

MICROSURGERY FOR VASECTOMY REVERSAL AND VASOEPIDIDYMOSTOMY

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ABSTRACT—Accurate microsurgical techniques developed by us have allowed a considerable improvement in success rate for vasectomy reversal, and a better understanding of the pathophysiology of obstruction. This has permitted us also to treat pathologic obstructive azoospermia more effectively.

Conventional techniques for reanastomosis of the vas have led to sperm leakage and granuloma, with poor alignment of the vas mucosa, causing total or partial obstruction (Figs. 1A, B, and 2).¹⁻¹⁰ These techniques have yielded a 30 to 70 per cent incidence of sperm in the ejaculate with 5 to 20 per cent of wives achieving pregnancy.⁹ Many of the cases in

these series were thought of as patent (because of the presence of sperm in the ejaculate) despite poor sperm counts with poor or no motility. The documentation of data in general has been weak. A few dead sperm per high-power field would be considered a sign of patency and technical success. It is no surprise that the pregnancy rate was low.

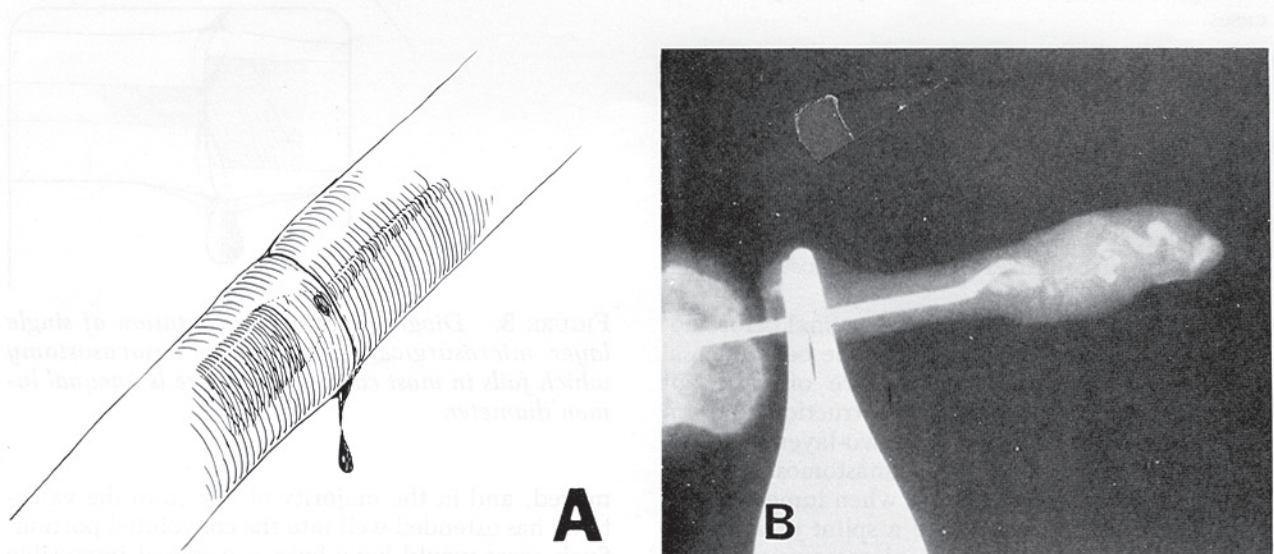


FIGURE 1. (A) Diagram demonstrates inaccurate approximation of mucosa which is achieved with most conventional methods of vas reanastomosis. Leaking of sperm fluid as well as muscle bridge which exists between two mucosal linings (rather than accurate mucosa-to-mucosa approximation) results in circular scar tissue which contracts and causes partial or complete blockage. (B) Vasogram of patient with 4,000,000 sperm/cc and poor motility after conventional vasovasostomy. This is anastomotic site. After this anastomotic site was resected and patient was reanastomosed with proper two-layer approach, sperm count went up to 80,000,000 sperm/cc with excellent motility, and his wife became pregnant.

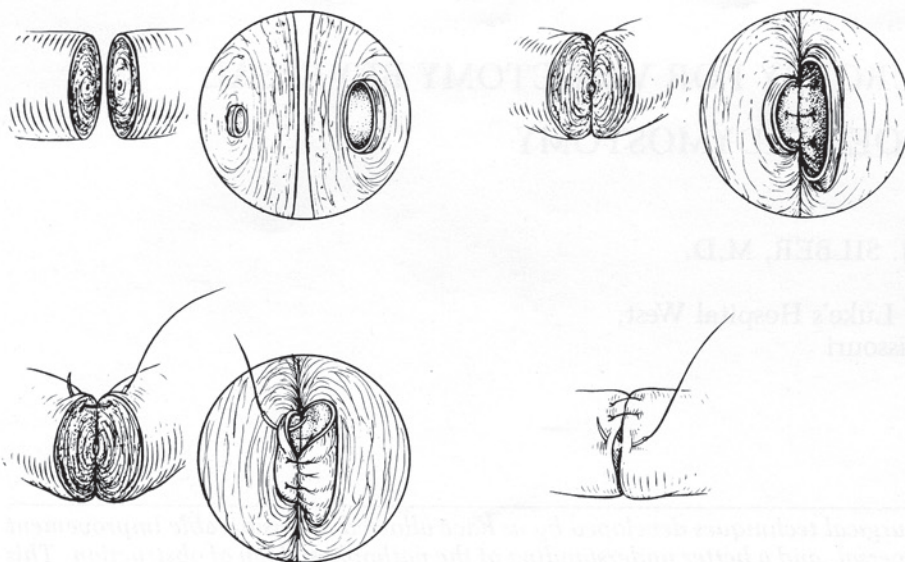


FIGURE 2. Diagram demonstrating two-layer microscopic reanastomosis of vas deferens originally described by me in 1975. It is particularly well suited for getting accurate mucosa-to-mucosa approximation in usual case where there is discrepancy in diameter of lumens.

An accurate microscopic technique for reconnecting the vas increases the fertility rate considerably and also clarifies what other factors may be affecting success. When one examines the anastomotic site after conventional vasovasostomy under the microscope, he ceases to wonder why success rates in the past were so low. Vasograms of failed vasovasostomy (as well as histologic sections) demonstrate strictures and blockage even though there may be some sperm present in the ejaculate (Fig. 1B). After reoperation with microsurgery, the sperm counts and motility of these patients improve dramatically in 90 per cent of cases.

Microsurgical approach to vasectomy reversal

I would like to discuss details and results of the microscopic technique which I originally described and have used in over 2,000 patients, and then elaborate what further steps may then be taken in patients who are failures after vasovasostomy (Fig. 2).¹¹⁻²²

It is advisable to practice in the animal before doing such surgery on humans. For the best mucosal approximation (when lumens are of different diameter because of chronic obstruction and increased pressure), a nonsplinted two-layer approach is advisable. With a one-layer anastomosis there is poorer mucosal approximation when lumen diameters differ (Fig. 3). Of course a splint of any kind should never be used, as it is only an excuse for not being certain one has obtained a good anastomosis. It results in sperm leakage, inflammation, and scarring.

It is unnecessary to determine preoperatively what type of vasectomy had been performed on the patient. We have learned to expect almost any kind of vasectomy. Often a large segment has been re-

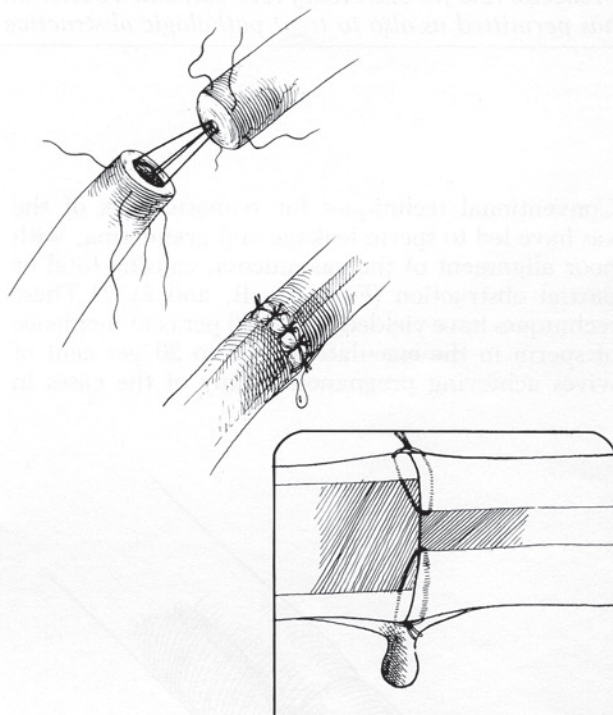


FIGURE 3. Diagrammatic representation of single layer microsurgical approach to vasovasostomy which fails in most cases where there is unequal lumen diameter.

moved, and in the majority of our cases the vasectomy has extended well into the convoluted portion. Such cases would have been considered impossible to approach with conventional techniques. With the techniques to be described, it means a little more dissection, but essentially no change from a standard routine.

The preparation of the two ends of the vas deferens for the microscopic anastomosis is best performed with $2.5 \times$ loupe magnification. The

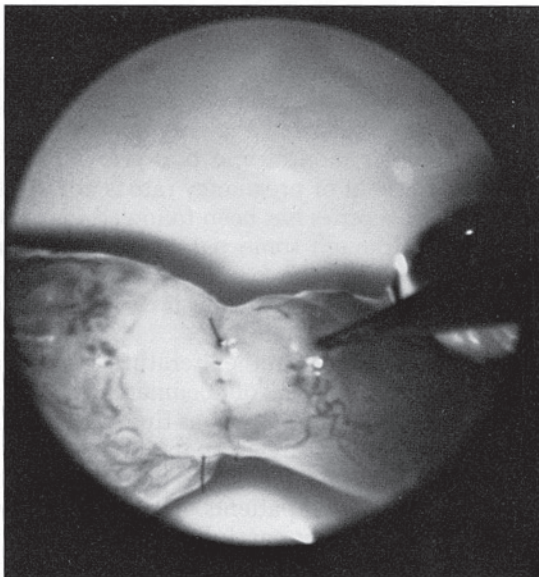


FIGURE 4. *Reviewing inside of anastomosis after first three posterior mucosa sutures have been placed one can verify accurate mucosa-to-mucosa approximation.*

healthy ends of the vas deferens above and below the fibrosis are freed up several centimeters (more than that if a large gap has to be bridged). The more one frees up the healthy vas deferens from surrounding attachments, the more easily the two ends will bridge any gap between them. It is critical to have a tension-free anastomosis. No effort at anastomosis should be made until the two ends above and below the obstruction have been adequately freed up. One generally need not fear devascularizing the vas deferens. The blood supply around the outer muscularis of the vas is extensive and we have found that freeing the vas a good distance will not injure the blood supply.

The beginner will immediately wonder why he cannot more easily perform the anastomosis with his loupes, avoiding the encumbrance of a microscope. Actually, the reason one uses a microscope is to make the operation easier and more accurate, not to make it more difficult. Loupes can at best provide 2.5 to 4 times magnification. To visualize adequately the inner lumen of the vas deferens for easy and accurate placement of stitches requires $16\times$ magnification. The advantage of the microscope, in addition to providing higher power magnification, is that the depth of focus is much deeper, light is constantly supplied directly to the subject, and the microscope is resting on a stand and is thus immobile. The surgeon can move his head or neck without in any way disturbing the steadiness of his view on the subject.

The fibrotic portion of the vas deferens is excised under the microscope until healthy lumen is reached. The testicular side lumen is noted to be dilated because of the chronic high pressure caused by

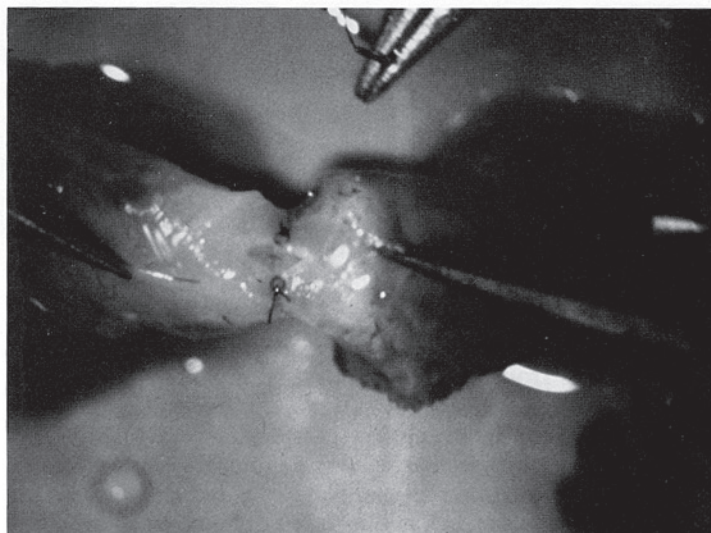


FIGURE 5. *Two ends of vas are retracted in preparation for placing three subsequent anterior mucosal sutures.*

obstruction. The abdominal side lumen will be tiny by comparison. A microcatheter is placed in the dilated end of the lumen on the testicular side of the vasectomy site allowing sperm fluid to enter by capillary action. This fluid is then examined under a laboratory microscope for the presence or absence, and characterization of sperm. Microscopic characterization of the sperm in the vas fluid is extremely accurate in predicting success or failure, and in alerting you to the likelihood of secondary epididymal blockage. Therefore, it is important to obtain enough fluid from the vas to be a representative sample of the sperm content of the entire length of the proximal vas. It is possible for a hasty smear to show no sperm even though a larger sample of fluid coming from more proximal in the vas may show many sperm.

Furthermore if the fluid is concentrated and a mediocre laboratory microscope is used to look at the specimen, it is common to mistake a smear that is in truth packed densely with nonmotile sperm, for debris. These mistakes have been made by well-meaning urologists and have led to some misleading reports in the literature. The motility of the sperm in the obstructed vas segment has no bearing on prognosis and when the sperm is non-motile and very densely packed, it can be mistaken for debris. Thus with nontransparent fluid, if there appears to be no sperm, the fluid should be diluted with saline and possibly stained with hematoxylin and eosin.

The inner mucosal anastomosis is performed under 16 to $25\times$ magnification. The object is to obtain an accurate mucosa-to-mucosa alignment despite the discrepancy in lumen diameter (Fig. 2).

The surgeon places the first mucosal suture anteriorly making sure that the suture includes the elastic layer directly next to the mucosa. By excluding as much muscularis as possible from this bite, a more precise mucosa-to-mucosa approximation can be obtained.

After the first three mucosal sutures are placed anteriorly in such a fashion, the entire vasovasostomy clamp is rotated around 180 degrees, and what was the posterior wall of the vas deferens is now visualized in the anterior position (Fig. 4).

At this point one can view the anterior row of sutures from the inside and inspect to see whether or not perfect mucosal alignment has been achieved (Fig. 5). If large bites of muscle were included in these mucosal sutures, the mucosal edges usually will not come together properly, and there will be a raw muscle bridge between the edges of mucosa (Fig. 3). This will subsequently scar down. There should be no tearing or inaccuracy in the line-up of the mucosal margins. The outer muscularis is then sutured separately.

Semen analysis is obtained every month for the first four months, then at eight months, one year, one and one-half years, and two years postoperatively. The sperm count and sperm quality gradually improve with time. If the anastomosis is poor, the sperm count may increase at first, but then eventually scar down to oligospermia or aspermia. If the patient is azoospermic at three months or more after vasovasostomy, then either the vas anastomosis or the epididymis is obstructed.

For patients who remain azoospermic after vasovasostomy, the interpretation of where the problem lies depends strictly on the quality of sperm that was found in the vas fluid at the time of surgery. The sperm findings in the vas fluid tell us whether there is continuity in the epididymis (with fresh sperm continuing to come through and reach the vasectomy site). As will be discussed in more detail, if there are numerous long-tailed sperm in the vas fluid, the epididymal tubule is intact, and the vasovasostomy should be successful.

We will now review the results of our studies on the effect of vasectomy on the testis and epididymis. This will allow us to help those patients who remain infertile even after a good vas anastomosis.

Results with two-layer microscopic technique for vasectomy reversal

Between 1975 and 1978, the first 400 patients subjected to this technique for vasovasostomy were studied carefully both pre- and postoperatively in an effort to determine what factors affect recovery of fertility.¹¹ The overall pregnancy rate after one and one-half years of follow-up on the first 42 unselected patients was 71 per cent. The five-year follow-up yielded an 82 per cent pregnancy rate. The causes for the failures will become apparent in the rest of this section. We now have performed over 1,800

such operations. Results are similar throughout our long series. Not many achieved pregnancy before three months, and most of the pregnancies begin to occur between six months and two years. Thus an accurate assessment of pregnancy rate is not usually possible before a series has been followed for two to three years. Although some patients achieve good sperm counts within the first few months and impregnate their wives quickly, this is certainly the exception.

Most of the confusion in the literature on vasovasostomy stems from the lack of documentation of preoperative sperm quality in the vas fluid, inadequate postoperative semen analyses, sparse observations of the epididymal ductal system, and poor testis biopsy studies in vasectomized patients. The group that we operated on were very carefully studied. Seminal fluid was sampled from the testicular side of the obstructed vas of each patient at the time of reanastomosis. The degree of dilatation of the vas lumen on the testicular side of the vasectomy site was measured in all patients. Appearance and quantity of vas fluid, as well as sperm morphology (electron and light microscopy), quantity, and motility were also recorded and correlated with postoperative results. The age of the patient, the time since the vasectomy, the type of vasectomy performed, and the area in which it was performed were correlated to subsequent sperm count and pregnancy of the spouse. Sperm counts were measured at monthly intervals after surgery for the first four months and then at intervals four months apart during the entire follow-up period. No patient was accepted for surgery who did not agree in advance to provide this careful follow-up.

Over 300 of the patients were subjected to a quantitatively meticulous testicle biopsy at the time of vasovasostomy, and the findings correlated with successful and unsuccessful postoperative results.²³ This was particularly important when there was no sperm in the vas fluid, and the patient remained azoospermic postoperatively despite a successful vas anastomosis.

Sperm counts were arbitrarily considered as normal when there was a concentration of more than 10 million sperm per milliliter, 50 per cent motility with good progression, and greater than 70 per cent normal forms, according to the criteria of MacLeod and Gold.²⁴ It is recognized that lower sperm counts can sometimes be found in fertile men and higher counts in infertile men. It turned out that the semen of most of the patients with "normal" counts actually had in excess of 30 million sperm per milliliter. All semen samples with good motility had greater than 70 per cent normal forms. The follow-up on these patients in most cases was superb.

With this kind of careful investigation in patients subjected to as meticulous a microsurgical anastomosis as possible, reliable physiologic data were obtained. *These data revealed that spermatogenesis is*

not significantly harmed by obstruction, and that failure to achieve fertility after an accurate vasovasostomy is caused by dilatation and then perforation of the epididymal duct with inspissation and subsequent secondary epididymal obstruction.

Thirty-two per cent of the early patients had an obvious sperm granuloma noted at the vasectomy site. That is a much higher incidence than we see in our more recent patients. This change is probably due to the increasing use of cautery for sealing the vas more effectively at the time of vasectomy. There were no particular symptoms of discomfort related to the sperm granuloma. The sperm granuloma represented a continual leakage of sperm fluid at the vasectomy site.

In the group with sperm granuloma all had abundant morphologically normal sperm in the vas fluid. Even when the vasectomy had been performed over ten years ago, none of the patients with sperm granuloma had poor quality sperm in the vas fluid. No matter how long ago the vasectomy had been performed, the presence of a sperm granuloma assured a high quality of sperm in the vas fluid at the time of vasovasostomy.

The internal diameter of the testicular side lumen of the vas deferens was almost always 3/4 mm or less in vasa with sperm granuloma. In patients without sperm granuloma, the internal diameter of the testicular side lumen was usually 1 mm or greater. Thus, the presence of sperm granuloma was associated with less dilation of the vas deferens on the testicular side of the obstruction.

In patients who had unilateral sperm granulomas, the sperm quality was always satisfactory on the side with the sperm granuloma but was usually of poorer quality on the opposite side. Thus a dramatic benefit was conferred on the side with sperm granuloma that was not conferred to the side without granuloma. These data favored the postulate that a failure to recover fertility after an accurate anatomic reconnection of the vas deferens is due to the local effects of high pressure created by the vasectomy. The presence of a sperm granuloma at the vasectomy site represents persistent and continual leakage of sperm, which alleviates the deleterious high intravasal and epididymal pressure which otherwise always occurs after vasectomy.

Effects of vasectomy on testis and epididymis

Despite the fact that vasectomy is one of the most popular operations performed in the United States, there has been a great deal of controversy in the scientific literature about its effects, both in humans and in animals. Many of the differences in experimental results in the early literature are related to the use of different animal models and different techniques of vasectomy. However, much of the controversy is simply a result of sloppy methodology. In

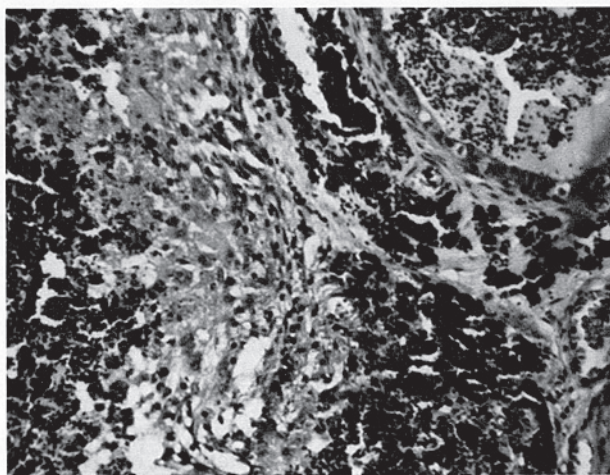


FIGURE 6. *Histologic sections of epididymal tissue at site of secondary obstruction usually reveals blow-outs with perforations of epididymal duct and sperm leakage into surrounding interstitium causing interstitial epididymal sperm granuloma.*

1978 I attempted to review all of the data available at the time and organize it according to species, in an attempt to understand the effect of pressure increase after vasectomy on the testis and epididymis.²² However, even grouping by species, there were many contradictory findings. In the last six years we have come to a clearer understanding.

There is no question about the marked dilation of the vas deferens and epididymal tubule that occurs consequent to vasectomy in all species. Observation of the epididymis in humans undergoing reversal reveals marked tubular dilation with blowouts and leakages in weak points of the epididymal tubule consequent to pressure buildup. This results in secondary epididymal obstruction. There is no longer any controversy about the presence of these epididymal changes in virtually all species.²⁵⁻²⁹

Bedford³⁰ studied in great detail the effects of vasectomy in four different species of animals. He noted rupture of the epididymal duct with leukocytic infiltration in all four species. He believed that sperm were continually produced after vasectomy, but no reabsorption occurred until there was an epithelial rupture somewhere within the ductal system. By eight months postvasectomy, the corpus epididymis began to show signs of dilation, and a series of ruptures and scars could be seen in the epididymis. Only after epithelial rupture had occurred did leukocytic infiltration and invasion appear, with reabsorption of sperm.

Although there is substantial pressure damage to the epididymis, there is no discernible effect on spermatogenesis or testicular architecture. The testicle biopsy showed normal spermatogenesis in all patients who had no sperm in the vas fluid, and therefore we believed the problem had to be epididymal

blockage. We reasoned that if the problem was secondary epididymal obstruction caused by rupture and sperm extravasation in the epididymis, much more sophisticated microsurgery bypassing epididymal blockage might restore fertility even in these least favorable cases.

To resolve this question, we explored patients who had azoospermia for at least two years following a patent vasovasostomy.³¹ These patients, of course, had no sperm (or only sperm heads) in the vas fluid at the time of vasovasostomy. However, normal sperm were found in the epididymal fluid despite absence of sperm in the vas fluid. Epididymal histology distal to this site revealed extensive interstitial sperm granulomas resulting from rupture of the epididymal duct, similar to what Bedford observed in four animal species (Fig. 6).

Once the epididymal rupture and subsequent blockage occurs, the fluid which had previously accumulated in the vas deferens is trapped and isolated. The sperm in that vas fluid die of "old age" and then eventually degenerate, the tails fall off, and the heads finally deteriorate into amorphous debris. Thus, the absence of sperm in the vas fluid just proximal to the vasectomy site indicates that there is secondary epididymal blockage from epididymal rupture and blockage. The presence of morphologically intact sperm in the vas fluid indicates an intact ductal system with relatively fresh sperm continuing to reach the vas. That is why the most accurate vasovasostomy cannot result in a success if there is no sperm in the vas fluid. To treat such failures successfully, one would have to bypass the secondary blockage in the epididymis.

We now have experience in over 2,000 cases and can state that in every case where there is no sperm in the vas fluid, and the patient is azoospermic after an accurate vasovasostomy, sperm can be found somewhere in the epididymal tract proximal to a point of secondary blockage. Occasionally this may be as high as the vasa efferentia. Fortunately, most of the blockages are limited to the region of the junction of the corpus and tail of the epididymis. Therefore, most bypass vasoepididymostomy procedures can be performed either to the distal, or the midcorpus, region of the epididymis where epididymal length is long enough for good maturation of sperm.³¹⁻³⁵

The duration of time since vasectomy correlates with the likelihood of pressure-induced rupture of the epididymis in these patients, just as it did in the laboratory animal studies of Bedford. In humans the time range, however, is considerably expanded. For example, in humans, whenever reversal was performed within one year of vasectomy, high-quality sperm were always found in the vas fluid, and normal semen analyses were obtained after surgery.

There was no sudden period of time after which an epididymal blowout would predictably occur. Rather, the risk of epididymal blowout on each side

gradually increases as the years progress. The chances of finding no sperm in the vas fluid on one side at ten years was 75 per cent. However, the chances of finding no sperm on both sides at ten years was about 50 per cent. At five years after vasectomy, the chances of finding no sperm on one side was 25 per cent, but the chances of finding no sperm on both sides was only 6 per cent. In every case where no sperm was found in the vas fluid, the testicle biopsy was normal. The absence of sperm was not caused by a disruption of spermatogenesis but rather by epididymal ruptures and secondary blockage.

Examination of the epididymal histology shows dilated epididymal ducts, sperm extravasation into the interstitium, sperm granuloma formation, and many macrophages, sperm heads, and inspissated protein within the tubular lumen (Fig. 6). Distal to this transition point the epididymal tubules are empty and devoid of sperm. There may be some macrophages noted in the fluid, and possibly some cellular debris, but no sperm.

The sperm count and the quality of motility gradually rise during the first twelve months after successful vasovasostomy as the epididymis recovers from the partial disruptions in the epithelium and the chronic dilatation. In most patients with high sperm counts after vasovasostomy normal motility eventually develops as the epididymis recovers. Patients who persist in having oligospermia and poor motility more than one year after vasovasostomy have continued blockage (partial) either at the vasovasostomy site or in the epididymis. Although this is usually clinically obvious, for uncertain cases, we have developed a useful tool which we call "simplified quantitative testicle biopsy."²⁴

Quantitative Interpretation of Testicle Biopsy

Testicle biopsy has been used by most clinicians in a nonquantitative fashion only. This has severely limited its usefulness and has led to many errors in its interpretation.³⁶⁻³⁹ However, the quantitative approach which we introduced has now made it extremely useful.

Heller and Clermont⁴⁰ first described the histology and kinetics of spermatogenesis in humans. They determined through radioactive tracer studies that the rate of spermatogenesis in humans, or in any species, is always constant, even when sperm production is reduced. Reduced sperm production is always caused by a reduced number of sperm "on the assembly line," not by reduced rate of production. Therefore, the amount of sperm being produced by the testicle should be reflected by what is seen at any moment in a fixed specimen of testicle biopsy.

Steinberger, Tjioe, and Paulsen^{41,42} then developed a method of quantitative interpretation of the

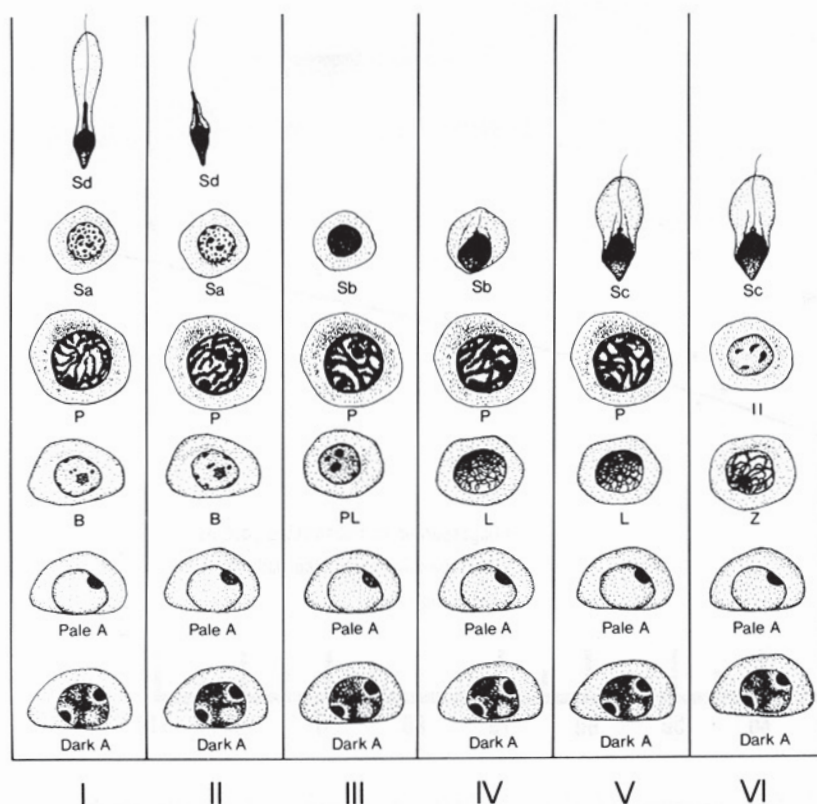


FIGURE 7. Demonstration of six different stages of human spermatogenesis. For quantitative interpretation of testicle biopsy we need only count mature spermatids at top of Stages I, II, V, and VI, since these are easiest to recognize to correlate closest to sperm count.

testicle biopsy. Unfortunately, they had a small number of patients, they did not concentrate on the mature spermatids, and were therefore limited in trying to make a precise correlation with sperm count. Furthermore, their technique was elaborate and time consuming.

In 1978 Zukerman *et al.*,⁴³ working with Steinberger, counted all components of spermatogenesis and found a reasonable correlation with sperm count. But the difficulty still remained that each quantitative testicle biopsy using the method they described was time consuming, and few fertility specialists had any inclination to put that much effort into photographing and analyzing the biopsy of each of their patients. Furthermore, their correlation fit was not as good as it could have been because they used a linear rather than an exponential curve.

A simplified method of quantitatively interpreting testicle biopsy, which any clinician can utilize precisely and with an expenditure of only ten to fifteen minutes of his time, was then developed by myself and Rodriguez-Rigau.²³ We analyzed patients with oligospermia, as well as patients with normal sperm counts, and showed that the testicle biopsy can accurately predict a patient's mean sperm count. We then showed that comparing the results of the sperm count predicted by quantitative testicle biopsy with the actual sperm count could document quite reliably whether or not the oligospermia is caused by partial obstruction.

In previous studies already alluded to, we showed that patients who were severely oligospermic after strictured vasovasostomy became fertile after reanastomosis. It is thus established that obstruction in the ductal system can cause oligospermia and poor motility. In fact, almost all cases of poor sperm motility and low sperm count after vasectomy reversal have been found to be due to obstruction. But when the patient's prior fertility is not known, or when documentation is necessary before embarking on a questionable case, a quantitative testicle biopsy can clarify whether blockage, or just poor spermatogenesis, is causing the poor semen quality.

The biopsy is performed with a careful "no touch" atraumatic technique under general anesthesia. The tunic albuginea is sharply incised with a scalpel. The protruding seminiferous tubules are excised with a wet, extremely sharp, microiris scissor, and then allowed to fall into Zenker solution without ever being touched. Specimens are carefully fixed, cut in thin sections, and stained with hematoxylin and eosin. The technique of biopsy is important. If the specimen is handled roughly or fixed in formalin, it is difficult to identify accurately the cellular components of the seminiferous tubule.

At least ten seminiferous tubules are included in the count on each side. The testicle biopsy is performed bilaterally. Thus, a total of twenty or more tubules are included in the biopsy evaluation on each patient. Only the mature spermatids (Stages I,

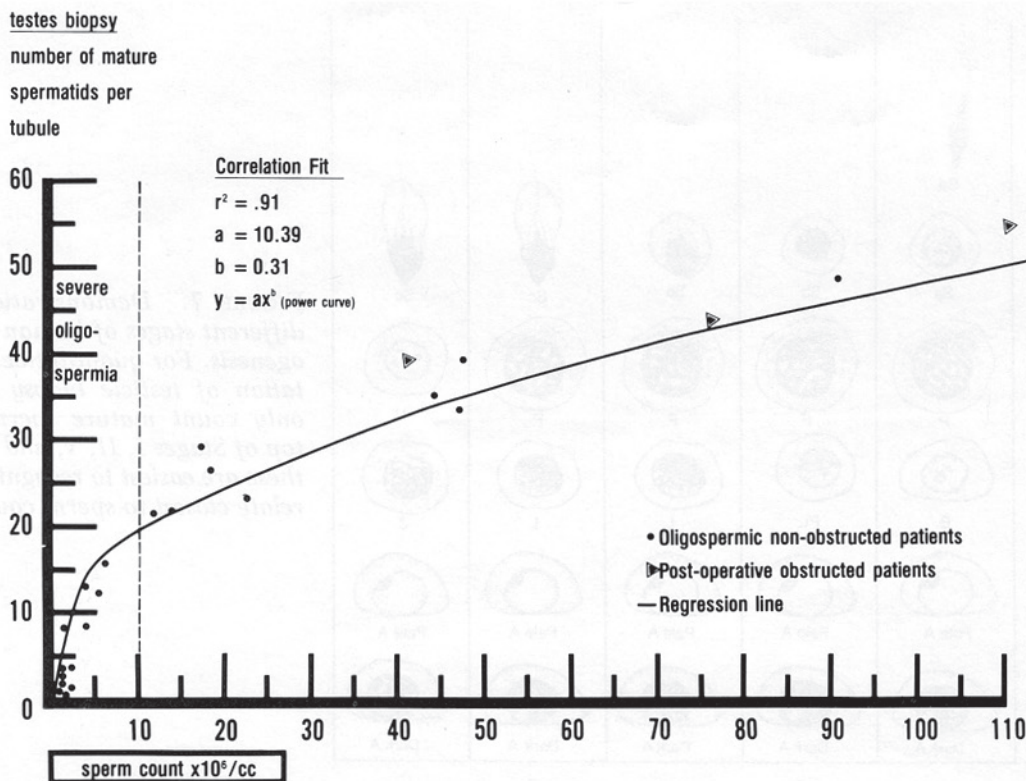


FIGURE 8. Exponential graph demonstrates relationship between number of mature spermatids per tubule in testicle biopsy and sperm count/cc. If one simply counts number of mature spermatids per tubule, he can figure fairly closely predicted sperm count of patient if there is no obstruction.

II, V, and VI) need be counted. Put in more simple terms, only the oval-shaped cells with dark, densely stained chromatin are counted. All of the complicated steps of spermatogenesis from spermatogonia through resting spermatocyte, leptotene spermatocyte, zygotene spermatocyte, pachytene spermatocyte, and early spermatids (S_A , S_{B1}) can be excluded from consideration (Fig. 7). Only the easy-to-recognize mature spermatids are included in the count. The reason for this choice is that for routine clinical use, we wanted to count only those cells which previous studies have shown will have the greatest correlation with sperm count and which are the easiest cells to recognize. We simply add up the number of mature spermatids in a minimum of twenty tubules and divide that by the number of tubules.

Using an exponential curve, summarized in the graph (Fig. 8), the number of mature spermatids per tubule can be used to predict what the sperm count should be. In the absence of obstruction there is a remarkably close correlation between the number of mature spermatids per tubule and the actual sperm count in the semen. For example, if the patient has forty mature spermatids per tubule, the sperm count should be just under 60,000,000/cc. If there are forty-five mature spermatids per tubule, the sperm count should be just over 85,000,000/cc. If the pa-

tient has a sperm count of only 3,000,000/cc, you would expect him to have only six to ten mature spermatids per tubule.

In unobstructed patients with less than 10,000,000 sperm/cc, there are always less than twenty mature spermatids per tubule in the testicle biopsy. In patients with over 10,000,000 sperm/cc, there are usually greater than twenty mature spermatids per tubule. The number of mature spermatids per tubule correlates very closely to the sperm count/cc.

The postoperative sperm count of patients who undergo microscopic vasovasostomy or vasoepididymostomy correlates with their quantitative testicle biopsy. A chronically low sperm count postoperatively is usually caused by continuing obstruction. This can be objectively determined by comparing the mature spermatid count in the testicle biopsy to the sperm count in the semen. For example if the patient is simply not manufacturing many sperm, his count could be low without continuing obstruction being the cause.

Quantitative testicle biopsy can accurately allow a firm diagnosis of obstruction prior to an involved scrotal exploration which could otherwise require a great deal of guesswork about whether or not there truly is epididymal blockage. Also, by comparing

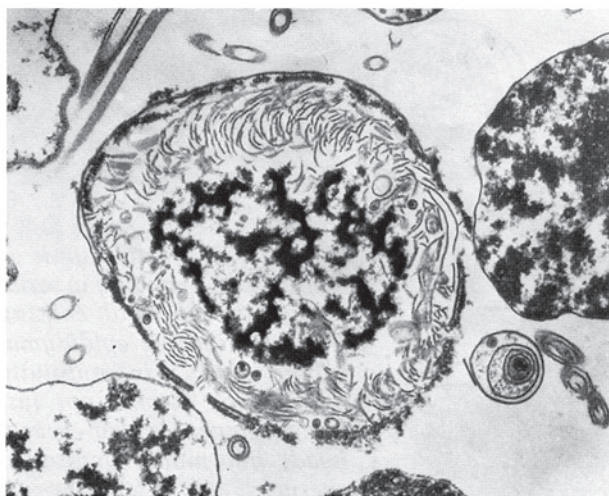


FIGURE 9. *Electron micrograph of vas fluid in patient who had "no sperm" but simply debris. Debris is simply broken down nucleoprotein from degenerating sperm head.*

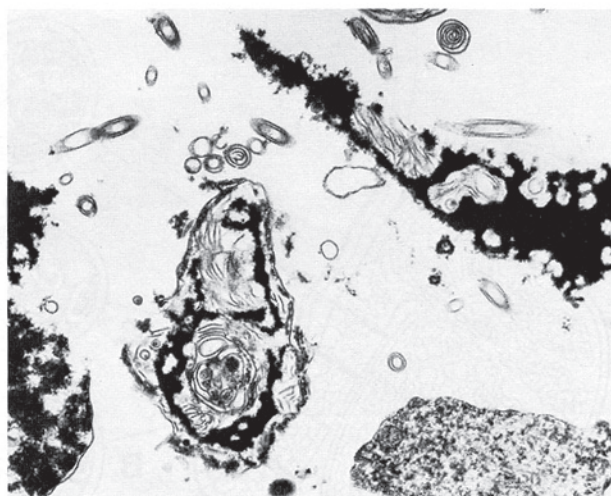


FIGURE 10. *Electron micrograph of vas fluid from patient who had only sperm heads and who definitely had secondary epididymal obstruction. Sperm in advanced stage of deterioration indicating no new fresh sperm have been able to reach vas fluid from epididymis.*

the number of mature spermatids per tubule to the patient's sperm count, unwarranted medical therapy will not be haphazardly administered to patients who have obstruction.

Frequently patients undergo vasoepididymostomy inappropriately because the pathologist qualitatively reports normal spermatogenesis. The pathologist's readings are usually not quantitative, but rather a general impression of tubules filled with spermatocytes and some mature sperm. This impression of normal spermatogenesis has led many nonobstructed patients into an unwarranted vasoepididymostomy. If the biopsy shows thick tubules with large numbers of spermatocytes but only three to eight mature spermatids per tubule, such a patient does not have obstruction as a cause of his azoospermia.

Some clinicians have attempted to use the serum follicle-stimulating hormone (FSH) level to monitor the amount of spermatogenesis. If an azoospermic patient were to have a normal FSH level, that would supposedly indicate obstruction. But this is simply untrue. The FSH level correlates very poorly with spermiogenesis.⁴⁴ FSH correlates most closely with the total number of spermatogonia and with testicular volume, but not with the number of mature sperm. The feedback mechanism is just not tuned finely enough for the serum FSH level to give any indication of what sperm count should be.⁴⁵

Ironically, it is the scattered mosaic arrangement of the various stages of spermatogenesis in the human seminiferous tubule (as opposed to the orderly wave of spermatogenesis moving across the seminiferous tubule in most other species) that allows quantitation of the human testicular biopsy to be so simple. In rats, a cut through any particular seminiferous tubule will show only one particular stage of

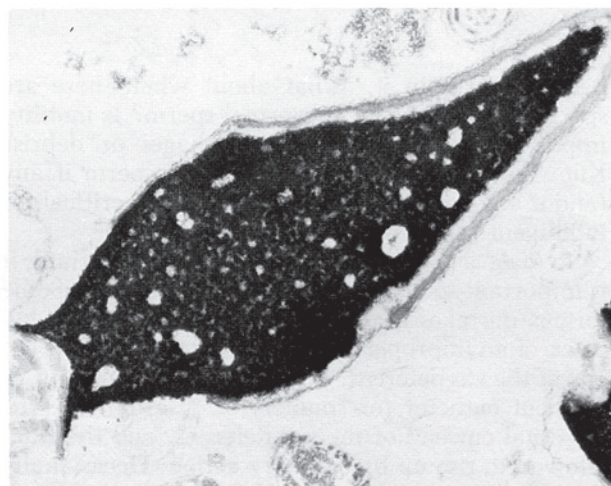


FIGURE 11. *Electron micrograph of fairly fresh sperm which can be seen in vas fluid when there is epididymal continuity.*

spermatogenesis. But in humans a cut through any area of the testicle will reveal a scattered array of all the various stages of spermatogenesis. Thus, in humans it requires only 20 tubules to get a good statistical sample of the total range of spermatogenesis in the entire testicle.

Interpretation of Vas and Epididymal Fluid

What to do when there is no sperm or poor sperm

The question now comes up as to what to do when the fluid from the vas at the time of vasovasostomy

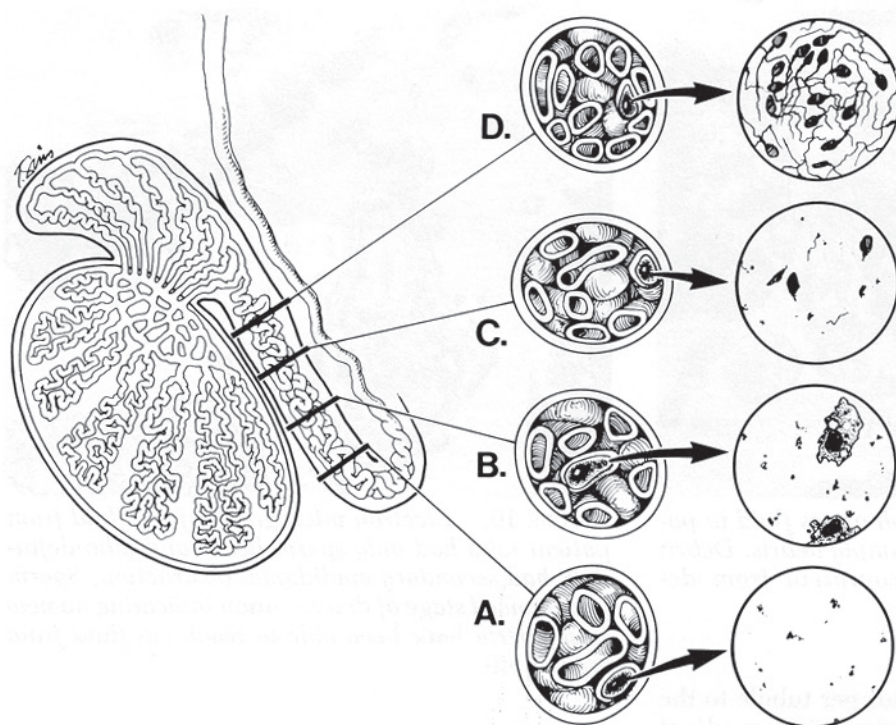


FIGURE 12. Epididymis is transected proximally in serial sections until fluid coming from one specific epididymal tubule which is in continuity with testes harbors not just macrophages or few sperm heads but many long-tailed sperm.

has no sperm in it. What about when there are sperm heads only but no normal sperm? Is motility important? What about macrophages or debris? Knowledge of the appearance of the sperm if any (and if any are present) in the vas fluid is critical for intelligent management of the case.

To make a proper interpretation of the vas fluid, it is important to obtain an adequate collection. Some serious mistakes in clinical judgment are made because of an improper collection of fluid from the cut end of the vas deferens. We recommend that a no. 22 Medicut catheter (or smaller) be inserted into the proximal cut end of the vas deferens, and the fluid allowed to rise up by capillary action. Occasionally this can be assisted by gentle fingertip massage of the vas. The fluid will be drawn into the tube, providing a copious specimen for examination and interpretation. It cannot be emphasized enough how important it is to obtain the specimen in this fashion. Not only will it give a clear answer as to whether or not the fluid is transparent, but it will provide enough fluid for the pathologist to view under phase contrast, dilute if necessary, or stain for greater detail, and it will provide a more representative sample of all the fluid that has built up in the vas.

There have been scattered reports of successful results from vasovasostomy in patients who supposedly had no sperm in nontransparent vas fluid. Upon talking directly to the physicians who had this experience, we found that the specimen was only smeared on a slide, not diluted when necessary, not stained, and often looked at under a poor quality

microscope on a table in the operating room. When these physicians began to obtain more complete collections of fluid from the vas and submitted these better specimens to a pathologist to characterize the sperm more professionally, they verified what we have observed in over 2,000 well-documented cases: when there is no sperm in the vas fluid, and the fluid is not transparent, the patient does not have a successful result after vasovasostomy. In these cases where there is no sperm in the vas fluid, if one is not prepared to perform a vasoepididymostomy, he can advise the patient of the poor prognosis after his vasovasostomy and refer the patient to someone who can perform vasoepididymostomy.

It makes no sense to open the tunica vaginalis and "look around" the epididymis to try to see if there are epididymal blowouts. One cannot tell whether or not there is epididymal ductal continuity by just observing the outside of the epididymal duct. The epididymis is always dilated and filled with inspissated material at various levels due to the pressure buildup created by the vasectomy, but this observation gives no clue to whether or not the epididymis is blocked; this decision can be reliably made only on the basis of interpretation of the vas fluid. To open the tunica vaginalis and explore the epididymis without actually being able to perform the vasoepididymostomy at the same time allows the formation of extensive adhesions which will make a subsequent vasoepididymostomy more difficult. A vasoepididymostomy should not be performed until the surgeon is extremely experienced with very advanced microsurgical technique.

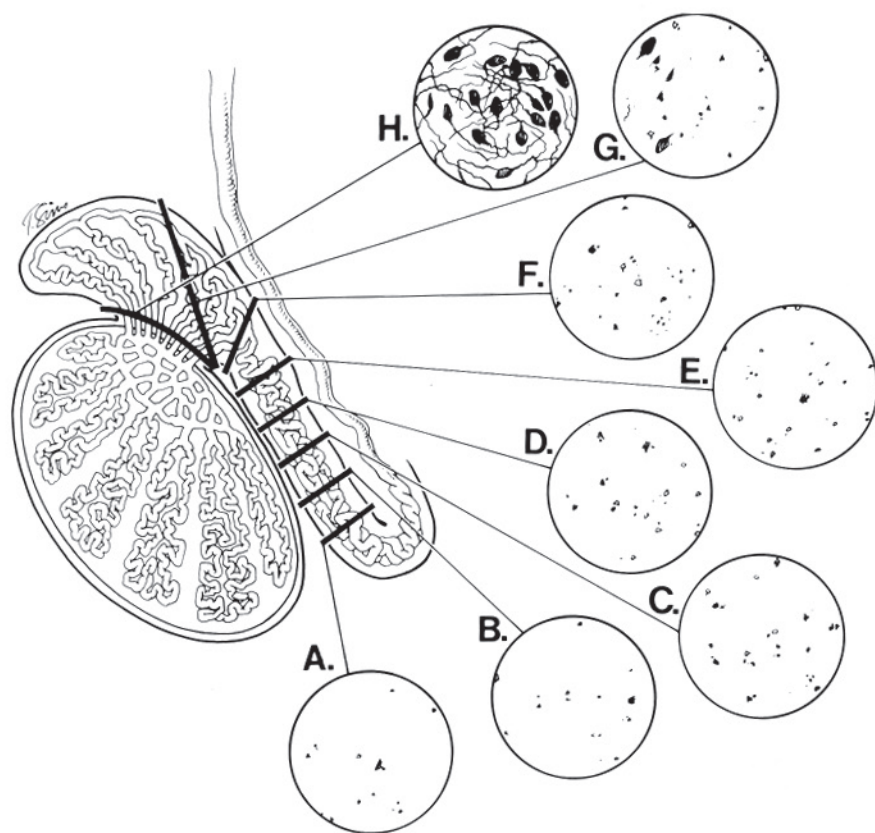


FIGURE 13. Sometimes transection has to be carried far proximally until normal sperm are found. In all cases eventually one gets beyond blowouts (even if it requires reaching vasa efferentia) to where there are normal sperm.

We have found that if the vas fluid has many sperm with long tails the prognosis for normal sperm count after vasovasostomy is over 90 per cent, depending on the skill and accuracy of one's microsurgical technique. Whether or not the sperm are motile has had no influence on the prognosis. If there are no sperm in the vas fluid (with the exception of transparent fluid), none of the patients will have a good result postoperatively. The only exception to this rule is that if the vas fluid is transparent and voluminous, the presence or absence of sperm is irrelevant. These patients can usually be expected to have a good prognosis if an accurate vasovasostomy is performed. The reason for this exception is not altogether clear. These are simply observations.

With our new understanding of the morphologic changes of senescent sperm, it becomes a bit easier to interpret findings in the vas fluid in cases that are not so clear-cut as simply "many long-tailed sperm" or "no sperm." The in-between findings are varying proportions of sperm with short tails, sperm heads only, sperm heads with macrophages and debris, or sperm heads with debris but no macrophages (Figs. 9, 10, 11). We have found that if there are no sperm with long tails, but only short-tailed sperm and sperm heads, the prognosis is very guarded; it is hard to say whether or not the patient will have a good result after vasovasostomy. If there are greater than 20 per cent sperm with normal long tails, then the presence of sperm heads and sperm with short

tails will not hurt the prognosis. If there are no long-tailed sperm, but only sperm heads, the prognosis is very guarded. To go a step further, when we have found only sperm heads in the vas fluid, and the fluid is curdy, then the patient will be sure to have an unsuccessful result after vasovasostomy. Only in cases where the fluid was still translucent or liquid in appearance did the patient with only sperm heads have a chance of having a good result after vasovasostomy. In a similar vein, the presence of a large number of macrophages or debris is worrisome and portends a bad prognosis in the presence of only sperm heads or short-tailed sperm. However, if there are many long-tailed sperm, we expect a successful result even if the fluid is loaded with macrophages and debris.

Sperm Antibodies

There has been a great deal of speculation about the role of autoantibodies to sperm in preventing subsequent restoration of fertility after an accurate vasovasostomy. Most studies report about a 60 per cent incidence of agglutinating antibodies and a 40 per cent incidence of immobilizing antibodies in the serum of vasectomized men.⁴⁶⁻⁵⁵ Despite the fact that many studies have demonstrated the formation of antisperm antibodies, most have not been able to show a strong association between the formation of antibodies and subsequent infertility.⁴⁶⁻⁵⁵

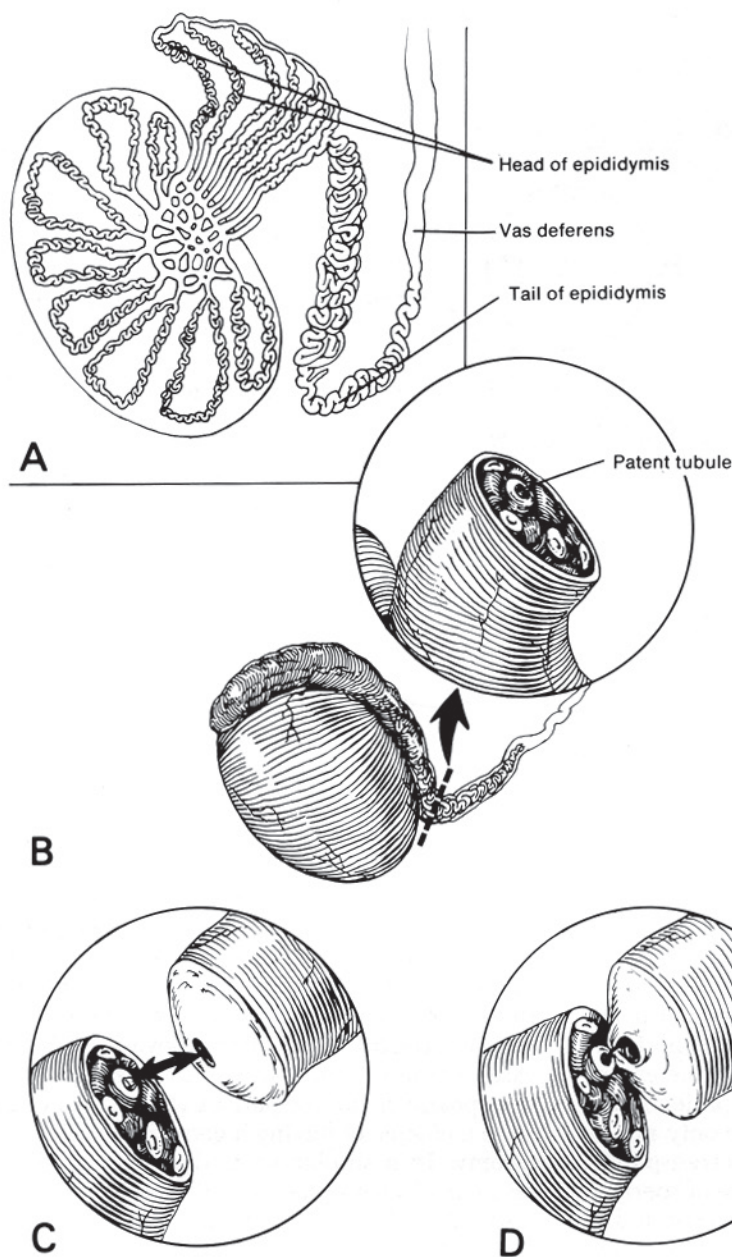


FIGURE 14. The diagram represents the single tubule approach to vasoepididymostomy which I first described in 1977 and published in 1978. Instead of simply joining the vas deferens to the epididymal tunic hoping for a fistula to form, the vas lumen is anastomosed to the specific epididymal tubule leaking the sperm fluid.

Bedford, on the basis of experiments in unilaterally vasoligated animals, has stated that he does not believe that sperm antibodies have any important role in subsequent fertility.^{30,56} In his classic article on the effects of vasectomy in four different species, Bedford showed in rats and rabbits that unilaterally vasectomized animals suffered no loss of fertility. He concluded that "vasectomy has no general immunologically mediated suppressive effect on the potential fertility of these species." In humans also, we know that unilateral blockage (as in inguinal hernia cases where one vas is ligated, vasectomy cases where only one side is patent, and vasectomy cases where only one side recanalizes) does not interfere with fertility.

It is clear that the pressure effects of obstruction on the epididymal system in humans are such that purely physical factors are probably the major ones (rather than autoimmunity) affecting the recovery of normal semen analysis and fertility. Thomas *et al.*⁵⁷ has found no correlation between pregnancy or lack of pregnancy and serum agglutinating or immobilizing antibody titers. Sperm antibodies were present in the semen in only 3.9 per cent of cases, but even in those cases there were pregnancies. But there may be some decrease in fertility in these cases.⁵⁸ Although more work needs to be done on this interesting problem of sperm antibodies, it is clear that the most frequent obstacle to recovering fertility is persistent obstruction, whether partial or

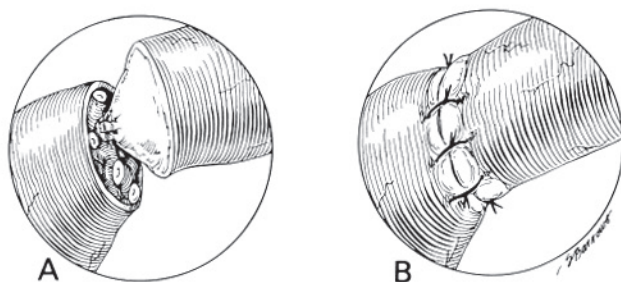


FIGURE 15. After inner mucosal layer is anastomosed, outer muscularis of vas is sutured to epididymal tunic for support only.

complete, either at the vasovasostomy site or in the epididymis.

Microsurgical Vasoepididymostomy

The epididymis represents one 20-foot long coiled tubule with myriads of intricate convolutions. It is squeezed into a two-inch length like the pleats of an accoridian. Because the epididymal tubule is so tiny, even by microsurgical standards, the results with conventional surgery for this type of obstruction have been poor. Schoysman has reported the best pregnancy rate with conventional vasoepididymostomy, (25%).^{59,60} Others have reported much poorer results.⁶¹ Hanley⁶² reported only one pregnancy after 83 vasoepididymostomies. The procedure utilized by Hanley and that described by Hotchkiss⁶³ formed the basis for the usual conventional vasoepididymostomy still performed by many urologists today.

Although this conventional procedure was the best that could be performed in the 1950s, modern microsurgical facilities have rendered it obsolete and these patients can now be given a much better prognosis with extremely exacting microsurgical procedures.

I first developed and described the microsurgical specific tubule technique for vasoepididymostomy in 1978 (Figs. 14 and 15).^{32,35} This idea had never been considered previously because anastomosing a specific epididymal tubule to the vas deferens required microsurgical expertise not yet developed. Over the past five years I have refined the microsurgical technique, and now have experience with over 800 such cases.

After the scrotal sac is entered, the vas is freed up, the tunica vaginalis is opened, and the testis and epididymis are exposed. The dilated epididymal tubule is usually about 0.1 to 0.2 mm in diameter. The epididymal duct is extraordinarily delicate, with a wall thickness of about 30 μ . If one were to employ the usual conventional approach and make a deep longitudinal incision into the outer epididymal tunic, he would see an illusion of many tiny cut tubules. Without the benefit of microscopic observation, there is an illusion that sperm fluid is welling up

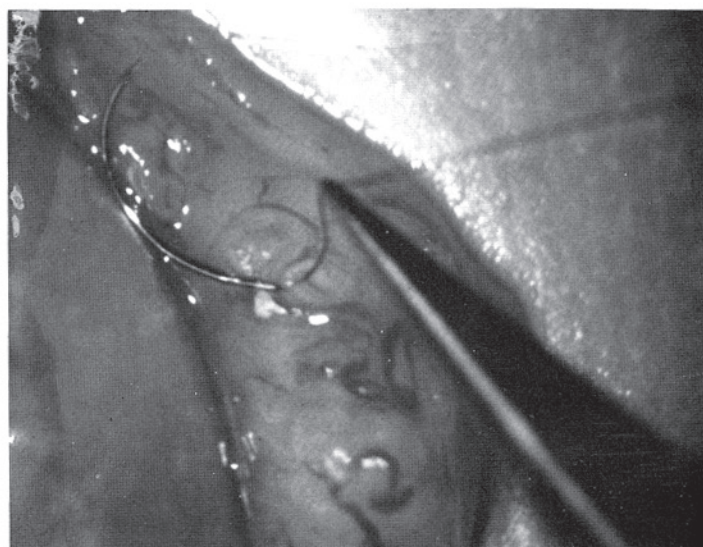


FIGURE 16. Placement of first mucosa suture in epididymal tubule.

from all of these tubules, but in truth the fluid is coming from only one of them. The other tubules are just blind loops disconnected from continuity with the testis by this incision. The ideal approach for reestablishing continuity of the ductal system is to anastomose directly the inner lumen of the vas deferens specifically to the one epididymal tubule that is leaking sperm.

Rather than making a conventional longitudinal incision, a transverse section of the epididymis is made at the most distal point, i.e., at the junction of the cauda and corpus epididymis. With this approach one can slice off portions of the epididymis more and more proximally until sperm are recovered at the most distal possible level but proximal to the area of obstruction (Figs. 12, 13). Under the operating microscope, three to ten cut tubules are usually visible on the transected surface of the epididymis, and all are carefully examined for efflux of sperm fluid. A slide is smeared on this cut surface of the epididymis and observed under a standard laboratory microscope or phase contrast microscope for the presence, and quality, of sperm as described in the previous section. Sometimes no fluid at all is observed, and in those cases one must transect more proximally. But the presence of fluid does not necessarily mean the presence of sperm. One must wait for the report on the fluid before deciding whether to do the anastomosis at that point or to transect more proximally. The anastomosis is performed at the distal most level where normal sperm are found in the epididymal fluid. This allows for the maximal possible length of epididymis.

For the first stitch the epididymis is held between the thumb and the forefinger, facing the microscope (Fig. 16). A slight milking action may sometimes be necessary to promote a continual efflux of fluid to

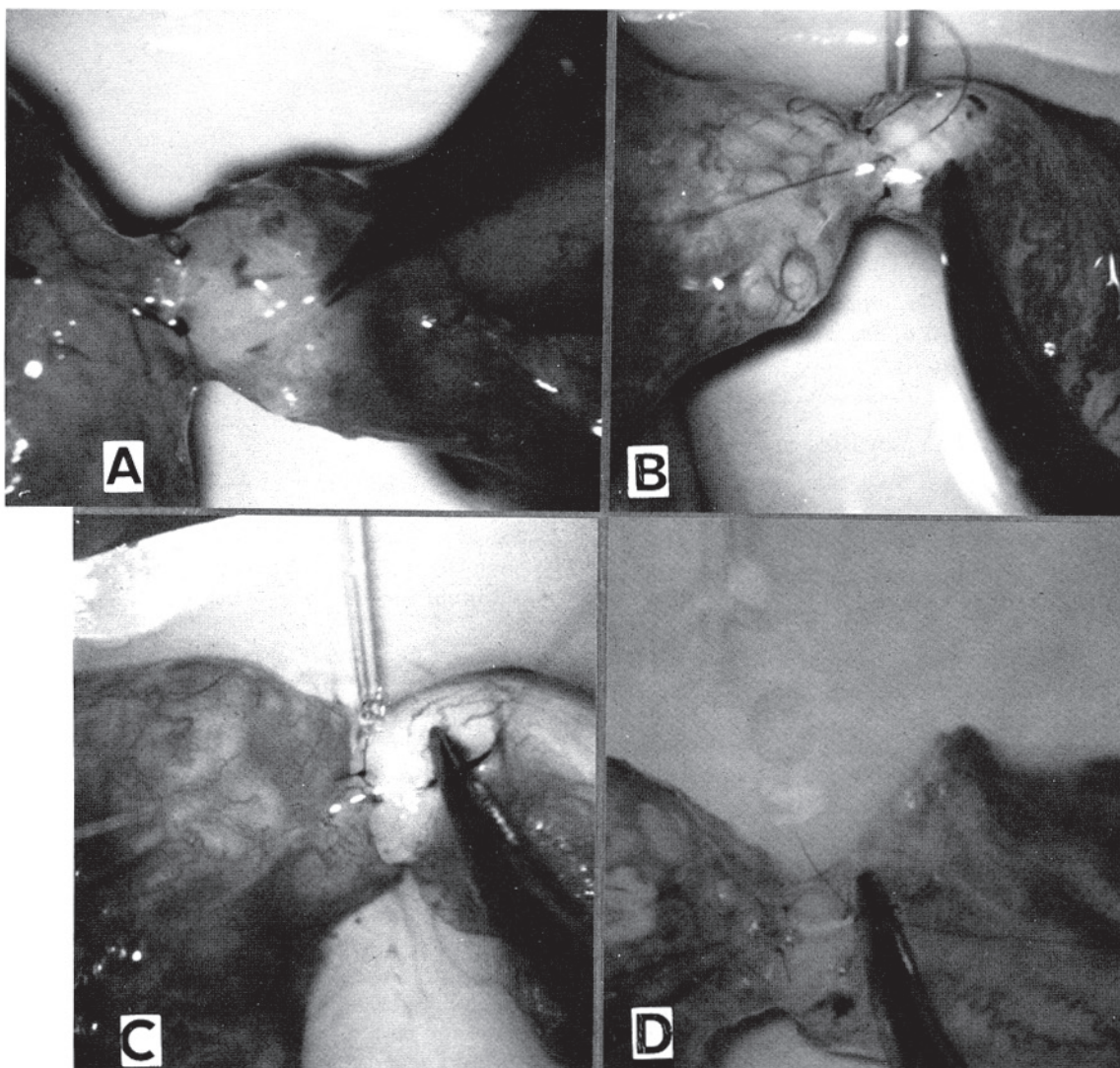


FIGURE 17. (A) After first two mucosal sutures of vas lumen to epididymal tubule have been tied, gap remains allowing room for two more subsequent sutures. (B) Third mucosal suture has been placed through lumen of vas and through lumen of epididymal tubule but has not yet been tied. (C) Fourth specific suture through vas lumen on right and epididymal tubule on left is being placed. (D) Similar view of fourth epididymal tubule suture being placed.

continue to see which is the correct tubule to anastomose. Either 9-0 or 10-0 monofilament nylon on a GS-16, a BV-9 needle, or the new "Silber" V-100 (Ethicon) is used. Ethicon designed the "Silber" V-100 needle especially for this procedure. The first suture is placed from the outside to the inside of the specific epididymal tubule that is leaking the sperm fluid. After the first suture has been placed in this fashion, the epididymis is put into one jaw of the Silber vasovasostomy clamp and the vas is inserted into the other jaw. A blue piece of plastic is then placed underneath the epididymis and vas, which are now held in the two jaws of the vasovasostomy clamp. From this point on, the specific anastomosis of the vas lumen to the epididymal tubule can be

performed in a fashion somewhat similar to vasovasostomy (Fig. 17). However, instead of six sutures as in vasovasostomy, four sutures are adequate.

The reason that the first stitch was placed before the epididymis was put in the jaws of the vasovasostomy clamp is that it is extremely difficult to locate the specific tubule leaking the fluid in any other way. The gentle milking action that the thumb and forefinger can provide is helpful in making sure that the fluid is continuing to flow from the tubule to which you have decided to suture. After placement of the first suture, the tubule is now easily identified at all times. Three more perfect mucosa-to-mucosa sutures are then placed to achieve an accurate anastomosis of the vas lumen to the epididymal tubule.

Fertility after vasoepididymostomy

What can be expected of sperm that have not progressed completely through the epididymis? It is certainly well known that sperm from the cauda epididymis are mature and capable of fertilizing the ovum. A large percentage of sperm from the corpus epididymis are also capable of impregnating and have good, active, progressive motility. However, in the head of the epididymis in animal models, sperm have not yet obtained maturity, and motility consists only of weak vibratory motions.

In the human, as long as the sperm has had an opportunity to pass through some small portion of the corpus epididymis, there are a good proportion of fertile sperm. Thus when the anastomosis is anywhere along the corpus epididymis, the prognosis is good. However, the prognosis is not as good in patients whose anastomosis was performed in the head of the epididymis. Schoysman⁶⁴ with long-term follow-up has had pregnancies in only 11 per cent of patients with a patent anastomosis at the head of the epididymis. But that is still greater than what would have been predicted from the animal studies.

All of the animal data on sperm maturity in the epididymis comes from sperm which were sampled from an intact epididymis. Whether or not remaining segments of the epididymal tubule and vas deferens would be able to promote sperm maturation in the physiologic context of vasoepididymostomy could not be answered by any of the animal experiments which form the basis of our understanding of epididymal function.

For example in the rabbit, Gaddum⁶⁵ sampled spermatozoa from the seminiferous tubules, the ductuli efferentes, and various levels of the epididymis to determine their intrinsic motility and fertilizing capability. Spermatozoa from the seminiferous tubules and ductuli efferentes showed only weak vibratory movements with no forward progress. Spermatozoa from the proximal head of the epididymis showed irregular, erratic motility with no forward progression. Traversing the corpus epididymis, however, increasing numbers of spermatozoa began to show forward movement with proper longitudinal rotation as they progressed distally toward the cauda epididymis. Similar studies have been performed by Bedford⁶⁶ in the rabbit and by Orgebin-Crist⁶⁷ in the rabbit.

In an effort to see whether or not the increase in spermatozoa maturity was merely a function of the time required for passage of spermatozoa through the epididymis, or whether it was dependent on specific areas of the epididymis, various authors ligated different portions of the epididymis in rabbits and examined samples of the spermatozoa from each portion at intervals after the ligation.⁶⁸⁻⁷⁰ After interruption of sperm flow, epididymal spermatozoa which had poor motility in the caput region of the epididymis now showed good motility.

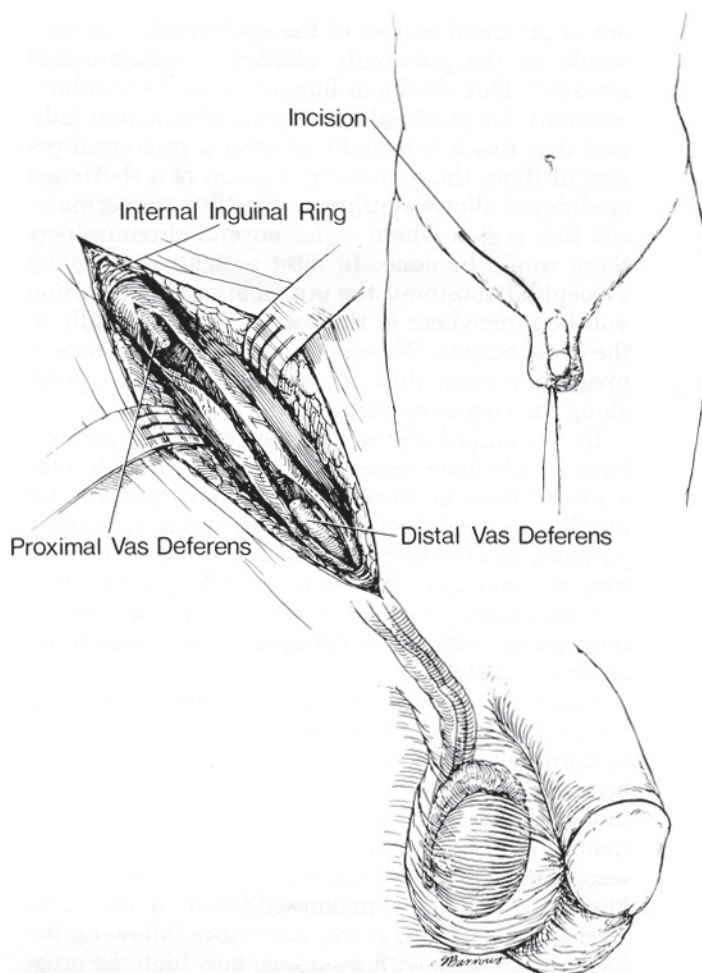


FIGURE 18. Diagram demonstrating the sometimes huge area of vas deferens missing in cases where vas was disrupted during infant herniorrhaphy repair.

However, because of the pathologic nature of the chronic obstruction created by such an experimental model, all of these sperm once again lost their motility by three weeks. These researchers believed that it was possible for spermatozoa to mature at any level of the epididymal duct, but their experimental approach created such an abnormal environment that this hypothesis could not be tested adequately. Obstruction to the flow of spermatozoa within the epididymis has been shown clearly to result in stagnation of epididymal spermatozoa. Thus the increased time allowed for maturation in that experiment was counterbalanced by the abnormal obstructed environment.

Orgébin-Crist⁷¹ first suggested to us that in cases of human vasoepididymostomy it was theoretically possible that sperm might be mature and fertile after vasoepididymostomy even to proximal portions of the epididymis. The remaining epididymal tubule might undergo compensatory changes, or spermatozoa might have more time to mature after coming

out of proximal regions of the epididymis than they would in the previously alluded to experimental models.⁷¹ Our results in humans with vasoepididymostomy for proximal epididymal obstruction indicate that this is true and that after a prolonged period of time, the remaining segment of a shortened epididymis allows motility and fertility to spermatozoa in a region where under normal circumstances there would be none. In most patients undergoing vasoepididymostomy, the proximal most obstruction will be somewhere in the corpus region, usually in the distal corpus. We see no significant difference in pregnancy rates thus far at any particular point along the corpus epididymis.

By one and a half years 90 per cent of the patients have an adequate semen analysis. It appears to take a longer time in vasoepididymostomy patients for motility to reach high levels than in vasovasostomy patients, and the sperm count may also require some time to come up to normal levels. We speculate that a considerable period of time is required in some of these patients for sperm transport mechanisms to recover completely.

About 60 per cent of these patients impregnate their wives within two to three years. It is too early to know what the eventual pregnancy rate will be because it does seem that fertilizing capacity per monthly cycle of sexual exposure is less in these patients than the normal population. Furthermore it seems that with time the fertility of these patients gradually increases, making difficult a life table type analysis. Thus, it will take more follow-up before we can state with assurance how high the pregnancy rate will be. Thus far it appears that there will be no serious discrepancy between good semen analysis and eventual pregnancy.

How to evaluate an infertility patient for obstruction not caused by vasectomy

The diagnosis of obstruction should really be simple. Adherence to a few simple principles will allow a proper decision to be made with great assurance, and minimal time or expense.

If a patient has a testicle biopsy which shows normal spermatogenesis, and if he is azoospermic, then he must have obstruction. That is all there is to it. Everything else is superfluous. If in addition to these two criteria he has a palpable vas deferens on physical examination, then this patient is a candidate for surgical exploration and probable vasoepididymostomy. All other data are irrelevant.

If the patient has a low semen volume (less than 1.0 cc), you might worry about ejaculatory duct obstruction. If the semen volume is over 1.0 cc, then you do not have to worry about ejaculatory duct obstruction, which is quite rare anyway.

If the patient has a normal FSH, this does not indicate that he has obstruction. As mentioned in the previous section on testicle biopsy, the serum FSH

level correlates most closely with the total number of spermatogonia, but does not correlate well with mature spermatids or with sperm count. Most patients with azoospermia and a normal serum FSH have maturation arrest (not obstruction) as a diagnosis. The FSH is in the normal range because the total number of spermatogonia in these cases is normal. It is true that if the FSH is elevated, this usually means there is inadequate spermatogenesis; but even that is not always true. Thus, semen volume and endocrine evaluations do not help one in the diagnosis of obstruction.

What about vasograms? A vasogram should only be performed as part of the whole operative procedure for correcting obstruction. It should not be used for diagnosing obstruction or for deciding to perform surgery for obstruction. Performing a vasogram as an isolated diagnostic procedure creates many problems. First, a scrotal exploration is not needed to ascertain that the vas is present; that should be easily discernible on physical examination. Second, any injection of the vas, or transection of the vas, to perform a vasogram could result in obstruction where there originally was no obstruction unless performed as part of a careful microsurgical procedure. Third, the vasogram data are not necessary for planning the operation ahead of time. Most importantly, the vasogram tells you nothing about the epididymis and can lead to a false positive diagnosis of obstruction as well as a false negative diagnosis of no obstruction.

If a diagnosis of obstruction is certain, based on testicle biopsy and sperm count, then a vasogram can be performed at the time of vasoepididymostomy (once the vas is transected) to make sure that the vas empties distally into the ejaculatory duct and prostatic urethra. There is no need to know this information ahead of time. The easiest, most logical time to perform the vasogram is at the time of vas transection when the vasoepididymostomy is being performed.

There is a mistaken notion that you can use a vasogram to make a diagnosis of epididymal obstruction. Nothing can be farther from the truth. Any retrograde injection of the vas deferens toward the epididymis can only result in potential damage to the epididymis. It cannot make a diagnosis of epididymal obstruction. Because the epididymis is a closed tubular system, the radiopaque fluid will come to a standstill somewhere in the epididymal duct. The point at which it will come to a standstill depends strictly on the difference in pressure between the fluid in the epididymal tubule and the amount of exertion the surgeon is willing to force on the plunger of the syringe. Thus, almost any vasogram fluid injected in the epididymal direction will give the illusion of epididymal obstruction at some point; but there is no obstruction. What usually happens with a forceful injection is that iatrogenic blowouts will be created, further complicating the

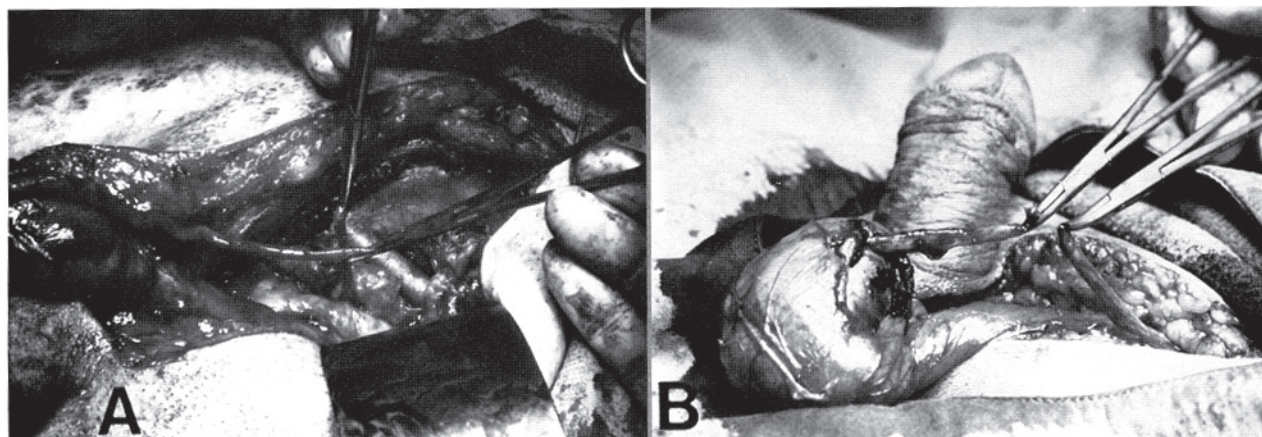


FIGURE 19. (A) Major abdominal dissection for freeing up vas deferens deep to internal inguinal ring where it was ligated during infant hernia repair. This operation twenty-eight years later must free up enough vas deferens and epididymis to bridge an enormous gap.

patient's problem. Thus, the only function of the vasogram at the time of microsurgical vasoepididymostomy is to make sure the distal vas empties properly into the ejaculatory duct.

Physical examination of the epididymis and testes, as well as history or lack of history of infection, can be misleading as well. Testicles producing a normal amount of sperm may sometimes be small, and testicles which are producing no sperm (that have maturation arrest) may often be large. History can be similarly confusing. At least half of the patients who were found to have epididymal obstruction from inflammatory causes gave no prior history of clinical epididymitis. We have to assume that whatever caused their epididymal obstruction must have been subclinical.

In conclusion, most of the ancillary medical information that we routinely consider in fertility patients is irrelevant to the question of whether or not they are obstructed. The physical examination is only relevant in that if there is no palpable vas deferens (i.e., congenital absence of the vas deferens), no surgical procedure is known to be effective. Except for that, history and physical examination, serum FSH, LH and testosterone levels, and vasography are irrelevant to the diagnosis of obstruction. All that is needed to schedule vasoepididymostomy is: (1) a palpable vas deferens on one or both sides, (2) a testicle biopsy showing quantitatively normal spermatogenesis, and (3) a semen analysis showing azoospermia.

Accidental Inguinal Disruption of Vas Deferens During Herniorrhaphy

We have encountered in routine evaluation of men with azoospermia the unsettling discovery that bilateral inguinal herniorrhaphy, particularly in infancy, carries a risk of causing iatrogenic obstruction

of the vas deferens. Such patients generally present in young adulthood with infertility. The semen analysis shows azoospermia, and the urologist usually performs a testicle biopsy and a vasogram. The vasogram reveals obstruction of the vas deferens near the external or internal inguinal ring on both sides and the testicle biopsy shows normal spermatogenesis. Such patients represent a major microsurgical challenge because an enormous segment of vas deferens has usually been removed throughout the inguinal canal along with the hernia sac, and the obstruction is of long duration, so that one can usually expect to find epididymal obstruction also (Fig. 18). These are very challenging cases requiring a great deal of meticulous freeing up of vas without damaging the blood supply to it (Fig. 19).

The approach which I take toward the patient who presents with azoospermia and a history of bilateral inguinal herniorrhaphy in the past is as follows: Perform a testicle biopsy first. If the biopsy is normal, the vasogram can be done operatively at the time of contemplated reconstruction. If the testicle biopsy is normal and there is a history of bilateral inguinal herniorrhaphy, there must be a high suspicion for interruption of the vas deferens in the inguinal area.

When there is no sperm in the proximal vas fluid, there is epididymal blockage also. In such cases, four anastomoses will be required, two on each side. A vasovasostomy must be performed in the abdomen, and a vasoepididymostomy in the scrotum. Extremely careful attention must be paid not to sever the deferential artery receiving retrograde blood from its connections in the epididymis to the blood supply coming down from internal spermatic artery. Otherwise the intervening segment of vas will be devascularized.

Our findings should give some concern to those pediatric surgeons who advocate bilateral inguinal

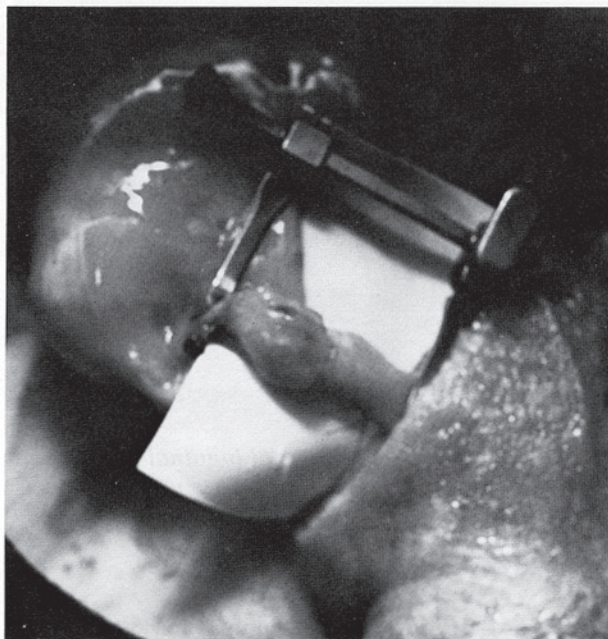


FIGURE 20. Completed crossover vasoepididymostomy with vas coming through median scrotal raphe to other side.

herniorrhaphy for infants with unilaterally detected hernias. The rationale for bilateral herniorrhaphy in those cases is that an undiscovered hernia probably exists on the other side and ought to be repaired also. However, when one considers that in some institutions as many as 15 per cent of infant hernia sacs may be found to contain vas deferens, a bilateral infant herniorrhaphy may conceivably sterilize as many as 2 per cent of children. Thus, these unsettling findings may give excellent argument to the concept of utilizing ocular loupes more routinely for certain pediatric procedures including inguinal herniorrhaphy. The vas deferens is so incredibly tiny in the infant that it would be very easy for an excellent surgeon accidentally to include it in his ligation of the sac at the internal ring.

Frequently one sees a case where there was inguinal disruption of the vas deferens only on one side, but the patient is still azoospermic because on the other side the testicle is atrophic. In this situation, the extensive operation of inguinal vasovasostomy or inguinal vasoepididymostomy can be avoided by using a simple crossover procedure. On one side the inguinal vas deferens is intact, but there is no sperm production. On the other side the testicle is normal, but the inguinal vas deferens has been obstructed by herniorrhaphy. In such a case, it is far easier to make an opening in the median scrotal raphe and pull the vas deferens through the raphe to the other side. Then the scrotal vas deferens (or epididymis) can be anastomosed in a crossover fashion to the inguinal vas deferens from the opposite side (Fig. 20). Such a crossover is much easier to perform

than trying to go through the area of the previous herniorrhaphy to bridge the gap of the missing vas deferens.

Ejaculatory Duct Obstruction

A rare cause of obstructive azoospermia is ejaculatory duct obstruction. This diagnosis is made when the patient has a palpable vas deferens, azoospermia, and a normal testicle biopsy, but has a low-volume semen with no fructose. If one is exploring a patient for epididymal obstruction and the semen volume is low, there must be a high index of suspicion for ejaculatory duct obstruction. But the vasogram which should always be performed routinely at the time of surgery is the only proper definitive way of making this diagnosis.

If there is normal sperm detected in the vas fluid and the vasogram shows ejaculatory duct obstruction, the patient is then placed in lithotomy position and undergoes a TUR of the ejaculatory duct orifice. If, however, the patient has no sperm in the vas fluid, he may have already suffered a blowout in the epididymis from this long-standing obstruction. In that event, a vasoepididymostomy is performed first and the incision closed before the patient is put in lithotomy position for TUR of the ejaculatory duct.

A TUR of the ejaculatory duct is not a difficult procedure for a competent resectionist to perform; but unless one has had considerable experience with transurethral prostatectomy, he had best not attempt this procedure.⁷²⁻⁷⁴ First one inserts a resectoscope through the urethra and inspects the prostatic fossa. A rectal sheath is used to allow an index finger in the rectum to palpate the posterior floor of the prostatic urethra. Using the finger as a guide, a hole is cut sharply into the floor of the prostatic urethra on either the right or the left side just proximal to the verumontanum but distal to the internal sphincter. I have been amazed to read about urologists resecting the "verumontanum" in these cases, thinking that the ejaculatory duct empties there. The verumontanum is simply the embryologic remnant of the müllerian duct—the fetal uterus and upper vagina. The ejaculatory duct does not enter it.

It is also important that the internal sphincter not be damaged, so that the ejaculate will still go out the urethra rather than retrograde into the bladder. It is important that the external sphincter not be damaged in order to prevent the complication of postoperative urinary incontinence. If the first tissue bite does not reveal the ejaculatory duct, then one can continue to resect deeper and deeper, much like drilling for oil. If it is truly a case of a blocked ejaculatory duct orifice, one should fairly soon after the first few bites come across a dramatically large dilated opening with an equally dramatic efflux of translucent seminal fluid.

The semen volume and fructose return to normal rapidly in these patients. One of the ancillary benefits of the procedure is that the patient now has a

reasonable volume ejaculate. A normal sperm count with good motility will return within three to eight months, and these patients are fertile. One problem is that they are more susceptible to the possibility of epididymitis because of urinary reflux up the vas deferens. The slightest hint of prostatitis or epididymitis should be treated aggressively with antibiotics. Adhering to this precaution, we have had good success with such cases.

There have been some unwarranted cases of TUR for ejaculatory duct obstruction in patients who do not have ejaculatory duct obstruction. Quite a few patients have been badly harmed by unwarranted or poorly performed TUR ejaculatory duct procedures. When there is no dilatation of the ejaculatory duct, the diagnosis of ejaculatory duct blockage is not warranted. A TUR in these cases may very well create blockage where there originally was none. Thus we caution that this rare condition not be diagnosed overfrequently.

For further details the reader is advised to refer to our revised and updated book on reproductive microsurgery.⁷⁵

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