EXAMINATION OF SEMEN WITH SPECIAL REFERENCE TO ITS GYNECOLOGICAL ASPECTS.*

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(With ten illustrations.)

THE frequency with which male sterility results from the lesser degrees of seminal defect is not realized; nor are the pathological conditions of the semen upon which sterility depends well understood. Proof of this is found in a review of the literature, which is very scant on this subject, especially in this country where the examination and study of semen has been much neglected. This may have been due, in part, to the unpleasant nature of the work, but more particularly to the difficulty encountered in securing properly collected specimens for examination. While always eager to claim his share of glory in the production of his offspring, a man is most reluctant to share any suspicion of responsibility for failure. In this feeling he has always been sustained by the attitude of the physician. Undoubtedly the mind of the medical profession has been prejudiced; and the study of this subject has been seriously handicapped by the almost universal assumption on the part of the laity that in the event of a childless marriage the wife is wholly responsible.

It is not difficult to understand why such an erroneous impression has prevailed so long. In the male, ability to copulate and the

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normal ejaculation of semen are regarded as sufficient evidence of his power to procreate; while in the female, the process of ovulation is an obscure one and therefore more readily suspected to be at fault.

It is significant that the more study and observation this subject receives, the higher is placed the percentage of male sterility. Two decades ago Matthews Duncan said, in a lecture on sterility, "Enlarged experience and inquiry make me more and more convinced of the greatness of the part played by the male." In countries where venereal diseases are more prevalent than they are here, observers have placed the proportion of cases in which the male is at fault at a surprisingly high figure. Thus Vedeler, of Christiania, reports that 70 per cent. of the childless marriages he investigated were due to the husband; while Kehrer reports a series of cases in which he found the male responsible in 40 per cent. These figures are too high for general acceptance. Most American writers place the male responsibility at from 15 per cent. to 25 per cent. I believe this to be a too conservative estimate. In cases of absolute sterility, the number in which the husband is at fault must be high at least one in three, for the sexual hygiene of the woman before marriage is usually better than that of her mate, and there is no real evidence to prove that the physiological processes involved in the production and delivery of the healthy ovum are more complicated or less often successful than is the secretion and emission of normal semen.

But at the present time it still seems advisable to seek first the cause of a sterile marriage in the female. *It must be stated, however, that to conduct long and exhaustive gynecological treatment and ultimately to offer a hopeless prognosis without having investigated the reproductive powers of the husband is neither fair nor scientific.* The opportunity to secure the semen for examination presents itself oftenest to the gynecologist and he should be equipped to make this examination as a routine part of the investigation of sterility. From such a viewpoint this study is contributed.

J. Marion Sims reasoned far in advance of his colleagues when in 1869 he wrote: "I insist that we have no right to perform any operation or to institute any treatment whatsoever solely with a view to the cure of sterility until we have settled the three propositions, above laid down, touching the presence and vitality of the spermatozoa." The propositions referred to were: (*a*) We must be sure that we have semen with spermatozoa; (*b*) we must ascertain if the spermatozoa enter the uterocervical canal; (*c*) we must determine whether the secretions of this canal are favorable or not to the vitality of the spermatozoa.

To-day, with superior opportunities for study at hand, we have no right to consider the study of the semen completed when we have demonstrated singly the presence or absence of active cellular bodies. Determination of the activity of the spermatozoa is not sufficient to assure us of their power to impregnate the ovum, neither is the absence of motion an infallible sign of their impotency. In general, the fertility of the semen depends upon the presence of:

1. Mature living spermatozoa (normal cells).

2. A normal secretion (liquor seminis) to convey the spermatozoa to the vagina and to maintain the vitality of the cells until such time as they may meet the ovum.

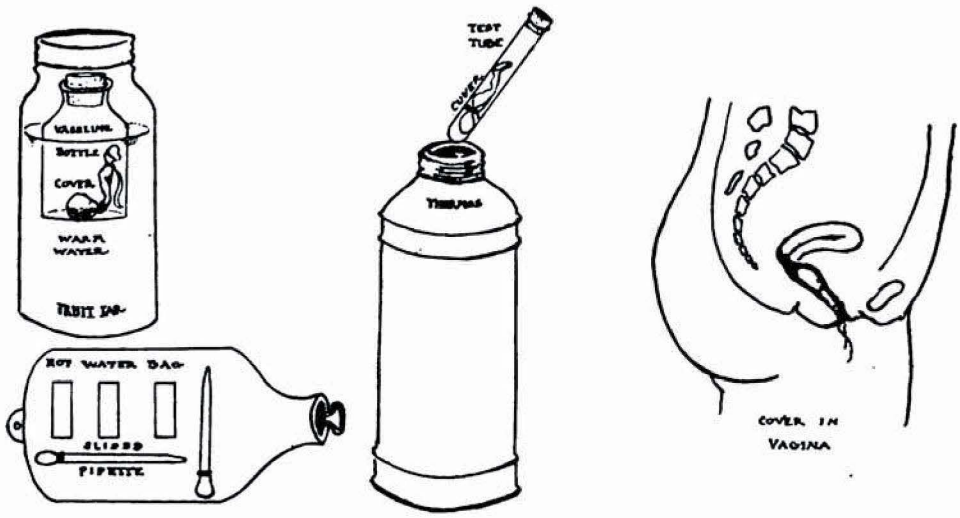

FIG. 1.

Finger and Saenger have divided male sterility into two groups: impotentia cœcundi and impotentia génerandi. Our subject pertains only to those conditions belonging to the second group, and will be confined to a consideration of the pathological conditions found in the semen, their etiology, and their treatment. Aspermia and conditions resulting from genital deformities will not be touched upon.

Method of Obtaining and Examining the Specimen.—In order to determine accurately the viability of the spermatozoa and the impregnating power of the semen great care must be exercised in preserving the specimen *en route* to the microscope. The most satisfactory arrangement for an examination in made by conveying the necessary implements to the home of the patient and making the observations immediately after conclusion of intercourse. The instructions here given apply more particularly to office observations.

The patient provides himself with the following articles: condoms, a wide-mouthed bottle like a vaseline bottle, and a jar which may be made water tight (Fig. 1). Upon the morning when the examination is to be made the doctor should be notified so that he may be prepared to work promptly. The specimen should be secured after three or four days of sexual rest. After intercourse the condom containing the specimen is placed in the wide-mouthed bottle and this is carefully corked. The bottle containing the condom should then

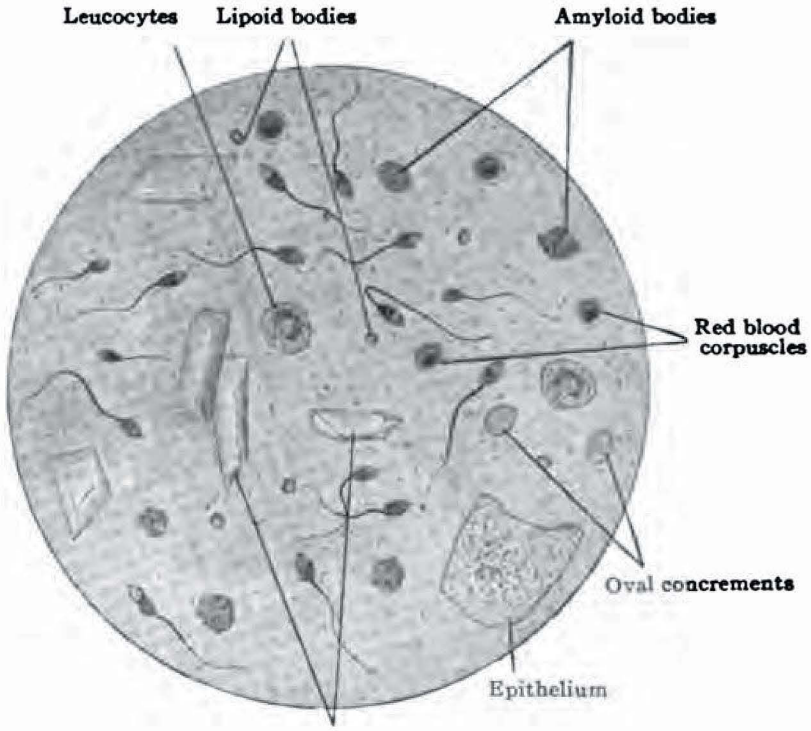

FIG. 2.—Elements which may be found in microscopical examination of the semen.

be placed in the jar which should contain water a few degrees warmer than body temperature. The jar is then immediately taken to the office of the physician. These precautions are necessary to maintain the warmth of the specimen. (If this method is refused by the husband, the semen may be secured from the genital tract of the wife who places a tampon after intercourse and reports at once to the doctor. Under the latter condition a normal finding only is of value as so many elements may enter to affect the condition of the specimen.)*

* Dickinson has developed the ingenious scheme of having the condom placed in the vagina and held there by the insertion of a tampon. The wife then comes to the office and the condom is removed and the examination proceeds. While this method assures the warmth of the specimen, the technic is not as readily carried out and is objectionable to some.

Upon delivery at the office, the bottle containing the specimen is removed from the jar and placed in a warm—but not—a hot bath. The examination should begin at once. The base of the condom is opened with scissors and the specimen is allowed to escape into a dry bottle or warm test-tube; the total amount of the specimen, the reaction, and the amount of sediment should all be noted. Also the temperature should be observed as well as the time that has

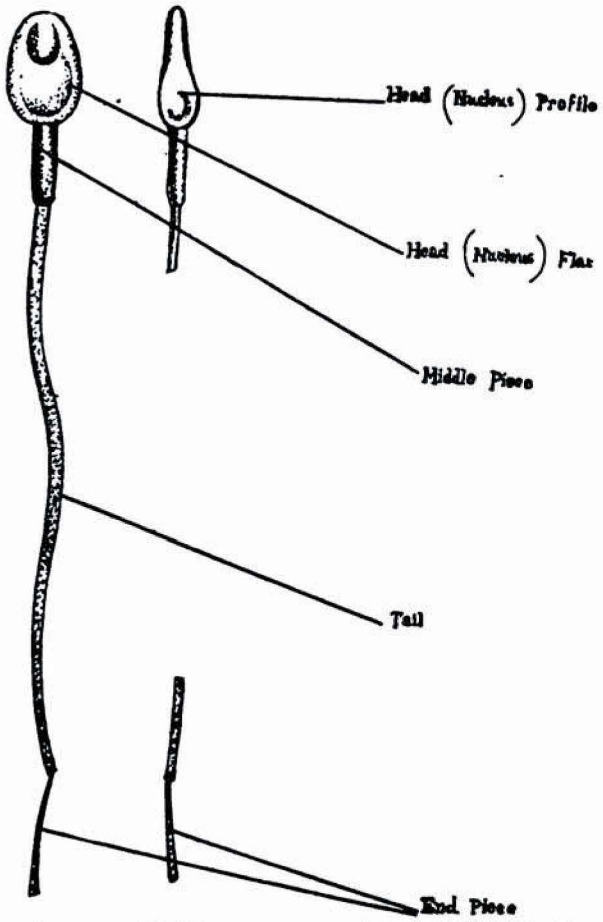

FIG. 3.—B. Human spermatozoa. (*Retzius.*)

elapsed since coitus. After remarking the gross appearance of the specimen, a drop of the semen is spread upon a warm slide, in very much the same way that is used for urine sediment, and examined with a high power lens. In this manner the best general and detailed study of the efficiency of the semen may be made (Fig. 2). I say this advisedly after trying the ordinary staining methods and the dark field apparatus.

If the semen is normal, and the instructions for its collection have been carefully carried out, the microscope will demonstrate a field

filled with active spermatozoa of fairly uniform size, shape, and activity (Figs. 3 and 4). If, however, the sediment is greatly reduced in amount and the microscope shows a diminution in the number of spermatozoa, or sluggishness and lack of motion, early crystal formation, or presence of pus the specimen is probably defective.

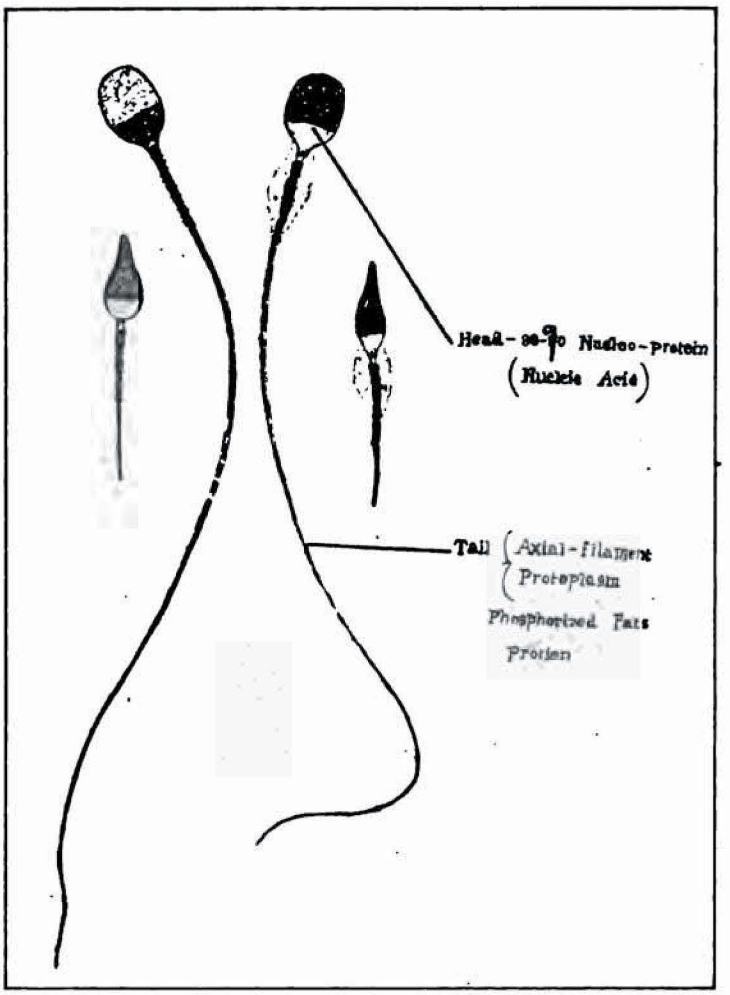

FIG. 3 A.—Human spermatozoa on the flat and in profile. (*Bramman, from Schaffer.*)

Under such conditions a more detailed examination must proceed. The sediment is covered with the thinnest cover-glass and examined with the oil immersion lens.

Semen devoid of its cellular elements is thin and usually coagulates rapidly, while the sediment, which normally constitutes two-thirds of the discharge, is very slight. The early formation of crystals is reported to denote a decrease in the number or the entire absence of spermatozoa. This commonly accepted sign, I have been

unable to confirm. These spermatic crystals, which are sometimes called after Boettcher who, with Van Deen, was the first to recognize them, are rhombic transparent bodies easily discerned under the microscope (Fig. 2). Fürbringer has demonstrated that such crystals occur exclusively in the prostatic secretion and indicate functional activity of that gland.

Ultzmann* describes the following varieties of semen in which spermatozoa are not found or are greatly reduced in number: (*a*)

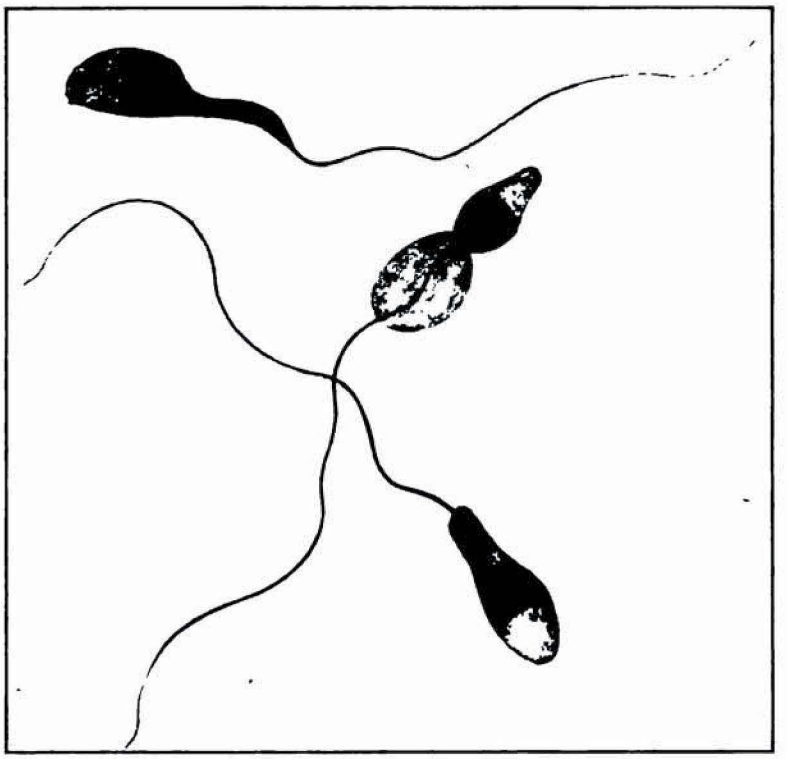

FIG. 4.—Normal forms and modifications of apparent importance.

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.

* If it should be desirable to stain a specimen the following method may be used. I quote from the book of Greene-Brooks: "The specimen may be spread upon a slide and fixed by heat, or by means of methyl alcohol, formalin 10 per cent., or alcohol. Slides so prepared may be stained by practically any of the chromatic dyes of which methylene blue, fuchsin, or gentian violet are best. When a sightly preparation is desired the specimen may be stained by Boehmer's hematoxylin and counterstained by eosin." Full directions are also found in an article by Martin, Carnett, Levi and Pennington, Univ. of Penna. Bull., March, 1902, p. 2.

After some practice variations from the normal will be readily noticed and their importance properly appreciated. A normal finding is conclusive, but if a pathological condition is present findings should be confirmed by subsequent examinations.

Etiology.—The most common cause of sterility in the male was formerly attributed to the absence of spermatozoa in the semen. Kehrer found this the cause in 21.3 per cent. of his cases. While many cases of azoospermia have been reported for which no cause was assigned, it is doubtful whether idiopathic azoospermia occurs. Hirtz reported two cases which he considered idiopathic but which have not been so accepted by subsequent investigators. The commonest cause of azoospermia is gonorrhea. In a very large proportion of cases this condition results from a unilateral or, more often, a bilateral epididymitis. One of the most valuable contributions to our knowledge of the part played by gonorrhea in sterility was made by Benzler, a German army surgeon. He was able to follow the history of 473 of his patients who afterward married. Of those with simple gonorrhea, 10 per cent. were childless; while 23.4 per cent. of those with unilateral epididymitis and 41.7 per cent. of those with both epididymes involved were without children. These findings have been generally corroborated. A few authorities, however, believe that gonorrhea is not so often a cause of azoospermia as these figures would indicate.

Another cause sometimes responsible for the disappearance of spermatozoa from the semen is exhaustion due to abnormal demands upon the sexual organs. In these cases the absence of the sperm cells is only temporary and the condition is classified as physiological azoospermia. Gross states that nervous exhaustion alters the character of the semen by causing perverted enervation of the sexual organs. It would seem that neurasthenia and the other neuroses which are prominent features of these cases and which are sometimes considered causative factors, are more often symptomatic, being, in common with azoospermia, a result of intemperate sexual habits. In a more recent contribution to the literature, Hoppe affirms that derangements of the nervous system cause sterility in the male only in those cases classed as impotentia cœundi with which our subject is not to be confused.

In modern times the *x*-ray has figured prominently as a cause of azoospermia. While it may yet be too early to state positively, those qualified to express an opinion believe that the *x*-ray is not likely to produce permanent sterility.

There is no question that the importance of syphilis and tubercu-

losis as causes of sterility was exaggerated by the early writers. In the work of Bangs-Hardway the statement is made that except as it causes cachexia or destroys the testes, it is doubtful whether syphilis influences the condition of the semen. Heidingsfeld, who reviewed the literature of this subject, and especially the work of Lewin and Hanc, beside making personal observations, is of the same opinion. The relation of tuberculosis to anomalies of the semen is a subject in regard to which widely different views are entertained. Not unlike syphilis, when tubercular processes attack the genitals or when the terminal cachexia is present, azoospermia results. It has been conclusively proved, however, by thorough investigations quoted at length by Gross that the semen of consumptives contains spermatozoa quite as frequently as that of normal persons. Great weakness occurring in the course of any chronic disease may result in impotency, and Hagner states, with reason. that the virility of the spermatozoa is often in direct proportion to the general physical condition of the patient.

Simonds examined the semen of several alcoholics at autopsy, and obtained results which led him to believe that in chronic alcoholics the function of the testes was at times suspended. In these cases the condition was apparently dependent upon a fatty degeneration of the testes.

Cases have been reported which would seem to indicate that the immoderate use of tobacco occasionally causes sterility. Such views were held by Peyer, Hanc, and Curling. It is reasonable to suppose that tobacco, like morphine and other sedatives, might, after a time, cause impotency by deranging the nervous mechanism of the sexual organs, but it seems highly improbable that it exercises any deleterious effect upon the production of spermatozoa.

There is little in the literature touching upon obesity as an etiological factor in male sterility. Kisch, who has done considerable work in this line, made frequent examinations of the semen of corpulent persons and reports that he found but few spermatozoa in many of the specimens, and that these were often not motile. He states that in 9 per cent. of his overcorpulent patients spermatozoa were entirely absent from the semen. Just what the pathological condition was that explained the azoospermia is not given.

Immature Cells.—In addition to azoospermia and other gross conditions there are cases in which the fertility of the semen is greatly diminished by immaturity of its fecundating elements (Fig. 7). This condition is indicated by morphological changes in the spermatozoa due to an arrest in their process of evolution. These irreg-

ular types of cells are difficult to classify. The condition as related to azoospermia might, however, be considered an intermediate stage. Accompanying the change in form, it is usual to find the sperm cells reduced in number (oligospermia), and macroscopically the semen assumes more or less the character of that described as azoospermia. If the reduction in the number of cells is marked it is, of course, quickly apparent, if not, an accurate estimation of the productiveness of the semen depends upon the recognition of the imperfect spermatozoon. To facilitate the study of these immature cells, it is well to take a moment to review the cycle of phenomena relating to the evolution of the spermatozoon.

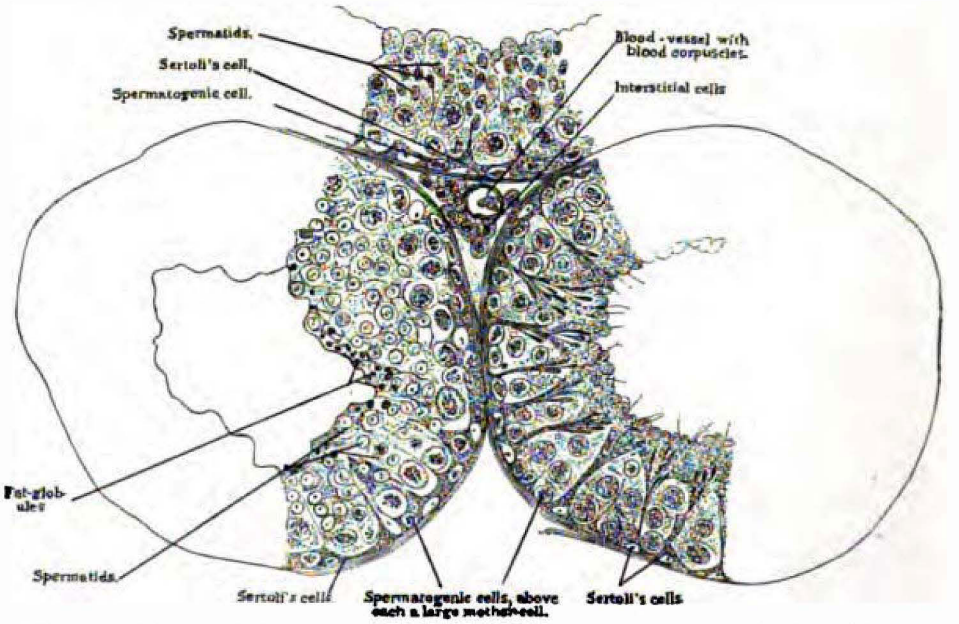

FIG. 5.—Cross-section of seminiferous tubules of a mouse. × 360. Observe that the nuclei of the spermatids (below on the left) at first round, become oval below and are transformed (below on the right) into the heads of the seminal filaments. (*Stohr.*)

The spermatozoa are formed by a process of division from cells which lie next to the basement membrane of the seminiferous tubules (Figs. 5 and 6). The ancestral (spermatogenic) cells which are naked epithelial cells come, by a process of indirect division, to be large cells which form a layer nearer the lumen of the tubule. These are the mother cells (spermatocytes), each of which, later on in the process, divides twice, thereby forming four cells known as the daughter cells. These daughter cells are really the spermatids or semen cells and are now in a zone still nearer the lumen of the tubule. The nuclei of these cells, which are primarily round, then become oval in shape, while the protoplasm of the cell forms the cau-

dal filament. The cells are then mature and as spermatozoa make up the secretion of the testicle. The semen as ejaculated is composed of the spermatozoa suspended in the secretion of the prostate and accessory glands (liquor seminis). The activity of the sperm cells is not manifested until this union has taken place. The head is the essential fecundating part of the cell. The tail, by the motion of its cilii, executes sinuous movements which result in the well-known

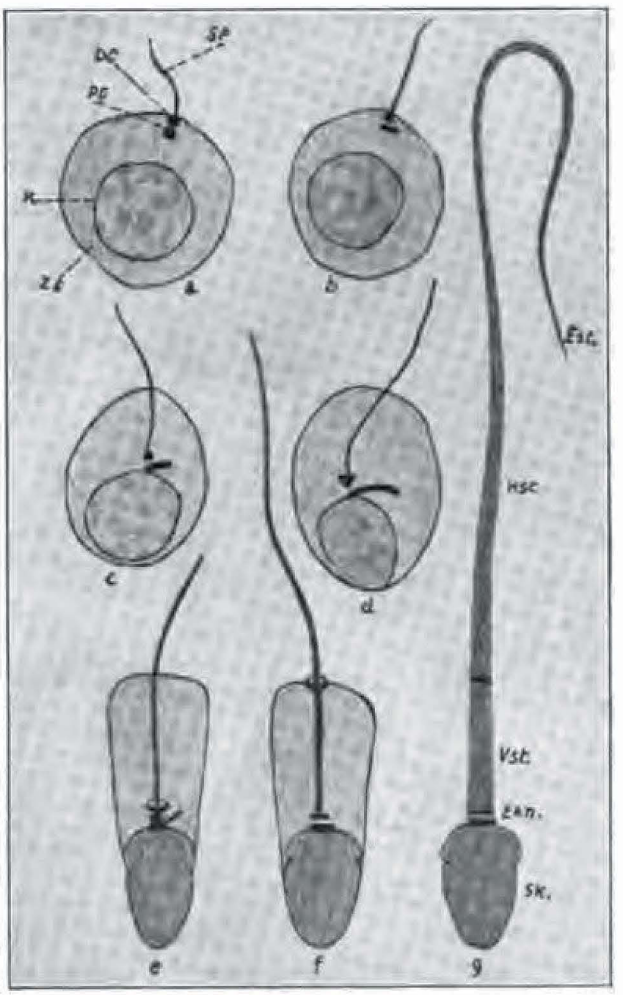

FIG. 6.—Seven stages of the conversion of a spermatic cell into a spermatozoan. *a* to *f*.—*Zs*, Cell contents; *K*, nucleus; *Pc*, proximal central body; *Dc*, distal central body; *Sp*, tail piece; *G*, head piece; *Ekn*, neck; *Est*, endpiece.

activity of the spermatozoa. Normally this is sufficient to liberate the cell from its medium and carry it to that part of the female reproductive tract where it will meet the ovum.

As stated above many of these irregularly formed spermatozoa are cells which have been cast off in the seminal discharge before they are fully developed. Evidences of immaturity are to be found

in abnormalities of both head and tail. The heads of these immature cells instead of being oval or pyriform, as in the normal specimen (Fig. 7), are round, corresponding in appearance to the nuclei of the spermatogenic cells while in the mother or daughter cell stage of transition. Not infrequently the heads of these cells are much increased in size being usually as large as red corpuscles and occasionally the size of the lymphocyte. The name megacephalic is desig-

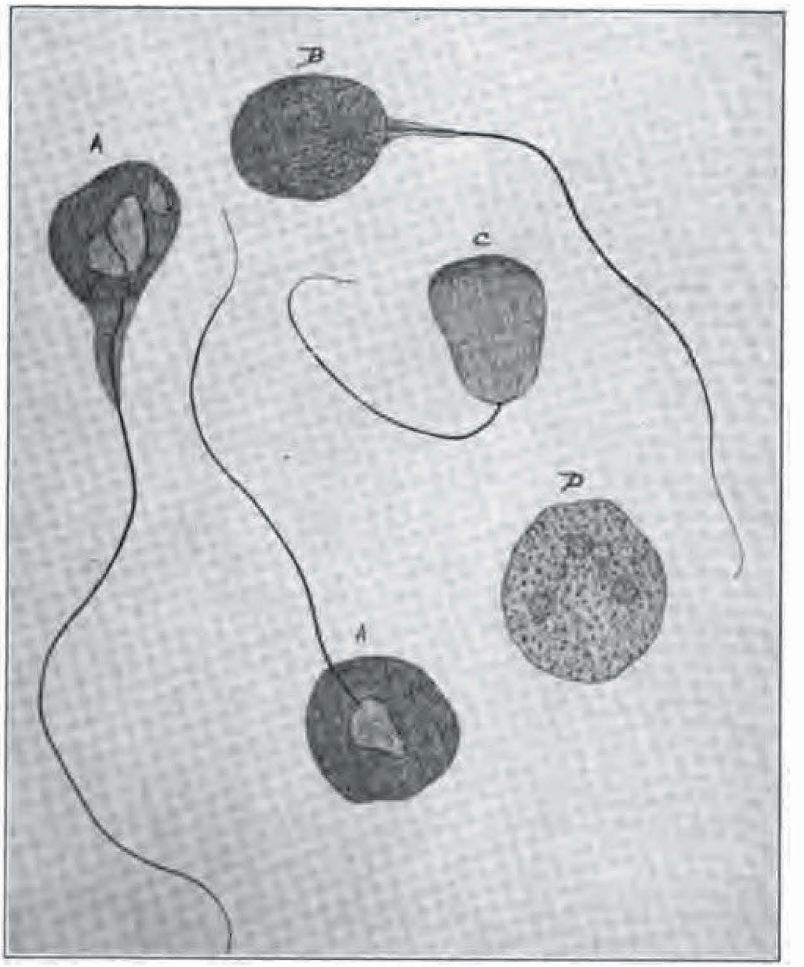

FIG. 7.—Immature types. *A*, Intermediate stage; *B*, large round head without nucleus; *C*, same type with blunt tail; *D*, leucocyte for comparison of size. Found in defective specimens due sometimes to too great sexual activity.

native of this type of spermatozoon. I have seen cells in which the protoplasm still surrounded the nucleus in ordinary cellular type with an active tail piece of some length. It is unusual, however, to find them deformed. Very often they are short and blunt, or, as occasionally occurs, the caudal extremity may be entirely lacking. These cells are easily recognized if the appearance of normal sperma-

tozoa is kept in mind. The majority of them are motionless and are not viable. Others are active but only for a short time and are probably incapable of impregnating an ovum. The production of these immature cells is an effort on the part of the testes to supply an abnormal demand, and when present, they indicate that the fertility of the semen is much impaired. If the excessive demand continues, azoospermia ultimately develops.

Deformities.—The fecundating power of the semen may be greatly lessened by the presence of many malformed spermatozoa (Fig. 8). Such cases are not rare. These abnormal cells cannot be properly placed under the immature class for they present none of the features peculiar to it. Their occurrence is due either to a functional derangement of the testes or to a degenerative process dependent upon some abnormality of the glandular secretion. In these cases, as in the preceding group, oligospermia is usually very pronounced and but few of the sperm cells are active. Ordinarily no one variety of deformity is peculiar to a given specimen; on the contrary, many different forms of faultily developed spermatozoa will be noticed. For the purpose of classification the deformities of the spermatozoa are best described under two general headings: (*a*) cephalic deformities; and (*b*) caudal deformities.

Cephalic Deformities.—A very common abnormality is the reduction in the size of the head. The term microcephalic has been employed to describe these spermatozoa. Such cells are surprisingly numerous in some specimens. Every degree of diminutiveness may be noted. In some instances the head is barely perceptible, appearing as simply a clubbed end of the tail. In these same specimens it is usual to find many caudal extremities with the head entirely absent or not distinguishable under the ordinary lens. Fig. 8 is a drawing taken from a specimen of this kind. At present it seems impossible to determine whether such deformed cells represent faulty development or are due to a degenerative process occurring subsequent to their formation. The fact that in a majority of the cells the tail is apparently fully developed and that in the normal process of evolution the tail is the last part of the cell to be exhibited tends to favor the latter theory. Other deformities of the head characterized by a ragged uneven outline of this extremity are not infrequent. The head of these inert cells may resemble a disintegrating corpuscle, while crescentic and other irregular shapes are not rare.

Caudal Deformities.—In the normal specimen, the tails of the spermatozoa are nearly uniform in size and are very active. Slight

variations in length occur but have little significance if the rest of the cell is normal and active. In defective specimens abnormalities are frequently present in the way the tail joins the head.

Instead of forming one extremity of the cell the head may be at the side of the caudal portion. In other spermatozoa there is a sharp angle in the tail near the cephalic end and sometimes the head and tail are disunited although each portion may in itself appear normal.

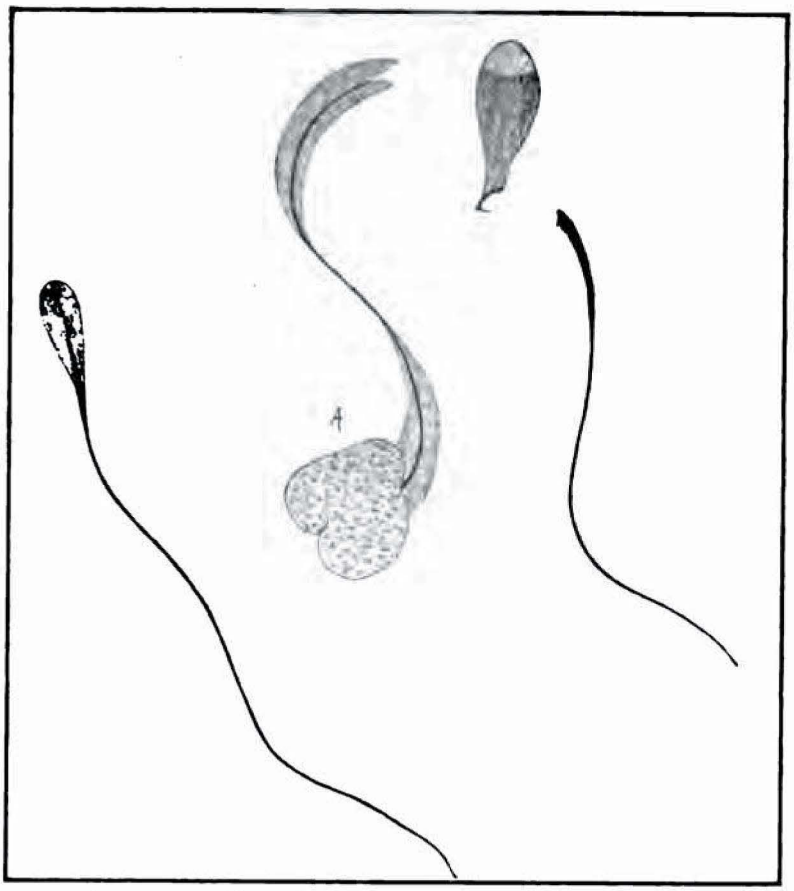

FIG. 8.—Headless and tailless forms found in great numbers in some defective specimens. Probably degenerative forms.

Sometimes the tail is rudimentary or entirely absent. In one specimen which I examined the last variety was very numerous (Fig. 8). It seems scarcely necessary to state that these cells with the deformed tails are inactive and unfertile.

Immature and deformed spermatozoa often occur in the same specimen and the extent to which the semen is impaired depends upon: (*a*) The degree of oligospermia; (*b*) the percentage of imperfect spermatozoa; (*c*) the percentage of cells that are motile and their degree of activity—whether sluggish or lively; (*d*) the length of

time activity persists under favorable conditions. Upon this basis a specimen may be said to be 25 per cent., 50 per cent. or 100 per cent. efficient; or it may be classified as sterile, poor, fair, or vigorous.

I have noticed the double-headed and multiple-tailed cells (Fig. 9) first described by Maddox and do not believe them to be rare. What their significance may be is not understood but their activity

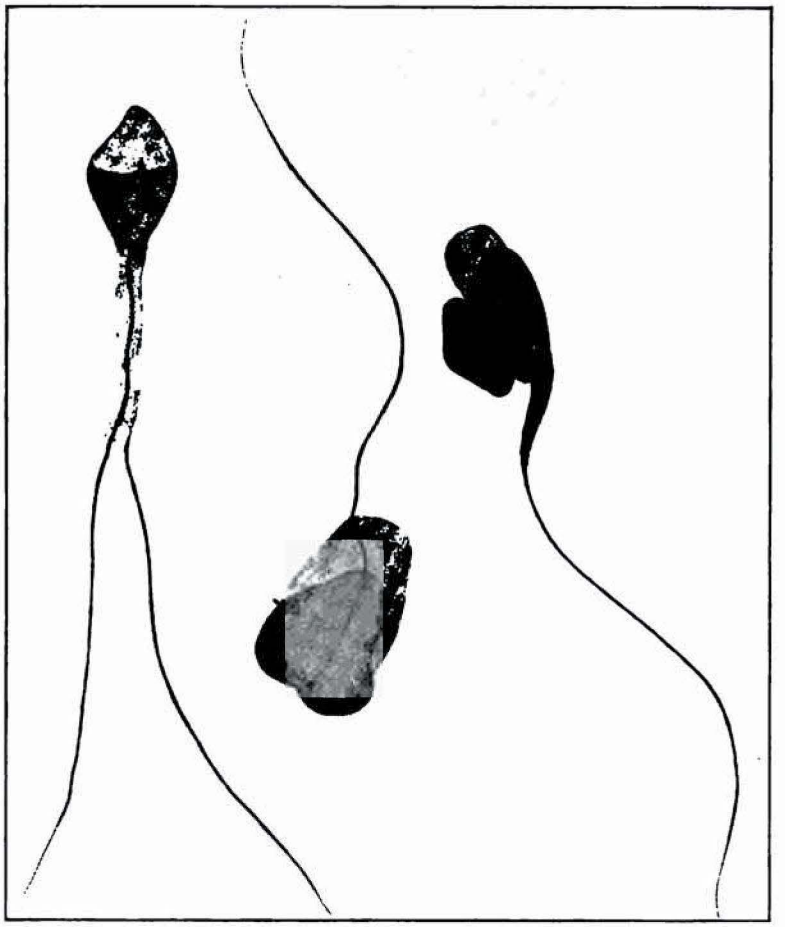

FIG. 9.—Double-tailed and double-headed forms. Their significance is unknown.

is as pronounced and as continued as in the normal type and I am inclined to believe them potent.

Viability.—Inasmuch as it is not determined definitely at what time the ovum is freed from the ovary, and in view of the physiology of ovulation it is obvious that the successful completion of the process of fecundation requires that the spermatozoa shall not only have the power to migrate to the interior of the uterus or tube, but that their vitality must be sustained until the ovum is presented. To this

end Nature produces thousands of fecundating cells that one may survive to perform its complete function.

While it is known that the testes furnish the fecundating elements of the semen, it is likewise important that we should recognize the complementary action of the seminal fluid. In addition to furnishing a vehicle for the spermatozoa, it contains properties that are essential to their vitality. As early as 1871 Kraus showed that in the absence of the prostatic fluid the spermatozoa would not live in the uterine mucous membrane. Later on Sims made the same observation.

Under normal conditions the vitality of the spermatozoa is remarkable. Gross, in discussing the microscopical examination of the semen, says that their motion should continue or be capable of being reëstablished for twelve hours. To state an arbitrary time is impossible, but we know that if proper conditions are afforded their motion continues much longer than this. Various references as to the duration of their motion are found in the literature (Biegel). It may be stated, first, that in their proper medium and at the body temperature the viability of the sperm cells may extend over a period of a few days; second, that their prolonged vitality is probably dependent upon the normal lime salts of the prostatic fluid, third, that the sustaining power of the seminal fluid is increased by its union with the normal secretion of the female genital tract.

The spermatozoa are, however, extremely sensitive. I have found that they perish promptly in tap water and in faint lactic acid mediums or under other minor changes in their environment. In the same study it was found that the sperm cells were adversely influenced by increased acidity of the vaginal secretions or by alterations in the cervical secretions. But normally these secretions are bactericidal and act as a chemical stimulant attracting sperm cell to cervix.

I have been much interested in an experiment made recently in which two specimens were obtained simultaneously. One was taken directly from the male, the other from the vagina where it was mixed with the secretions incident to normal intercourse. This revealed that while the specimen taken directly appeared poor it showed an exaggerated activity when mixed with the vaginal secretions. Such an experience suggests that to make our study thorough we must not neglect to determine the degree of physiological affinity existing between the male and female secretions.

One of the less common forms of seminal defect is that resulting from too great density of the semen. The spermatozoa being com-

posed of suspended bodies, their activity is naturally inhibited by any abnormal increase in the specific gravity of the seminal fluid. Such a specimen when placed under the microscope shows normal cells but their motion is sluggish and of short duration or entirely suspended. If, to such a specimen, a few drops of normal saline solution be added the cells will at once become active. If they fail to do so they are probably no longer viable. Similar conditions may be found where an altered state of the prostatic secretion causes an increased coagulability of the semen. Here, as in the former condition,

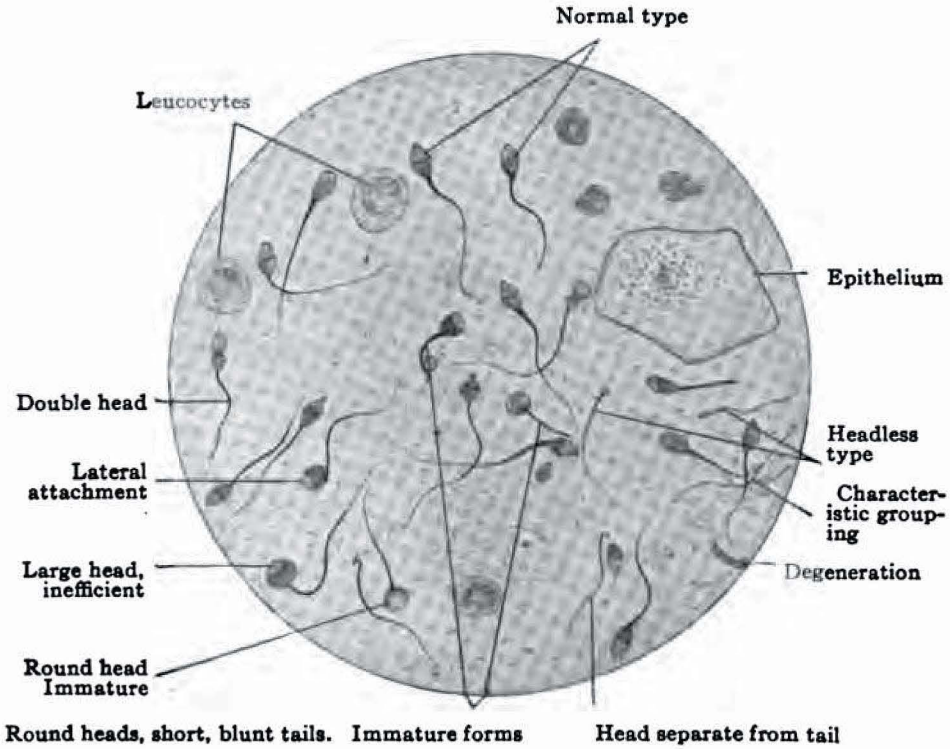

FIG. 10.—Defective specimen sketched two hours after emission; well preserved. Thin and little sediment. Total number of spermatozoa reduced; one in three active. Deformed, immature, and degenerate forms.

the semen, soon after deposit in the vagina, becomes a gelatinous mass from which the spermatozoa are unable to escape. Leigois, in one of his cases which is often quoted, believing this condition to explain the sterility of a patient ordered that coitus should be followed by an injection of saline solution into the vagina, and pregnancy actually resulted.

Of still rarer occurrence are those cases where the fertilizing elements of the semen are destroyed by the presence of pus and blood in the seminal fluid. These foreign substances are found in the semen in inflammations of the epididymes, the seminal vesicles, the

vas, and the prostate. The available data justify the assertion that pus is destructive to the evolution and life of the sperm cells, and probably explains in part the sterility of women who suffer from endocervicitis and endometritis. Sims states that catarrhal conditions of the cervix cause sterility by increasing the density of the semen rather than by any chemical action. A tenacious mucous plug is often found in the cervical canal of sterile women, mechanically obstructing the entrance of the semen.

There is some difference of opinion in regard to the injurious effect blood exerts upon the seminal elements. My observations confirm those of Robin who demonstrated that spermatozoa would live four or five hours in blood, while Dieu showed that when blood had mixed for some time with the contents of the seminal vesicles, the sperm cells were reduced in number or entirely absent. The findings of these investigators represent the opinion now generally accepted, which is that while blood in the semen exercises a very harmful effect upon the vitality and fecundating powers of the spermatozoa the semen must, however, have contained the blood for some time before such changes are produced. It is evident, therefore, that hemorrhage within the seminal vesicles would be the only way in which blood could affect the virility of the semen before emission. In instances where blood appears as one of the elements of inflammation destruction of the spermatozoa occurs because of toxicity.

Treatment.—The treatment of male sterility has been less studied and has received less attention in the literature than any other part of the subject. This may be explained by the fact that the major part of the investigation of these cases has been carried on in foreign countries where the treatment of disease does not receive as much attention as the other branches of medical science.

Many of these cases can be helped. Others are hopelessly incurable. The percentage of the favorable cases is large enough, however, to warrant careful study of each case. Unless dependent upon obviously incurable conditions, sterility in the male justifies the same effort in its correction as when it occurs in the female. If success is to be attained, a thorough knowledge of the etiology and pathology of the individual case is imperative.

A comparison of the statistics of other countries with our own demonstrates the important rôle played by venereal disease as an etiological factor in sterility. This at once introduces the subject of prophylaxis, which is much too broad a subject to be taken up in this paper. Suffice it to say, that if it is made possible to educate the mature members of society in this matter as they are being

instructed with regard to tuberculosis, venereal disease would fast decrease and sterile marriages would become a much less common occurrence. Another means of accomplishing much along similar lines would be a disposition on the part of the general practitioner to refer these cases to those qualified by special study to treat them. Prostatitis, epididymitis, and inflammation of the vesicles often result from unskilled treatment or urethritis and are responsible for sterility in no small proportion of cases.

If, after careful study of the pelvic condition of the wife, it be suspected that the cause of the sterility is to be found in the husband, a detailed history must be secured, and much further study of the case is often required before the tentative diagnosis may be confirmed or denied. If by such study it is found that the patient is sterile, classification of the case either under impotentia cœundi or impotentia génerandi will not be difficult. The treatment of those conditions of the second group which have been discussed under the foregoing headings will alone be considered here.

A class of cases amenable to treatment is that in which sterility has resulted from too frequent intercourse Such hygienic errors are at times made by young married people and occasionally they occur later in life. Similiar results may follow excessive sexual indulgence by those who erroneously think that they may thereby increase the likelihood of pregnancy. Very much like these are the cases in which the fertility of the semen is impaired by involuntary emissions and faulty habits. In the conditions cited, the spermatozoa may either be absent or much decreased in number. In the latter event, variously deformed and immature spermatozoa will be present which are fairly characteristic of this class of cases. Motion of the spermatozoa may be suspended or an occasional cell may show activity.

The treatment of these cases consists chiefly in regulating the sexual life, correcting unwholesome habits, or adopting measures to check involuntary seminal loss. A frank, friendly explanation by the family physician will usually be sufficient. When such excesses are stopped the testicles may be relied upon to resume their normal function unless atrophy has occurred.

Sterility due to defective semen may exist in men in whom there is no apparent cause other than a much debilitated condition incident to an overactive business career. Such men are aware that they are exhausting their energy. Evidences of it are obvious in various neuroses and digestive disturbances. It is not difficult to believe that the reproductive system shares in the general depression, and

that similar methods must be adopted in its correction as in the treatment of nervous and digestive disorders. Accordingly a shorter business day is recommended, or a vacation is ordered for the more serious cases. Systematic exercise is prescribed—golf, sailing, swimming, etc., on certain days for a fixed number of hours. In winter fast walking and well-regulated gymnasium work are excellent, while the cold shower and brisk rub which should follow are not the least helpful part of the prescription.

The sexual habits of these patients must be investigated. Drugs play a very small part in the treatment of these conditions. Sometimes tonic treatment is required, while sedatives may be indicated in others. In the treatment of impotency and some forms of sterility, the choice between stimulation and sedative treatment is an important and difficult one. If the reproductive power of these men is to be reëstablished, details as to their manner of living must be diligently studied and such changes must be made as are conducive to the betterment of their general health. In excessive smokers, stopping the use of tobacco or restricting its amount may be followed by happy results. In others, the prohibition of alcohol or the interdicting of drugs may be necessary to secure good results.

Some cases of sterility occurring in the overcorpulent may be cured by treatment of the obesity. If it be true that in some cases obesity results from a disturbance of an internal secretion of the testicles and is in that event only a symptom of tissue change in the testes, as is azoospermia, treatment directed to the obesity will be without effect.

Azoospermia resulting from chronic inflammations or exudates due to a remote gonorrhea is very unsatisfactory to treat. A few of these cases will improve and may be cured if placed in the hands of the genitourinary specialist. A cure has been reported as long as two years after a double epididymitis. If the defective state of the semen be dependent upon the presence of pus or other inflammatory elements local treatment directed to the inflammation of the prostate or seminal vesicles may be curative. Azoospermia, when present in patients with a negative venereal history should excite a suspicion of some chronic constitutional disorder. It must not be forgotten that absence of spermatozoa may occur in such rare conditions as cryptorchidism, congenital absence of the testes, congenital deficiencies of the excretory passages, and malignant disease of the genitals. When dependent upon such conditions, except in rare instances, azoospermia is absolute and permanent. Tubercular disease of the testes and syphilitic orchitis render the

prognosis very unfavorable. If the syphilitic condition be diagnosed early, mercury and the iodides may reëstablish the spermatogenic power of the testes. Delayed development of the testes does not necessarily produce permanent sterility. Full development with the establishment of normal functions may occur under proper sexual influences.

Summary.—In the study of sterile marriages, to conduct exhaustive gynecological treatment and ultimately to offer a hopeless prognosis without investigating the reproductive powers of the husband is neither fair nor scientific.

Semen examination, by reason of its intimate character and the vital relation which it bears to the general subject of sterility, is best performed by the gynecologist.

Selection of the method of collection and transportation of the specimen to the office of the examiner must be made to suit the individual conditions, with special regard to maintaining the warmth of the specimen and appointment for immediate examination.

Examination is best made with the high power lens. In addition to noting the general physical properties, the determination of efficiency depends on the degree of oligospermia; the percentage of imperfect spermatozoa—whether immature or deformed; the percentage of the cells that are motile—whether sluggish or lively; and finally, the length of time activity persists.

Recent experiments have shown that a specimen obtained directly from the male, which appears to be poor, may reveal an exaggerated activity when obtained from the vagina where it has been mixed with the secretions incident to normal coitus. Such experience suggests that before an unfavorable prognosis can be made complete study must include an inquiry into the physiological affinity of the male and female secretions.

Observations show a direct relation between the vigor of the individual and the potency of the semen.

Treatment is usually a genitourinary problem. A large proportion of cases is improved by measures which better the general health and sexual hygiene. Twenty-five per cent. efficiency warrants artificial impregnation; fifty per cent. efficiency justifies correction of definite pathology in the female.

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