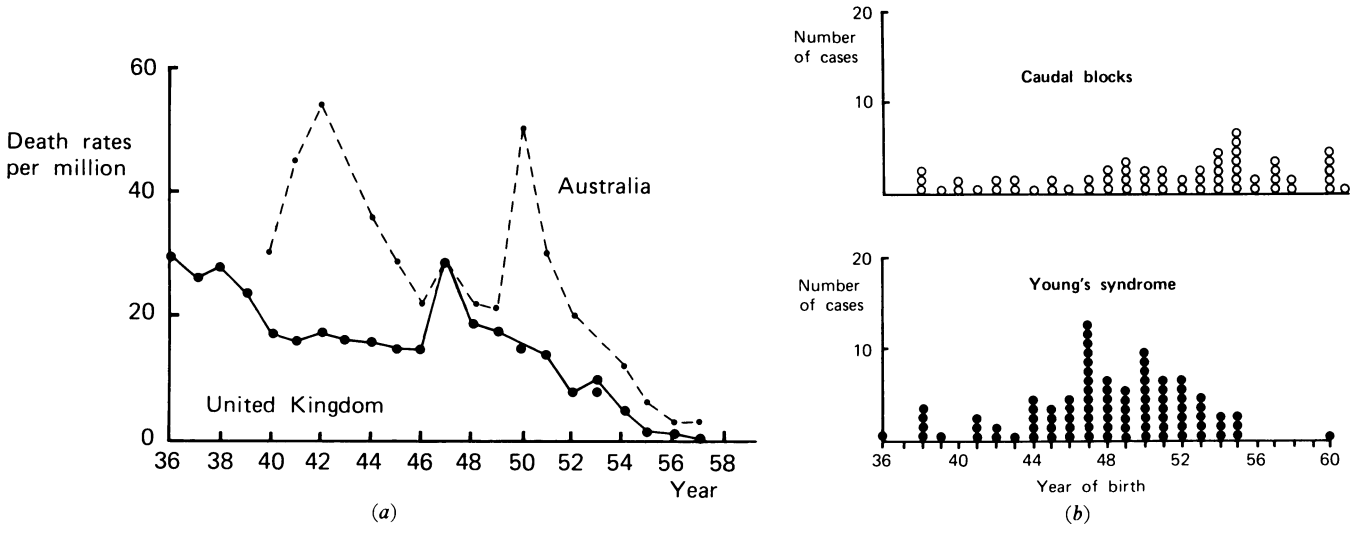


Figure 8. (a) Death rates from Pink Disease in Australia and UK. (Redrawn from Clements *(40)* by courtesy of *Medical Journal of Australia.*) *(b)* Years of birth of men with caudal blocks and those with Young's syndrome. Note that Pink Disease and Young's syndrome declined in incidence in those born after 1955, when calomel-containing teething powders were withdrawn.

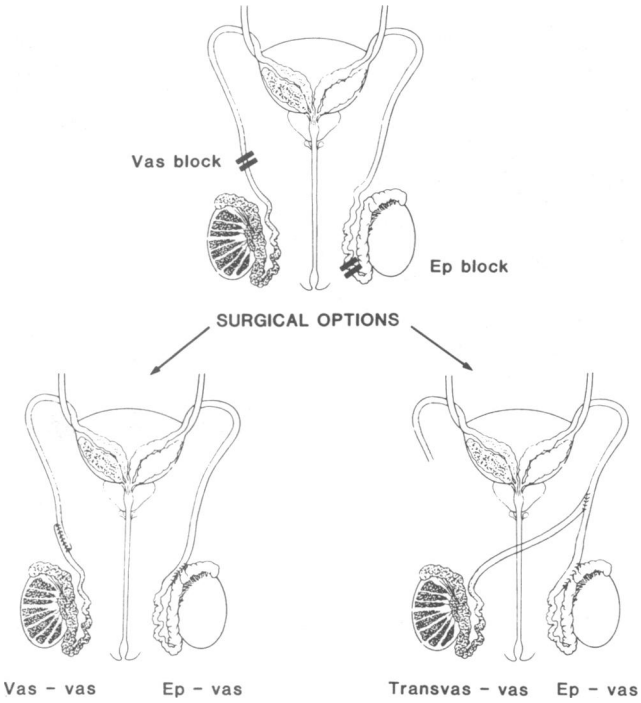


Figure 9. With asymmetrical blocks there is a choice between total anatomical reconstruction and transvasovasostomy; the former gave better results (Table I). (By courtesy of the *British Journal of Urology (16).*)

most commonly found; in four cases the opposite epididymes were empty, in three the contralateral testis was atrophic; there was one capital block and the situation was not defined in four cases. The most common cause of vasal blocks was previous genital or urinary infection (16 cases) and there was a history of tuberculosis in seven, of which three were active, significantly different from capital blocks ($\chi^2 = 69.8$, $P < 0.001$; no significant difference from caudal blocks). Nine vasal blocks resulted from surgery in infancy or childhood, two from landmine injuries, and the cause was unknown in six.

Various methods and combinations of repairs were used with these testicles (Fig. 9). These included vasovasostomies (10), epididymovasostomies (15), transvasovasostomies (15) and insertion of sperm reservoirs (3) *(41)*. No repair was possible in 37 testicles. The results are shown in Table I. It can be seen that transvasovasostomy gave much poorer results than complete reconstruction.

Table I. Results of surgery for vasal blocks (excluding vasectomy reversal). See Fig. 9 for techniques of reconstruction

Reconstruction	*Treated and followed up*	*Sperm count >10 m/ml*	*Pregnancies*
Vas–vas	11	8	3
±ep–vas		(73%)	(27%)
Trans–vas–vas	11	1	0
±ep–vas		(9%)	
Reservoirs	3	0	0

Ejaculatory duct obstruction (14 cases; 3.8%)

Vasography revealed four Mullerian duct cysts (Fig. 10), three Wolffian duct malformations (Fig. 11), and one example of megavesicles in a diabetic. The obstruction was due to surgery in infancy (Fig. 12) in two (one imperforate anus, one cystectomy), tuberculosis in one, and cause unknown in one. After endoscopic incision of Mullerian duct cysts, the sperm count returned to normal in two cases and one produced a pregnancy.

Antisperm antibody results

The incidence of antisperm antibodies in the various categories is shown in Table II. It can be seen that such antibodies occurred significantly more often with caudal than with capital epididymal blocks, and with blocked compared to absent vasa. Postinflammatory disease was, therefore, most likely to be associated with development of antisperm antibodies.

The relationship between presence or absence of antisperm antibodies and production of pregnancy by 66 men

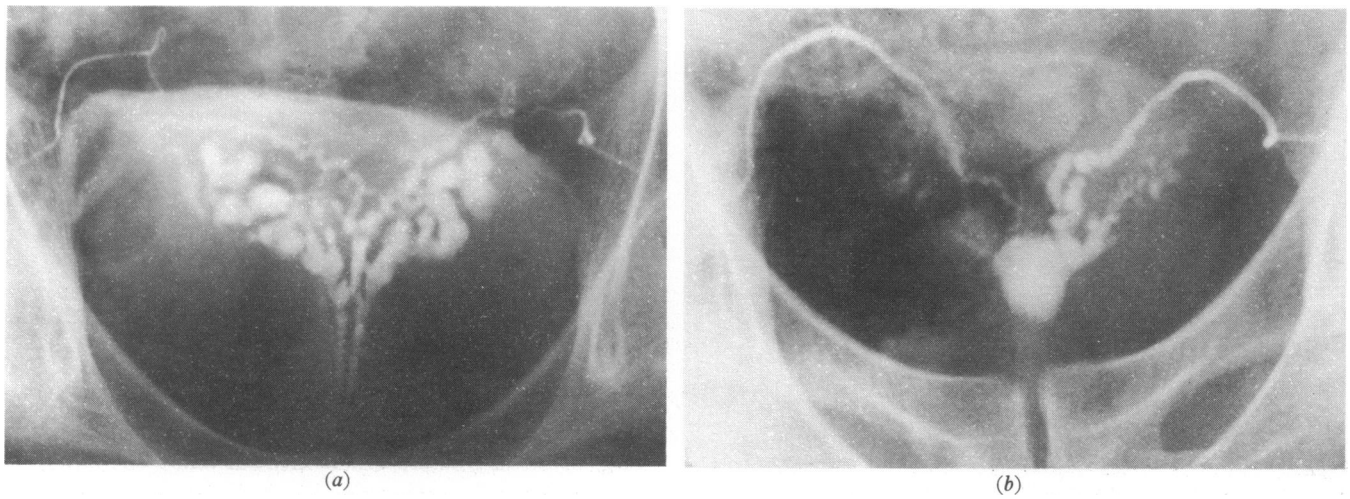


Figure 10. (*a*) Normal vasogram. (*b*) Vasogram showing Mullerian duct cyst and marked dilatation of vasa.

whose obstruction had been corrected (sperm counts ≥10 m/ml) is shown in Table III. Pregnancy was significantly more likely to occur if the man had not produced such antibodies.

Figure 11. Vasogram showing malformation of right Wolffian duct; the left vas was absent.

Table II. Incidence of serum antisperm antibodies in 369 men with obstructive azoospermia related to site of obstruction

Site of problem	*Number*	*Serum GAT or TAT positive (%)*
Epididymis		
Caput	106	29 (27.4)
Cauda	69	36 (52.2)
Vas deferens		
Absent—bilateral	67	11 (16.4)
—unilateral	19	6 (31.6)
Blocked	40	19 (47.5)
Ejaculatory duct	14	1 (7.1)
Totals	369	112 (30.4)

(Caput *vs* Cauda $\chi^2 = 9.43$; $P < 0.01$
Absent *vs* blocked vas $\chi^2 = 17.99$; $P < 0.001$)

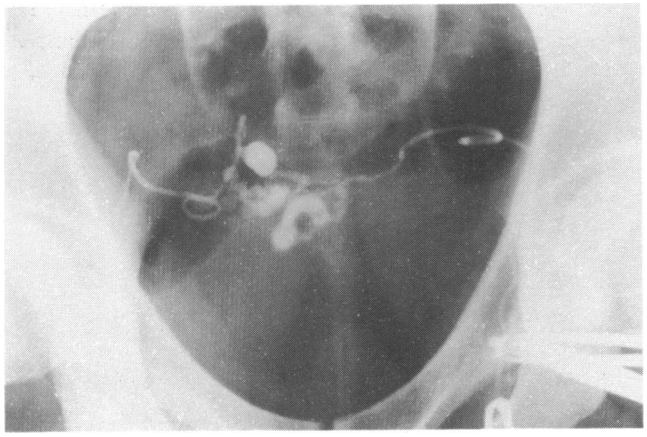


Figure 12. Vasograms showing obstruction after surgery for imperforate anus in childhood.

Table III. Relationship between serum antisperm antibodies and production of pregnancy after correction of obstructive azoospermia (sperm counts ≥10 million per ml)

Pregnancy produced	*Serum GAT or TAT*		*Total*
	Positive	*Negative*	
Yes	11	20	31
No	22	13	35
Total	33	33	66

(By Fisher's Exact Test $P < 0.05$)

Unilateral testicular obstruction

The findings in these 80 cases have been described in detail elsewhere *(42)* and so only the salient points will be summarised here. The most common sites of obstruction were cauda epididymis after previous genital infection (40 cases), or in the vas after surgery in infancy or childhood (24 cases). Half of these patients had severe oligozoospermia despite normal testicular biopsies, and three-quarters had antisperm antibodies, often in high titres. After surgical reconstruction, and prednisolone therapy *(43,44)* if necessary, 32% of 60 patients with adequate follow-up produced pregnancies, with the best results occurring in those who started with the lowest sperm counts (Table IV). Removal of irreparably blocked testes in 10 men led to profound falls in antibody titres (Fig. 13). Histological examination showed evidence of mononuclear cell infiltration in epididymes and seminiferous tubules, similar to that seen in azoospermic men with empty epididymes (see above). In one case with a demonstrable high vasal block and empty epididymis, pronounced mononuclear cell infiltration of rete testis was found (Fig. 14).

Table IV. Unilateral testicular obstruction: pregnancies produced/number treated in 60 patients with adequate follow-up related to initial sperm counts and surgical treatment. From Hendry *(42)*

Surgical treatment	*Initial sperm count m/ml*			
	<5	*6–20*	*>20*	*Total*
Ep–vas	7/18	1/10	2/5	10/33 (30%)
Vas–vas	2/5		1/1	3/6 (50%)
Orchidectomy	1/2	0/2	1/6	2/10 (20%)
None (steroids)	2/5	1/2	1/4	4/11 (36%)
Total	12/30 (40%)	2/14 (14%)	5/16 (31%)	19/60 (32%)

Vasectomy reversal

The results of bilateral microsurgical vasovasostomies in 130 cases have been described in detail elsewhere *(45)*. In essence, patency was restored in over 90% of cases, and

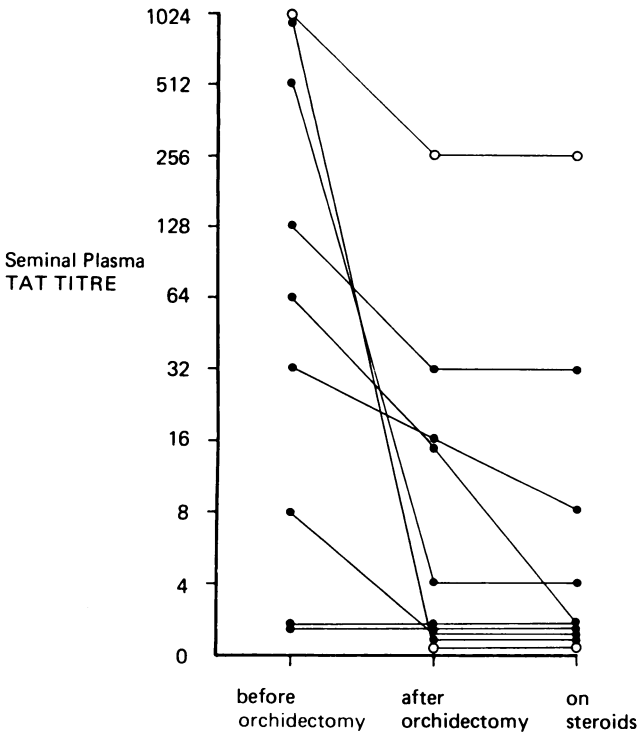


Figure 13. Seminal plasma antisperm antibody titres in men with incorrectable unilateral testicular obstruction before and after orchidectomy, and subsequently on prednisolone therapy. (By courtesy of *British Journal of Urology (42).*)

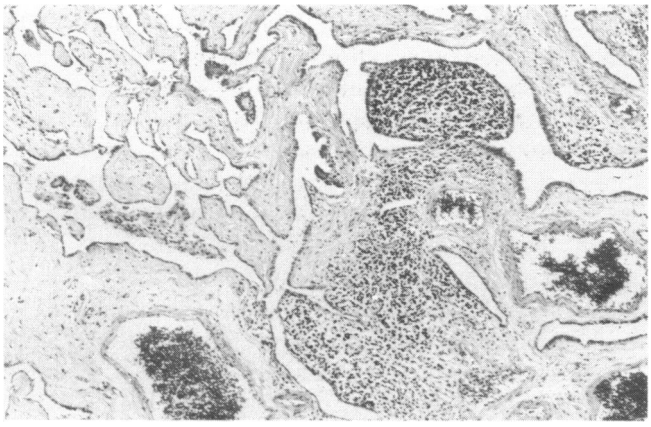


Figure 14. Rete testis in patient with a high vasal block, empty epididymis and high titres of antisperm antibodies. Note mononuclear cell infiltrate supporting a diagnosis of autoimmune orchitis (H + E × 80).

similar pregnancy rates were observed in the two collaborating centres (Brighton 44%; St Bartholomew's 45%). Antisperm antibodies were found in the sera of 95 (79%) of 121 men tested. Significantly fewer pregnancies (25%) were produced by men with very high serum antisperm antibody titres (≥512) (Table V).

Failure of vasectomy reversal was studied in 23 azoospermic men (Table VI). Microsurgical revision of the vasovasostomies was required in 12, whereas four required epididymovasostomies for secondary epididymal blocks due to ruptured tubules. Overall, 37% produced pregnancies, although there was no success in those with very high antisperm antibody titres (≥1024). Unilateral obstruction was found and corrected in eight of nine oligozoospermic men, leading to normal sperm output in four. These results have also been reported in detail elsewhere *(46).*

Discussion

Experience gained from more than 600 patients with testicular obstruction allows some conclusions to be drawn, and some comments to be made on the pathophysiological processes associated with this condition.

Vasectomy reversal, provided it is carried out carefully and accurately, gave reliable results in over 90% of cases. Microsurgery is clearly essential; the surgeon can choose between a two-layer end-to-end anastomosis as recommended by Silber *(24–26)*, or a one-layer side-to-side technique as described above. The latter technique provides a large opening between the vasa, and is not affected by any disparity in diameter of the two ends. Pregnancy rates of around 50% have been reported in many series *(23)*. If the reversal procedure fails, the most likely cause is fibrosis at the previous anastomoses; however, if no flow of milky fluid can be obtained from the testicular end of the vas, epididymal blow-out should be suspected, and epididymovasostomy carried out at a level where free flow of fluid containing spermatozoa can be obtained from the epididymal tubules *(47)*. Antisperm antibodies are seldom a problem in this setting until serum titres of 512 or more are reached, and even then titres may fall following the reversal procedure. If pregnancy does not occur with a good sperm count and high antibody titres are present in serum or seminal plasma, cyclical prednisolone therapy may be considered *(43,44)*.

Table V. Results 1 year or more after vasectomy reversal. From Parslow *(45)*

Serum TAT titre	*Total no.*	*Information available*	*Sperm count*			*? Pregnancy*	
			0	*<20*	*≥20 m/ml*	*Trying*	*Success*
≤16	40	32	5	7	20	29	15 (52%)
32–256	40	36	1	14	21	32	17 (53%)
≥512	41	31	2	9	20	28	7 (25%)

Table VI. Results of redo vasectomy reversal in 23 men who were azoospermic following a previous attempt. From Royle and Hendry *(46)*

			Follow-up sperm counts			
Category	*Treatment*	*No.*	*0*	*<20*	*≥20*	*Pregnancies**
1 Blocked vas	Redo vas–vas	12	1	3	7	5/8 (62%)
2 Epididymal block	Ep–vas	4	1		3	2/4 (50%)
3 Antisperm antibodies (≥1024)	Surgery + prednisolone	7	1	3	3	0/7
Total						7/19 (37%)

* Trying for 6 months or more

Epididymovasostomy for postinfective obstruction is the next most successful procedure. These blocks are usually situated at the tail of the epididymis or in the vas, sometimes in both sites. The surgeon should be aware that a history of genital infection may not always be forthcoming. However, the appearance of epididymes with bulging tubules from top-to-bottom is unmistakable. Vasal patency can be established, or co-existing vasal blocks recognised, by vasography which should be carried out routinely. Accurate, tension-free anastomosis is essential, making sure that a free flow of milky fluid is obtained from the epididymis *(47)*. If the surgeon has the time and the facilities, tubule-to-tubule anastomosis can be performed, and there is some evidence that this may give improved results *(48)*. If the sperm count remains negative at 6 months, the scrotum should be re-explored and the anastomoses redone—this is often a relatively easy procedure since the structures are already in apposition and a tension-free join is easy to obtain. Vasal blocks are most often postinfective, and can usually be dealt with by vasovasostomy through a separate incision. Sometimes the blocks are asymmetrical; for example, a caudal epididymal block on one side, a vasal block on the other. Under these circumstances a scrotal transvaso-vasostomy offers an temptingly easy solution, but the results shown in (Table I) indicate that it is better to take the time to repair each side, seeking perfect anatomical reconstruction. Not uncommonly, a great length of vas may have been removed at the time of groin surgery in infancy or childhood, and transvasovasostomy may be the only way of reconnecting such a testicle. However, this may put the patency of the contralateral vas at risk, so unless the opposite testis is atrophic, removal of the obstructed testicle is probably the treatment of choice, especially if there is a high titre of antisperm antibodies. Once the testis is removed, antibody titres fall rapidly, although some patients require supplementary prednisolone therapy *(43,44)*. It is the authors' practice to discuss this option fully with the patient and his spouse, explaining why it is necessary, and offering to insert a prosthesis, before proceeding with this option.

Capital blocks are associated with chronic chest disease in three-quarters of these patients (Young's syndrome). There appears to be failure of flow through the ductuli efferentes, of which 10 or 12 lobules make up the caput epididymis, before joining to form the ductus epididymis at the junction of the head and body of epididymis *(49,50)*. The ciliated columnar epithelial lining of the ductuli efferentes is similar to the lining of the nasal and respiratory passages. Since the cilia are ultrastructurally normal in Young's syndrome, with normal beat frequency *(38)*, increased viscosity of the fluid within the tubules seems the most likely explanation for the demonstrably impaired mucociliary clearance *(39)*. Fat stains have revealed excess neutral lipid in the epithelial cells and in the lumina of the ductuli efferentes (Fig. 7). Comparison with normal epididymes shows that this is abnormal in young men, although it is seen increasingly often in older men (unpublished observations). Indeed, patchy hold-up in the ductuli efferentes has been observed commonly at autopsy in old men *(51)*. In Young's syndrome there is abnormal accumulation of lipid, leading to impairment of flow in the ductuli efferentes and respiratory passages.

There was a definite past history of Pink Disease (mercury intoxication) in childhood in 10% of our patients with Young's syndrome. The relatively common occurrence of Young's syndrome in England and Australia, where Pink Disease was quite common up to 1955 when mercury-containing teething powders were withdrawn *(40,52)*, and the virtual disappearance of Young's syndrome in men born after this date, make it possible that mercury intoxication in childhood may have had a part to play in its aetiology as well. By contrast, Young's syndrome is rare in the USA and it is noteworthy that sale of calomel (mercurous chloride) was actively discouraged by the Food and Drug Administration in the USA in 1932 and on several occasions thereafter *(53)*. Mercury is well recognised as an enzymatic poison which can leave permanent damage *(54,55)*; other examples of poisons which affect function in the head of the epididymis include α-chlorhydrin and

6-chloro-6-deoxyglucose, which interfere with sugar transport across the epithelium *(56)* and block glycolysis, leading to epididymal cyst formation and sterility in rats *(57)*. Carbocisteine may be exerting its beneficial effect by feeding sulphydryl groups into the glutathione shunt mechanism (for dealing with superoxide radicals), thus providing some indirect evidence that lipid peroxidation may be contributing to the problem. Certainly deficiency of vitamin E (a potent antioxidant) leads to damage in the ductuli efferentes *(58)*. If the hypothesis connecting Young's syndrome with mercury intoxication in childhood is correct, this condition should disappear.

Absent vasa are commonly found, and may be diagnosed by finding a small volume ejaculate with low pH and absent fructose. Hitherto considered untreatable, recent technical developments have allowed pregnancies to be produced by *in vitro* fertilisation using motile spermatozoa obtained by micropuncture from the distended epididymal tubules *(59)*.

Routine vasography revealed a number of congenital malformations in the ejaculatory duct region. Although uncommon, these are worthy of recognition since they may be correctable by simple endoscopic incision.

When a testicle is obstructed, spermatozoa are redirected to the lymphatics *(60)*, and have been observed in para-aortic lymph nodes in man following vasectomy *(61)*. It is therefore not surprising that antisperm antibodies are produced by many patients with testicular obstruction. The effects of these antibodies on subsequent fertility will depend on how much antibody production is provoked. This is likely to vary according to the individual's immunological responsiveness, and we have shown that it is also modified by the age at which the obstruction develops—antibodies being much less common with congenital absence of the vasa than with acquired vasal blocks or following vasectomy. Secondly, the class of antibody produced is most important; locally produced immunoglobin (Ig)A has a much more potent inhibitory effect on sperm function than circulating IgG *(62)*. Stimulation of antibody production has been demonstrated after urethritis *(63)*, and so it is not surprising that antibodies are common with postinflammatory epididymal and vasal blocks, and that a deleterious effect on fertility after surgical reconstruction in these cases can be demonstrated (Table III). This is unfortunate, as this is the most favourable group among subfertile males with azoospermia; however, treatment with prednisolone may be effective *(43,44)*.

The finding of an empty epididymis was recognised as an unfavourable sign by Hagner 50 years ago *(7)*. Although this is often a sign of defective spermatogenesis, which will be demonstrated by testicular biopsy, it may sometimes be associated with normal spermatogenesis and very high antisperm antibody titres ($\geq$512). Histologically, there may be evidence of mononuclear cell infiltration in and around seminiferous tubules, and although this is not an uncommon finding *(64)* it may support a diagnosis of autoimmune orchitis (Fig. 4). This condition, well-recognised in experimental animals *(65–67)*, had not been described before 1979 as a spontaneous occurrence in man *(68)*. Typically, it occurs in a very patchy distribution, with maximal cell infiltration round the rete testis—the weakest part of the blood–testis barrier. As a result of its local distribution, the absence of mononuclear cell infiltration on testicular biopsy should not be interpreted as evidence that this condition is not present. This is particularly important as appropriate steroid therapy (such as prednisolone 5 mg three times a day for 6 months) may lead to normalisation of the sperm output (*see* Fig. 5). This response demonstrates that antisperm antibodies in high titre, when associated with severe oligozoospermia or even azoospermia *(69)*, may be due to a combination of autoimmune orchitis with rete testis hold-up. This mechanism may play a part in the production of the oligozoospermia seen with unilateral testicular obstruction, associated with normal spermatogenesis and positive serum antisperm antibodies—a syndrome that was first described over 20 years ago *(70)* but still often escapes recognition in the subfertile male.

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Notes on books

Current Critical Problems in Vascular Surgery edited by Frank J Veith. 587 pages, illustrated. Quality Medical Publishing, St Louis. 1989. $79.00. ISBN 0 942219 01 5

160 authors contribute 109 chapters to this extensive text on vascular surgery. As the title implies, the authors debate controversial topics and try to put them into a context that is meaningful to practising surgeons.

Ultrastructure of the Extraparietal Glands of the Digestive Tract edited by A Riva and P M Motta. 278 pages, illustrated. Kluwer Academic Publishers, Boston. 1990. £95.75. ISBN 0 7923 0303 2

A textbook of advanced anatomy. Numerous electron microscopic photographs and a text that covers immunocytochemistry and autoradiography relating to the salivary glands, pancreas, liver and gallbladder.

Fundamentals of Obstetrics and Gynaecology by Derek Llewellyn-Jones. 5th edition. **Volume 1: Obstetrics**. 488 pages, illustrated. **Volume 2: Gynaecology**. 301 pages, illustrated. Faber and Faber, London. Vol. 1, £15.00; Vol. 2, £12.99. ISBN 0 571 14227 3 and 0 571 14228 1

This 5th edition of a well-established textbook has been fully updated to include recent knowledge. Although principally designed for undergraduates and house officers, there is much in both volumes that will interest more senior trainees in the specialty of obstetrics and gynaecology. Both volumes are well illustrated and clearly written and there seems little doubt that they will uphold the reputation of the earlier editions.

Complications of Spine Surgery edited by Steven R Garfin. 399 pages, illustrated. Williams and Wilkins, Baltimore. 1989. £63.50. ISBN 0 683 03435 9

As surgical audit has begun to be accepted and practised on a wide scale, so there has arisen a number of books on the various complications of surgery. This volume contains twenty chapters on problems that may arise after spinal surgery. These range from excessive blood loss, through spinal cord injury, to complications associated with the use of the halo fixation device that is used for stabilisation of the cervical spine.

Atlas of Automated Percutaneous Lumbar Discectomy by Antonio Solini. 143 pages, illustrated. Springer-Verlag, Vienna. 1989. DM138. ISBN 3 211 82172 4

Removal of a prolapsed intervertebral disc through a percutaneous approach was first introduced in 1975 in Japan. Since then the technique has been refined. In 1984 Onic introduced a technique which is carried out under local anaesthesia and uses a probe only 2 mm in diameter. This procedure has now been performed over 35 000 times without documented instance of major morbidity. This clearly laid out atlas shows how it should be performed. Yet another example of the development of minimal invasive surgery.