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## Sterility in the male

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STERILITY IN THE MALE

Senior Thesis

by  
L. Joe Ruzicka

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Presented to the  
College of Medicine,  
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## INTRODUCTION

"From biblical times until the present century the wife has borne the burden of blame for failure to produce offspring. Until twenty years ago no serious studies of male fertility had been undertaken" (11).

"One of the old laws states that a woman who does not bear an heir in a year or eighteen months, can be cast out from her husband's bed and board" (53).

Due to such remarks as above I have taken this topic of "Male Sterility", not as a primary reason to protect the female sex, because I feel that they are well protected in the present day and age, but to bring out those male faults that are responsible for sterile matings.

Until recent years and in some cases till the present day, in considering a sterile marriage, the wife is considered first and often solely. Thus, all the tests and examinations for fertility have been done on the wife, before the husband had been considered, if at all. I will endeavor to show in this paper the importance of a relatively complete history of both the husband and wife before any tests and examinations for sterility are undertaken. Having the etiology of the male faults in mind, a history of the husband in the presence of a negative history of the wife, may often reveal the male faults and save the expense and possible embarrassment of one or the other, or both.

Sterility is said to be the pathological condition characterized by the capability of performing the sexual act without being able to fecundate (10). To define it further, Kleegman qualifies sterility as the inability to initiate the reproductive process on the part of the couple who have desired and attempted to do so for the period of one year.

In considering the sterility in the male alone it is said to be that condition of loss of procreative power which implies the absence of living spermatozoa, since these are the elements essential to impregnation, but, it does not imply failure of power in sexual congress (40).

Sterility in general is commonly classed and defined as follows:

Primary sterility is applied to those marriages from which no pregnancy has resulted (45)(25)(50).

Secondary sterility is the term used in those cases in which one or more pregnancies have occurred but further conception is apparently impossible (45)(25). This definition makes husband and wife equally responsible and it excludes all the accidents of pregnancy, which destroy the fetus before viability (25)(50).

Relative sterility or lowered infertility are

terms expressing a condition where various factors interfere with pregnancy, but it is possible (45)(50).

Absolute sterility is the term designating the state or condition in which anatomic or congenital defects or pathologic changes make pregnancy impossible (45)(50). Certain of these conditions are correctable.

Moench (35) had coined another term, selective fertility, which he believes is more descriptive of those areas in which a man may impregnate one fertile woman and fail to do so with another.

#### Statistics

Statistics of sterility in the male are rather inaccurate and not wholly dependable. This is so because a relatively small percentage of men come to the physician complaining of sterility and a large percentage are ignorant of the fact that the male may be sterile. Another large percentage of men who may suspect themselves of being sterile disregard it because they are fearful of a physical examination, while others do not believe it worth the possible humiliation by exposing their past.

However statistics of male faults have been computed by various authors from cases of reported sterile matings.

F. S. Patch found that out of fifty-one cases of a series of fifty-three, forty-five cases or 88.3% represented husbands who were to blame for the unsuccessful issue of the marriage. Other authors place the male faults at 50% (45), 25% (18)(8)(32), and 20% to 40% (8)(33).

F. R. Hagner (15)(16) states that the male is at fault in one out of six cases. He also quotes Grosse to say that "the male was deficient in 18% of a series of 192 cases", and Noeggeroth to say that "eight out of fourteen sterile marriages were due to the male".

In another series of cases, in which the husband was found guilty, 31.3% were due to developmental defects and 59.6% to misconduct (8).

### Etiology

The etiology of male sterility varies somewhat in its classification with the various authors. Samuel R. Meaker (33) presents a "Working Classification of Sterility" which does not use the exact terms of most other authors. I have segregated the male from the female faults from Meaker's classification and present them here in an outline form.

#### A. Defective production of spermatozoa

##### 1. Testicular underdevelopment

- II. Testicular underfunction
  - a. Exhaustion from sexual excess
  - b. Depressed constitutional states
    - 1. Metabolic faults of extrinsic origin
    - 2. Debility
    - 3. Intoxication
  - c. Endocrine failure
- B. Obstruction or hostility in the male passages
  - I. Obstruction in epididymitis
  - II. Hostility of prostatic-vesicular secretion
    - a. Acidity
    - b. Viscosity
    - c. Infection
- C. Faults of delivery
  - I. Intercourse lacking or incomplete
    - a. Male faults
      - 1. Malformation
      - 2. Impotence
  - II. Intercourse without cervical insemination
    - a. Maladjustment or disproportion
    - b. Male faults
      - 1. Premature ejaculation
      - 2. Hypospadias
      - 3. Stricture



Some of the other authors classify sterility in the male as follows (10)(15)(40)(2)(48):

A. Azoospermia, a term for a complete absence of spermatozoa.

B. Oligospermia, a term for a marked diminution in the number of spermatozoa present.

C. Necrospermia, a term for the presence of dead spermatozoa or of spermatozoa devoid of motion.

D. Aspermia, a term for complete absence of semen.

Although the latter classification seems to be the one most often used, the former is the more complete and more definite. This discussion will be based on Meaker's classification and the two will be considered simultaneously.

Defective production of spermatozoa:--A very evident and unquestionable cause of sterility is the absence of the testicles (42). The absence of the testicles may be either congenital or acquired; acquired as in castration, atrophy following orchitis or in the case of ectopic testicles (10). It is stated that men with double undescended testicles are almost invariably sterile (16). Chapman S. Moorman finds that azoospermia in most cases is due to the congenital condition of cryptorchism which was not repaired surgically in time

to prevent injury and destruction of the generative cells. However, a cryptorchid is not necessarily sterile except where there are pathological changes present, resulting from abnormal position. The testes are often fibrosed or atrophied in sterile cryptorchids (42).

Numerous factors are found which tend to inhibit the function of the testicles in spite of their being well developed. A very frequent cause of this disturbance, found especially in the young married couples, is a too frequent coitus and seminal emission (10)(43)(49)(2)(50)(8). Too frequent or too prolonged intercourse frequently overstimulates the secretory structure of the testicle leading to exhaustion of it (16). This results in decreased number and imperfectly formed spermatozoa although the quantity of the fluid may be normal (16)(35). F. S. Patch contends that too frequent intercourse, on the other hand, will result in less quantity and consistency of the semen. He continues to say that finally the secretion ejaculated will consist only of that from the accessory genital glands. In this type of ejaculation, what spermatozoa may be present are slow in movement and die early. All practices which are departures from the usual

physiological and normal sexual act are without doubt outstanding factors in causing a diminution in the number of spermatozoa (40). A common one, the practice of withdrawal to prevent conception, is found guilty through the action of exhaustion of higher nervous centers.

Depressed constitutional states carry a great deal of weight as factors in sterility. It is generally thought that diet is an important consideration (45). Dietetic deficiencies such as protein and vitamin E are stressed as important factors (49). Reynolds and Macomber have done quite extensive work on rats, employing the various dietetic factors. They found that a mere moderate decrease in the percentage of fat soluble vitamin, protein, or calcium which are contained in an otherwise excellent diet, produces a definite decrease of fertility in individual rats. They have also concluded that a slight decrease in fertility of both partners produces a sterile mating. How much this can be applied to human beings is rather uncertain but other authors seem to include this as a factor in human sterility. Another of their (44) conclusions is that dietary deficiencies produce a lowered fertility varying in degree with different individuals even though they may

be of the same age and same parentage. It is found however, that these individuals which are sterile when mated, may reproduce freely when remated to partners of higher fertility. Cases of sterility have been found where the wearing of suspensory straps or too warm clothing around the genitalia was a factor; undue warmth interfering with the spermatogenic function of the testicles (50). Metabolic faults causing sterility are often due to a lack of physical exercise (33). This is quite often the cause of sterility in business and professional men who also have a tendency to become fat.

Spermatogenesis is the most delicately poised of all glandular mechanisms. Thus, there are but few pathological changes either in the testis itself or elsewhere in the body, that do not affect it (51). Debilitating diseases have a retarding effect upon the testicles, some of which go as far as to cause a definite injury and atrophy of the glands. Disturbance of the secretory function of the testicles may come about in acute fevers and during a prolonged convalescence (10). Any diseases such as syphilis, tuberculosis, or malignancies when involving the testicles, especially when bilateral, destroy completely the

parenchyma of the organs (10)(16)(48)(40). These conditions will manifest themselves in azoospermia and necrospermia. Bilateral gummata of the testes in a syphilitic patient are invariably a cause of sterility, in which cure may follow specific treatment (16)(42). A very frequently found cause of sterility is an epididymo-orchitis following a case of complicated mumps (16)(8)(42). This is, however, permanent. Large hydroceles, scrotal hernias, and varicoceles often cause sterility through atrophy as a result of long continued pressure (42). Guiteras states that a severe contusion of the testicle is always followed by atrophy. In severe contusions the tunica albuginea is torn through; blood and spermatic fluid mix which may develop into an abscess.

It may help, to make the patient's history more significant, to mention a few diseases that may cause an acute orchitis. It may be caused from such diseases as mumps as mentioned before, typhoid fever which causes phlebitis of spermatic veins, small pox, scarlet fever, or malaria (12).

Further, under depressed constitutional states, intoxication must be considered as a factor in causing underfunction of the testicles. Usually a sterility

complication of a gonorrhoeal infection is thought of as an inflammatory condition in the epididymis resulting in a stricture. It has been suggested that, besides the above mentioned, damage is done by the gonorrhoeal toxin; an endotoxin which deranges the cells secreting the spermatic fluid and perhaps injures the quality of the spermatozoa themselves (43)(40). The result here would be a lessened penetrating power of the motile spermatozoa. Prolonged or toxic doses of x-ray and other irradiations to the testicles have been found to cause the condition of azoospermia in many cases. This toxic condition is usually brought about when inadequate protection is given to the testicles in treating some dermatological conditions in or around the genital area with x-ray (40)(17). Different testicles react differently to the influence of the x-rays, some being more sensitive than others, so that what might be adequate protection in some cases would not be at all in others (22). However, the reduced fertility due to this condition is temporary (53) and removal from an x-ray atmosphere usually restores spermatozoa in the semen after about six months (10). In another case, only inactive spermatozoa were found in the semen of a patient who had a mild

lead poisoning, and this persisted until the poisoning was corrected (49). Likewise, chronic alcoholism results in a depression of the spermatozoa. It may be that other toxic diseases or conditions as such afore discussed may be equally responsible in male sterility.

A relatively recent problem in sterility has been that of endocrine failure. Some workers in sterility problems do not give much credit to endocrine failure but several authors make quite definite claims. Chute points out that sterility occurs in such extreme endocrine abnormalities as gigantism, acromegaly, pituitary dwarfism, pituitary cachexia (Simmond's disease), cretanism, Addison's disease, and tumors of the adrenal cortex. Clinically, the two most important glands in the male causing an endocrine sterility, are the thyroid and the anterior pituitary (45)(7). Charny adds the adrenal and the testis hormones to the dicussion.

Normal thyroid activity has an important influence on spermatogenesis (50) and thyroid dysfunction either in the form of hypoactivity or hyperactivity not infrequently is a cause of sterility (2). An example of this is a case of a sterile mating. The wife was

without any cause for infertility. The husband's basal metabolism was found to be -29, and under appropriate thyroid treatment his hypothyroidism improved and pregnancy occurred (45).

Thyroid and pancreatic hormones stimulate the testes to growth, but this effect is probably exerted through the pituitary gland. Thyroid increases the pituitary content of the gonad stimulating hormone. Hyperthyroids are sterile because of a lack of the male sex hormone, due to its hastened elimination in a heightened metabolic process. Hypothyroids are sterile because of a failure of the gonad stimulating hormone of the anterior pituitary gland (6).

At or after puberty, when body growth has nearly reached its maximum, the sex hormone of the anterior pituitary becomes prominent. This hormone stimulates the testis to activity, both in germ cell production and in hormone stimulation, but the germ cell stimulation is not sufficient to produce spermatozoa. Although the male sex hormone does not stimulate nor is a factor in spermatogenesis, it is an essential element in keeping the spermatozoa alive (6). Here, Charny must consider spermatogenesis as beginning from the spermatid stage, because in speaking of the effects of the hormone



he says that he has found definite evidence of differentiation of the tubular epithelium into spermatids, but no actual spermatogenesis.

The relation of the pancreas to testicular function is not fully understood but may be affected through the pituitary gland. Some of Charny's cases of diabetes mellitus show normal spermatogenesis while others show a deficiency of varying degrees.

Testis hormones have no direct effect upon the testes themselves, but have to do with the development of the secondary sex hormones (6). For this reason, they have nothing to do with sterility directly.

Obstruction or hostility in the male passages:--  
The most frequent causes of sterility in the male are inflammatory occlusions of the epididymis or vas deferens (14)(53), resulting from a bilateral epididymitis, Neisserian in origin (10)(40)(42)(48). Gonorrhoea is a prepondering cause in both primary and secondary sterility (2). However, a similar obstruction may also be caused by tuberculosis and syphilis (10).

It may be well, at this time, to show the preponderance of gonorrhoea in resulting sterility. Patch presented a series of cases of sterile men in which gonorrhoea had been present in 49% of the cases. Of

this series 43% had uncured gonorrhoea. Thompson and Walker go on to quote Benzla in finding that soldiers of the German army who had suffered from gonorrhoea, 10.5% were childless although they had no epididymitis. Of those who had unilateral and bilateral epididymitis, the childless percentages were respectively 23.4% and 41.7%.

One-sided epididymitis does not necessarily reduce procreative power, providing the other organ is sound and no other conditions causing sterility exist; and not all cases of bilateral epididymitis cause a sterility (42). Total absence of spermatozoa in the sexual ejaculate, when it follows obstruction of the vas deferens from epididymitis following gonorrhoea, does not mean the testicles are not producing spermatozoa, but that their escape is being prevented (40)(50).

In the course of gonorrhoeal epididymitis the healing process leads to the formation of scar tissue. This scar tissue causes an occlusion of the efferent ducts of the epididymis and prevents the egress of normal spermatozoa (14). The globus minor is the portion of the epididymis most involved, which is most dangerous since there is but one efferent duct, while in the globus major there are numerous ducts (14).

The globus minor becomes the seat of a chronic fibrous nodule which surrounds and compresses the duct of the epididymis (48). Huhner (23) describes a form of epididymitis or vasitis in which the acute pain and swelling of the ordinary form of epididymitis are not present. However, there is just enough inflammation to cause an agglutination of the walls of the epididymis or vas.

Obstructive sterility may be caused by other means than those resulting from an epididymitis, gonorrhoeal in origin. Lesions of the prostate gland, due to adenomatous or cancerous enlargement, abscess formation, fibrosis, calculus, tuberculosis, trauma, and operation may also cause obstruction of the ducts (42)(48).

Wolbarst has worked on the male sterility problem from the aspect of a post-influenzal complication. From his work he infers that an influenzal infection of the vas may occur and produce stenosis and permanent sterility unless relieved by vasotomy. He has found that organisms recovered in the secretion found in the vas are similar to those recovered in the focal influenzal infections of the prostate, seminal vesicles and epididymis.

Atresia of the ejaculatory ducts, a definite cause of an absolute aspermia, may be either congenital or

which will carry the seminal fluid forward by a continuous wave of muscular contraction. Although the sensation of ejaculation is present, there is no actual discharge. The fluid remains in the prostatic cavity and is voided out in the urine at the next micturation. A similar explanation of this is also given by Thompson and Walker. Of the series of seventy-five, two patients, ages 58 and 69, remarried and had children thus showing that not always is sterility a resultant (13). Thompson and Walker (48) claim that aspermia follows suprapubic prostatectomy in 32.5% of cases.

Another similar condition is, that following a lumbar sympathectomy, sometimes used in the treatment of Hirshprung's disease. Learmonth showed that when the presacral nerve is stimulated, a cloud of seminal fluid and prostatic secretion is poured into the prostatic urethra as a result of the contraction of the seminal vesicles, ejaculatory ducts, and muscular septa of the prostatic gland. Consequently lumbar sympathetic neurectomy in males results in the abolition of ejaculation, although the sexual act is otherwise normal and the physiological orgasm is unaffected (24). Lumbar sympathectomy should only be performed in males when a condition is present which is likely to receive suf-

ficient benefit to compensate for the unavoidable sterility which will develop (24).

Anesthetic aspermia is also to be considered in sterility where no ejaculation takes place. This type of aspermia results when the sensibility of the penile skin is disturbed, as a result of which reflex action of the peripheral nerves on the ejaculation center is impossible (42). Destruction through ulceration of the prepuce and dorsum of the penis causing the formation of massive insensitive scar tissue is the pathology of this condition (42)(10). Insensitive condition of the glans penis may be brought about by injury to the spine and consequently complete anesthesia of the skin of the genitals (10).

Patch also speaks of a psychic aspermia. He claims that in this form various influences of a psychic character can irritate the inhibitory center in the brain so powerfully, as to suppress the working of the ejaculatory center in the cord.

Hostilities of prostatic-vesiculitis secretion are factors in sterility in several different ways. In the secretion, a deviation of the degree of acidity or pH., out of bounds of a normal range, has a definite reaction on the spermatozoa contained. The optimum range lies

between the hydrogen-ion concentration values of 8.5 and 9.5 (41) and 7.7 to 8.5 (21). A change toward acidity causes inhibition of motility, no motility occurring below hydrogen-ion concentration of 6.0. Reactions higher than hydrogen-ion concentration of 10.0 also cause inhibition of motility. Spermatozoa made inactive by rendering the medium acid (pH 4.0) regain their motility after realkalinization (41). If there is lessened motility of the spermatozoa, but good morphology, this is probably due to an acidity of the secretions of the seminal vesicles and prostate. Behind this picture may be a chronic focal infection such as bad teeth, tonsils, sinuses, etc., or chronic gonorrhoea of the prostate (18).

The subject of increased viscosity comes into question as a factor of sterility. If the semen is increased in coagulability or viscosity, sperms can not escape (5). Hotchkiss (21) on the contrary says it is likely that variations in viscosity have little or no clinical significance.

Inflammations of the seminal vesicles, either acute or chronic, by altering the secretion of the seminal vesicles, and therefore normal semen may be a cause of necro-spermia (10). This not only happens in prostato-

vesiculitis but also in epididymitis, vas defferentitis, and urethritis (42). When any portion of the seminal tract is inflamed, the products of inflammation, pus and blood, may mix with the semen and result in pyospermia and haemospermia. Pus and blood mixed with semen do not in all cases exercise a deleterious effect upon the spermatozoa, though they may reduce their vitality or even completely destroy them (42). Lespinasse agrees with the above statements in saying that pus in prostatic or vesicular fluids in some cases is stimulating or irritating to the spermatozoa. The usual effect of pus in these secretions, however, is detrimental to the sperm and the sperm is usually dead or very feebly motile when pus is present. Hagner and Fuller (17) also agree that pus inhibits sperm motility, but Sangree claims that in the majority of his cases of varying grades of chronic prostatitis, the spermatozoa in the ejaculate were actively motile, but all contained many abnormal forms. Huhner (23) disagrees somewhat by saying that pus itself does not interfere with the vitality of spermatozoa, but that it is the pathological condition in the genitalia which is responsible for the sterility as well as the pus. Mason cites where small amounts of semen from specimens of virile

sperms were mixed with larger amounts of semen in which sperms were found dead. These virile sperms lived as long as those in the control. Therefore he claims there is no hostility of prostatic or seminal fluid as is so often given as the cause.

Of a series of twenty-two cases of prostatico-vesiculitis, eleven had their semen examined and only three showed healthy spermatozoa (42).

Faults of delivery :--There are certain malformations and deformities which prevent the approach of the penis to the vulva. Intercourse becomes impossible when there is an absence of the penis, either congenital or acquired, or when the penis is distorted extremely by neoplastic growth, trauma, or inflammatory damage to its erectile tissue. Some other factors here are ankylosis of the hips in flexion, great obesity, and any form of large scrotal tumor such as hydrocele, hernia, or elephantiasis (34).

Impotence serves as a definite factor in aspermia. Impotence is the inability to obtain erection and to maintain it sufficiently for intromission and mature ejaculation (34). In some cases, though there is ability to copulate, the semen is later emitted during sleep (10). Causes of impotence are too numerous, being local,



constitutional, nervous, and psychic. More than one may operate in the same case (34).

Conditions exist where there is actual intercourse but there fails to be cervical insemination. Walker modifies this statement by saying that, while the projection of semen onto the cervix is favorable to conception, it is not essential, for many cases have been reported of pregnancy following the deposition of semen on the labia.

If cervical insemination is necessary for pregnancy to occur then surely a premature ejaculation on the part of the male stands out as a factor in sterility. This would be a factor if the ejaculation was so premature as to lose the semen before complete penetration of the vaginal orifice is affected. Meaker (34) describes ejaculatio praecox which he says is in a sense a lesser degree of impotence and usually a result of similar underlying causes.

There are cases where there is a disproportion between the male and female organs, either the vagina is too long for the penis or the vagina is too short and the ejaculate falls way beyond the cervix (34).

Cases of hypospadias or epispadias make cervical insemination impossible (18)(49)(34). Patch calls this

condition a false aspermia or a malemission. This male fault is however a congenital defect and unless treated during childhood it very likely remains a permanent condition.

Stricture of the urethra during coitis is an inhibitory factor in male sterility. The mucous membrane of the urethra becomes swollen and, if the stricture be a tight one, its orifice will become entirely closed, thus preventing the escape of the semen which, however, remains imprisoned between the verumontanum and the stricture. When the congestion of the verumontanum subsides and it no longer closes the vesicle outlet, the semen escapes into the bladder to be discharged later mixed with the urine (10)(50). This may be due to a chronic gonorrhoea (42). The condition of phimosis as a congenital cause is given in this class of sterility factors (42).

One more classification of sterility not mentioned by Meaker (34) is that of the so-called obscure causes. Kurzrock and Miller describe what they consider an obscure cause. This case deals with a sterile woman whose past history is negative, whose physical condition is good, who presents no endocrine disturbance, has no pelvic pathology, has patent fallopian tubes and whose

husband shows normal motile spermatozoa, and the patient remains sterile in spite of the usual forms of treatment. They believe that there is an absence or diminution of the lytic substance in the semen, since they found that a normal specimen of semen should dissolve cervical mucus, which enables the sperms to penetrate it. It has also been shown that the semen of a certain man will do this to mucus of one woman but not another.

Another case reported by Moench (35) has to do with the acidity of vaginal secretions and the alkalinity of the ejaculate. The normal vaginas are the most acid and thus have no factor in the sterility. But if the reaction of the seminal fluid is reduced in alkalinity below normal in amount, the acid reaction of the vaginal cavity may be strong enough to kill the sperms.

Finally, Hagner (15) concludes, in those cases in which no specific cause can be found in either sex, we are forced to consider the theory of sexual incompatibility, or an apparent lack of affinity of one cell for another, as demonstrated in the experience of a childless first marriage followed by later fertile marriages on the part of both members. However, Seymour has a case more final and obscure. This case has to do with a couple, the husband being a college professor of an

intelligence quotient of 140 and the wife herself a college graduate. They had been married eleven years and all this time failed to produce offspring. Both husband and wife were found entirely negative in their histories and physicals. They both received plenty of exercise and plenty of rest. The sperm count on the male revealed 94 million sperms per cubic centimeter, abnormal forms 3%, motility index 18 hours and pus absent. The Huhner test showed viable sperm in the cervical canal. There was no endocrine dysfunction in either of the partners.

They were both subjected to biweekly and then weekly ultraviolet irradiations for a period of seven months. Their diet was drastically changed. Both retired at an early hour and had afternoon rests. Both were given daily doses of irradiated ergosterol and also, the wife had weekly doses of testicular extract intragluteally. After the seven months no pregnancy ensued.

Then, a change of environment was advised. They were sent to the country for three months. No pregnancies ensued.

Since the husband was of the genius group, children by him were desirable. Sixteen childless women, ages 22 to 40, whose husbands were sterile and wanted

children, were found. These were artificially inseminated with semen of the professor. No pregnancies resulted.

A man, intelligence quotient 120 and father of children, was found. The wife of the professor and the sixteen other women were artificially inseminated with this man's semen. Pregnancies resulted in all the women.

### Diagnosis

There are three primary conditions in male sterility (8) on which the diagnosis procedures can be based. These are as follows:

- a. No spermatozoa, a deficient amount, or a decrease in their fertility rate
- b. Anatomical or developmental defects somewhere in the genital tract
- c. Pathological conditions which destroy or render infertile the spermatozoa at the origin or on the way through the ejaculatory route

The order and method or technique in diagnosis of male sterility seems to vary with the various men working in this particular field. Again I wish to emphasize the importance of a complete history, especially genitourinary history, of the male before any further tests and means of diagnosis are made. Titus (49) makes men-

tion of this fact in saying that the history for example, may show dietetic deficiencies (protein and vitamin E starvation), previous debilitating or damaging diseases (mumps with orchitis, scarlet fever, recurrent tonsillitis, anemia, nephritis or gonorrhoea). Excessive use of alcohol, too frequent intercourse, the use of acid lubricating jellies with coitus, coitus interruptus followed by chronic passive congestion of the tubes, and similar factors may be important in the history.

The next in order in this diagnosis may be the general physical examination of the patient. Titus (50) stresses that blood pressure readings, blood counts and urine analysis are important. For example, especially low blood pressure may suggest deficient thyroid function, or may result from anemia, either of which can be a strong factor in causing sterility. A urological examination should disclose any anatomic abnormalities such as epispadias or hypospadias, undescended testicles, or insufficient development of the penis (50). The testicles should be firm rather than soft. According to Lespinasse, congenital absence of sperm formation may be found clinically by the testicles being a little smaller than normal, slightly more globular, and consid-

erably harder than a normal sperm-producing testicle. There should also be no nodules nor any infiltrations in the vas or epididymis. Thus orchitis, or epididymitis may be identified by physical examination. Prostatitis should be revealed by means of a urological examination. The prostate gland should be smooth and firm instead of enlarged, softened, and irregular as in the case when it is the site of a chronic infection of any type (50). The prostate should be massaged and the secretion examined for leukocytes, mucin globules, corpora amylacia and lecithin bodies (18). To differentiate between a stricture and an atrophy or degeneration of the generative tract, Huhner (23) aspirates the testicles, in every case of azoospermia or aspermia. An absence of sperms in the fluid would indicate a degeneration while presence of sperm would indicate an obstruction.

Before any semen appraisal is done on the ejaculate of the male, Huhner (23) advocates the "Huhner test", a procedure devised by himself. In this procedure the wife comes into the office or place of examination within a few hours after coitus, and then the male ejaculate is aspirated from the vagina. This is then examined microscopically. Huhner (23) says if only dead sperms are

found on the cervix, a microscopic test is made on the male ejaculate directly, by having the husband deposit his semen directly into a condom. If microscopically the sperms of the direct ejaculate are found dead also, one may be certain sterility is due to the husband; if live mobile sperms are found in the condom, one would suspect the wife at fault.

Although most authors advocate seminal appraisal directly from a condom specimen, Huhner (23) still insists that this appraisal should be done by aspiration of the semen from the vagina post-coital. Quoting him, he says, "Many times I have removed live active spermatozoa from the female cervix several days after coitus, only to see them die very rapidly under the microscope".

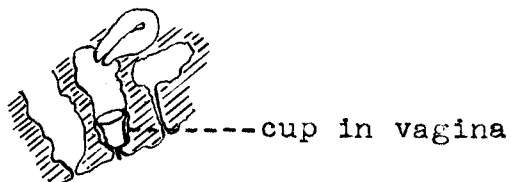
Often spilling or contamination is encountered before the woman appears for the Huhner test. Krigbaum specifies three methods by which specimens of post-coital contents of the vagina can be obtained. These are namely:

- a. To call at the patient's home to secure the sample
- b. To provide facilities in the office for the intercourse of the patient and the husband.
- c. Or make the office examination of the patient



after intercourse at home with the use of a bell-shaped cup inserted into the vagina to prevent gravity loss of coital contents.

The third method is illustrated here:



This cup is boiled sterile to be free from chemical compounds destructive to sperm, such as are often found in condoms.

Krigbaum suggests that two hours be the maximum interval between intercourse and the patient's arrival for examination.

There are also various methods or techniques employed in the collection of specimens directly from the male. Hotchkiss (20) has three methods by which this can be done:

- a. A self-produced specimen at the office of the examiner.
- b. Collection outside of the office by external emission. After collection into a graduate, it is allowed to liquefy and is then transferred to a one-ounce wide-mouthed bottle.

c. Condom technique. The patient is told to wash the preservative powder off of the side of the condom that is to receive the semen, since there is a chance to harm the specimen by the various ingredients of these powders. The condom is then wrapped in a handkerchief and placed next to skin and delivered to examiner.

Carey in 1916 described a method which is now obsolete. He advised that ejaculation should be made into a condom at intercourse. The condom be then put into a wide-mouthed bottle. This bottle then was put into a jar which contained water a few degrees above body temperature. However, he had one principle of present day caution by stating that this specimen should be obtained after a three to four days rest from intercourse.

A condom specimen will not do, because a preservative is used in their manufacture and destroys the sperm motility. The patient should bring the specimen in a wide-mouthed jar kept at room temperature or less and delivered in less than two hours (18). Hotchkiss (20) says examination should be made not later than one hour after ejaculation.

Contrary to this first method of collecting specimen proposed by Hotchkiss (20), Lespinasse insists that

actual intercourse must be indulged in, in obtaining a reliable specimen. If the self-produced specimen at the office of the examiner is to be used, it must be put aside in a heating box for one-half hour to allow the temporary thickening of the semen to liquefy (20).

Before going on with the diagnosis by means of semen appraisal it may be well to summarize the contents of normal semen. D'Oronzio gives the normal semen as consisting of (a) spermatozoa from the testicles; (b) spermatid cells; (c) secretions from the seminal vesicles, prostate, Cowper's glands and urethra. Any alteration in its composition may bring about pathological changes causing sterility.

Semen appraisal is both macroscopic and microscopic. Macroscopically, the total volume of the ejaculate is determined. The normal volume is given as three to eight cubic centimeters (29) and three to four cubic centimeters (21); while variations from one or two drops to ten cubic centimeters are encountered. Specimens of less than one-half cubic centimeter fail to produce an adequate seminal pool, which ordinarily provides a medium for the survival and protection of the sensitive sperm (21). Carey (5) quotes Ultzman in describing varieties of semen in which spermatozoa are

not found or are greatly reduced in number. These are: (a) watery transparent semen, which is normal in amount but contains slight sediment and in which crystal formation begins early; (b) colloid semen, that is semen containing epithelium which has undergone colloid degeneration; (c) purulent semen.

The technique of spermatozoa count is the use of an ordinary blood counting chamber and a white blood cell pipette. The procedure is the same as that employed in a white blood cell count. The diluent is 5% solution of sodium bicarbonate to which 1% formalin is added. The bicarbonate dissolves small amounts of mucus and the formalin stops the activity of the cells. In case there is too much mucus, the specimen may be diluted 1-20 with bicarbonate-formalin solution and counting without further dilution (30). Belding believes that the blood pipette method is an error of faulty sampling from the use of small quantities and thus advocates the bulk method. The same dilution of 1-20 is used in both methods, but larger quantities, 0.1 and 0.5 cubic centimeters of seminal fluid are employed for the bulk method. Experimentally, Belding found that from ten counts with each method on the same individual specimens, extremes of standards of deviation ranged from 6.32 to 17.01

with blood pipette and 4.24 to 13.53 with bulk method. He says, that for proper counting, various degrees of total count should be examined in different dilutions. He gives the optimum dilutions for counting to be: 200 million sperms--1:80; 150 million--1:60; 100 million--1:40; 50 million--1:20; 25 million--1:10. This may be done by using .5 cubic centimeter seminal fluid plus 9.5 cubic centimeter diluent. The various optimum dilutions are then made from this.

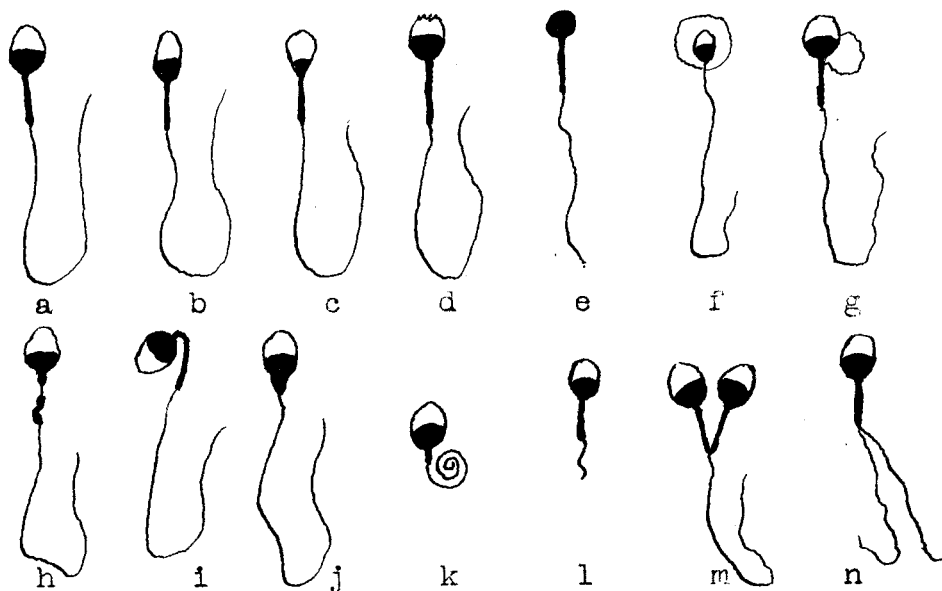
Microscopically, the number, morphology, motility, and viability of the spermatozoa are determined. The average fertile male will produce 100 million to 150 million spermatozoa per cubic centimeter of ejaculate (21). Macomber and Saunders report an average of 100 million sperms per cubic centimeter from 294 cases, while Belding, with the bulk method finds 119 million as a normal count and 70 million as for sterile matings. Hotchkiss (20) claims 70 million per cubic centimeter are found in fertile specimens providing the sperms are normal otherwise. Macomber and Saunders give some statistics from their own cases. In their cases only four matings showed a resulting pregnancy where the sperm count was less than 60 million per cubic centimeter; seven pregnancies with counts between 60 million

and 70 million; five pregnancies with counts between 70 million and 80 million; and thirty-six pregnancies with counts over 80 million. Hotchkiss (21) and Macomber both agree that the more reliable and consistent cell counts have been on the basis of cells present in that total volume of the ejaculate rather than in units of cubic centimeters. Macomber and Saunders give the average count in the total volume as 400 million to 600 million.

Moench (36) believes that the abnormalities of sperm heads will explain the apparent paradoxical situation wherein some men with relatively few spermia were normally fertile while other men with many more spermia, were relatively infertile. For the study of morphology of the sperms, Moench (36) uses a particular stain which is a filtered mixture of two-thirds Ziehl-Neelsen's carbol-fuchsin and one-third concentrated alcoholic eosin (bluish) solution. As a counter stain he uses Loeffler's methylene blue with two parts of distilled water. In this stain the heads of the spermia take varying shades of blue, whereas the bodies and tails are red. To remove the mucus before staining, he treats the fixed smear with a 1% chlorazene solution for ten to twenty minutes. However, Hotchkiss (21)

believes the Gram stain gives a fair visualization of the cell structure after proper fixation.

Mason described what is thought to be the normal morphology of the spermatozoa. The definite majority of 500 consecutive sperms of a series was always five by three microns in size and of regular ovoid contour, with tails approximately fifty-five microns long. The heads were almost filled with dense nuclear material, except for an area of cytoplasm from one and five-tenths to two microns at the proximal end. The following is Meaker's (34) illustration of the various forms of spermatozoa:



(a) normal spermatozoon; (b) narrow head; (c) tapering head; (d) roughened head membrane; (e) small head solidly staining; (f) immature head with cytoplasm not cast off, body lacking; (g) immature head with cytoplasm

partially cast off; (h) irregular body; (i) bent body; (j) short thick body; (k) short thick body, curled tail; (l) rudimentary tail; (m) double headed, double body; (n) double tail.

Carey believes the double-headers to be potent.

Of the millions of spermatozoa that are present in a given ejaculate, Williams doubts if more than a fraction of 1% are ever capable of union with the ovum. "Naturally", he says, "the larger the population of normal appearing cells, the greater is the probability of the fertilizing ability." Moench (38) states that a normally fertile male always ejaculates less than 20% abnormal sperm heads. If the head abnormalities rise to between 20-25%, impaired fertility is to be assumed, and above 25% there is always sterility. In appraising seminal specimens one must deduct the percentage of abnormal forms found from the total cell count (20). The average of abnormal forms in the specimens from sterile marriages were found somewhat higher than those counted from specimens from husbands of fertile marriages, being 9% and 5% respectively (31).

Motility and vitality may be considered at the same time, as one is dependent upon the other. For this examination a thick moist smear of the specimen is used. Motility of the sperm cells is a requirement without which, in animals with internal fertilization, preg-



nancy can not occur. Absence of all motility in repeated, absolutely fresh, and uncontaminated semen specimens establishes the cause of sterility. Slight changes in motility, as to the degree and length of time the cells remain active, unless such time is unduly short, must be judged very cautiously. Often such changes mean nothing, being due to purely temporary and accidental local condition (37).

A normal speed of sperm motility is difficult to determine because they do not travel in a straight line across the microscopic field. Normal sperms will survive in appreciable numbers for at least twenty-four hours, under ordinary conditions of temperature (21)(31). It is not necessary to keep the specimen at a temperature corresponding to that of the body.

To conclude, according to Belding, the spermatozoa count is an index of spermatogenic activity and as such is correlated with fertility. A low count in itself does not necessarily indicate sterility or low fertility. Individual variations renders single counts of questionable value. Moench (38) claims neither a normal morphology alone nor a normal biometric result alone means the semen is normal. On the other hand a disturbance in either of these two factors means dis-

turbed spermatogenesis and thus impaired fertility. Before any unfavorable prognosis can be made, complete study must include inquiry into the physiological affinity of the male and female secretions (5).

### Treatment

If treatment of the sterile male is indicated, it is done in one of three ways, educationally, medically, or surgically, as is required in individual cases.

Educational therapy has to do with such conditions as sterility due to coitus interruptus, too frequent intercourse, psychic aspermia and improper position for cervical insemination. This therapy consists of changing the psychology and philosophy of the marital partners.

Medical treatment of the sterile male is, of course, directed toward the cause of his sterility (18). Factors of sterility due to dietary deficiencies must be treated with adequate balanced diet. The giving of high protein diet, diet rich in vitamin, is essential in treating the cases of impaired motility of the sperm due to a low grade sepsis or any infectious disease (29). In the male, a diet rich in vitamin E is more important than in the female. Wheat germ oil constitutes one of the highest known sources of vitamin E (2). Besides

high vitamin and protein, calcium lactate should be included in the diet and enough exercise to assure assimilation is recommended (2).

Prostatic massage even though the prostate is not infected is another means of therapy. Prostatic massage appears to stimulate better function (18).

Recently, an improved treatment was found to be that of giving stimulating doses of x-ray to stimulate spermatogenesis (34)(18).

Cases of interstitial orchitis due to syphilis, and syphilitic gumma, when treated by antiluetic remedies, frequently can be restored to fertility.

At the present, we are quite concerned with the endocrines, in their being factors of diseases and also in the treatment of numerous conditions. Likewise, much consideration is given to the endocrines in the treatment of male sterility. Meaker (34) gives seven principles of endocrine treatment of sterility. These are: an actual endocrinopathy must exist; non-endocrine causative factors must be eliminated; the primary focus of failure must be identified; conditions inhibiting endocrine function must be removed; glandular medication must be given in potent form; the glandular dosage must be adequate; and treatment must be continued

for a sufficient time. The different investigators, both clinical and experimental, do not agree on the number or manner in which these hormones influence gonad physiology (19).

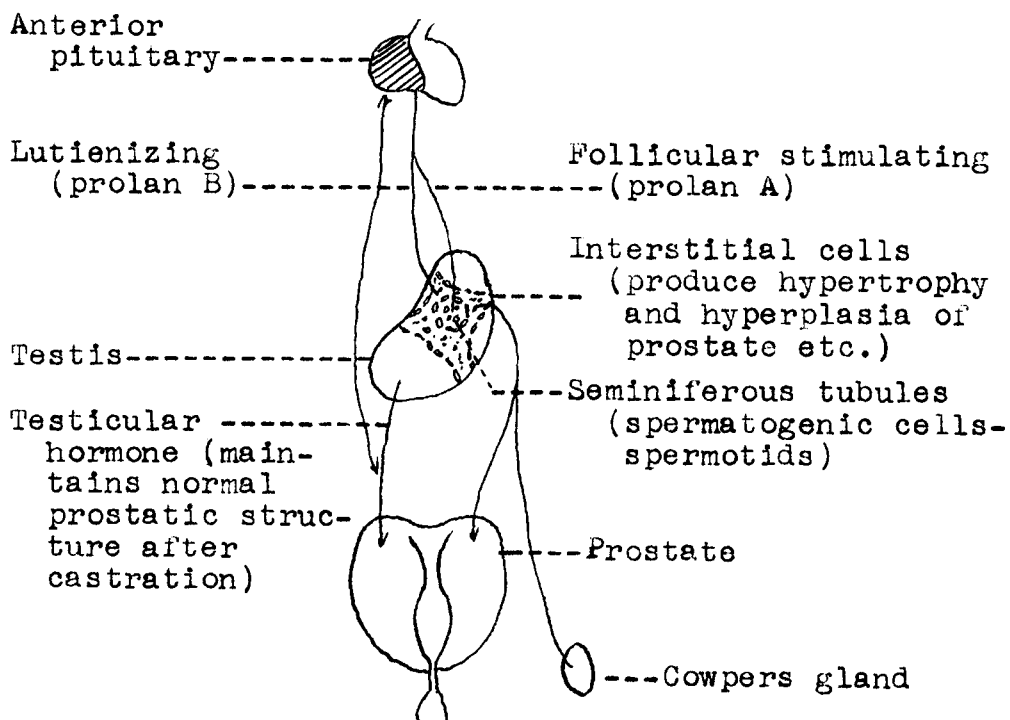
Brosius and Schaffer (4) have worked with animals, using a gonad stimulating extract from human urine of pregnancy. Their results on laboratory animals were the same as in one human case they treated. This was a case of complete aspermia with bilateral testicular atrophy, following orchitis as a complication of mumps. Spermatogenesis repeatedly followed the administration of the extract and aspermia returned on the withdrawal of the treatment. Brosius (3) finds from his cases that the so-called P.U. factor from the human urine of pregnancy stimulates spermatogenesis and hastens testicular descent in the human. The same year Brosius, quoted by Heckel, reported findings on twelve cases of male sterility in which experimentally, he felt that the anterior pituitary-like hormone obtained from the urine of pregnancy did stimulate spermatogenesis. If the anterior pituitary-like hormone does stimulate spermatogenesis, then it is evident that great harm might be done in its clinical application in the treatment of undescended testicles in young individuals before puberty (19).

Sexton treated thirteen boys with intramuscular injections of anterior pituitary-like hormone of pregnancy urine. These boys were between the ages of ten and twenty-one years and suffered with genital underdevelopment of varying degrees. Eleven of the boys responded to treatment by an increase in the size of the external genitalia and the appearance of secondary sex characteristics. Of the thirteen boys, six were cryptorchids and in four of these the testicles localized in the scrotum after treatment. Sexton added that the dosage and duration of treatment with anterior pituitary-like hormone was dependent on the degree of underdevelopment and the ages; those in early adolescence responded most favorably.

From experimental evidence up until 1935, Heckel claims that the follicular stimulating hormone might offer better results in the treatment of cases of sterility that are caused by dysfunction of the primitive sperm cell (spermatogonium). His illustration is presented on the next page.

Experimental work on laboratory animals shows that the fundamental biologic function of the male sex-hormone is the conditioning of the accessory reproductive organs to handle the sex products (germ cells) in a

manner to insure their proper meeting, and the conditioning of the animal to respond characteristically and at the proper time in the sexual act (9). It is not a testicular stimulant (39), since this function belongs to the anterior pituitary (9). Although it is not a testicular stimulant in sex-hormone production nor spermatogenesis, it is an essential element in keeping the spermatozoa alive (6).



According to Day, sex-hormone production is greatly diminished or disappears at the prostatic age. Testosterone (male sex-hormone) may be expected to control and hold in check or temporarily arrest the hyperplasia

and overgrowth of the prostate. Day continues to say that the synthetic testosterone is the most preferred and potent of the known substances. It should be administered in adequate dosage and at sufficiently frequent intervals.

Thyroid is to be administered in cases of low basal metabolic rates, or even in normal metabolic rates but with clinical symptoms of lowered thyroid activity. In addition to the thyroid therapy, the administration and occasional readministration, in courses, of gonadotropic hormone or the anterior pituitary-like hormone derived from urine of pregnant women, often has a striking effect (50).

Surgical treatment is employed in conditions such as obstructions in the genital tract, varicoceles which cause a passive congestion of the testes, cryptorchidism, orchitis due to mumps, hypospadias and epispadias.

Obstructions between the vas and the globus major as the result of epididymitis are treated by resection and anastomosis of the vas deferens and the globus major. Doctor Edward Martin was the first to do this type of operation (14). Such an operation offers the only chance of recovery. According to Hagner (14) there are two conditions necessary for a successful

result in sterility of this type: first, the vas must be patulous above the point of the anastomosis; second, the globus major, or the upper part of the body of the epididymis, must contain live spermatozoa. Hagner and Fuller (17) described this operation in 1907. At that time they made the following remarks: (a) the condition of obstruction can not possibly be made worse by operation; (b) there is double chance for success, as patency on one side is sufficient; and (c) the operation has been found feasible.

In cases where the occlusion is found high on one side or testicle and low on the other side, Hagner (14) describes an operation involving a cross anastomosis, that is, joining the vas from one testicle to the globus major of the other testicle.

As quoted by Hagner (14), Hotchkiss, in doing anastomoses, has had no successful cases except those in which silver wire was used as a suture. It is his impression, and also that of Hagner, that there is less tissue reaction from silver wire than any other type of suture.

When the obstruction is in the lower portion of the epididymis, Lespinasse had performed vaso-epididymostomies, or a joining of the vas to one particular



epididymis tubule loop. The obstruction is excised and an end to end anastomosis is done. He has had 60-70% successful results with this type of operation. When the obstructions in the vas are surgically inaccessible, deep down in the pelvis, Lespinasse does a sac operation. Here, he converts the tunica vaginalis into a spermatocele. When it is desirable to obtain sperm, the spermatocele is tapped and then the sperms are injected into the wife's uterus. Other obstructions to be treated are such as hypertrophied verumontanum and polypoid tissue in the posterior urethra. These polyps should be burned out with monopolar current, and the verumontanum shrunk in the same way (34).

Surgical removal is the only treatment in the case of varicoceles of the testes affecting the proper function of the glands. Care should be taken not to remove too great a portion of the venous plexus (34).

A surgical procedure, orchidopexy, is employed in the treatment of undescended testicles. This is a suturing of the testicles to the scrotal sac. In cases successfully operated upon during as early childhood as possible, the testicles usually go on to normal gross development and a considerable number produce satisfactory spermatozoa (34). All urologists agree that

the operation should be done before the beginning of adolescence. Some perform this as early as the third year (34).

Meaker (34) advocates prompt surgery to minimize local tissue damage in the case of orchitis as a complication of mumps. His method is that of multiple incisions through the tunica albuginea to relieve pressure and render necrosis less likely. At the same time the operation decreases pain and shortens the whole course of the affection.

If hypospadias or epispadias are to be treated surgically, the operation should, as a rule, be performed during childhood (34). However, in sterility cases, the easiest means of overcoming epispadias is usually artificial insemination.

### Conclusions

It must be borne in mind that several factors of male sterility may exist in the same individual simultaneously and that any factor may overlap another.

It must be concluded that there exist such cases, obscure in nature, for which there is no diagnosis or remedy and also some which are not obscure for which there is no remedy.

It must also be concluded that, in considering the diagnosis and treatment of male sterility, the urologist is not the only one concerned as may primarily be thought, although the majority of cases are urological problems. In viewing all the different types of factors playing their part in sterility, we can see that it is necessary to have the cooperation of the internist, neuro-psychiatrist, urologist, gynecologist and the surgeon. Therefore it would hardly be fair to place the entire burden of the male sterility upon the shoulders of the urologist alone, nor would it be fair to place the burden on any one of the other specialties.

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