# Male Reproductive Dysfunction

## Diagnosis and Management of Hypogonadism, Infertility, and Impotence

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# 22

# Diagnosis and Treatment of Obstructive Azoospermia

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## I. VASECTOMY REVERSAL

There are two major aspects to the problem of vasectomy reversal. The first concerns techniques for obtaining a reliable reanastomosis of the vas deferens. With modern microsurgery, accurate reanastomosis should be achievable in almost every case. The second aspect of the problem relates to the detrimental secondary effects of vasectomy, i.e., the pressure-induced epididymal damage occurring secondarily as a result of the vasectomy.

This section of the chapter will describe the scientific work clarifying that damage, and the microsurgical approach, not only to vas reanastomosis but also to bypassing secondary epididymal obstruction as well.

The data we have obtained on the reversible and irreversible effects of vasectomy were obtainable only because of the refined anastomosis which microsurgery allows. In the past it has always been difficult to tell whether the failure of a vasovasostomy was due to a poor reconnection or other more obscure problems. With accurate microsurgical techniques we have been able to avoid technical problems such as leakage, sperm granuloma, stricture formation, and scarring down of the anastomosis.

## Conventional Approaches to Vasectomy Reversal

Conventional techniques for reanastomosis of the vas have involved placing a splint of nylon or polyethylene tubing into the two ends of the vas deferens and stitching the vas muscularis inaccurately with three to eight large 4-0 sutures (see Fig. 1). This sort of crude approach leads to sperm leakage, granuloma, and poor alignment of the vas mucosa (1-10). These conventional techniques have yielded a 30-70% incidence of sperm in the ejaculate with only 5-20% of wives achieving pregnancy (9). Most of the conventional cases inappropriately referred to in the literature as patent, or as surgical successes (because of the presence of sperm in the ejaculate) had very poor sperm counts with poor or no motility. So it is no surprise that the pregnancy rate was so low. Furthermore, in most of these previous series, the documentation of data was very weak.

In most of the literature, the discrepancy between patency, i.e., the presence of any sperm whatsoever in the ejaculate, and the lack of fertility as evidenced by no pregnancy in the spouse after vasovasostomy, has been a source of great confusion. Most authors have made the mistake of considering the operation as technically successful whenever there is any sperm in the ejaculate. A single dead sperm per high-power field was mistakenly considered a sign of patency and technical success. Very few studies in the past utilizing conventional techniques have actually performed accurate semen analyses over many years, and all have claimed greater technical success in the reanastomosis than is justified.

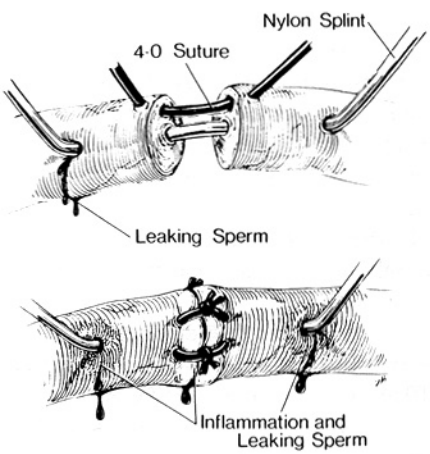



**Figure 1** A diagram demonstrating the typical, conventional technique for vasovasostomy, which results in inaccurate approximation of mucosa, sperm leakage, and subsequent obstruction. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

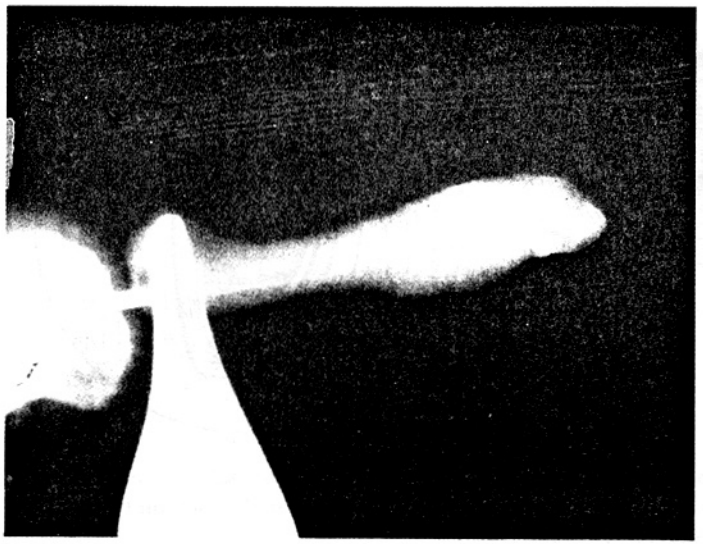


**Figure 2** A vasogram of a conventionally performed anastomosis showing a severe stricture and only partial patency. This patient was oligospermic with only 4,000,000 sperm/ml and very poor motility. After microsurgical reanastomosis, this patient's sperm count went up to over 80,000,000 sperm/ml with normal motility. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

An accurate microscopic technique for reconnecting the vas increases the fertility rate dramatically, but also clarifies what other factors may be operating. When one examines the anastomotic site after conventional vasovasostomy under the microscope he finds a ghastly array of errors and ceases to wonder why the success rates in the past have been so low. Vasograms of anastomotic sites after conventional vasovasostomy confirm severe stricture formation and fistulas (see Fig. 2). After reoperation with microsurgery, the sperm counts and motility improve dramatically.

### Microsurgical Approach

I would like to discuss details of the microscopic technique used in a series of over 1800 patients which appears to provide as satisfactory an anastomosis as is presently possible, and then discuss success rates, giving our views on what factors other than a technically accurate vas reanastomosis influence the recovery of fertility, and what further steps may then be taken in patients who appear to be failures (11-22).

It is advisable to begin practice in the animal before doing such surgery on humans. For the best mucosal approximation in the human situation where lumens

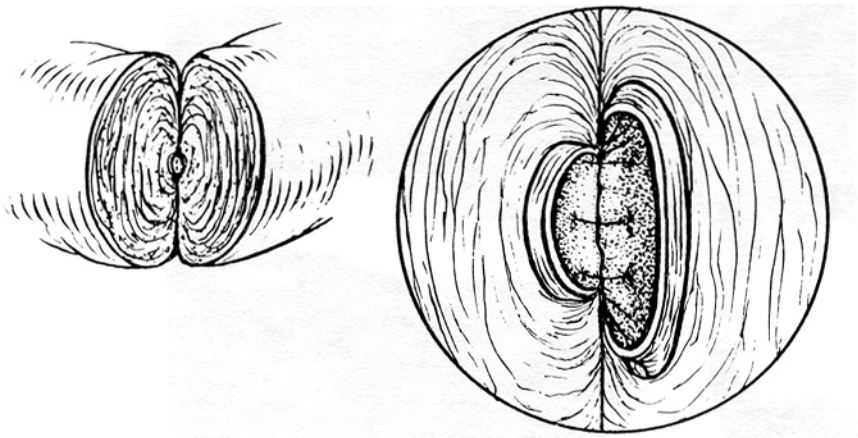


**Figure 3** A diagrammatic representation of the inner mucosal anastomosis, the small lumen on the abdominal side of the vas to the dilated lumen on the testicular side of the vasectomy site.

are of different diameter (because of chronic obstruction and increased pressure), I recommend a nonsplinted *two-layer* approach (see Fig. 3). With a one-layer anastomosis there is poorer mucosal approximation when lumen diameters differ. A splint of any kind should never be used and is only an excuse for not being certain one has obtained a good anastomosis. It results in sperm leakage, inflammation, and more 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 and the correlation between the doctor's recollection and what one finds is very poor. Often a very large segment has been removed and in the majority of cases that we have come across, 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 merely 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 2½ X loupe magnification. The healthy ends of the vas deferens above and below the fibrosis are freed up several centimeters and often 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 quite

extensive and, contrary to conventional thinking, we have found that freeing the vas a good distance will do nothing to injure the blood supply.

The beginner will immediately wonder why he cannot more easily attempt to perform the anastomosis just keeping his loupes on and 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 2½ to 4 times magnification. To adequately visualize the inner lumen of the vas deferens for easy and accurate placement of stitches requires 16X magnification. The advantage of the microscope, in addition to providing higher power magnification, is that the depth of focus is much clearer, your light is constantly

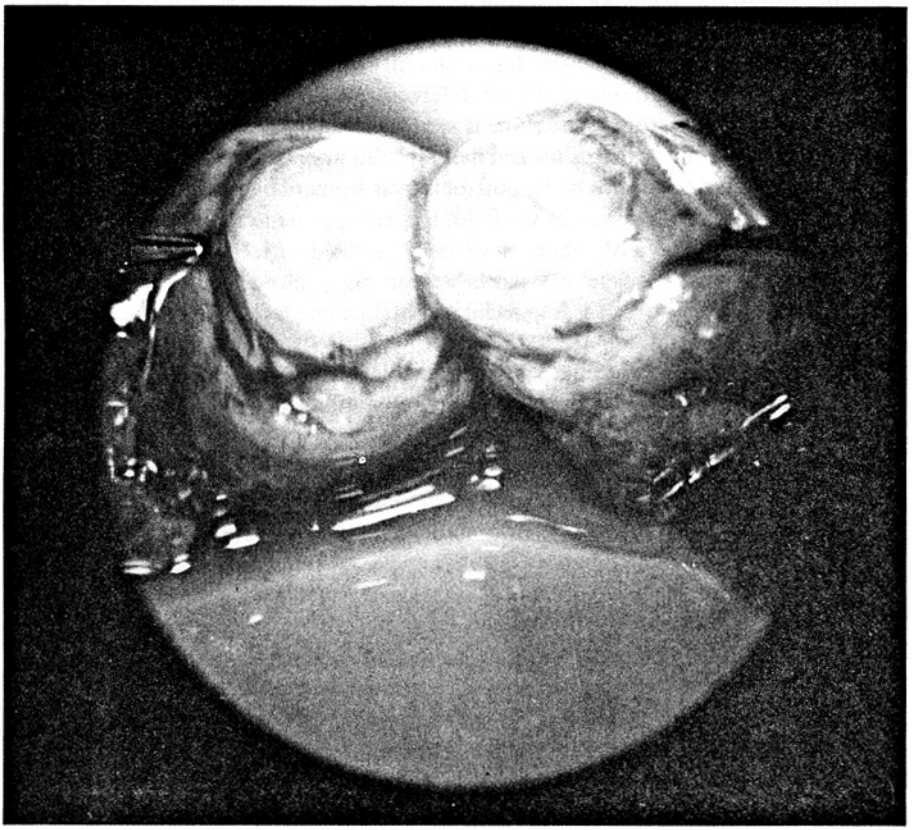


**Figure 4** A photograph demonstrating the lumenal disparity prior to vas reanastomosis.

supplied directly to the subject, and the microscope is resting on a stand and is thus immobile. One can move his head or neck from time to time 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 obstruction (see Fig. 4). The abdominal side lumen will be extremely tiny by comparison. A tiny 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 content.

Microscopic characterization of the sperm in the vas fluid is extremely accurate in predicting success or failure after a properly performed vasovasostomy, and in alerting you to the likelihood of secondary epididymal blockage. It is best not just to smear a slide on the cut end of the vas to obtain the fluid specimen, as this may dry quickly and give an inaccurate impression of the sperm content within the length of the obstructed side of the vas deferens. If the fluid is very concentrated, and a mediocre laboratory microscope is used to look at the specimen, it is very easy to mistake a smear that is packed densely with nonmotile sperm for debris.

The motility of the sperm in the obstructed vas segment has *no* bearing on prognosis. Thus, with nontransparent vas fluid, if there appears to be no sperm, the fluid should be diluted with saline, and possibly stained with H & E, as this may reveal large numbers of sperm in what otherwise might have appeared to be a negative smear. Furthermore, it is important to obtain enough fluid from the vas to be a representative sample.

The inner mucosal anastomosis is performed under 16-25X magnification. The object is to obtain as flawless as possible a mucosa-to-mucosa alignment despite the discrepancy in lumen diameter (see Fig. 5). 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. The suture is pulled through separately and then placed into the mucosa of the testicular side lumen. It is then pulled through, an instrument tie is performed, and the suture is cut.

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

At this point one can easily view the anterior row of sutures from the inside and inspect to see whether perfect mucosal alignment has been achieved. If large bites of muscle were included in these mucosal sutures, the mucosal edges do not come together properly and there is a muscle bridge between the edges of mucosa. There should be no tearing or inaccuracy in the line-up of the mucosal margins. Finally, the outer muscularis is sutured separately.

Semen analysis is obtained every month for the first 4 months, then at 8 months, 1 year, 1.5 years, and 2 years postoperatively. The sperm count and quality tend

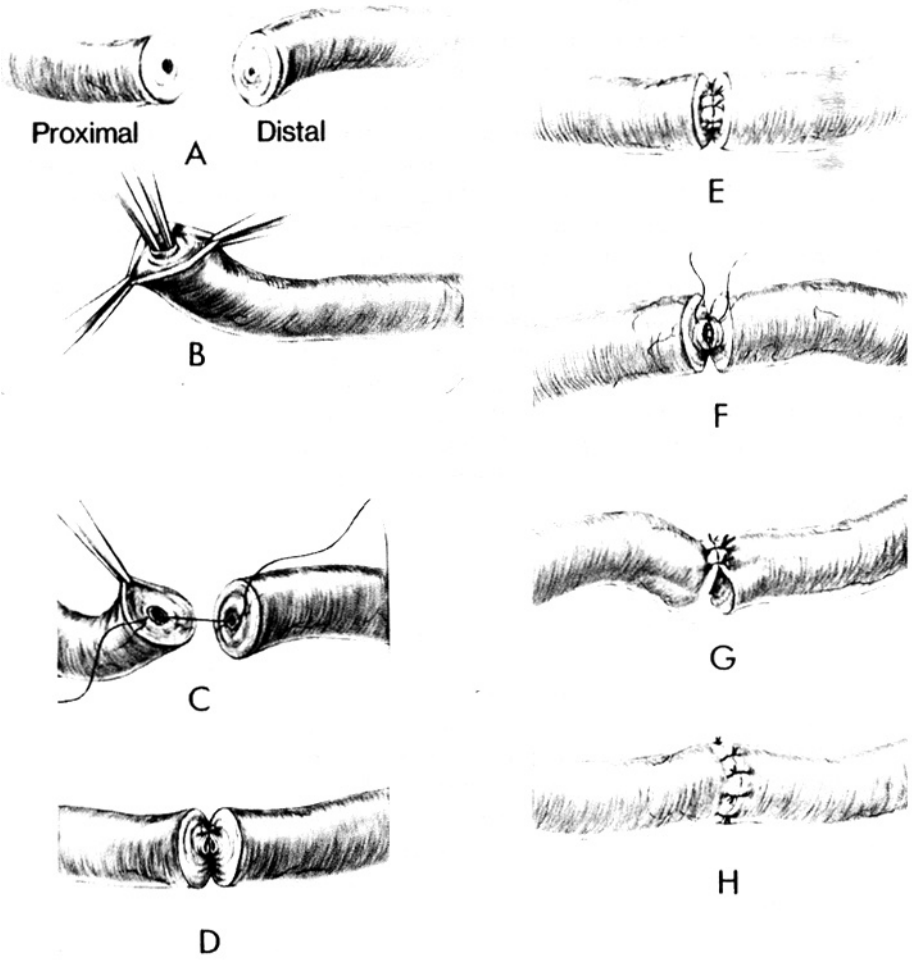



**Figure 5** A diagram demonstrating the steps of the microscopic two-layer vasal anastomosis. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

to gradually improve with time. If the anastomosis is poor, however, the sperm count may increase at first, but then eventually scars down to oligospermia or aspermia. If the patient is azoospermic at 3 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 upon 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), or whether there is a blockage in the epididymis which prevents sperm from reaching the vas. 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 go into the results (and the conclusions which we can draw from these results) of our studies on the effect of vasectomy on the testis and epididymis. We will then delve into practical consequences of this information that may allow us to help those patients who would otherwise remain infertile despite an excellent vas anastomosis.

### Results with Two-Layer Microscopic Technique

From 1975 to 1978, over 400 patients subjected to the two-layer microscopic technique for vasovasostomy were carefully studied both pre- and postoperatively in an effort to determine the factors which affect recovery of fertility (11). The overall pregnancy rate after 1.5 years of follow-up on the first 42 unselected patients was 71%. The 5-year follow-up yielded an 82% pregnancy rate. The causes for the failures will become apparent in the rest of this section. We now have performed over 1800 such cases. Results are similar throughout our long series. Very few patients get pregnant before 6 months and most of the pregnancies begin to occur between 8 months and 1.5 years. An accurate assessment of pregnancy rate is not possible before a series has been followed for at least 2 years. Although some patients achieve normal sperm counts within the first month and impregnate their wives immediately, 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 upon were very very carefully studied. Seminal fluid was sampled from the testicular side of the obstructed vas for each patient at the time of reanastomosis. 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 4 months and then at intervals 4 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. 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.

Sperm counts were arbitrarily considered as normal when there was a concentration of more than 10 million sperm/ml, 50% motility with good progression, and greater than 70% normal forms, according to the criteria of MacLeod and Gold (23). 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/ml. All semen samples with good motility had greater than 70% normal forms. The follow-up on these patients in most cases was superb.

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 (24). This was particularly important when there was no sperm in the vas fluid, and the patient remained azoospermic postoperatively despite a perfect vas anastomosis.

With this kind of careful investigation in patients subjected to as meticulous a microsurgical anastomosis as possible, physiological data of greater reliability were obtainable. These data have led us to conclude that spermatogenesis is not significantly harmed by obstruction (as evidenced by quantitative testicle biopsy in over 300 patients), and that failure to achieve fertility after an accurate vasovasostomy is caused by dilatation and then perforation of the epididymal duct with subsequent secondary epididymal obstruction. We noted that there appeared to be an improved quality of sperm in the vas fluid in patients who had minimal dilation of the testicular side lumen and in patients who had a sperm granuloma at the site of the vasectomy.

Fifty-nine of the first 184 vasa examined had an obvious sperm granuloma noted at the site of vasectomy (an incidence of 32%). That is a much higher incidence than we presently 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 (Fig. 6).

In the group with sperm granuloma all had abundant morphologically normal sperm in the vas fluid. Even when the vasectomy had been performed over 10 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 0.75 mm or less in vasa with sperm granuloma. In patients without sperm

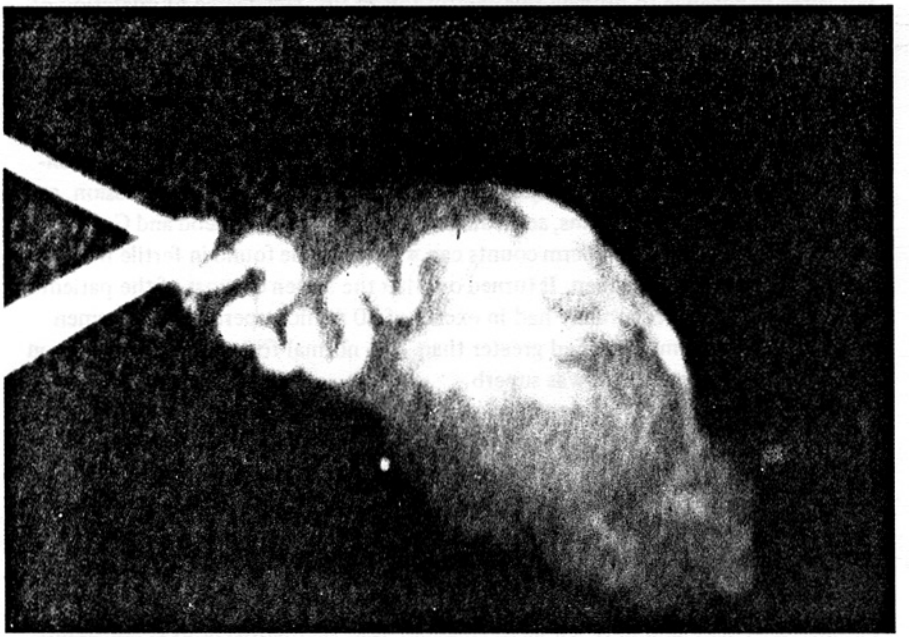


**Figure 6** A vasogram of a sperm granuloma at the vasectomy site demonstrating that this represents continual sperm leakage into a multiplicity of diffuse channels from which sperm fluid can be reabsorbed.

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 anatomical 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 the Testis and Epididymis in Animals

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 1975, 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 6 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 blow-outs and leakages in weak points of the epididymal tubule consequent to the 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.

Howards et al. (25) using micropuncture studies in the rat, demonstrated that all vasectomized animals either had leakage at the vasectomy site or in the epididymis. Pressures were enormous in the epididymis but low in the seminiferous tubules except in the few cases where no sperm leakage had occurred (26).

Galle and Friend (27) noted marked distention of the epididymis and vas in vasectomized guinea pigs with no reabsorption of spermatozoa or spermatozoa parts by epithelial cells or leukocytes within the ductal system. There were a great many degenerating spermatozoa. They postulated that intralumenal disintegration of sperm following vasectomy appeared to be the mechanism for sperm processing rather than sperm reabsorption (28).

In the rat, MacMillan (29) noted that the head of the epididymis became massively distended with spermatozoa and there appeared to be ruptures of the tubules of the head of the epididymis with extravasation of their contents and encapsulation to form an artificial spermatocoele which enlarged progressively up to the time of sacrifice. Eventually the efferent ductules also exhibited changes due to stasis of sperm. In some animals the efferent tubules were totally obstructed, and in others a disorganization of the junctional region between the vasa efferentia and the initial segment of the head of the epididymis was noted. There was little if any damage to the germinal epithelium.

Bedford (30) studied in great detail the effects of vasectomy in four different species of animals. He noted that there was no leukocytic infiltration after vasectomy *unless* rupture occurred somewhere in the duct. He believed that sperm were continually produced after vasectomy, but no reabsorption occurred until there was an epithelial rupture somewhere within the ductule system. By 8 months postvasectomy, the corpus began to show signs of dilation; and a series of lesions, ruptures, and scars could be seen in the epididymis. Interestingly there was less distention and damage in rhesus monkeys than in all the other animals he studied (rabbit, hamster, and rat). Only after epithelial rupture had occurred did leukocytic infiltration and invasion appear, with reabsorption of sperm.

All these studies indicated that there were pressure changes induced by vasectomy that could affect subsequent restoration of fertility even after accurate vas reanastomosis. If the problem was secondary ductal obstruction caused by rupture and sperm extravasation in the epididymis, more sophisticated microsurgery could restore fertility even in the least favorable cases.

Although there is substantial pressure increase in the epididymis, there is no discernible effect upon spermatogenesis or testicular architecture. Since the testicle biopsy showed normal spermatogenesis in all patients who had no sperm in the vas fluid, we felt the problem had to be in the epididymis.

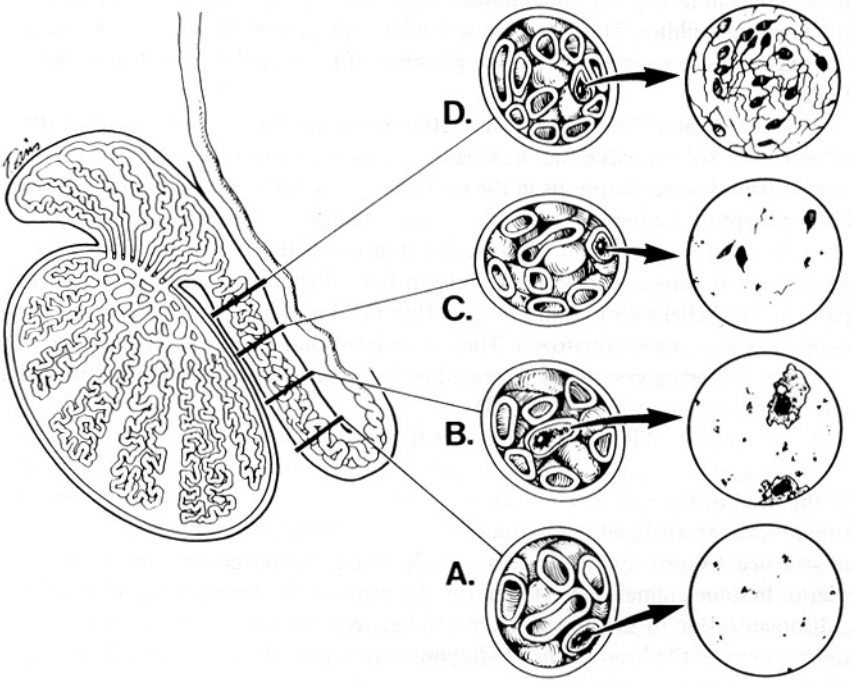



**Figure 7** Stepwise transection of the epididymis from distal to proximal in the course of determining the site of epididymal obstruction. At point A, there is still no sperm or cells seen in the epididymal fluid. At point B, we begin to see macrophages meaning that we were getting close to at least one site of epididymal perforation. At point C, we see some sperm heads and debris, and at point D, we have finally gotten beyond all areas of obstruction and see a multiplicity of normal sperm. Histological sections between point C and point D demonstrate epididymal inflammation, interstitial sperm granuloma, and tubular obstruction. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

To resolve this question, we explored patients who had azoospermia for at least 2 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. In 33 of the 39 such cases first explored, normal sperm were found in the epididymal fluid of the corpus epididymis despite absence of sperm in the vas fluid (see Fig. 7). Epididymal histology distal to this site revealed extensive interstitial sperm granulomas resulting from rupture of the epididymal duct, similar to what Bedford (66) observed in four other species.

Once the epididymal rupture and subsequent blockage occurs, the fluid which had previously accumulated in the vas deferens is trapped there and isolated. The sperm in the vas fluid then eventually degenerate, the tails fall off, and then the heads finally degenerate into amorphous debris (Fig. 8). Thus, the absence of sperm

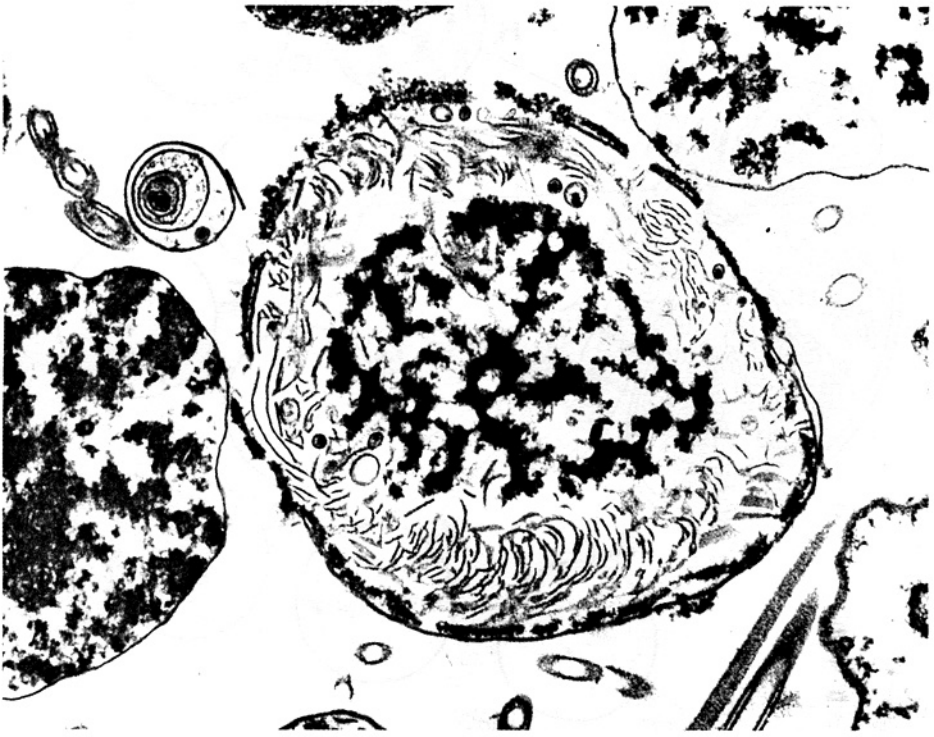


**Figure 8** A degenerated sperm head in the vas fluid of a patient who on light microscopy showed only debris. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

in the vas fluid just proximal to the vasectomy site indicates that there is secondary epididymal blockage from epididymal ruptures caused by the pressure buildup which occurs after vasectomy. That is why the most accurate vasovasostomy cannot result in a success if there is no sperm in the vas fluid. To successfully treat such cases, one would have to bypass the secondary blockage in the epididymis.

We now have experience in over 1800 cases and can state that in every case where there is no sperm in the vas fluid, 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 (see Fig. 9). Fortunately, most of the blockages are limited to the region of the junction of the corpus and tail of the epididymis.

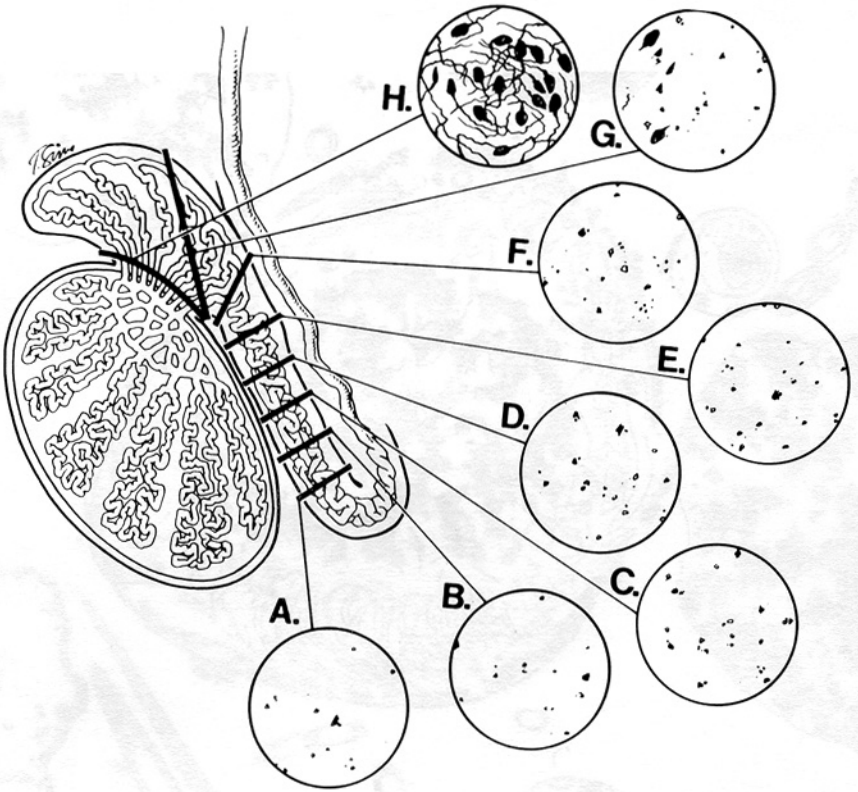



**Figure 9** Similar to Fig. 7, stepwise transection at the epididymis in which no normal sperm are found until the level of the vasa efferentia (H). (From Silber, S. J., *Reproductive Microsurgery*, Williams and Wilkins, Baltimore, 1984.)

Therefore, most of the bypass vasoepididymostomy procedures can be performed either to the distal, or the mid-corpus region where the 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 (66). In humans the time range, however, is considerably expanded. Whenever reversal was performed within 1 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 afterwhich an epididymal blow-out could always be found. The risk of epididymal blow-out on each side gradually increased as an independent variable as the years progressed. The chances of finding no sperm in the vas fluid on either side at 10 years was 75%. However, the chances of finding no sperm on both sides at 10 years was about 50%. At 5 years after vasectomy, the chances of finding no sperm on one side was 25%, but the chances of finding no sperm on both sides was only 6%. 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 and sperm heads within the tubular lumen. 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.

Much of the poor sperm quality seen after vasovasostomy in patients who were failures can be explained by secondary epididymal damage caused by pressure-induced disruptions of the epididymal duct. Of course, the sperm count and the quality of motility may gradually rise over 12 months in vasovasostomy cases who appeared at first to have oligospermia or poor motility in the early postoperative period. But the patients who persist in having oligospermia and poor motility more than 1 year after vasovasostomy usually are found to have partial blockage either at the vasovasostomy site or in the epididymis.

Although this is usually clinically quite obvious, for uncertain cases, we have developed a useful tool which we call simplified quantitative testicle biopsy (24).

## II. 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).

Heller and Clermont first described the histology and kinetics of spermatogenesis in the human (40). 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 cuased by a reduced number of sperm "on the assembly line," but not by reduced speed 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 then developed a method of quantitative interpretation of the testicle biopsy (41,42). Unfortunately, they had a small number of patients, 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., working with Steinberger, counted all components of spermatogenesis, and found a good correlation with sperm count (43). But the difficulty still remained that each quantitative testicle biopsy using the method they described was time consuming, and very few fertility specialists had any inclination to put that much effort into photographing and analyzing the biopsy of each of their patients.

A simplified method of quantitatively interpreting testicle biopsy, which any clinician should be able to utilize with an expenditure of only 10-15 min of his time, is now available (24). We analyzed patients with oligospermia, as well as patients with normal sperm counts, and showed that the testicle biopsy can predict a patient's mean sperm count. We then showed that comparing the results of quantitative testicle biopsy with the sperm count could document whether the oligospermia is caused by a partially obstructed anastomosis.

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 patients' prior fertility is not known, or when documentation is necessary before embarking on a questionable case, this approach should clarify whether or not there is blockage or just poor spermatogenesis 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's solution without being handled. Specimens are carefully fixed, cut in thin sections, and stained with H & E. The technique of biopsy is important. If the specimen is handled roughly or fixed in formalin, it is difficult to accurately identify the cellular components of the seminiferous tubule.

At least 10 seminiferous tubules are included in the count on each side. The testicle biopsy is performed bilaterally. Thus, a total of 20 or more tubules are included in the biopsy evaluation on each patient. Only the mature spermatids (stages I, II, V, and VI) need be counted (see Fig. 10). Put in more simple terms, only the

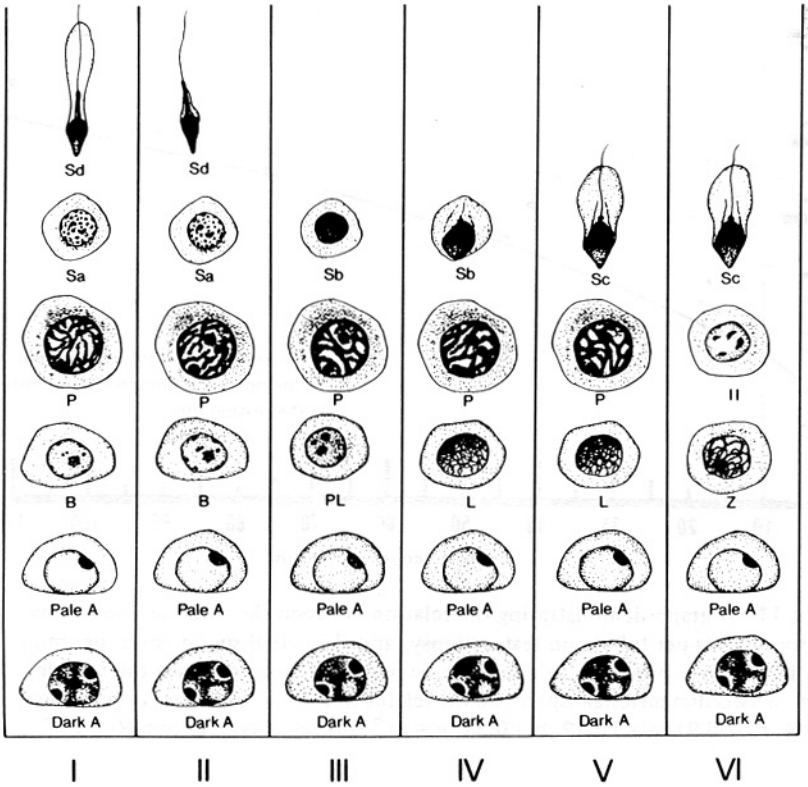



**Figure 10** The six stages of human spermatogenesis in tabular form. The mature spermatids are only seen in stages I, II, V, and VI. Note that the mature spermatids are the easiest cells to denote, and require very little time for quantitation.

oval-shaped cells with dark, densely stained chromatin are counted. All of the steps of spermatogenesis from spermatogonia through resting spermatocyte, leptotene spermatocyte, zygotene spermatocyte, pachytene spermatocyte, and early spermatids ($S_A$, $S_{B1}$) are excluded from consideration. 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 20 tubules and divide that by the number of tubules.

In unobstructed patients with less than 10,000,000 sperm/ml, there are always less than 20 mature spermatids per tubule in the testicle biopsy. In patients with

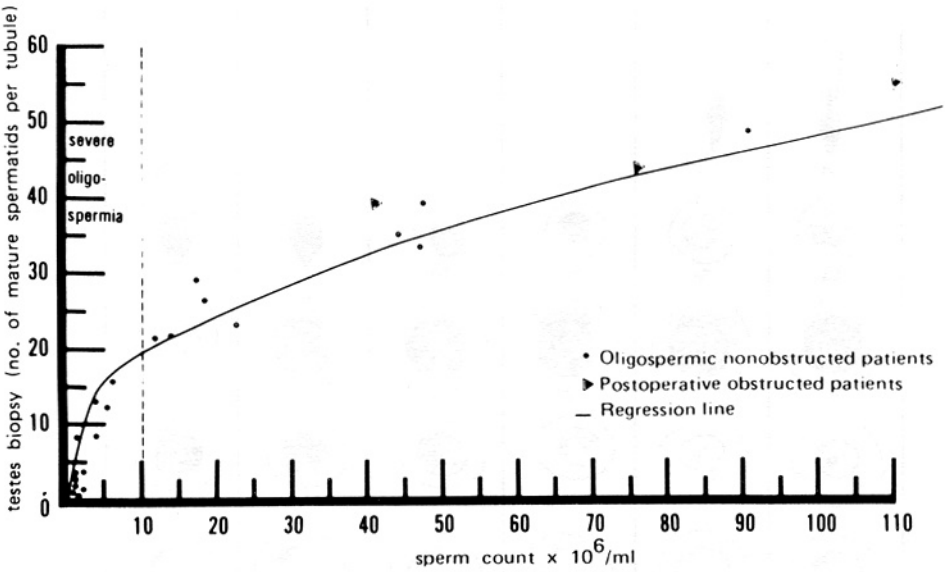



**Figure 11** A graph demonstrating the relation between the total number of mature spermatids per tubule on testes biopsy, and the actual sperm count per milliliter. This is an exponential relationship, as one would expect, since the testicle biopsy is two dimensional. Sperm count relates to a volumetric function of the testicle. $r^2 = 0.91$; $a = 10.39$; $b = 0.32$; $y = ax^b$ (power curve). (From Ref. 24.)

over 10,000,000 sperm/ml, there are usually greater than 20 mature spermatids per tubule. The number of mature spermatids per tubule correlates very closely to the sperm count per milliliter.

Using an exponential curve, summarized in the graph (see Fig. 11), 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 40 mature spermatids per tubule, the sperm count should be just under 60,000,000/ml. If there are 45 mature spermatids per tubule, the sperm count should be just over 85,000,000/ml. If the patient has a sperm count of only 3,000,000/ml, you would expect him to have only six to 10 mature spermatids per tubule.

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. These patients usually have adequate motility because their low count does not reflect any pathology, but just a low count.

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 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 there are tubules filled with spermatocytes and some mature sperm. This impression of normal spermatogenesis has led many nonobstructed patients into an unwarranted vasoepididymos-

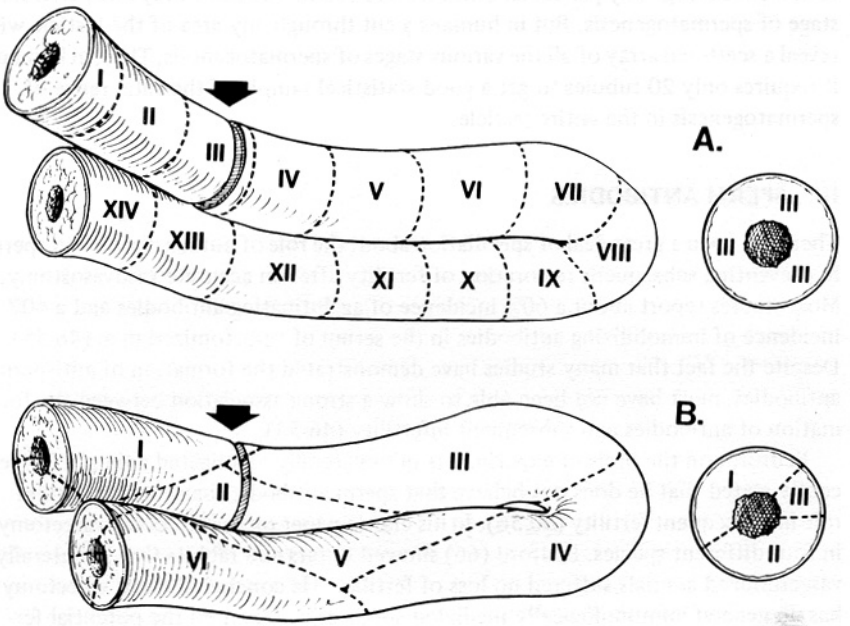



**Figure 12** As seen in A, most animals' spermatogenesis proceeds in an orderly wave across the seminiferous tubule from one stage to another. As seen in B, in the human there is a mosaic, scattered arrangement of the stages of human spermatogenesis which does not proceed in an orderly wave. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

tomy. If the biopsy shows thick tubules with large numbers of spermatocytes but only two or three 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. Unfortunately, the FSH level correlates very poorly with spermiogenesis (44). Patients with maturation arrest causing azoospermia have a normal FSH. Rather 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 (see Fig. 12). In rats, a cut through any particular seminiferous tubule will show only one particular stage of 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.

## III. 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% incidence of agglutinating antibodies and a 40% 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).

Bedford, on the basis of experiments in unilaterally vasoligated animals, has recently stated that he does not believe that sperm antibodies have any important role in subsequent fertility (30,56). In his classic paper on the effects of vasectomy in four different species, Bedford (66) 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 has found *no* correlation between pregnancy or lack of pregnancy and serum agglutinating or immobilizing antibody titers (57). Sperm antibodies were present in the semen in only 3.9% of cases (58). Even in those cases there are pregnancies, but possibly in these few cases there may be some decrease in fertility. Although more work needs to be done on this interesting problem of sperm antibodies, it is clear that the major obstacle to recovering fertility is persistent obstruction, whether partial or complete, either at the vasovasostomy site or in the epididymis.

## IV. MICROSURGICAL VASOEPIDIDYMOSTOMY

The epididymis represents one 20-foot long coiled tubule with myriads of intricate convolutions (see Fig. 13). It is squeezed into a 2-in. length like the pleats of an accordian. Because the epididymal tubule is so tiny, even by microsurgical standards, the results with conventional surgery for this type of obstruction have been very poor. Schoysman has reported the best pregnancy rate with conventional vasoepididymostomy, (25%) (59,60). Amelar and Dubin have reported 22 cases of azoospermia due to epididymal obstruction (61). Thirty-six percent achieved some sperm in the ejaculate after vasoepididymostomy, and only 18% had good semen quality. There were very few pregnancies. Hanley reported only one pregnancy after 83 vasoepididymostomies (62). The procedure utilized by Hanley and that described by Hotchkiss formed the basis for the usual conventional vasoepididymostomy performed by most urologists today (63). 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 described the microsurgical "specific tubule" technique for vasoepididymostomy in 1978 (32-35). After the scrotal sac is entered, the tunica vaginalis is opened and the testis and epididymis are everted from the hydrocele sac. The dilated epididymal tubule is usually about 0.1-0.2 mm in diameter. The epididymal duct is extraordinarily delicate, with a wall thickness of about 30 $\mu$m. 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 as many as 20 or 30 microscopic size tubules (see Fig. 14). Without the benefit of microscopic observation, there is an illusion that sperm fluid is welling up 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 directly anastomose (end-to-end) the inner lumen of the vas deferens specifically to the one epididymal tubule.

Rather than making a conventional longitudinal incision, a *transverse* transection of the epididymis is made at the most distal point; i.e., at the junction of the cauda

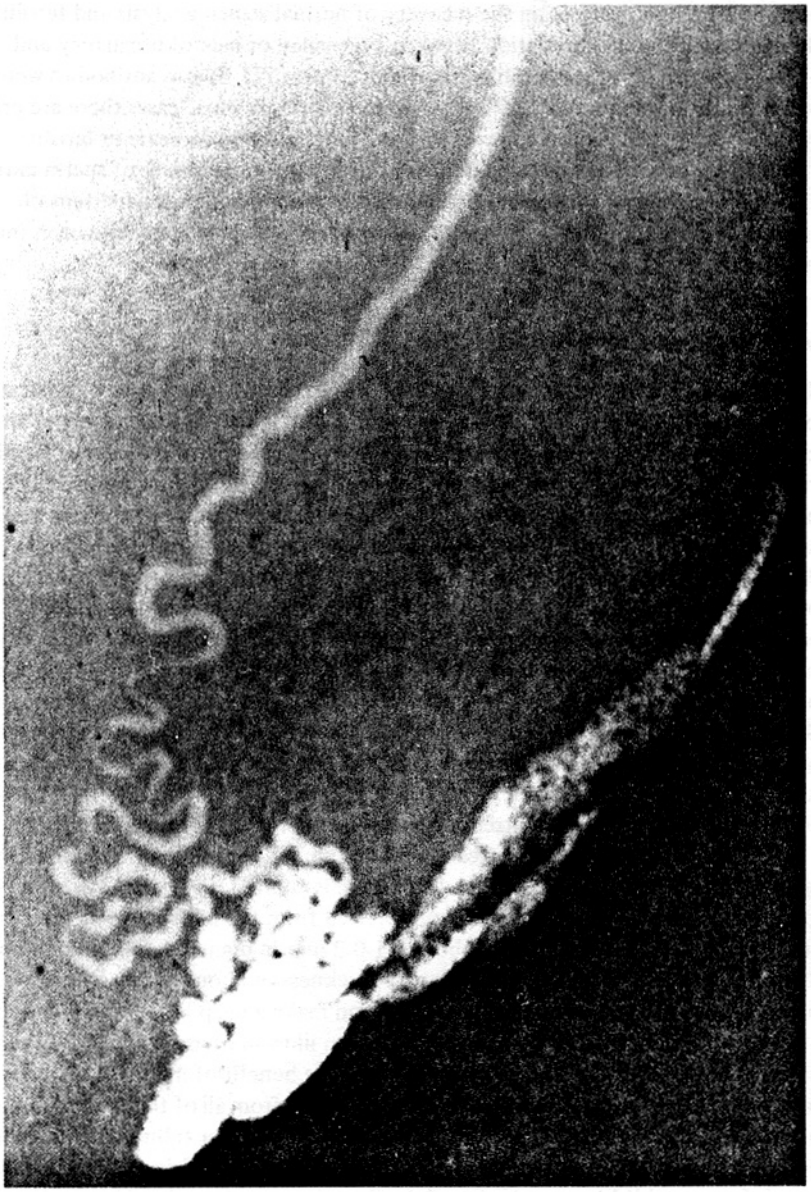


**Figure 13** A retrograde vasogram of the vas deferens and epididymis demonstrating that the epididymis is simply one long, intricately coiled tubule which is continuous with the convoluted region of the vas deferens.

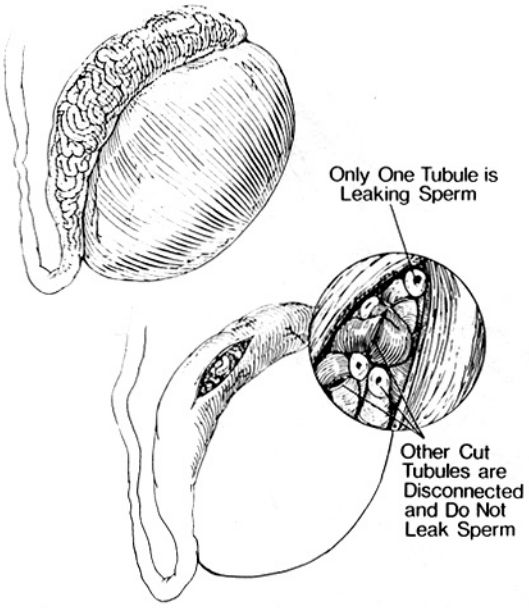



**Figure 14** A view of the first stage of a conventional approach to vasoepididymostomy in which a longitudinal slit is made in the tunic of the epididymis, cutting the one epididymal tubule in so many different places that it looks like many cut tubules. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

and corpus epididymis (see Fig. 15). 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. Under the operating microscope, three to 10 cut tubules are usually visible on the transected surface of the epididymis, and all are carefully examined for the 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 distalmost level where normal sperm are found in the epididymal fluid. This allows for the maximal possible length of epididymis.

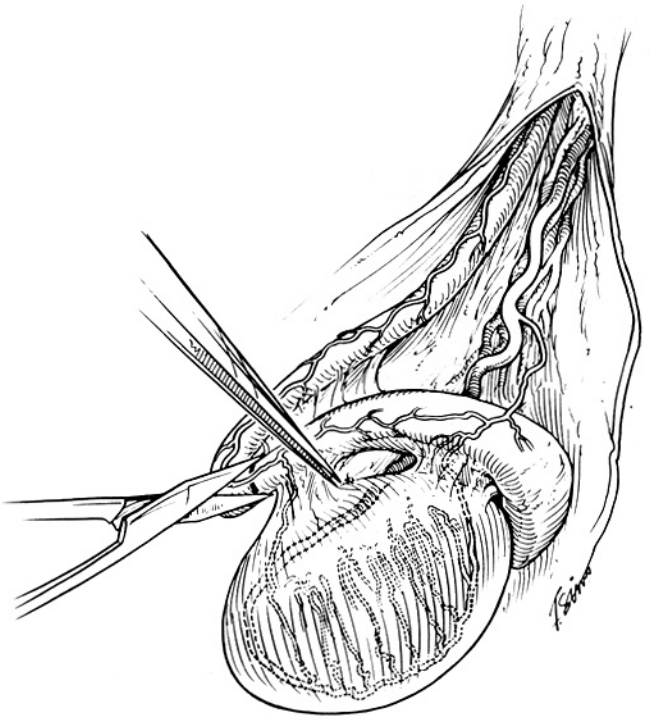


**Figure 15** The demonstration of the freeing up of the epididymis without damaging the blood supply in preparation for a transverse sectioning of the epididymis in preparation for a specific tubule-to-tubule vasoepididymostomy. (From Silber, S.J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

For the first stitch the epididymis is held between the thumb and the forefinger, facing the microscope. A slight milking action may sometimes be necessary to promote a continual efflux of fluid in order to continue to see which is the correct tubule to anastomose; 9-0 or 10-0 monofilament nylon on a GS-16 or BV-9 needle is used. The first suture is placed from the outside to the inside of the specific epididymal tubule which 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 in the other jaw. A blue piece of plastic is then placed underneath the epididymis and vas, which are 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 (Figs. 16 and 17).

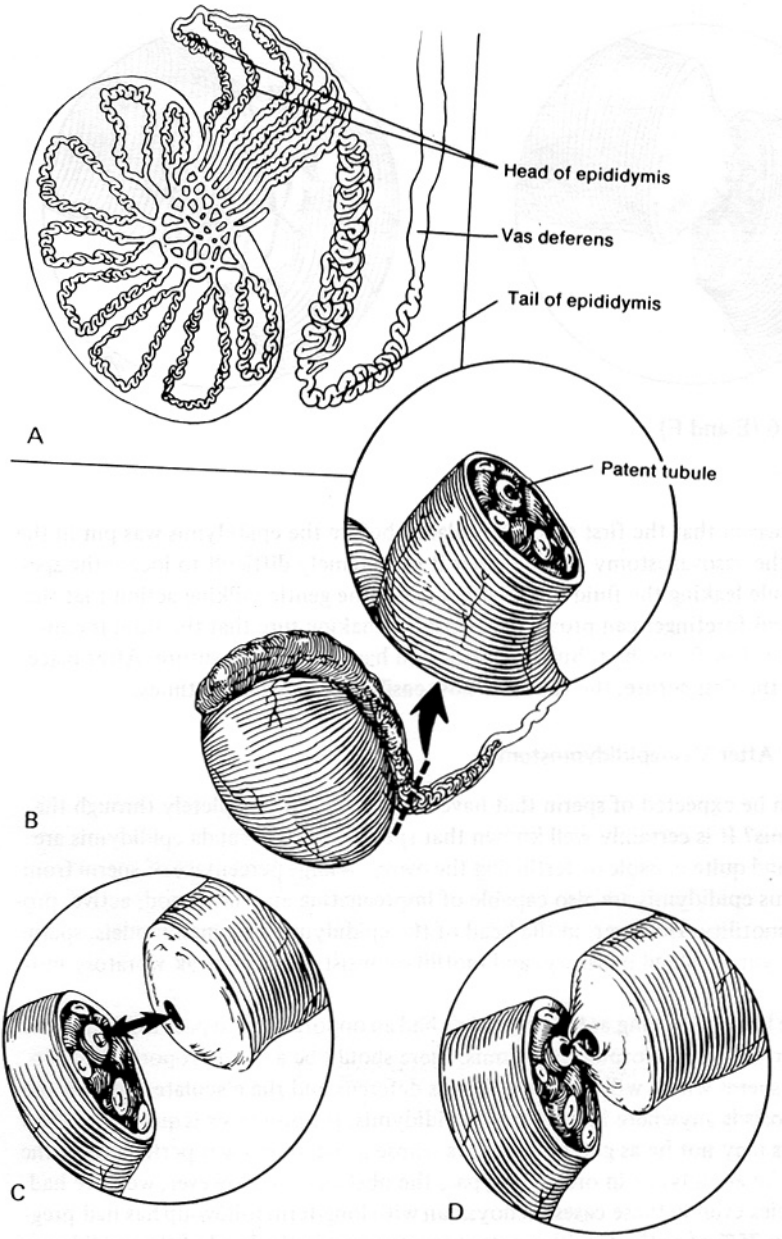



**Figure 16** Diagrams of the specific tubule technique for vasoepididymostomy which we first described in 1978. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

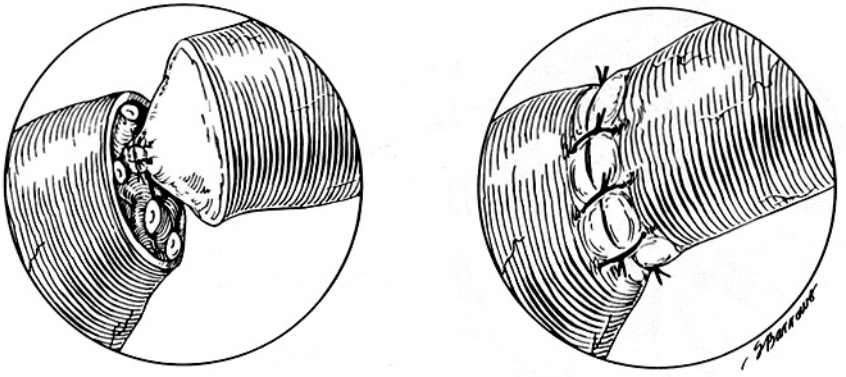


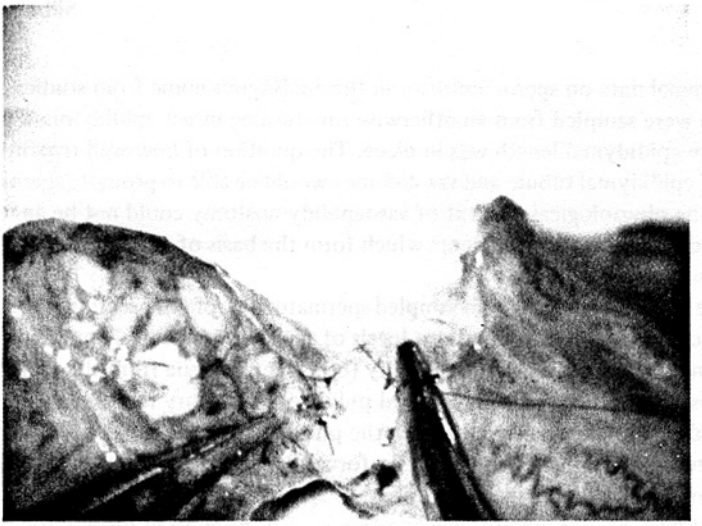
Figure 16 (E and F)

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.

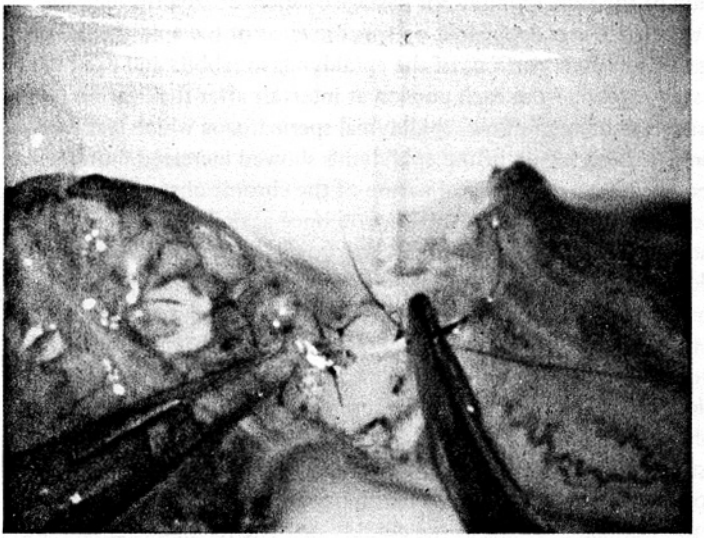
### Fertility After Vasoepididymostomy

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 quite 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 should be a good proportion of fertilizable sperm which will pass into the vas deferens and the ejaculate. Thus, if the anastomosis is anywhere in the corpus epididymis, the prognosis is quite good. The prognosis may not be as good in patients whose anastomosis was performed in the head of the epididymis in order to bypass the obstruction. However, we have had pregnancies even in these cases. Schoysman with long-term follow-up has had pregnancies in 25% of patients with a patent anastomosis in the head of the epididymis (64).



(a)



(b)

**Figure 17** Photographs of the final sutures being placed for an anastomosis of the vas lumen to the specific epididymal tubule. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

All of the animal data on sperm maturity in the epididymis come from studies in which sperm were sampled from an otherwise functioning intact epididymis in which the entire epididymal length was in place. The question of how well remaining segments of epididymal tubule and vas deferens would be able to promote sperm maturation in the physiological 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, Gaddum sampled spermatozoa from the seminiferous tubules, the ductuli efferentes, and various levels of the epididymis to determine their intrinsic motility and fertilizing capability (65). Spermatozoa from the seminiferous tubules and ductili efferentes showed only weak vibratory movements with no forward progress. Spermatozoa from the proximal head of the epididymis showed very 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 Bedford in the rabbit and by Orgebin-Crist in the rabbit (66,67).

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 been poorly motile in the caput region of the epididymis showed increased motility.

However, because of the pathological nature of the chronic obstruction created by such an experimental model, all of these sperm once again lost their motility by 3 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 adequately be tested. 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.

Orgebin-Crist, first suggested to use that in human vasoepididymostomy it was theoretically possible that sperm might be mature and be 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 after a prolonged period of time, the remaining segment of the vastly shortened epididymis in some compensatory fashion may restore motility to spermatozoa in a region where under normal circumstances they would not be motile.

In most patients undergoing vasoepididymostomy, the proximalmost obstruction will be somewhere in the corpus region, and usually in the distal corpus. We see no significant difference in pregnancy rates thus far at any particular point along the corpus epididymis. Ninety percent of patients undergoing vasoepididymostomy with this technique have a semen analysis consisting of a sperm count of greater than 10 million/ml and directional motility of greater than 50%. It appears to take a longer time in many cases for motility to reach high levels in vasoepididymostomy patients than in vasovasostomy patients, and the sperm count also may not always come up to normal levels until a year and a half postoperatively. This indicates that a considerable period of time is required in some of these patients for sperm transport mechanisms to completely recover. But nonetheless, by 1.5 years 90% of the patients have an adequate semen analysis.

About 60% of these patients impregnate their wives within 2-3 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 somewhat less in these patients than the normal population. Thus, it will take 5 years of follow-up before we can state with assurance how high the pregnancy rate will eventually be. Our impression, however, is that the results with vasoepididymostomy will parallel those with vasovasostomy in that there will be no serious discrepancy between good semen analysis and eventual pregnancy.

Our very high pregnancy rate with vasovasostomy patients (82%) correlates well with semen findings. In similar fashion we expect with longer follow-up that the vasoepididymostomy pregnancy rate will also correlate well with semen findings. Some of our earliest vasoepididymostomy patients had normal semen analyses for as long as 3 years before the wives finally became pregnant. With longer follow-up, therefore, we suspect that although there may be decreased fertility in many of these vasoepididymostomy patients, so long as the semen parameters are normal, the majority of them will eventually impregnate their wives.

### How to Evaluate an Infertility Patient for Obstruction Not Caused by Vasectomy

The diagnosis of obstruction should really be quite simple. However, it is sometimes approached in a confusing way which can lead to embarrassing situations; e.g., where one finds himself attempting to do a vasoepididymostomy on a patient who has no obstruction. But adherence to a few simple principles will avoid these difficulties and allow a proper decision to be made preoperatively rather easily.

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 also 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 ml), you might worry about ejaculatory duct obstruction. However, if the patient does have ejaculatory obstruction, this can be handled by transurethral resection (TUR) after vasography.

If the semen volume is over 1.0 ml, then you do not have to worry about ejaculatory duct obstruction, which is quite rare anyway.

If the patient has a normal FSH, that 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 it 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 vasogram as an isolated diagnostic procedure creates many problems. Firstly, a scrotal exploration is not needed to ascertain that the vas is present; that should be easily discernible upon physical examination. Secondly, 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. Thirdly, the vasogram data is 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.

Many urologists mistakenly feel that they 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 injected in the epididymal direction will give the illusion of epididymal obstruction at some point; but there is truly no obstruction. What usually happens with forceful injection like this is that iatrogenic blow-outs will be created, further complicating the 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.

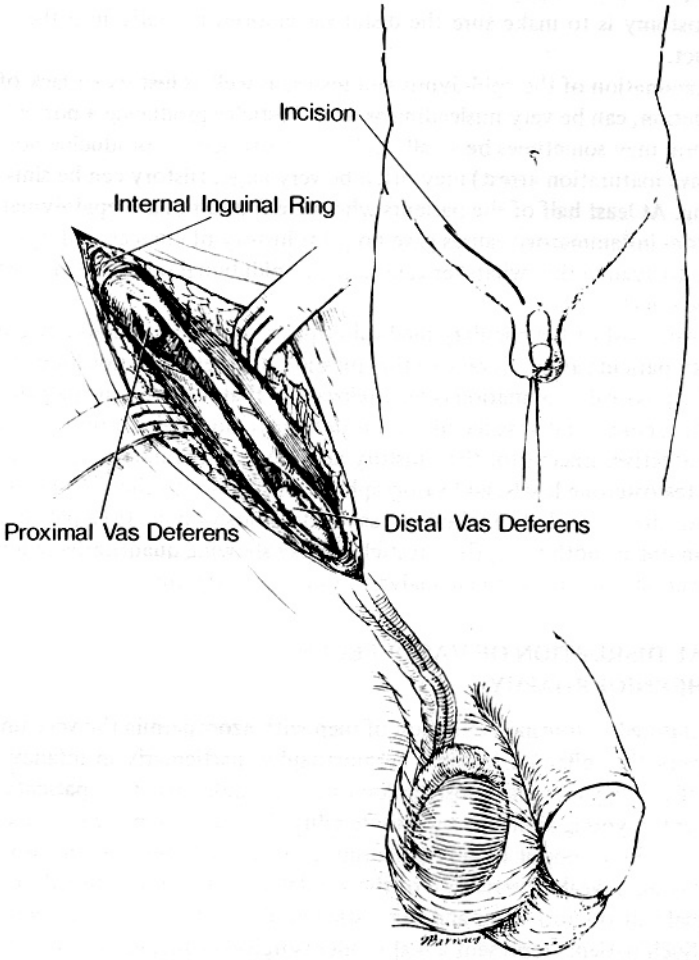
Physical examination of the epididymis and testes, as well as history or lack of history of infection, can be very misleading as well. Testicles producing a normal amount of sperm may sometimes be small, and testicles which are producing no sperm (that have maturation arrest) may often be very large. 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 are 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: (a) a palpable vas deferens on one or both sides, (b) a testicle biopsy showing quantitatively normal spermatogenesis, (c) and a semen analysis showing azoospermia.

## V. INGUINAL DISRUPTION OF VAS DEFERENS AFTER HERNIORRHAPHY

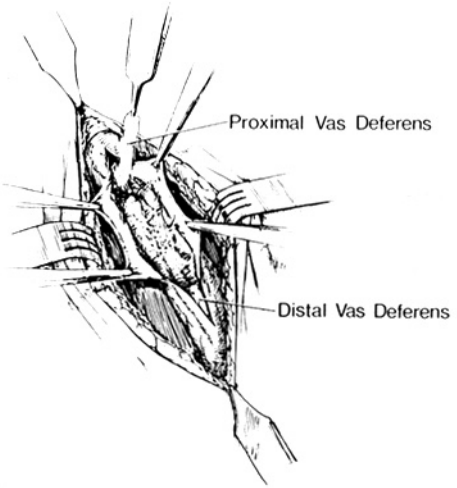
We have encountered in routine evaluation of men with azoospermia the very unsettling discovery that bilateral inguinal herniorrhaphy, particularly in infancy, carries a risk of causing iatrogenic obstruction of the vas deferens. Such patients generally present in young adulthood with infertility. The semen analysis shows azoospermia and the urologist usually performs a testicular biopsy and a vasogram. The vasogram reveals obstruction of the vas deferens near the external or internal inguinal ring on both sides and the testicular biopsy shows normal spermatogenesis. Such patients represent a major microsurgical challenge because of 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 (see Fig. 18a). One can usually expect to find either no vas deferens in the inguinal canal or some length of vas missing, and he must free up enough vas deferens to bridge the gap (see Fig. 18b).

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 at a separate time. 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 very high suspicion for interruption of the vas deferens in the inguinal area.




(a)

**Figure 18** Demonstration of the problem encountered when attempting to anastomose a vas deferens that has been severed accidentally at the time of a herniorrhaphy repair. (From Silber, S. J., *Reproductive Microsurgery*, Williams and Wilkins, Baltimore, 1984.)




(b)

Our findings should give some concern to those pediatric surgeons who advocate bilateral inguinal 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% of infant hernia sacs may be found to contain vas deferens, a bilateral infant herniorrhaphy may conceivably sterilize as many as 2% of children. In addition, 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 to accidently include it in his ligation of the sac at the internal ring.

### Crossover Vasoepididymostomy

Frequently one sees a case where there was inguinal disruption of the vas deferens caused by herniorrhaphy on one side only, but the patient is still azoospermic because on the other side the testicle was atrophic. In this situation, the extensive operation of inguinal vasovasostomy or inguinal vasoepididymostomy can be avoided by using a simple crossover procedure (Fig. 19). On one side the inguinal vas deferens is completely intact, but there is no sperm production from the testicle on that side. On the other side the testicle is normal, but the inguinal vas deferens has been obstructed or destroyed by herniorrhaphy. It is far easier to make an opening in the median scrotal raphe and pull the vas deferens from the healthy side through the raphe to the other side. Then either the scrotal vas deferens (or epididymis) can be anastomosed in a crossover fashion to the inguinal vas deferens

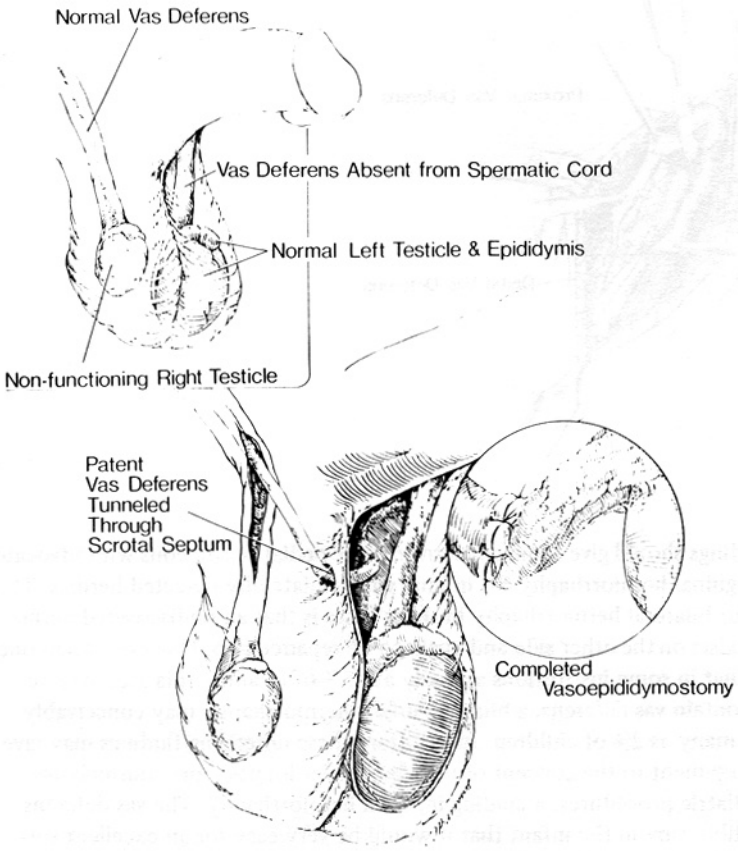



**Figure 19** A diagram of the crossover vasovasostomy or vasoepididymostomy. (From Silber, S. J., *Reproductive Microsurgery*, Williams & Wilkins, Baltimore, 1984.)

from the opposite side (see Fig. 19). These procedures are highly successful and much easier to perform both for the surgeon and the patient than trying to go through the area of the previous herniorrhaphy to bridge the gap of the missing vas deferens.

## VI. EJACULATORY DUCT OBSTRUCTION

A rare cause of azoospermia is congenital 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 very 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 blow-out in the epididymis from this longstanding obstruction. In that event, a vasoepididymostomy is first performed 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 (72,73). 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 embryological remnant of the mullerian duct—the fetal uterus and upper vagina. The ejaculatory duct does not enter it.

It is very 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 very rapidly in these patients. One of the ancillary benefits of the procedure is that they now have a reasonable volume ejaculate. A normal sperm count with good motility will return within 3-8 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 these cases despite their rarity. One usually does not make the diagnosis definitively before surgery but comes across it at the time a vasography is performed while the patient is undergoing exploration for probable epididymal obstruction and low semen volume. However, one can be suspicious of ejaculatory duct obstruction

when there is normal spermatogenesis on testicle biopsy, azoospermia, a palpable vas deferens, and low semen volume with absent fructose.

There have been some unwarranted cases of TUR for ejaculatory duct obstruction in patients without ejaculatory duct obstruction. When there is no cystic 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. Furthermore, we have not seen ejaculatory duct obstruction to ever cause oligospermia. Thus, we caution that this rare condition not be diagnosed over frequently.

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