VASECTOMY FOR CONTRACEPTION—PHYSIOLOGICAL PERSPECTIVES

In many parts of the world today, bilateral vasectomy easily tops the list amongst all elective surgical procedures as far as numbers are concerned. Not only in India, where 2.5 million vasectomies are proposed annually, but even in an advanced country like the U.S.A., as many as 700,000 males underwent this operation last year. By any surgical standards vasectomy is a minor operation, practically free from any complications. The patient rarely takes the trouble of reporting back to the surgeon except when the wife becomes pregnant despite the vasectomy, and then it is the surgeon who is really in trouble. But it is for us physiologists to enquire whether vasectomy merely interrupts the seminal outflow or, in addition, produces any subtle and remote structural, functional, immunological or psychological changes. The problem assumes serious practical significance today with an ever increasing number of males choosing this simple highly effective and least bothersome contraceptive procedure.

The first clear suggestion that vasectomy can lead to significant changes in the endocrin part of the testis came from the Viennese endocrinologist Eugene Steinach (10), in the early twenties, when he reported the exciting discovery that ligation of the vas deferens in ageing animals could produce remarkable rejuvenation. So convincing was his argument that a urologist friend, Robert Lichtenstern, started performing vasectomies on the prosperous elite who were losing their youthful vigour and falling out of the fast and gay life. All this brought great fame to Steinach and a sizeable fortune to Lichtenstern but, alas, the enthusiasm was only short lived. Subsequent workers failed to substantiate Steinach’s claim unequivocally and the operation fell into disrepute till it was revived again, but now (or the altogether different purpose of birth control).

How does vasectomy affect the structure of the testis? Opinions have differed markedly, ranging from no change at all (2) to widespread degeneration of seminiferous tubules and hyperplasia of the Leydig cells (10). There appear to be two main reasons for this discrepancy:

1. All previous work has been on different laboratory animals in which wide species variation seems to occur, related perhaps to the absorptive capacity of the epididymis behind the obstructed vas. There have been hardly any studies directly on man.

2. In the absence of any precise quantification, it is difficult to visualise changes in the total size of the endocrine tissue of the testis as the Leydig cells lie scattered in between the tubules.

We have recently been examining the whole problem all over again, circumventing these two difficulties. Studies have been undertaken not only on animals but also directly on human subjects by repeated open testicular biopsy and other biochemical investigations. A method
Preliminary work on dogs has shown widespread degeneration of the seminiferous tissue with thickening of the tunica propria and complete arrest of spermatogenesis within 2 weeks of vasectomy. The epididymis is distended with flattening of its epithelium but shows no sperms. There is, however, no evidence of any inflammatory reaction or vascular lesion anywhere, indicating that the observed changes are entirely due to the obstruction of the vas. Corresponding changes have been observed histochemically also. Thus, alkaline phosphatase and lipids decrease in the seminiferous tubules while the PAS positive-diastase resistant tunica propria becomes much thickened. At the same time the Leydig cell clusters become more prominent with increase in cholesterol and neutral lipids.

In both young and elderly men (above 50 years) vasectomy has led to a similar but partial degeneration of the seminiferous tissue with some inter-tubular fibrosis within one month. The basal cells, however, withstand the degeneration effectively, upholding the possibility of regeneration later on. Occasional tubules still remain normal in contrast to the much more widespread degeneration in the dog. When examined 2 to 3 years after vasectomy, the seminiferous tubules in man look almost normal (at least histologically) except for some intertubular fibrosis. It is, therefore, clear now that regeneration of the seminiferous tissue occurs spontaneously in man even without recanalisation. Restoration of fertility has, of course, been achieved by vaso-vasostomy in innumerable cases by now.

Whether functionally effective sperms are present while the testicular outflow tract is obstructed remains unverified. We have a rare opportunity to study this in a healthy young man who has complete absence of the vas on both sides but whose testes appear to be histologically almost normal; he has also no clinical evidence of any endocrine or genetic disorder. All necessary investigations are completed on the couple, it is proposed to attempt artificial insemination with fluid drawn from the epididymis. What the outcome would be is anybody's guess but it could be highly rewarding for the couple and very interesting scientifically.

The testosterone secreting Leydig cells have been found to constitute about 15% of the testis in young adults (1.2 to 3.1 ml per testis). It is interesting to note that elderly males of 50 years of age have approximately 30% greater Leydig cell mass as compared to the young. This may well reflect a pituitary gonadotropin stimulus, under a failing testosterone feedback but it needs further elucidation. After vasectomy young adults have shown a modest but significant increase in the Leydig cells, from 2.2 ml/testis to 2.5 ml/testis on an average, within 2 months (8). This is the first such quantitative estimate in man and would require further substantiation. A somewhat greater proliferation of the Leydig cells has been found in vasectomised dogs during the same period (4). The possible mechanism by which the degenerating seminiferous tubules could influence the Leydig cells, has, however, never been clearly spelt out.
Psychological problems following vasectomy have not so far received adequate attention except by way of some casual surveys. Recently Uehling and Wear (11) found increased libido in 32% and no change in 65% subjects interviewed more than 1 year after the operation; adverse reactions were found in none. But in our society one does come across a vasectomised individual who feels completely run down with all sorts of vague complaints including loss of his masculine vigour. Surgeons would tend to dismiss him lightly (and leave him miserable) but the best thing to do is to recanalise him and advise alternate contraceptive measures. Once the patient is convinced that he has been restored to his original condition, he quickly recovers. It is obvious that persons who already have some sexual problems genito-urinary abnormality or are emotionally unstable ought to be screened out before vasectomy is performed.

As vasectomy becomes more and more popular, many sociological as well as legal problems are also likely to crop up. (For instance: the young daughter telling her alarmed mother "But mammy, he had definitely told me he was vasectomised"). Unfortunately, the social and ethical aspects are receiving hardly any attention in our country so far. Similarly, the immunological consequences of vasectomy like the presence of sperm agglutinating/immobilising antibodies needs much attention particularly in relation to the long term effects on the testis (1).

Taking a broad view of the whole situation, J.H. Roberts had concluded in 1968 that "the long term medical complications of vasectomy have yet to be critically evaluated". Certainly, there are still a number of interesting problems which could be profitably taken up by any interested person. For instance:

1. What is the exact dynamics of epididymal function in man and animals? It may well be that the difference in the response to vasectomy is related to the varying absorptive capacity of the epididymis.

2. Is there a possible feed-back from the degenerating seminiferous tubules to the anterior pituitary which could influence gonadotropin output and, ultimately, the Leydig cells? Or is there some direct influence of the gametogenic component of the testis on the endocrine component?

3. Is there any change in the blood testosterone level after vasectomy on a long term follow-up? How does it correlate with changes in the total Leydig cell volume of the testis.

4. How would a ligation between the testis and the epididymis (Steinach Operation II) affect the testis?

5. What is the factual basis for a post-vasectomy rejuvination? Is it only a myth or has it some definite endocrinological or psychological foundation, at least in certain circumstances.

6. How does sterility and ageing relate with the structure of the testis, particularly the Leydig cells?

In this year, while discussing some of our work, Dr. Clive Wood (3) has very wittily summed it up by saying that 'if nothing else, it helps to further confuse an already confused situation'.

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But with the basic clinical material available in such large amount, and particularly in India, the confusion should not take long to clear up.

REFERENCES


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