THE SURGERY OF MALE SUBFERTILITY

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by
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THE INCREASING REALIZATION that the husband is probably at fault in a subfertile marriage quite as often as his female partner, has recently led to a much more thorough investigation of the male genital tract, while the widespread establishment of new subfertility clinics points to the general public's growing interest and demands.

This lecture is concerned only with the *surgical* aspects of *male* subfertility, while still keeping in mind the fact that both an ovum and a spermatozoa are necessary, and that they must come into contact at the right time and place. The observations are based upon a study of 148 cases of azoospermia, and over 300 cases of oligozoospermia, but no attempt will be made to draw any statistical conclusions from figures because the groups are highly selected.

A majority of the patients in this review were well built virile, athletic men with normal external genitalia, the size of the testicles providing no reliable information about the quality of the spermatogenesis, and one has come to regard consistence as being more important than size (apart from frankly atrophic testicles). The small firm testicle can be just as effective as a very large one, and I have yet to encounter a normally firm testicle which showed no evidence of spermatogenesis on histological section. In other words—if a testicle feels clinically normal it is nearly always capable of producing spermatozoa, even if they are reduced in number, or absent from the ejaculate.

Following the ordinary clinical examination, one of the most useful preliminary investigations of a subfertile couple, is a post-coital test, performed at the estimated time of ovulation. If there are very few, or no actively motile sperms in the cervical mucous plug 12 to 18 hours after intercourse, it is quite unjustifiable to start tubal insufflations or curettage until the test has been repeated and confirmed, and the male partner more fully investigated. Conversely if the cervix is filled with actively motile sperms, the male is unlikely to be at fault.

Any surgeon should be able to confirm the presence or absence of spermatozoa under a microscope, but the correct interpretation of morphology requires expert knowledge. A laboratory unused to reporting on such material can give valueless and even misleading information, and I consider myself very fortunate that the great majority of the sperm counts referred to in this review have been performed by Dr. H. A. Davidson, who is an accepted authority in these matters.

It should be obvious that the full investigation of the subfertile couple can only be undertaken by a team of clinicians each interested in one aspect of the problem, but working closely together.

Special investigations in the male

Two investigations which are often carried out in order to obtain information about spermatogenesis and possible obstruction, are testicular biopsy and catheterisation of the ejaculatory ducts. Personally I think that although these two procedures are of great interest from an academic and research standpoint, their scope and value in the diagnosis and treatment of subfertility are extremely limited.

Testicular biopsy

This is a time-honoured procedure and I am aware that my case against its value as a routine procedure will have to be a good one. In the first place, in cases of azoospermia I have invariably found that a testicle which feels clinically well developed will show a relatively normal biopsy picture. If it feels atrophic, a biopsy is hardly necessary to confirm the fact, while in the borderline cases where one is in doubt I have not found that a biopsy has helped in deciding upon any particular line of treatment.

In cases of oligozoospermia, the biopsy may well support the findings of a reliable sperm count, but gives little more indication of the correct line of treatment. In addition, the patient does not always regard a testicular biopsy under local anaesthesia as a minor operation, since the procedure is not without morbidity. Scrotal haematomata can occur, hydroceles are not infrequent, while we have a record of one patient who developed an atrophic testicle within three months of a biopsy.

For the above reasons I have given up taking biopsy specimens before surgical exploration, being content to wait until the time of operation, and have not yet found that the result of such a biopsy would have influenced the line of treatment in any way had it been performed before operation. I was very gratified to learn last year that so experienced an authority on this subject as Dr. Vincent J. O'Connor of Chicago, felt the same way about testicular biopsy.

Seminal vesiculography

In the same way this procedure is of great academic interest, and may reveal some unusual congenital anomaly, but it gives little help in deciding upon treatment in the subfertile male. I have spent many hours catheterising ejaculatory ducts and have one or two very creditable vesiculograms, but success or otherwise depends too much upon luck, since a second chance rarely occurs if the patient or the X-ray apparatus have to be moved in any way. Inability to demonstrate a vesicle by this method means nothing, and I no longer try to do so.

A far more certain technique of demonstrating the vesicles when necessary, is to inject opaque medium down the vas from a small scrotal

incision. However, although we do not hesitate to do this during the course of an exploratory operation on the testicle, it is no longer used as a pre-operative investigation, unless the ejaculate volume is greatly reduced. One would think that the detection of a blockage in the vesiculo-prostatic area would save the patient an unnecessary testicular exploration, but in fact an obstruction in this region which causes an azoospermia without a marked decrease in ejaculate volume must be very rare.

In a review of 123 azoospermic patients, O'Connor (1953) did not find one ejaculatory duct obstruction. In the present series, all but one of the obstructions in this region were tuberculous, and they were all associated with a marked reduction or absence of ejaculate volume.

Classification

Once having established the fact that the *male* partner is at fault, and that there are no difficulties with the sexual act, two or more semen analyses will place him into one of two main groups. Oligozoospermia or azoospermia. I repeat, two or more sperm tests. No reliance should be placed upon one test unless it is well above average.

Oligozoospermia

Much has been written on the causes and treatment of oligozoospermia but we still do not know why so many millions of spermatozoa are necessary to enable one of them to effect fertilisation. Opinions vary about the numbers which constitute the lower limit of fertility, but for the purposes of this review, a count of under 20 millions per c.c. has been regarded as subfertile (Macleod, 1950). However, fertilisation can and has occurred with counts even under a million (Moore, White and Barton, 1951). I have a record of a man whose count ranged from 800,000 to 3 millions on four occasions, although a count of 5 millions was once noted elsewhere. Artificial insemination (husband) resulted in a normal pregnancy at the second attempt. Quite apart from these exceptional cases the count can vary enormously over a short space of time (Kennedy, *et al.*, 1951 ; Davidson, 1952), and it is indeed a foolhardy clinician who tells a man possessing any spermatozoa at all, that it is impossible for him to make his wife pregnant. Unlikely by all means—but not impossible.

Testicular temperature control

Spermatogenesis can be affected by many factors, among which are small variations in testicular temperature. The classical experiments of Moore and Oslund (1924) and Phillips and McKenzie (1934) showed that characteristic seminal degeneration occurred in rams whose scrotums were insulated from the external temperature. Gunn and his colleagues (1942) confirmed this work and found that the effects were not apparent in the semen unless the scrotum had been insulated for at least five days. However, quite recently in Cambridge, Glover (1955) has shown that the elevation of testicular temperature by 5°C. for only 24 hours resulted in the sudden appearance of tailless spermatozoa 19 to 24 days after the experiment.

Davidson (1954) has shown that the wearing of Y-front underpants which hold the testicles in close contact with the body surface has a depressant effect on sperm maturation, while repeated cold sponging of the scrotum has the opposite effect.

Interference with the temperature regulating mechanism of the scrotum in man occurs in two surgical conditions, namely, delayed or incomplete testicular descent, and varicoceles or hydroceles. The problem of delayed descent in relation to underdeveloped testicles will be discussed later with azoospermia, but one obvious difference between the undescended and the scrotal testicle is that of temperature. Badenoch (1945), using a simple mercury bulb thermometer, recorded an average difference of 2·2° C. between the peritoneal cavity and the scrotal sac. Experimental work being carried out at the moment is not suitable for inclusion in a surgical lecture other than to say that, using a needle thermo-couple, I have recorded a difference of as much as 4·5° C. between a testicle held within the external ring and its normally descended fellow.

Minor degrees of maldescent are relatively common and although we have only been aware of the importance of this factor for a short time, we have seen several cases in the last year, where the testicles have been held high up in the scrotum by congenitally short cords. Even when these men are examined emerging from a hot bath, both halves of the scrotum may be seen hanging below the testicles as two empty folds, while the taut, short cords are easily palpable under such conditions. I have explored several of these cases, and on occasion have been able to lengthen the cord by dissection of the fibrous bands which are often present. The most clear-cut example of this phenomenon is shown in Fig. 1. After simple division of the fibrous strand the right testicle lay in the bottom of the scrotum, and within four months the sperm count had risen from one to 14 millions per c.c., without any other form of treatment. Unfortunately this was the only testicle present.

One of the main functions of the scrotum would appear to be the regulation of testicular temperature. The cremasteric and dartos muscles are capable of very marked and rapid contraction or relaxation, by which means they can alter the position of the testicles, thereby maintaining their temperature below that of the body generally. Anything which interferes with the heat loss from the scrotum results in elongation of the muscle fibres. A *varicocele* acts in this way and accounts for the long flabby scrotum and low testicles found in this condition. Whereas there may be a difference of up to 4° C. between the intrascrotal temperature and the rectal temperature in the normal male, I have recorded a difference of as little as 0·2° C. in a large varicocele, even with the testicle hanging four inches below the symphysis.

The pain of a varicocele is due to the weight of the testicle pulling on the cord, which was never designed as a suspensory apparatus. The older varicocele operations performed for this dragging pain, were often

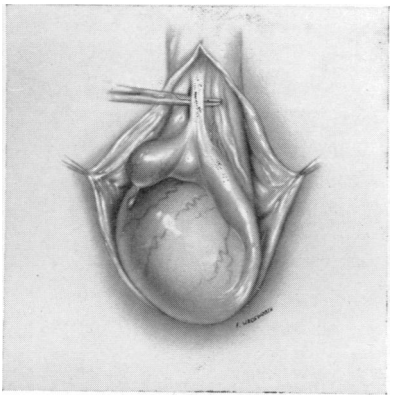

Fig. 1. Testicle retained high in scrotum by a fibrous band.

combined with a shortening of the pampiniform plexus so as to raise the testicle up in the scrotum. In actual practice this is quite unnecessary, since when the varicosities are removed, the dartos and cremaster muscles will contract and take the weight of the testicle again normally.

In the same way a *hydrocele* will also prevent loss of heat from the scrotum. An important feature about hydroceles is that small ones are extremely difficult to detect since they may not transilluminate, but even 5 to 6 c.c. of fluid are enough to raise the temperature by 2° C.

Minor degrees of these two surgical conditions are relatively common in normal young men and usually have no demonstrable effect on their fertility. On the other hand there is convincing experimental and clinical evidence in *some* men, that a varicocele or hydrocele can cause severe depression of spermatogenesis (Davidson, 1954*a*; Russell, 1954; Tulloch, 1952) and that this can be improved or cured by adequate surgical treatment of the varicocele or hydrocele (Davidson, 1954*b*).

Much valuable work on the blood supply of the testicle, etc., has recently been published by Harrison (1949) and Macmillan (1953, 1954) who pointed out that in the inguinal portion of the cord, the arteries are still large channels, easily seen and easily avoidable, so that resection of the pampiniform plexus should be theoretically safer if performed through an inguinal than a scrotal incision. Indeed, the classical operation for varicoceles consists of resecting a third, or other fraction, of the thickness of the pampiniform plexus *via* an inguinal incision. In practice, however, the varicosities round the epididymis (which are the ones of importance for our present purposes) cannot be dealt with except by individual dissection and resection, and are quite unaffected by tying off or excising a portion of the pampiniform plexus above. In fact, high ligation appears to favour hydrocele formation—the last thing we want.

In the great majority of the large varicoceles I have excised through a scrotal approach—the offending veins have been accessory to the main pampiniform plexus, the sheath of which has not even been opened in most cases.

The temperature changes which occur, and the detailed clinical results obtained following the excision of varicoceles in a large series of subfertile men, are in the process of publication elsewhere, but a few general remarks can be made here.

The sperm counts in men with subfertility due to a failure of temperature regulation are fairly typical. There is normally, though not always, an oligozoospermia well below 20 millions per c.c., while Davidson (1954*b*) describes "an increased semen volume, impaired morphology and motility and an excess of desquamated precursors of spermatozoa." If this picture is found in a physically normal man with reasonably developed testicles, whose only other clinical finding is a varicocele or hydrocele, one can state that he will certainly be no worse following excision of his varicocele while he may well be rendered fertile. In actual fact where these standards have been adhered to, operation has improved the counts in 70 to 80 per cent. of the cases and has resulted in many pregnancies in previously sterile couples. It should be clearly understood that deficiencies of testicular temperature regulation form only one small facet of the problem of oligozoospermia, while the excision of a varicocele or hydrocele is regarded by some as largely empirical treatment. However, the really dramatic improvement which frequently follows such a procedure makes further research essential, and amply justifies the performance of the operation in selected cases.

Causes of azoospermia

Complete absence of spermatozoa in the ejaculate can be due to two things. Bilateral failure of spermatogenesis or some bilateral abnormality of the conducting system.

BILATERAL ABSENCE OF SPERMATOGENESIS

If sperms are not being produced by the testicle, no known form of surgery will alter the situation.

I have no experience of azoospermia due to clinically obvious endocrine disturbances since these patients are generally under the care of the endocrinologists, and all of the men in this review showed a good average physique with normally developed secondary sexual characteristics.

When a testicle felt firm and of moderate size, it was always capable of forming at least some spermatozoa. This is contrary to some reports, but was the finding in this series, where the only biopsy reports showing complete absence of spermatogenesis came from clinically small, soft atrophic testicles.

Maldescent in relation to atrophic or underdeveloped testicles

The causes of testicular atrophy may be congenital or acquired, but it is difficult to decide whether to call failure of normal testicular descent, a congenital anomaly or an endocrine phenomenon.

A relatively normal endocrine balance is required to produce the normal secondary changes at puberty, but there is no doubt that unless a testicle descends well before this time, it will not produce spermatozoa. Failure to descend is unlikely to be a hormonal effect, because the opposite testicle if it descends, will develop normally, and it is unlikely that if the correct hormones are in circulation they will only influence one testicle. Occasionally one sees men who are normally developed in every way except for two small atrophic testicles. This could be due to hormonal deficiency, but the commonest cause of bilateral atrophy or poor development in this series appeared to be maldescent (Table I, p. 183). Seven of the 15 had undergone a bilateral orchiopexy, while two of them had testicles just protruding from the external ring. Of the remainder, three showed old gross inflammatory changes, two had associated absence of the vasa or epididymis and one was due to mumps.

Unilateral atrophy was encountered 14 times in the azoospermic group, and the pathology in the opposite testicle which accounted for the complete sterility is shown in Table II, p. 183. It is interesting to note that acute inflammatory reactions, either gonorrhoeal or non-specific, accounted for bilateral testicular atrophy in three men, and for unilateral atrophy in 11 men, being relatively more dangerous than mumps.

Unilateral ectopia was encountered 13 times in the 148 cases of azoospermia. All of these ectopic testes were very small if not atrophic, while a majority of them showed some congenital anomaly.

Two men clearly remembered the so-called normal testicle coming down, while three others were told by their parents that neither testicle was down in the scrotum when they were small boys. It is interesting to record that all five of these testicles which were late in descending, but which were now reasonably normal, showed some congenital anomaly at operation which accounted for the azoospermia. In fact an undescended testicle is so frequently maldeveloped that one cannot help suspecting that its failure to descend is due to its being abnormal.

In the oligozoospermia group there were seven men with very small testicles and sperm counts persistently under 5 millions, who could well remember that up to the time that pubic hairs began to grow, their testicles were not permanently down in the scrotum, but tended to disappear up into the inguinal canal for varying periods of time.

I have been able to trace 21 married men who had undergone bilateral orchiopexy at about 10 to 12 years of age. None of them has produced any children, while five of the six who were fully examined were azoospermic. The sixth man had a bilateral orchiopexy combined with

herniorrophy at the earlier age of six. He is the only one I have seen with reasonably normal testicles, but even he has a subfertile count of 12 millions per c.c., and I believe that if any operation for a retained testicle is performed at, or only a few years before puberty, it will give purely cosmetic results.

Mumps orchitis

Out of a total of nearly 500 men examined, bilateral testicular atrophy due to mumps occurred only once and unilateral atrophy 12 times. The atrophy in mumps is thought to be due to intra-testicular tension for which incision into the tunica albuginia was recommended by Wesselhoeft and Vose (1942) but Charny and Meranze (1948) from their pathological investigations did not consider that pressure was to blame. Much more research is required into this problem, if only to discover why, every so often, a mumps orchitis is *not* followed by atrophy. From the treatment point of view, incision or multiple puncture of the tunica as recommended by Kenneth Walker (1948) should be seriously considered since immediate clinical relief is afforded and at least no harm appears to be done.

An orchitis followed by atrophy clinically similar to mumps, may develop without any parotid swelling, while certain cases of inexplicable orchitis occurring in infants, unassociated with parotid swelling, may also be due to mumps. I have observed three such cases which progressed to atrophy. Two of them were explored in the hope of finding a torsion, but the epididymis was not involved in any way, and would fit into this category.

Connolly (1953) has reported three well documented examples supported by serological tests, so that it would appear unwise at the present time to assert too dogmatically that mumps before puberty can have no effect upon fertility.

Having said this, it must be emphasised that *torsion of the testicle* in small children is relatively common and is very difficult to diagnose, so that from the subfertility aspect it would appear safer to explore the scrotum in all cases of doubt. Most unfortunately, the attitude that there is still one on the other side prevails in many quarters.

OBSTRUCTIVE AZOOSPERMIA

The commonest cause of azoospermia in this series was some form of obstruction in the conducting system.

Vesicles and ejaculatory ducts

Congenital lesions of the seminal vesicles and ejaculatory ducts are probably much more common than realised, particularly since they are amongst the most difficult of all urological anomalies to detect. Even to the experienced posterior urethroscopist, catheterisation of the ejaculatory ducts is a delicate manoeuvre, where success in achieving a

satisfactory X-ray record is largely governed by luck. The vesicular anomalies which can occur (Dockerty, 1951 ; Pereira, 1953 ; Rolnick, 1954) include absence, fusion, re-duplication, cyst formation and fusion with the lower end of the corresponding ureter. The majority of recorded examples are from post-mortem reports and although all these interesting deformities are said to have been found by urologists during subfertility investigations few of them appear to have been recorded in the literature.

In the absence of both vesicles there would probably be no ejaculate volume. This series contains one such case with proven absent vasa, and I suspect absent vesicles. Absence of only one vesicle would be very difficult to prove, since any critical urologist would agree that inability to demonstrate it by endoscopy means nothing. However, in general, if the ejaculate volume is normal (2·5 c.c. or over) the vesicles and ejaculatory ducts are most unlikely to be responsible for an azoospermia, and although it may occur, we have not yet seen a case of total azoospermia due to blockage in the prostato-vesicular area which had a normal ejaculate volume.

Apart from the man with bilateral absence of vasa and vesicles referred to above, my experience of congenital lesions in this area is confined to a left ureter opening into the common ejaculatory duct ; a left ureter opening onto the trigone but also continuing down into the ejaculatory

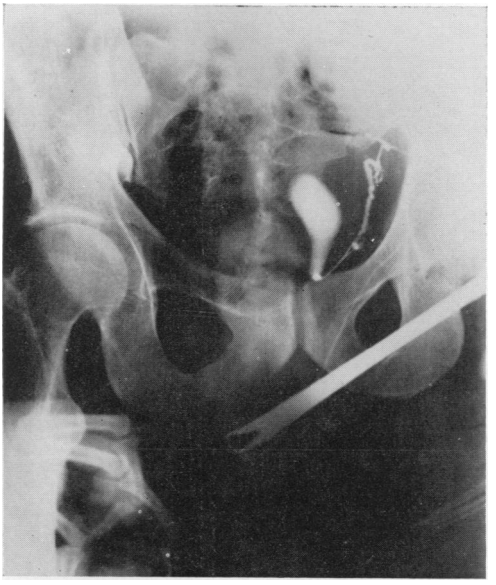

Fig. 2. Descending vesiculogram showing large cyst of left seminal vesicle.

duct, and a large golf ball size cyst of the left vesicle, which could be emptied into the urethra by palpation, but which could not be demonstrated radiologically from below after repeated attempts (Fig. 2). None of these cases, however, was associated with azoospermia.

In the same way, excluding tuberculosis, inflammatory lesions in the prostato-vesicular region, though accounting for pyo- and haemospermia, premature ejaculation or painful ejaculation, rarely seem to cause obstruction and azoospermia.

One case of azoospermia followed a diathermy loop resection of the bladder neck, but with this exception, all 14 men with azoospermia due to obstruction in the ejaculatory duct area were tuberculous, and all of them had a greatly reduced ejaculate volume.

Seminal Tuberculosis is much more common than we realise, and can remain active for 15 years or more without producing any symptoms and without affecting fertility even though the ejaculate is full of pus cells and tubercle bacilli (Hanley, 1954). Involvement of the vesicles may produce an early reduction in ejaculate volume, but reduction of the sperm count is a late development, and in some cases which have been followed over several years it would appear that azoospermia was finally due to the absence of any ejaculate fluid, rather than the sperms. In one particular case proven fertility was retained until the ejaculate volume was reduced to practically nothing. By the time azoospermia occurs the diagnosis is usually obvious on rectal examination.

Treatment, so far as fertility is concerned, is obviously conservative but promises to be much more hopeful in the future. The semen can now be rendered culture negative for tubercle bacilli with antibiotic treatment, while quite recently an improvement in ejaculate volume has been noted in two men with advanced prostato-vesiculitis undergoing a sanatorium and antibiotic régime.

It will be seen therefore that, excluding the tuberculous cases, this series contains only one obstruction of the ejaculatory ducts, and we have now come to expect a lesion in the region of the scrotum in any azoospermic patient who has normally developed testicles and who has a good ejaculate volume.

Vasa deferentia and epididymes

The commonest sites of obstructive azoospermia are at the testicular end of the conducting system, but although the literature, especially in America and France (Bayle, 1952) quotes gonorrhoea as heading the list of causes, this is not the experience in this country to-day, where the occurrence of epididymoorchitis, particularly bilateral, is an extremely rare complication of gonorrhoea, and has been for some time past (Harkness, 1955 ; Mascall, 1955).

LESIONS OF THE VAS

Trauma

Traumatic division of the vas can be accidental or deliberate. This series contains three accidental injuries which occurred presumably at the time of a hernia operation in childhood. According to one history a bilateral hernia operation was performed at the age of three when one retained testicle was removed. On examination the remaining testicle was well developed, but on exploring the inguinal canal, the vas became a thin fibrous thread in the region of the external ring. The fibrous tissue at this site was very rigid, and it was impossible to trace any further extension of the vas as far as the internal ring. In the other two men the vas on both sides was merely a solid cord between the external and internal inguinal rings. Reparative surgery was impossible in these three cases. It should be noted that these three men had bilateral accidents resulting in azoospermia, and one cannot help speculating as to how many unilateral injuries occur and escape unnoticed.

Deliberate section of the vas

Five cases of deliberate section of the vas carried out on German or Polish Jews during the war have been encountered. In three instances the vas would seem to have been isolated at the neck of the scrotum and divided. There was very little fibrous tissue resection, and after cutting across the stenosed ends of the vas, sperms were detectable after compression of the tail of the epididymis. Anastomosis was established in the simplest possible way over a nylon splint, using 000 plain catgut. The splint was left *in situ* for nine days in one case, five days in another and withdrawn in 24 hours in the third, a successful anastomosis resulting in all three cases. In a fourth man a large piece of vas had obviously been resected, so that apposition was very difficult. Fixation broke down on the right side, but the left side remained intact, and a successful anastomosis was achieved. Three patients, who were unmarried, returned to Israel, but all had sperms in the ejaculate within eight weeks of operation. The fourth man's wife was delivered of a healthy child within two years.

The fifth patient has so far been a failure. In his case, the original operation was performed in Germany before the war because of a family history of insanity. He was twelve at the time, and remembers very little of the affair. However, both scrotal incisions were very low down and one can only conclude that a clamp had been applied to the lower end of both epididymes. This necessitated re-implanting the vasa into the mid-zone of the epididymis. No sperms have appeared after nine months.

Deliberate ligation or section of the vas is sometimes carried out to prevent a tuberculous infection in the remaining epididymis after removal of its fellow. I do not wish to discuss this problem in detail here, but the fact that reconstitution is not only possible but likely, should be more widely known. I have repaired one such vas, with a resultant

pregnancy which must have started within a few days of the man's return home. This may be an example of the wish being father to the child rather than the thought, but it is interesting.

Richard Handley reported a case in the *Archives* of the Middlesex Hospital in 1951, where there was a three-quarter inch gap filled with fibrous tissue between the cut ends. Approximation was achieved with fine nylon but no internal splint was used. This patient's wife became pregnant within four months of operation. Although only one side was repaired the sperm count eventually rose to 80 millions per c.c.

In spite of the encouraging reports by O'Connor (1948) and Humphreys (1953), the good prognosis to be expected after surgical repair in these cases is not generally appreciated. The vas possesses a remarkable tendency to recanalise if given a reasonable chance, and it would seem that if the cut ends are approximated without tension, it matters little what technique or suture materials are used, or whether a splint is left *in situ* or not.

Inflammatory lesions of vas

Inflammatory strictures in the scrotal portion of the vas have been encountered several times. They have always been multiple with areas of patent lumen separating them, and there has always been a history of a severe epididymitis. Such strictures made epididymo-vasostomy impossible on five occasions.

One particular case is of interest. The right testicle was atrophic following a severe gonococcal epididymo-orchitis, but over a 10-year period there had been one miscarriage and one baby which died soon after birth. A further gonococcal infection then resulted in azoospermia of four years' duration, although there was no definite history of an epididymitis. The left testicle was explored and the vas was found to be so convoluted and kinked that opaque medium could not be injected towards the vesicle. Sperms however were present in the testicular end. Careful sharp dissection enabled the vas to be more or less straightened out, after which it was possible to inject saline with ease. A nylon splint was left *in situ* for 10 days and the man's wife became pregnant within two months of the operation.

Congenital lesions of vas

During quite recent years, an increasing number of congenital obstructive lesions have been recorded in the literature, and it is obvious that these cases will mount in number and importance as more interest is taken in the problem of male subfertility (Nelson, 1951 ; Young, 1951 ; Walker and Boyd, 1951 ; Merren and Kelley, 1952 ; O'Connor, 1953).

The " normal " diameter of the lumen must vary from one individual to another as in all anatomical structures, but usually it will admit one number 8 strand of nylon suture with ease. Unfortunately it is not

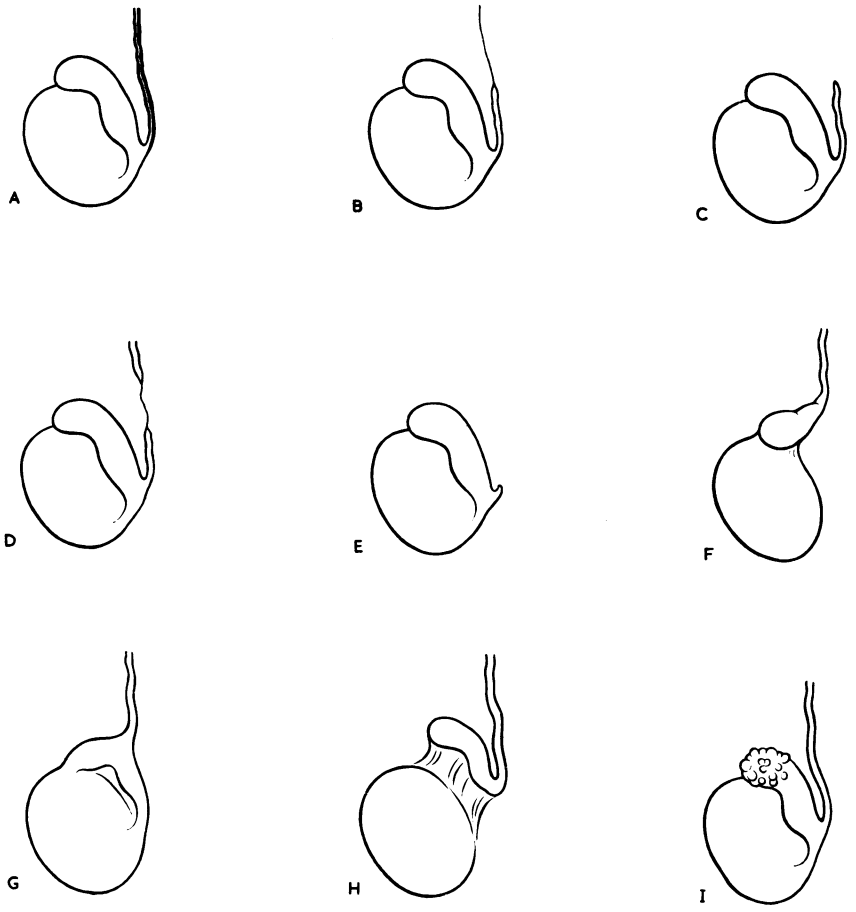

Fig. 3. Congenital variations in vasa deferentia and epididymes.

uncommon in azoospermic cases to find that, regardless of the external appearance, the lumen may be markedly reduced or even obliterated, while the whole vas may vary in diameter from the average down to a mere fibrous strand, or may be absent altogether (Fig. 3A, B, C). Walker and Boyd (1951) consider that these phenomena are due to " disappearance as the result of some previous disease," rather than to congenital maldevelopment, but I cannot subscribe to this view since degrees of defect in the vas range from complete absence, to the various anomalies shown in Fig. 3, which are so obviously congenital anatomical variants.

Complete agenesis of the vas on both sides is reputed to be very rare, and Weyeneth (1954) could only find ten cases in the literature. However, it is not such a rare finding in practice, and there are nine examples in this present series.

It is clear that epididymo-vasostomy is impossible in the absence of a vas (Fig. 3C nine instances) or where it is merely a fibrous cord (Fig. 3B 11 instances) but the vas may have a relatively normal external appearance and yet possess an extremely narrow lumen, or in fact no lumen at all, and this latter condition has been met with on four occasions (Fig. 3A).

The situation depicted in Figure 3D, where an apparently normal vas simply failed to unite with the epididymis was present on three occasions, while other anomalies are shown in Figures 4 and 5.

A short stump of vas arising from the epididymal tail, Figure 3E, was found to contain active spermatozoa in four patients, and a similar case to this was described by Young (1951) where he attempted to make an artificial spermatocele using a small cellophane envelope.

A method of treating these cases has yet to be devised. I have been encouraged by the fact that it was sometimes possible to recover spermatozoa for a period of several days when the vas was attached to the skin surface following vasostomy and prostatectomy. O'Connor (1953) reports three attempts to form a skin pocket from which sperms could be collected, but my own attempts to form a permanent fistula or a subcutaneous artificial cyst using ammion, must be described, to quote the late Professor G. Grey Turner, as " instructive failures."

Lesions of the epididymis

Although embryologically the same structure as the vas, the epididymis will be discussed separately since the surgical problems are different. This present series contains 71 azoospermic men where it was considered that the fault lay in the region of the epididymis. That is to say, in spite of adequate spermatogenesis in the testicle itself no sperms were entering the vas deferens.

The great majority of reported successes after empididymo-vasostomy in France and America have been in gonococcal cases (Bayle, 1952) where the inflammatory obstruction starts at the vaso-epididymal junction and progresses along the epididymal tubules towards the testicle. In such cases the vas is usually patent, and although the lower epididymal pole may be nodular, the head of the epididymis contains living sperms, so that anastomosis is a practical procedure.

Epididymo-vasostomy

Before proceeding further I will discuss this operation of epididymo-vasostomy which has assumed Hagner's name since his published results in 1931. In principle, a minute eliptical opening in the vas is anastomosed to a similar opening in the epididymis, at a site where live sperms have been recovered. Hagner used various suture materials, but only achieved success with silver wire. While I fully agree with Hagner that non-irritating sutures are essential in such a situation, I have found that these fine metal wires are really too frustrating for use, and that the

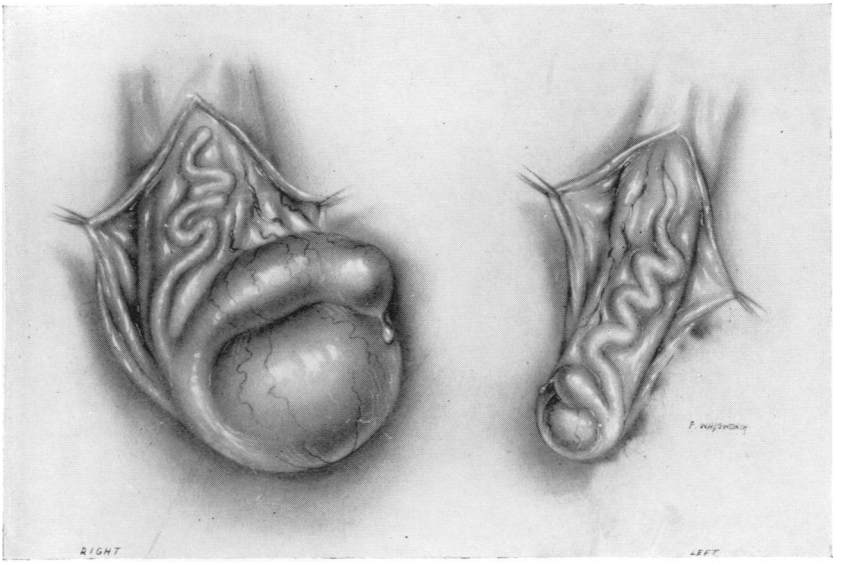

Fig. 4. Operative drawing showing a blind ending stump of vas high up in the cord on the right side. Living sperms in epididymal head. Ectopic left testicle with similar anomaly of vas except that instead of ending abruptly it continued as a thin fibrous strand.

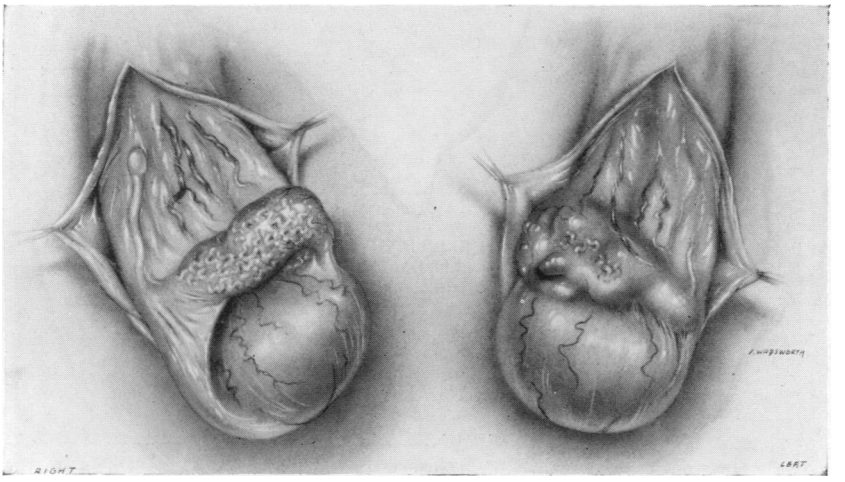

Fig. 5. Operative drawing showing distended epididymal tubules and a small thick walled cyst at termination of vas filled with actively motile spermatozoa. On the left the vas is represented by a thin fibrous strand running upwards, while the epididymis was distorted with multiple cysts. No sperms were detected in the cysts and it is probable that there was a failure of union at the vasa efferentia.

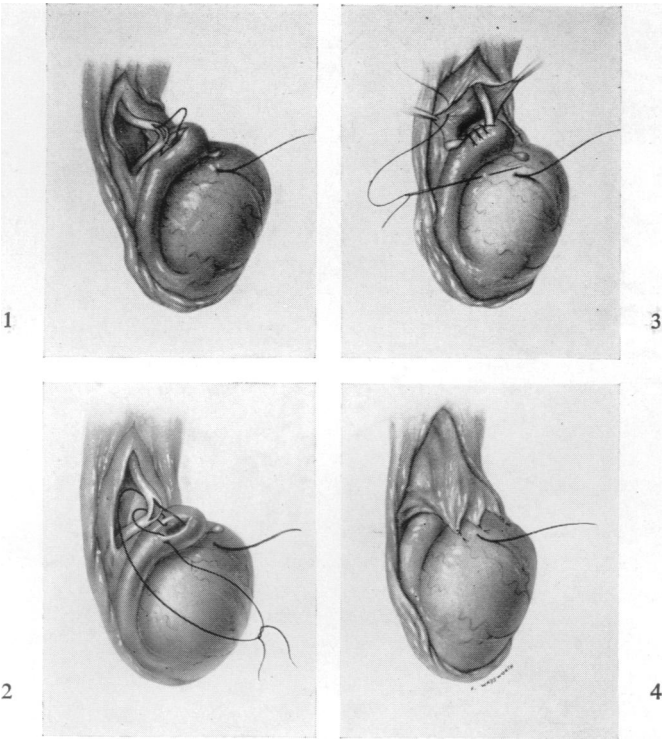

Fig. 6. Author's modified epididymo-vasostomy operation. No sutures are used at the actual stoma.

formation of an anastomosis even loosely resembling the artist's drawings is an utopian ideal rather than an achievement in my hands. I have therefore devised a modification of this operation, whereby sutures of any kind are unnecessary at the actual site of anastomosis.

Film

(Operative details were demonstrated with a colour film).

Figure 6 is self explanatory. The splint consists of number 8 nylon which is passed down to the vas for four or five inches. The other end is anchored to the scrotal skin by a button. 3/0 plain catgut sutures are used to bury the stoma.

Once again I must stress the fact that the majority of successes following epididymo-vasostomy in the literature, have been in cases of gonococcal epididymitis, but unfortunately I have seen very few of these. I say unfortunately because in such cases the prognosis is relatively good.

Out of 148 azoospermic men in this series only 15 gave any real or even presumptive evidence of old gonococcal, or other inflammatory infection of the epididymis. Anastomosis was impossible in three of these owing to strictures of the vas, and in three others owing to testicular atrophy. Of the remaining nine men who underwent epididymo-vasostomy, four pregnancies have followed operation, while sperms have appeared in the ejaculate in six men. In other words a permanent anastomosis was established in 66 per cent. cases. This compares very favourably with results reported by other workers.

Congenital anomalies of the epididymis

We are now left with a group of azoospermic men where it was decided that the fault, though confined to the scrotal area, was not due to any traumatic or inflammatory lesion in the epididymis. While some mistakes may have occurred in this over simple classification, the operative findings enabled these men to be put into two clearly defined groups: 1. Those with obvious congenital anomalies, and 2. those with relatively normal anatomy or concealed congenital anomalies.

1. Obvious visible congenital anomalies

The size and contour of the epididymis and its method of attachment to the body varies slightly from one patient to another, so that it is difficult to decide when a variation of the normal becomes a definite congenital anomaly, but quite obvious structural alterations were found in 64 testicles in 148 men in this review.

The most important attachment between epididymis and body contains the vasa efferentia as shown in Figure 3F. An attachment of this sort is frequently seen in undescended or abdominal testicles in small children, and the subsequent folding and creation of a globus major and minor would appear to be a later stage in development.

Numerous testicles were found to have variations of attachment of this type while in the most extreme examples (four cases) the epididymis failed to unite with the body at all, being attached merely by a thin transparent vascular " mesentry " (Fig. 3H, and Fig. 7).

Badenoch (1946) reviewed the subject of complete failure of union between epididymis and testis and found three examples in 42 undescended testicles. He thought that this condition only occurred in undescended testicles, but the four examples recorded above were normally descended organs, although two of them were late in coming down.

The peculiar anomaly shown in Figure 3G, has been met with once. The vas merged into normal looking epididymal tissue which then bifurcated and appeared to be firmly attached to the upper and lower testicular poles. No sperms were detected anywhere in this epididymis.

So far, therefore, we have seen a series of congenital anomalies of the vas and epididymis ranging from the normal down to atrophy, or complete absence of union of any, or several portions, of the conducting system.

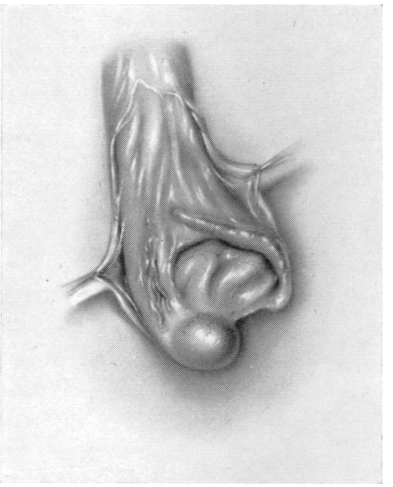

Fig. 7. Failure of union between epididymal head and body of testicle. Not infrequently seen in undescended testicles.

If such gross macroscopical defects can occur, it would not be surprising if less obvious, even microscopical lesions could also be present, and it has been assumed that such was the case in the largest group of testicles in the series.

2. Relatively normal anatomy or concealed congenital anomalies

In general these 139 testicles appeared to have a relatively normal anatomy except that the epididymes were sometimes smaller than usual. The vasa were always patent and the biopsies, when taken always showed spermatogenesis, and yet there was some mechanical obstruction or physiological failure of conduction between the testicle and the vas.

The importance of this group is obvious, not only on account of its size, but because so far its existence as a clinical entity has not been appreciated.

The obstruction or conduction failure appears to vary in site from the tail of the epididymis up to the vasa efferentia, but two broad clinical groups emerge.

A. Those with spermatozoa in the epididymis, and B. those without spermatozoa in the epididymis. An attempt at such a distinction must be made, because, if spermatozoa enter the head of the epididymis, a short circuit anastomosis should be theoretically possible, but if they do not, such an operation is unscientific and a waste of time.

Group A. Cases with sperms in the head of the epididymis

In this group it was not uncommon to see distended tubules underneath the glistening capsule of the epididymal head, filled with yellow fluid containing millions of spermatozoa, while the body and tail would be small and collapsed.

Although it is impossible to be absolutely certain that the obstruction was not inflammatory it has been considered to be congenital because there was no history of any previous genito-urinary infection, nor was there any clinical sign of infection, adhesions or hydrocele at the time of the operation. In support of this is the fact that although such a condition was bilateral in eight men and was the only pathology detected, it was associated in the remaining cases with some other obvious congenital anomaly on the opposite side. In addition many testicles in this group showed other unmistakable evidence of structural defects. In the head of a normal epididymis one may occasionally see minute cysts about 2 - 3 mm., in diameter, but in some of these azoospermic cases, the whole head has been replaced by multiple small cysts which fill but may not distort or enlarge the outline. No sperms could be obtained from the tail of the epididymis in such cases, though some of the cysts were full of them (Fig. 3I and Fig. 5).

A variant of these small multiple cysts was one large cyst, filling, but not enlarging or distorting the epididymal head, and which continued to provide millions of spermatozoa on aspiration for five months.

It should be noted that these cysts, which do not distort the epididymal outline and are seldom palpable clinically before exploration, are quite distinct from the larger discrete cysts which we often feel, and sometimes remove. The embryology is doubtless the same, but the latter do not necessarily affect fertility. They have been fully described by Badenoch (1946) and Ogier Ward (1922).

Before leaving this group it must be admitted that the presence of an obstruction in the epididymis has been assumed when there is an azoospermic ejaculate associated with living sperms in some portion of the epididymis. But since we still do not know where the bulk of the sperms are stored prior to ejaculation, it could well be that in the type of case just described, there might be a free passage for the sperms from the testicle to the posterior urethra, but that the muscular mechanism for ejecting them is deficient.

However, an epididymo-vasostomy was performed in 83 out of these 89 testicles, but in only four men were sperms ever seen in the post operative ejaculate, although one pregnancy has resulted five years after the operation.

Group B. Cases without sperms in the head of the epididymis

I am assuming that cyst formation at the junction of epididymis and testis represents a congenital failure of union at this site rather than an inflammatory or degenerative change. This failure of union can also probably occur without cyst formation.

If all of the vasa efferentia are affected no sperms will enter the epididymis. Fifty such testicles have been placed in Group B, but this group may be too large since some epididymes may have contained sperms which one did not detect. When the epididymis is incised in these cases the tubules appear dry and lack the yellow fluid which generally oozes out of them, but although biopsy specimens are not grossly abnormal, they contain no spermatozoa. On other occasions the head appears to be filled with fatty tissue, in which case sections show very few if any tubules.

I have now seen three epididymes, which on superficial examination appeared to be normally attached to the body of the testis in the region of the vasa efferentia, but which on closer inspection showed that, apart from the capsular attachments, the actual epididymal tissue did not appear to make contact with the tunica albuginia (Fig. 8). The difference between Figure 8 and Figure 9 is obviously only one of degree.

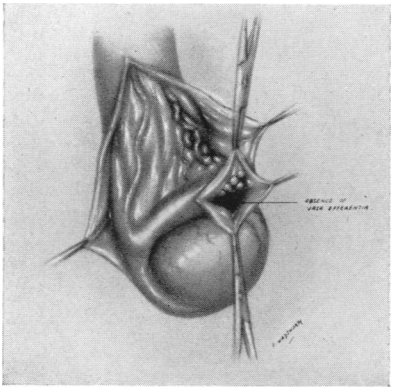

Fig. 8. Congenital absence of union at site of vasa efferentia.

Discussion

If we now consider the results of surgical exploration of the epididymis in this series, we find nine old inflammatory cases with a good prognosis, and 25 men with such gross congenital anomalies that epididymovasostomy was impossible. The remaining cases all had patent vasa, but were divided into groups according to whether or not spermatozoa could be found in the tubules of the epididymal head.

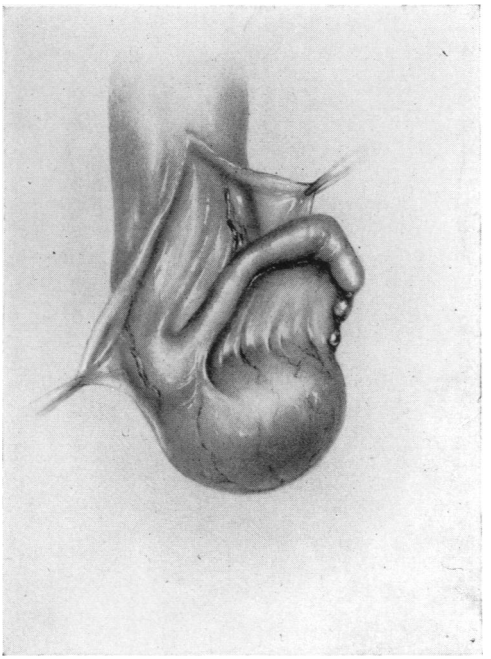

Fig. 9. Failure of union between epididymis and testicle.

Eighty-three epididymo-vasostomy operations were performed on 89 testicles in the group with sperms detectable in the head of the epididymis, with one resultant pregnancy. Only four other men subsequently showed an occasional sperm on ejaculation. These four at least had some continuity of the conducting system.

In the second group where no sperms were recovered from the head, epididymo-vasostomy was performed on 48 testicles but without success. Nine anastomoses were made to the region of the vasa efferentia themselves, but were still unsuccessful.

In a majority of the testicles with sperms in the epididymal head there was no proof that an actual mechanical obstruction existed, while experimental work in radiology of the epididymes which we have quite recently started, forces one to the conclusion that an unobstructed channel from the vasa efferentia to the ejaculatory ducts, may be of no value if the propulsive mechanism which forces sperms along the channel is deficient.

It is therefore necessary at this stage to discuss the theories of sperm conduction.

Sperm Conduction

It is now accepted by most authorities that the sperms are not normally stored in the seminal vesicles, which are hollow structures only in man and anthropoid apes, being solid glanduli seminales in other mammals.

However, it is difficult to explain why opaque medium injected down the vas fills up the vesicles so well if the sperms are not supposed to reside in the vesicles. It could well be that the sperms only enter the vesicles in any numbers just before ejaculation. It is also unlikely that the sperms are normally stored in the ampulla, because it is impossible to express any great numbers of spermatozoa from either the vesicles or ampulla by rectal massage, even under anaesthesia. On the other hand the epididymal tubules are twenty feet long and could easily contain the requisite numbers.

However, in spite of the fact that spermatozoa are highly motile on ejaculation, they are non-motile at their site of origin in the testicle, and it is not known how they traverse the twenty-odd feet of the epididymal tubules and the vas.

Rolnick (1954) in a recent paper on the seminal ducts asserts that spermatozoa are non-motile in the head of the epididymis, possibly motile in the tail, non-motile in the vas, but actively motile in the ampulla and the seminal vesicles.

I can only state that these have not always been our findings. When there have been visibly distended yellow tubules in the head, the sperms have often been motile, but were usually non-motile when the tubules were collapsed. In all of the cases where the vas was repaired after previous deliberate section, live sperms have been recovered from the testicular end at operation. Even so, it is unlikely that the sperms could travel more than twenty feet along the ducts by their own motive power, and Rolnick thinks that they are forced down the vas by secretory pressure from above, aided by muscular contractions of the vas which are greatly increased during coitus.

I have no views on the secretory pressure theory, but feel that muscular contractions of the epididymis and vas must play a large part in the process.

Weisman (1941) found that most of the sperms in spermatocele fluid were immature, and considers that the epididymal secretions are necessary for the maturation of the sperms, which are therefore not fully mature until they have traversed the full length of the epididymis.

Rolnick asserts that sperms from spermatoceles, which are usually at the head of the epididymis, are smaller than normal and have only a short life on a microscope slide. He therefore considers that artificial insemination using these sperms is unscientific, and moreover, that the accepted method of short-circuiting obstructions in the tail of the epididymis by anastomosing the vas to the head of the epididymis has little merit, since the sperms will not have matured.

In spite of this I can only say that on five occasions I have performed insemination using sperms removed at operation from the distended tubules in the epididymal head, and in two of these women, living motile sperms were recovered from the cervical plug after 20 and 25 hours respectively. In other words an apparently normal post-coital test.

Of far greater importance, however, is the fact that all of the five epididymo-vasostomies, which have resulted in pregnancies, were made between the vas and the head of the epididymis, not as low down as possible, as recommended by Bayle (1952) and others. These sperms can only have traversed a few inches of epididymal tubules in the head before entering the vas, but the acid test of their morphology is their ability to fertilise the ovum, and produce a normal pregnancy.

I have devoted time to this problem because a choice of the various surgical procedures available in azoospermic cases depends upon the views we hold concerning the morphology of the semen in the epididymis and the physiology of the conducting mechanism. Personally I believe that in the presence of such conflicting data, we, as surgeons, should regard the sperms in the epididymal head as potentially capable of effecting fertilisation, and should continue with our efforts to overcome the anatomical and physiological obstructions which we encounter, until further knowledge proves that we are wasting our time.

Artificial Spermatoceles

Before closing I will refer to some purely experimental work which may be worthy of continued trial. This entails artificial insemination using sperms aspirated from artificially created spermatoceles in the epididymal head. Until quite recently I have only attempted such measures where the vas was obstructed or absent, but have now started to form cysts as well as perform an epididymo-vasostomy, in the hope of achieving a second chance. This is purely experimental work, but has been welcomed by patients who have nothing to lose, and who will go to almost any lengths rather than adopt or consider donor insemination.

Various techniques have been used but the most successful results have followed the insertion of a small loosely folded ball of ammion, $\frac{1}{4}$in. in diameter, under the capsule of the epididymis (Fig. 10).

Eleven such attempts have been made in the past nine months and so far a cyst from which sperms could be aspirated has resulted in six cases. One cyst has been aspirated on five occasions and each time it delivers up to 1 cc. of fluid laden with spermatozoa which remain actively motile in the cervical mucous for 24 hours.

The other cysts are not so large, and two of them do not fill up fast enough to perform an insemination at each ovulatory phase, while one of the earlier cysts to form has now dried up after repeated aspirations.

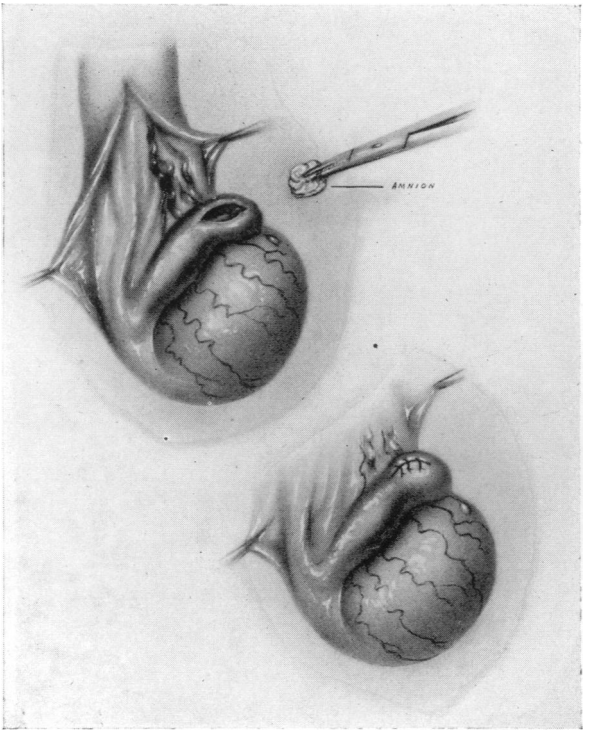

Fig. 10. Formation of artificial spermatocele by insertion of a loosely folded ball of ammion.

No pregnancy has yet resulted, but the experiments appear to be worth continuing, and I feel that the great John Hunter, in whose name this lecture is given, would not have abandoned the problem without further effort.

ACKNOWLEDGMENTS

I should like to thank my colleagues on the staff of St. Peter's and St. Paul's and St. Philip's Hospitals, for referring various patients to me; also Lt.-Gen. Sir Frederick Harris and Col. J. E. Snow for permission to include Service cases; and particularly Dr. H. A. Davidson. Not only did the majority of the patients come from Dr. Davidson, but he was responsible for my initial interest in this problem and has given me constant expert advice and encouragement.

I am most grateful to Miss Freda Wadsworth for her patience and skill with the operative drawings, and to Mr. E. Stride for his expert photography.

TABLE I.—FIFTEEN AZOOSPERMIC PATIENTS WITH BILATERAL TESTICULAR ATROPHY

7	Bilateral orchiopexy.
2	Testes at external ring.
3	Gross bilateral inflammatory changes.
2	With obvious congenital anomalies of vas.
1	Bilateral mumps.

TABLE II.—FOURTEEN PATIENTS WITH UNILATERAL TESTICULAR ATROPHY SHOWING PATHOLOGY ON OPPOSITE SIDE RESPONSIBLE FOR THE AZOOSPERMIA

G.D.	L	atrophy, high testicle	R	intra-abdominal testicle
M.W.	R	atrophy, high testicle	L	cystic epididymis
D.M.	R	atrophy, high testicle	L	removed for ectopia
A.D.	L	atrophy, late descent	R	congenital lesion of vas
P.J.W.	R	atrophy, gonorrhoea	L	G.C. epididymitis
D.B.	R	atrophy, trauma, herniorrhaphy	L	trauma to vas at herniorrhaphy
E.J.R.	R	atrophy, mumps	L	epididymitis non-specific
H.W.	R	atrophy, mumps	L	G.C. epididymitis
H.C.	R	atrophy, after epididyectomy	L	epididymitis non-specific
I.M.	L	atrophy, epididymitis non-specific	R	epididymitis non-specific
J.M.	R	atrophy, gonorrhoea	L	G.C. epididymitis
P.M.N.	R	atrophy, gonorrhoea	L	G.C. epididymitis
L.J.I.	L	atrophy, late descent	R	congenital absence of vas
A.C.	R	atrophy, late descent	L	T.B. epididymitis

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