Review Article

Reversal of Vasectomy and the Treatment of Male Infertility

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In the last 125 years the population of the earth has increased from one billion to four billion. Some authorities predict that by the turn of the century the population may reach ten billion. In the face of diminishing resources, this staggering population growth is a major crisis.

One of the most popular and effective solutions to the problem of birth control and family planning is vasectomy. Its only disadvantage has been its irreversibility. Until very recently, the surgical techniques for reuniting the severed vas have been clumsy and fraught with failure. Furthermore, most clinicians performing vasectomy have not been aware of the associated pressure-mediated effects on the epididymis and testis.

The first step in solving the problems associated with reversing vasectomy was the development of a very accurate technique for microsurgically reuniting the vas deferens (Silber, 1977a). This new technique resulted in a dramatic improvement in postoperative semen quality and pregnancy rate. Indeed, of the patients who had had unsuccessful vasectomy reversal operations by competent clinicians, 88% had semen analysis within the normal range after a second operation with microsurgery. Seventy-one percent achieved pregnancy.

Once the technique of reuniting the vas deferens had been improved, we were able to carefully study the secondary effects of the vasectomy itself on the likelihood of recovering fertility (Silber 1977a,b; 1978a,b,c; 1979; Silber and Cohen, 1978, 1979). In a previous article (Silber 1978c), we reviewed the many conflicting studies regarding the effects of vasectomy on the epididymis and testicle. It was clear that reversibility was related to the chronic pressure changes caused by vasectomy. Thus, the second step in improving vasectomy reversal was to define the pressure-mediated changes attributable to vasectomy and to devise a procedure by which these effects could be eliminated.

We identified the pressure-mediated effects of vasectomy on the epididymis in over 300 cases and determined how microsurgery of the epididymis may be used to solve this problem in many cases. This review deals with results obtained in these patients and addresses the problem of modifying the technique of vasectomy itself to limit the pressure effects and thus make vasectomy more reversible. Finally, it will be mentioned how this new understanding has helped to improve surgical approaches to obstruction not caused by vasectomy.

Effects of Pressure Increase After Vasectomy

Because vasectomy is essentially a painless and symptomfree condition, urologists have been unaware of some rather obvious physiologic changes in men after vasectomy that profoundly affect reversibility. After vasectomy, there is a pressure increase transmitted back to the epididymis that causes substantial dilation and distention. In over 1000 vasovasostomies performed under an
operating microscope, we have always observed some degree of dilation of the lumen of the vas deferens (which often cannot be seen with the naked eye) as well as congestion of the epididymis and resultant dilation of the epididymal tubule.

A great deal of fluid is secreted by the testis into the epididymis. The majority of this fluid is reabsorbed by the epididymis. Despite this reabsorption, every patient who is vasectomized develops varying degrees of epididymal dilation and distention. However, studies in several species indicate that there is no easily discernible effect upon the testis (Turner et al., 1977; Van Wagenen, 1924, 1925; Smith, 1962; Neaves, 1973; Setchell, 1971; Bedford, 1976; Pardanani et al., 1976).

We observed that the pressure changes were less pronounced when a sperm granuloma developed at the vasectomy site. Furthermore, sperm quality in the vas fluid was always superior when a sperm granuloma occurred at the vasectomy site. When there was no such sperm granuloma, frequently the sperm quality was not very good, or sperm were absent. The likelihood of finding normal sperm in the vas fluid at the time of vasovasostomy decreased as the duration of time since the original vasectomy increased. The best results with vasovasostomy were obtained when morphologically intact sperm were seen in the vas fluid at the time of vasovasostomy. The worst results occurred in patients who had no sperm in the vas fluid at the time of vasovasostomy.

Even though the aforementioned observations had been made, we still had to identify the physiologic mechanism by which a pressure increase after vasectomy decreased the likelihood of successful vasectomy reversal. We also had to determine how there could be no sperm in the vas fluid despite normal spermatogenesis. Thus, we performed microsurgical epididymal explorations on all patients who had azoospermia after an anatomically perfect vasovasostomy and found secondary epididymal obstruction to be the cause of failure.

The duration of time since vasectomy correlates with the likelihood of pressure-induced rupture of the epididymis. In all cases in which the vasectomy reversal was performed within one year of the vasectomy, high quality sperm were always found in the vas fluid and normal semen analyses were obtained after surgery. (In these cases, the request for reversal was usually prompted by a crib death.) On the other hand, 50% of patients who underwent vasectomy more than ten years prior to reversal did not have sperm in the vas fluid on either side at the time of vasovasostomy. Postoperatively, these 50% remained azoospermic. When reversal was performed within ten years of vasectomy, there were often no sperm found on one side, but since the other side did have sperm, the patient usually recovered a normal sperm count. Thus, the duration of time since the vasectomy is a very important factor in influencing the likelihood of a successful reversal.

The presence of a palpable sperm granuloma at
the vasectomy site was always associated with good quality sperm in the vas fluid. In cases in which a unilateral sperm granuloma was present, sperm were always found in the vas fluid on the affected side (even though there may not have been sperm present on the contralateral side). Furthermore, in the presence of a sperm granuloma, the vas lumen was rarely dilated to more than $\frac{3}{4}$ mm in diameter, whereas in the absence of a sperm granuloma, the vas lumen was usually dilated to over 1.0 mm in diameter. Thus, a sperm granuloma is a site of continuing leakage with reabsorption of vas fluid, and acts as a safety valve, decompressing the vas and preventing too great a build-up of pressure.

Even when no sperm were found in the vas fluid and the patient remained azoospermic after vasovasostomy, the testicle biopsy was always normal. Therefore, we looked more closely at the epididymis. In patients in whom there were no
sperm in the vas fluid, pressure-induced epididymal ruptures were observed. As a result, sperm leaked from the epididymal tubule into the interstitium, causing secondary obstruction. In over 80% of the cases, this epididymal disruption caused by pressure-induced ruptures occurred at the junction of the corpus and tail of the epididymis, just where the relatively muscular caudal epididymal tubule thins out into the very delicate tubule of the corpus. In some cases, however, disruptions were found farther up the corpus epididymis or in the head of the epididymis. In every case in which there were no sperm in the vas fluid, we were able to locate a point in the epididymis at which normal sperm were found in the fluid (even if we had to go up to the vasa efferentia).

These secondary obstructions were difficult to locate. Only by transecting the epididymis serially and subjecting the sections to histologic examination could the specific focal areas of epididymal sperm granuloma and secondary obstruction be discovered. Gross examination of the epididymis rarely revealed any useful information, except that, as with any vasectomized patient, there was epididymal dilation. But in patients who had secondary epididymal disruption, this dilation appeared no different to the naked eye than in patients who had no secondary epididymal disruption.

Thus, we concluded that the secondary effects of pressure build-up on the epididymis after vasectomy can prevent fertility even after an accurate vasovasostomy. The longer the duration of time since vasectomy and the greater the pressure build-up, the greater is the likelihood of epididymal extravasation and secondary obstruction. The presence of a sperm granuloma at the vasectomy site, indicating continual leakage and reabsorption, eliminates the risk of epididymal rupture.

**Microscopic Vasoe epididymostomy: Specific Microanastomosis to the Epididymal Tubule**

We now realize that in the special cases in which there are no sperm in the vas fluid or in which fertility does not result after a perfect vas reanastomosis, bypass of the area of the secondary epididymal obstruction affords a chance for successful reversal. The problem in the past was that vasoe epididymostomy with most conventional techniques yielded low success rates (from 2–10%). The epididymal tubule is so tiny and so delicate that the gross techniques available were rarely successful. The fallacy in the conventional approach to vasoe epididymostomy is the notion that if one merely makes a slice in the epididymal tunica, there will be multiple tubules, all of which will be oozing sperm. Of course, the epididymis is just one 20-ft-long coiled tube. Therefore, no matter how large an incision one makes longitu-
dinarily in the epididymal tunic, only the distal end of the tubule that remains intact and connected with the efferent ductules is the genuine source of leaking sperm. Without well-controlled microscopic observation, it is difficult to identify this particular section because it appears as though the sperm fluid is welling up from all of the cut ends of the tubule. The only rational surgical approach is to suture the inner lumen of the vas deferens directly to that one cut end of the epididymal tubule that is leaking the sperm fluid.

Conventional gross vasoepididymostomy is a very crude operation that, for success, relies upon the formation of a sperm fistula. Instead of relying on the formation of a sperm fistula, we prefer an accurate anastomosis. The technique involves serial sectioning of the epididymis until a level proximal to the obstruction is reached, where intact sperm are found leaking from the epididymal tubule. When one first observes the transversely sectioned epididymis, there may appear to be eight or ten cut tubules, but of course only one of those tubules is leaking sperm. The other openings are merely cut ends of segments of this one convoluted tubule that have been disconnected from the proximal portion of the tubule. Thus, by close observation using the operating microscope, one can see the fluid emerging from only one of these cut ends of the tubule. This is the one end of the tubule that is directly anastomosed to the inner lumen of the vas deferens. Then the outer muscularis of the vas deferens is sutured to the epididymal tunic for support.

It is not merely the introduction of an operating microscope that is important in our approach to vasoepididymostomy. If we were to make a longitudinal slit in the epididymis in the traditional fashion, even the aid of an operating microscope would not allow us to identify the specific site to which the vas lumen should be anastomosed. In fact, it is rather irrational to make a longitudinal slit, since what is needed is to locate a site proximal to the level of epididymal obstruction and to conserve as much epididymal length as possible. Thus, it makes more sense to serially section the epididymis, going from the caudal region proximally until one crosses that transition zone from finding no sperm in the epididymal fluid to finding the epididymal fluid loaded with sperm. If only a longitudinal incision were made, it would be very difficult to locate this transition point.

This operation is extraordinarily delicate. Unlike microscopic vasovasostomy, vasoe epididymostomy requires more than laboratory practice. It requires a great deal of experience and seasoning with all kinds of microsurgical techniques. If a vasectomy reversal fails, it can always be performed again with a more accurate technique. But if a vasoe epididymostomy fails, the scarring that develops around this delicate structure makes subsequent operations extremely difficult.

About 80% of patients upon whom we have performed a bilateral vasoe epididymostomy have recovered normal semen characteristics. When the obstruction was in the distal corpus, sperm motility quickly returned to normal in most cases. When the vasoe epididymostomy had to be performed in proximal regions of the corpus or in the head of the epididymis, motility initially did not return to normal. However, prolonged follow-up (two years) disclosed that normal motility did eventually develop.

Thus, we now have a method for successfully reversing vasectomy even in patients who otherwise have a poor prognosis because of pressure damage created by this condition. However, this technique is very difficult and should be attempted only by one experienced in microsurgical techniques. It is more reasonable to perform vasectomy in such a way as to minimize this pressure-mediated damage in the first place.
Vasectomy Technique Assuring Greatest Likelihood of Reversibility

Sperm granuloma (with subsequent spontaneous recanalization) has classically been considered a complication of vasectomy (Shapiro and Silber, 1979). Yet, if spontaneous recanalization does not occur, the formation of a sperm granuloma at the vasectomy site may be beneficial in that it reduces the amount of pressure in the epididymis and eliminates the likelihood of eventual epididymal rupture. When cauterization is used to seal the vas at the time of vasectomy, the incidence of sperm granuloma is around 1%, and recanalization is rare. On the other hand, when ligature techniques are used for sealing the vas deferens, sperm granulomas will form in up to 30% of cases, and the spontaneous recanalization rate is about 1–2%. The mechanism for spontaneous recanalization is that sperm leak out through the cut testicular end of the vas deferens and swim through connective tissue, grinding a pathway to the other side. Most spontaneous recanalizations result in a low sperm count and poor motility. Such recanalizations usually become completely blocked by scar tissue, resulting in azoospermia. However, an occasional spontaneous recanalization will result in permanently restored fertility. Thus, the formation of a sperm granuloma at the vasectomy site, although desirable for later reversibility, cannot be taken lightly unless there can be some assurance that spontaneous recanalization will not occur.

However, a great deal of emotionalism has clouded our feelings about sperm granulomas. Certainly, in performing a vasectomy one must accept the almost inevitable occurrence of sperm granulomas. If a sperm granuloma does not form at the vasectomy site, then it will eventually form in the epididymis at the site of a rupture of the epididymal tubule induced by the pressure build-up. It is simply a question of where one would rather have the sperm granuloma, at the vasectomy site or in the epididymis. In the context of vasectomy reversal, it is better to have the sperm granuloma at the vasectomy site.

As to whether or not such a sperm granuloma causes pain, we would like to cite our experience with an objective review of 1000 men upon whom we performed vasovasostomy from one month to 28 years after vasectomy. In over 1000 such patients, we have very rarely found a sperm granuloma to be a source of discomfort. In fact, patients with a sperm granuloma were less likely to have epididymal tenderness than patients who had no sperm granuloma at the vasectomy site. Furthermore, in patients who had a sperm granuloma on only one side, the side with the sperm granuloma usually had no epididymal tenderness, and the side without the sperm granuloma frequently had epididymal tenderness. Oddly enough, pressure-induced epididymal rupture, discovered at the time of vasectomy reversal, was not a source of much pain either. Patients with the most troublesome epididymal tenderness usually did not have epididymal rupture. It appears to be the pressure build-up within the epididymis that causes most of the mild intermittent orchialgia that sometimes occurs in postvasectomy patients.

Of ten patients who were referred to us because of persistent discomfort many years after vasectomy and who did not wish to be fertile again, only two had a somewhat tender sperm granuloma. The other eight had no sperm granuloma but did have marked epididymal tenderness. After performing vasovasostomies on these eight patients to relieve the pressure, the symptoms of discomfort and the epididymal tenderness subsided (Shapiro and Silber, 1979).

Thus, it would be inappropriate for us to suppose that one can perform vasectomies without the risk of some scrotal discomfort in a very small proportion of patients. However, a sperm granuloma at the vasectomy site does not cause any increased risk of scrotal discomfort. Furthermore, it ensures the continued integrity of the epididymis, making successful reversal much more likely.

A method of vasectomy specifically designed to encourage the formulation of a sperm granuloma and yet minimize the risk of spontaneous recanalization was tried in over 750 patients by Shapiro in Ottawa, Canada (Shapiro and Silber, 1979). Shapiro did nothing to the testicular cut end of the vas deferens at the time of vasectomy. He merely let it leak freely into the scrotal tissue. In an early group of his patients, he sealed the lumen of the abdominal portion of the vas with hot wire cautery (vasector). In a subsequent, larger group of patients, he used a hemoclip on the abdominal side lumen along with fascial interposition. A sperm granuloma failed to form in 3% of patients. In all of the others, however, a sperm granuloma
formed, which never progressed to more than 4.0 mm in diameter. None of the granulomas were tender or required surgical treatment. Therefore, the results of clinical studies carried out by Shapiro do not support the findings of Schmidt, who reported that almost 50% of patients with sperm granuloma after vasectomy have such severe and persistent pain that they require surgical intervention (Shapiro and Silber, 1979).

The somewhat unsettling aspect of Shapiro’s work is that in the first group of patients in whom the abdominal lumen was treated with hot wire cautery, 7% had spontaneous recanalization. However, in the second group of over 500 patients who had a hemoclip applied to the abdominal end of the cut vas deferens, and who also underwent fascial interposition, none had a recanalization. This work is very encouraging, but further studies are needed to determine the simplest method of sealing the abdominal side of the vas with no recanalization.

There is no doubt that development of procedures for the prevention of spontaneous recanalization by proper treatment of the abdominal side of the cut vas deferens is essential before open-ended vasectomy can be recommended, despite its greater reversibility. Yet it is clear that this problem should be solved rather easily, and answers may be available in the next year or so. Presently, instead of performing cautery with the standard 0.5-cm hot wire length, we have persuaded the company (Concept, Clearwater, Florida) to make a 1.5-cm hot wire. With this simple change in our technique, we have thus far seen no recanalizations. Thus, “open-ended” vasectomy seems to be the ideal approach to making reversal much easier, and with the new, longer “vasectomy” unit available from Concept, spontaneous recanalization is not likely to be a problem.

Any emotional outcry against sperm granuloma, whether at the vasectomy site or in the epididymis, is really an uninformed indictment against vasectomy itself, since sperm granuloma is an inevitable and unavoidable consequence of vasectomy. Furthermore, a certain tiny percentage of vasectomized men are going to experience some pain no matter what technique is used and whether or not a sperm granuloma forms at the vasectomy site. The symptoms are rarely more than just a minor nuisance, and they are usually transient.

Needless to say, we recommend vasectomy only for a man who wishes to have no more children; however, the death of a child or wife may change his views radically. Therefore, it would be inhumane not to consider reversibility when we perform vasectomy.

Obstructive Azoospermia Not Related to Vasectomy

When there are no sperm in the ejaculate of patients in whom the FSH is normal, only one additional diagnostic test should be performed—namely, testicular biopsy. If this shows “normal spermatogenesis,” the diagnosis is obstruction, and microsurgery should be planned. The chance of success in these cases is over 80% with properly performed microsurgery. Over half of these patients have had no prior history of clinically detectable epididymitis. Yet they apparently have had sufficient inflammation of the epididymis to produce complete occlusion. We should not be surprised that azoospermia caused by epididymal obstruction is usually not related to any discernible history of epididymitis, because all vasectomized patients have “congestive” epididymitis with minimal or no symptoms. Epididymitis has to be very severe before painful swelling is apparent to the patient. It may well be that subtle degrees of epididymitis are even responsible for many cases of severe oligospermia. By comparing the quantitative production of sperm seen on testicular biopsy to the degree of oligospermia, we may be able to identify those cases of oligosperma that may respond to microsurgical correction.

References


Van Wagenen G. Degeneration of germinal epithelium in the testes of the rat as a result of efferent duct ligation. Anat Rec 1924; 27:189.