obvious benefit to the rectal condition. Before recommending this method of treatment we should require a body of evidence that cure of the rectal disease can really be anticipated in a fair proportion of cases. When the rectum is affected above the peritoneal reflection, and is associated with abscess, evacuation of the abscess by the intraperitoneal route is to be recommended, but except for the treatment of this complication operation has no service to offer.

4. Lastly, in selected cases, operation gives good results in limited tuberculous disease of mesenteric glands. According to the extent and stage of the focus this will take the form either of enucleation or of excision of the mesentery involved, together with associated bowel.

SPHENOIDAL EMPYEMA AND EPIDEMIC CEREBRO-SPINAL FEVER.*

BY

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THERE are four varieties of the clinical course taken by cerebro-spinal fever.

First, there is the type which, commencing as an acute illness, may then go on to recovery or may result in death.

There is, secondly, the type of case that, after having shown considerable improvement from the acute stage, suffers from a recrudescence, both in general symptoms and in the condition of the cerebro-spinal fluid. This usually occurs from the fifth to the twelfth day, and may subsequently lead to recovery or death.

Thirdly, there is the type of case that, after convalescence from the acute stage, still retains a tendency to suffer from malaise, headaches, and vomiting, which gradually become more pronounced. The patient becomes extremely emaciated and death occurs from hydrocephalus.

Fourthly, there is the case that, having entirely recovered from the preliminary attack, months later has a relapse which may prove fatal.

Thirty-four *post-mortem* examinations were made in the Netley district between 1915 and 1919, on cases of cerebro-spinal fever dying as a result of one of these four types of the disease. These are all grouped in the accompanying tables under three of these types:

- A. Those dying during the acute illness ... 21 cases
- B. Those dying during a recrudescence ... 6 "
- C. Those dying from hydrocephalus during convalescence ... 7 "

One case only (22) died as a result of a relapse occurring months after the preliminary attack, but as he suffered from an apparently typical attack of the disease with a recrudescence he is grouped under heading B for convenience.

Of these 34 deaths only 11 occurred within the first week of illness. In this observation we differ from Rolleston, who states that "more than half the fatal cases occur during the first week of the disease." A possible explanation of this difference is that at Netley we were fortunately able to insist that every case in which the least suspicion of cerebro-spinal fever was entertained should be immediately admitted to the isolation hospital. On admission, lumbar puncture was performed as a routine within a few minutes of arrival, antimeningococcic serum being administered there and then to cases showing turbid cerebro-spinal fluid. A bacteriological diagnosis was only waited for when there was middle-ear disease or a head wound. This very early administration of serum, often within an hour or two only of the suspicion of cerebro-spinal fever being present, probably saved many cases who would otherwise have died during the first week.

In a previous paper² it was shown that of 36 cases in which it was possible to obtain cultures from both the nose and the cerebro-spinal fluid, in 35 the types of meningococci in the two sites were identical, as proved by both agglutination and saturation tests. The irregularity shown by the remaining case has been discussed.

In no case was any inflammatory process discoverable about the cribriform plate of the ethmoid by naked-eye examination. The contents of the pituitary fossa showed

TABLE I.—A. *Acute Cases.*

No. of Case.	Days from Onset to Death.	C.S.F. Type.	Nasal Type.	Sphenoidal Sinus.		Sphenoidal Bone.	
				Empyema.	Meningococci.	Inflammation.	Meningococci.
1	1	N.O.	N.O.	Serous effusion	Not found	Nil	—
2	1	II	N.O.	Yes	Not found	Yes	Not found
3	2	II	N.O.	Yes	II	Nil	—
4	2	II	II	Yes	II	Nil	—
5	3	Pos.	Pos.	Yes	Pos.	Yes	Pos.
6	3	Pos.	Pos.	Yes	Not found	Nil	—
7	3	II	N.O.	Serous effusion	II	Nil	—
8	3	I	I	Yes	Mening.	Yes	Not found
9	4	I	N.O.	Yes	Mening.	Nil	—
10	5	Pos.	N.O.	Yes	Not found	Nil	—
11	7	Pos.	N.O.	Yes	Not found	Nil	—
12	9	I	I	Yes	I	Nil	—
13	10	Pos.	Pos.	Yes	Not found	Yes	Not found
14	11	I	N.O.	Yes	Not found	Nil	—
15	16	II	II	Yes	Mening.	Yes	II
16	19	Pos.	N.O.	Yes	Mening.	Nil	—
17	27	III	N.O.	Normal	—	Nil	—
18	34	Pos.	Pos.	Yes	Pos.	Yes	Pos.
19	40	Pos.	N.O.	Yes	Not found	Nil	—
20	47	Pos.	Pos.	Yes	Not found	Nil	—
21	68	Pos.	N.O.	Yes	Mening.	Yes	Not found

N.O. = Culture not obtained. Roman numerals denote Gordon's four types of meningococcus as proved by serological tests. Pos. = Meningococcus grown, but serological tests not performed. Mening. = Organism having the morphological and staining characteristics of the meningococcus seen, but not grown.

TABLE II.—B. *Cases with Known Recrudescences.*

No. of Case.	No. of Days to		C.S.F. Type.	Nasal Type.	Sphenoidal Sinus.		Sphenoidal Bone.	
	R.	D.			Empyema.	Meningococci.	Inflammation.	Meningococci.
22	8	8	II	II	Yes	Mening.	Nil	—
23	5	9	I	I	Yes	I	Nil	—
24	9	14	I	I	Yes	I	Nil	—
25	12	16	III	I or III	Yes	Mening.	Nil	—
26	42	48	II	N.O.	Normal	—	Nil	—
27	Rr.	99	Pos.	Pos.	Yes	Mening.	Nil	—

R. = Recrudescence. Rr. = Repeated recrudescence. D. = Death.

TABLE III.—C. *Convalescent Cases Dying from Hydrocephalus.*

No. of Case.	Days from Onset to Death.	C.S.F. Type.	Nasal Type.	Sphenoidal Sinus.		Sphenoid Bone.	
				Empyema.	Meningococci.	Inflammation.	Meningococci.
28	87	Pos.	II	Yes	Not found	Nil	—
29	90	Pos.	Pos.	Yes	Mening.	Nil	—
30	93	II	II	Yes	II	Nil	—
31	109	Pos.	N.O.	Yes	Mening.	Nil	—
32	133	Pos.	I	Yes	I	Nil	—
33	147	Pos.	II	Yes	Not found	Nil	—
34	199	I	I	Yes	Not found	Nil	—

Note.—In no case was the cribriform plate of the ethmoid affected. The contents of the pituitary fossa were inflamed in every case. In Case 8 there was inflammation of the middle ear, and in Case 9 pus; in all other cases the ear was not affected.

* A paper read before the Pathological Section of the Royal Society of Medicine.

inflammation in every case. The middle ear was seen to contain pus in one case, and there was congestion of the bone but no pus in another case.

There was an empyema of the sphenoidal sinus in

A. Acute case deaths	...	20 out of 21 cases
B. Recrudescence deaths	...	5 out of 6 "
C. Hydrocephalus deaths	...	7 out of 7 "

An empyema of the sphenoidal sinus is taken to mean a cavity filled with pus, muco-pus containing fresh and autolyzed pus cells, or glairy fluid showing pus cells, and the mucous membrane of the sinus showing congestion.

In 34 necropsies an empyema of the sphenoidal sinus was present in 32 cases. In the pus from the sphenoidal sinus—

Meningococci were proved serologically in	...	8 cases
Meningococci were cultured but were not tested serologically in	...	2 "
Meningococci were seen but not grown in	...	10 "
No meningococci were seen or grown in	...	12 "

Inflammation of the sphenoid bone over the empyematous sphenoidal sinus was not found at all in recrudescence and hydrocephalus deaths, but was found in 7 cases of deaths from the acute illness. In these 7 cases the meningococcus was only found three times, and the type of the meningococcus was only able to be proved once, but was in this case the same as that found in the nose and cerebro-spinal fluid.

The association of sphenoidal sinus inflammation with cerebro-spinal fever was first noticed by Westenhoeffer,³ who only found it to occur in one-third of his twenty-nine necropsies. Other workers have also reported cases showing empyemata of the sphenoidal sinus, but have generally considered that this condition is not the cause of the disease, either because they have been unable to find an empyema in all cases or because they have been unable to trace the meningococcus in the walls of the sphenoidal sinus or in the body of the sphenoid bone. Very great improvements have recently been made by Mervyn Gordon in the culture medium for the propagation of the meningococcus, so that a meningococcus can now, under suitable circumstances be made to grow as rapidly and luxuriantly as *Bacillus coli*. These improvements were not available for most of the workers in this connexion. In spite of these advantages the meningococcus was not demonstrated in the sphenoidal empyema in 12 of these cases. This was possibly due to the length of time that elapsed between death and the necropsy in many cases, and also to the chilling of the plates during transport from a distant camp to the laboratory in very cold weather. The majority of deaths occurred during cold weather, and meningococci are difficult to cultivate in primary culture unless precautions against chilling are taken.

Thus in 34 deaths from cerebro-spinal fever empyemata of the sphenoidal sinus were found 32 times; meningococci were found 20 times in the sinus and 3 times in the sphenoid bone. In no other diseases were empyemata of the sphenoidal sinus observed, except during the epidemic of "Spanish influenza," when empyemata of the sphenoidal sinus were frequently found.

The relationship between an empyema of the sphenoidal sinus and cerebro-spinal fever may now be considered. The primary site of a meningococcus infection in the human body is undoubtedly the nasopharynx. The meningococcus has been demonstrated in this position not only early in the acute attack, but also in the incubation period,⁴ in carriers who never develop meningeal symptoms, in meningococcal pneumonia, etc. The infection is propagated from person to person, as is a common cold, from nose to nose. The frequency of the carrier condition and the relative infrequency of the disease is noteworthy. It is probable that the whole population of England has been a carrier at some time or other during the last five years, and yet the total number of cases is relatively small.

At the time when contacts to cases of cerebro-spinal fever were examined for the carrier condition, it was always noticed that the people under examination were suffering from coughs and "colds." Many of the contacts gave pure cultures of the meningococcus in the nasopharynx. This suggested that the "colds" were in some cases due to the meningococcus. Also several carriers, when first isolated, suffered from a profuse watery nasal discharge, from which only the meningococcus could be isolated. Chronic carriers nearly always had a gluey nasal discharge, which was apparent on examination of a nasal

swab. It thus appears that the meningococcus can give rise to a nasal catarrh. It seems probable that this is the natural disease produced by this organism, and it is only when some other factor comes into play that meningitis or septicaemia results. This other factor does not seem always to be a rise in virulence of the meningococcus, for of the camp epidemics about Southampton and Winchester during the war it was only in one epidemic that all the cases of meningitis were due to one serological type of meningococcus. Usually two or three serological types of meningococcus were recognized in the cases of one epidemic, the carriers found in the camp corresponding with the serological types of meningococcus in the cases.

Thus, in the Winchester epidemic of the spring of 1917 there were 8 cases, 3 due to Gordon's Type I coccus, 2 due to Gordon's Type II, 2 due to Type IV, and one in which the type was not proved. The carriers found were all infected with one of these three types of meningococcus; no Type III carriers were found.

On another occasion two cases occurred in one ward in a hospital within four days of one another. If serological tests had not been performed, it would have been felt certain that either one case infected the other, or that both were infected from the same source. This was not so, as the first case was due to Gordon's Type II, and the second due to Gordon's Type I or III coccus.

Some epidemics in other parts may have been produced by a particularly virulent meningococcus of one serological type, but it is evident that this is by no means a necessary condition for the outbreak of an epidemic.

The frequency of the appearance of a sphenoidal sinus empyema suggests that this might be the determining factor in the onset of the meningitic form of the disease at any rate. The empyema may be the result of an inflammatory reaction to the meningococcus on the part of the mucous membrane lining the ostia. The fact that the meningococcus was several times isolated in pure culture from the sinus suggests that for the inflammatory closure of the ostia a mixed infection is not necessary, although streptococci and staphylococci were also found on occasions to be present. Thus a sphenoidal empyema might be produced by a meningococcal infection of the nasal mucosa of an individual whose resistance to the meningococcus was such that the infection resulted in a vigorous inflammatory reaction. Anatomical or pathological peculiarities in this region might also favour the production of an empyema.

In cases dying from cerebro-spinal fever an empyema of the sphenoidal sinus was common, in only 2 cases (17 and 26) in this series, out of 34, was this condition not found. Both these cases died at a considerable number of days from the onset, 27 and 48 respectively; 47 completely recovered cases, on the other hand, were examined by rhinological experts, Dr. Peters and Dr. Bryant, who found that no sphenoidal empyemata were present. Chronic carriers⁵ were also carefully examined bacteriologically, and no meningococcal infection of the sinus was found. Thus in dying cases an empyema was frequently found; in completely recovered cases no empyemata were discovered. A series of five cases showing symptoms of hydrocephalus were operated on for drainage of the sphenoidal sinus.⁶ Of these cases all showed the presence of a sphenoidal empyema. The meningococcus was only cultivated and proved serologically in one of these cases from the sphenoidal pus obtained at the operation; a typical Gram-negative diplococcus was seen only, but not grown, in another case. But in two other cases, subsequent to the operation, the meningococcus reappeared in the throat in pure culture, after it had entirely disappeared for some weeks previously. Subsequent to the operations all cases appeared temporarily worse, three made complete recoveries, two died with typical hydrocephalus *post mortem*. Of the recovered cases one went through a typical relapse after the operation, with reappearance of the meningococcus and pus in the theca, subsequently completely recovering.

From this it would seem that if early and vigorous serum treatment be given, and if the sphenoidal empyema disappear, recovery will probably occur. If, however, the empyema persist, the case will, if it recover from the acute illness, either relapse or go on to hydrocephalus.

In order to see if draining the sphenoidal sinus during the acute stage of the illness would accelerate recovery, three cases of cerebro-spinal fever were operated on; a sphenoidal empyema was found in each case, but each case died. This discouraged further operative procedure during the acute stage.

The next point under consideration is, how do the meningococci get from the nasal mucosa to the meninges?

There are many possible routes:

1. The arachnoid prolongations round the olfactory nerves.
2. The perineural lymphatics.
3. Direct inflammatory extension.
4. The pituitary.
5. The systemic lymphatics.
6. The blood stream.
7. The middle ear.

1. The Arachnoid Prolongations Round the Olfactory Nerves.

This route is favoured by Netter and Debré and Flexner. Netter and Debré show that dyes, Indian ink, and meningococci can get from the cerebro-spinal fluid into the nose by this route, but they do not show that the reverse can take place. Their experiments show that the natural direction of the current is from the cerebro-spinal fluid to the lymphatics; it is thus difficult to see how the meningococci could progress against the stream to gain access to the meninges except by inflammatory extension. In none of this series of *post-mortem* examinations was there any naked-eye appearance of inflammation about the cribriform plate of the ethmoid.

2. The Perineural Lymphatics.

In papers by Orr and Rows,⁷ Teale and Embleton,⁸ it has been shown that not only dyes and toxins, if injected into a nerve, will travel up the perineural lymphatics to the cord, but also particulate material, such as washed spores. These spores can be traced up the perineural lymphatics into the substance of the cord itself. In this way it might be possible for the meningococci to travel up the perineural lymphatics to the brain substance. Cerebro-spinal fever does not, however, appear to start as an encephalitis, so, although this route would be a satisfactory one for polio-encephalitis, it does not seem suitable for cerebro-spinal meningitis, especially as it has been shown that neither toxins, dyes, nor particulate material, when passing up a perineural lymphatic, get into the cerebro-spinal fluid.

3. Inflammatory Extension.

It has been suggested that meningococci could gain access to the cerebro-spinal fluid by a process of inflammatory extension through the bone separating the nose and accessory sinuses from the meninges. In this series of necropsies there is evidence of direct spread from the sphenoidal sinus to the sphenoid bone, and so to the overlying meninges. In seven cases there was inflammation, and the meningococcus was found three times in the bone. In the other cases no evidence, as viewed by the naked eye, of inflammatory extension could be discovered.

4. The Pituitary.

An infection from Luschka's pharyngeal tonsil along a possibly patent connexion between this and the pituitary gland has been suggested. Luschka's tonsil is one of the sites in the nose most richly infected with the meningococcus. In this series of necropsies the pituitary body always showed infection, but it is obvious that this would certainly occur anyhow from its intimate relationship with the cerebro-spinal fluid.

5. The Systemic Lymphatics.

The ordinary flow of lymph from the nasal passages is away from the brain, and in connexions between the subarachnoid space and the lymphatic system the current is away from the meninges. If bacteria⁹ are injected into the arachnoid space they rapidly appear in the lymphatics of the head and neck. If, however, a process occurs, such as an empyema of the sphenoidal sinus, the ordinary direction of lymph flow might be reversed, and bacteria be thus forced from the lymphatics of this neighbourhood towards the meninges, without showing any sign of inflammatory extension. In the case of a sphenoidal empyema large numbers of meningococci would get into the lymphatics, and by this means would also get into the blood stream. It would only be necessary for a sphenoidal empyema to exist for a very short time to produce these effects, possibly only for half an hour. If the sphenoidal empyema discharged at the end of a short time into the nose, and if the meningococci that had reached the meninges were destroyed, a recovery would ensue. If, however, the sphenoidal sinus empyema persisted, and kept on pouring fresh infection into the meninges, the case would probably die.

6. The Blood Stream.

Many workers have considered that the infection of the meninges occurs by way of the blood stream, the meningococci gaining access to the blood from the nasopharynx. This suggestion has been based partly upon the early appearance of the meningococci in the blood, and partly on those cases of meningococcal infection where no meningitis occurs. The blood stream, however, would receive meningococci very early in the infection of the nasal mucosa, at any rate as soon as an empyema of the sphenoidal sinus occurred. The meningococci would reach the blood stream from this region via the deep cervical lymphatic trunks, which join the thoracic duct just before it enters the venous system in the neck. Also, as soon as meningococci gained access to the cerebro-spinal fluid, they would appear⁹ in the blood stream, both direct, via the Paccionian bodies, and also by way of the lymphatics, through their junctions with the subarachnoid space. The blood then

would, from the very earliest stages of the disease, receive repeated and heavy showers of meningococci from the sphenoid region and the cerebro-spinal fluid. Thus it can be said that the early finding of the meningococcus in the blood stream is no indication that the meninges are primarily infected by this route.

In the recent outbreaks of "Spanish influenza" empyema of the sphenoidal sinus was a common finding in the fatal cases, but meningitis was rarely found. It is not necessary, then, that an empyema should be followed by meningitis. It is thus possible that the meningococci can gain access to the blood stream from a sphenoidal empyema, and so be carried round to the meninges and set up inflammation.

7. The Middle Ear.

Inflammation in this region was only found in two necropsies in this series. It is unlikely that this is the portal of entry of the meningococcus to the meninges.

Thus it would appear improbable that the meningococci gain access to the meninges through the arachnoid prolongations round the olfactory nerves, or by way of the perineural lymphatics. It is probable that the meningococci pass by way of the lymphatics from the sphenoidal sinus direct to the meninges, with or without naked-eye signs of inflammation. It is possible also that they can be conveyed to the meninges by the blood stream.

The relationship between a persistent sphenoidal empyema and hydrocephalus may now be considered.

In the seven deaths from this condition an empyema was found in each case. Five cases were operated on, and pus was found in the sphenoidal sinus each time; two of these cases died, and are grouped in with the *post-mortem* cases. There were thus a total of 10 cases of hydrocephalus, and in each case a sphenoidal empyema was found. The cerebro-spinal fluid in the ventricles of each of the cases which died contained pus cells. The cerebro-spinal fluid taken from the lumbar region was usually quite clear.

This suggests that there is a close relationship between empyema of the sphenoidal sinus and the hydrocephalus that develops after an attack of cerebro-spinal fever. The hydrocephalus develops as a result of a chronic infection about the foramina of Luschka and Magendie. This chronic infection also appears to exist in the ventricles. The exact path by which the meningococci in the sphenoidal sinus empyema reach the interior of the ventricles is not clear, unless it be by the blood stream. But there would appear to be some relationship between the two positions, inasmuch as the only three cases who recovered from the hydrocephalus condition did so after an operation for drainage of the sphenoidal sinus. It might be argued that these three cases who recovered were not examples of hydrocephalus at all. Against this it is urged that all five cases operated on were advanced cases with typical symptoms; the two that died showed typical necropsies, and all other cases showing these symptoms died with typical necropsies.

The course of events in a case after infection of the nasal mucous membrane with the meningococcus might be as follows:

1. A simple catarrh, followed either by recovery or a chronic infection.
2. A vigorous local reaction in the nasal mucosa followed by sphenoidal empyema. This may produce a general blood infection, with or without infection of the meninges.

If the sphenoidal empyema disappear, the body, with or without the aid of antiserum, may be able to deal with the meningococci that have gained access to it, or death may result. If the sphenoidal empyema persists, and very heavy discharges of meningococci are poured into the body, death will probably result. If, however, the empyema remains quiescent, improvement may occur, but at any time a recrudescence or a relapse may supervene. A quiescent empyema may also keep up a smouldering infection which will lead to hydrocephalus.

Operation on the sphenoidal empyema always produced an increase in general symptoms. During the acute stage of the disease this increase in symptoms appeared to accelerate, at any rate, a fatal termination. In hydrocephalus cases recovery did occur in three cases, though one case went through a severe relapse.

The author wishes to acknowledge his indebtedness to Miss Iris Harmer for her valuable work in connexion with this research.

Summary.

1. In thirty-four necropsies on persons dying from cerebro-spinal fever, empyema of the sphenoidal sinus was found thirty-two times.

2. In ten cases of hydrocephalus following cerebro-spinal fever an empyema of the sphenoidal sinus was found in every case.

3. In forty-seven completely recovered cases of cerebro-spinal fever no sphenoidal empyemata were present.

REFERENCES.

- ¹ Rolleston: Lumleian Lectures, *Lancet*, April 5th, 1919. ² Embleton and Steven: Persistence of Cerebro-spinal Fever Cases as Carriers of the Meningococcus during Convalescence, *Lancet*, May 10th, 1919. ³ Westenhoeffer: *Berl. klin. Woch.*, 1905, lxii, 737. ⁴ Fildes: Report to the Medical Research Committee, ⁵ Embleton and Bryant: Site of Carrying in Chronic Meningococcus Carriers, *Lancet*, October 17th, 1919. ⁶ Peters: *Proc. Roy. Soc. Med.*, vol. xii, No. 9, August, 1919. ⁷ Orr and Rows: *Edinburgh Medical Journal*, vol. xvii, 1916, pp. 78, 89. ⁸ Teale and Embleton: *Journal of Pathology*, In the Press. ⁹ Teale and Embleton: Infection Paths of Spread in Bacterial Infection, *Proc. Roy. Soc. Med.*, vol. vii, Path. Sect., 1914.

STERILITY IN THE MALE.

BY

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WHEN a marriage is infertile it is still sometimes too readily assumed that the woman is at fault, although all writers on the subject recognize that the defect may rest with the husband. The object of the following summary is to emphasize the fact that no operative procedures on the wife are justifiable until the male has been properly examined, and until it can be definitely stated that his semen contains live, healthy, and vigorous spermatozoa.

It is difficult to obtain reliable statistics as to the frequency with which the male is at fault in childless marriages. The figures given by different authorities show great discrepancies, but at any rate they indicate that the fault of the childless marriage lies far oftener with the man than is commonly supposed. Hubner, in his investigation of 129 childless marriages, discovered a male sterility of 59 per cent., and Kehrers, amongst 40 similar couples, one of 35 per cent. Gross, in figures collected from various authors, convicts the husband in 17 per cent. of childless marriages, whilst Vedder places it as high as 70 per cent. Barney, in investigating 40 childless unions, found 20 sterile husbands, and, what is still more suggestive, discovered that the wives of 4 of these (20 per cent.) had undergone more or less severe operations for sterility! Even if the lowest of the above percentages be taken as correct, it is sufficient to support the dictum that "the investigation of childlessness should begin not with the curettage of the wife, but with the microscopic examination of the husband's semen" (Belfield).

The investigation of sterility in the male necessitates no small amount of labour and skill. Such gross lesions as epispadias, hypospadias, cryptorchism, malignant disease, bilateral tuberculosis, and gummata of the testis, require no consideration beyond the noting of the fact that tuberculous epididymitis, even although it be unilateral, causes azoospermia in 85 per cent. of cases. This fact depends on the frequent association of a unilateral tuberculous epididymitis with disease of the prostate. It is the case where no gross lesion of the male genitalia is present that calls for careful investigation.

Whereas with the female it is Nature that plays the chief rôle in producing sterility, with the male the condition is more often the legacy of disease. A careful history of previous illnesses is the first step in the investigation. The orchitis of mumps, especially when it occurs about the age of puberty, is, as a rule, followed by atrophy. Fortunately such an orchitis is generally unilateral. Alcoholism, exposure to x rays, debilitating diseases, and sexual excesses, whether from natural coitus or masturbation, produce an azoospermia or oligospermia which is temporary in nature. Prolonged and absolute continence has been said to have a similar result (Barney). Non-venereal infections of the testicle and prostate, whether by typhoid, *Bacillus coli*, or staphylococci, may similarly result in azoospermia, oligospermia, or necrospermia, as the case may be. In a few cases it would appear that the otherwise healthy testicle of a normally active and sexually vigorous man may be entirely unproductive of spermatozoa (Janet).

But although non-venereal causes may sometimes be responsible for sterility in the male, by far the commonest

cause is a previous attack of gonorrhoea. When it is remembered that in many countries the incidence of this disease is from 50 to 75 per cent. of the total adult male population, its importance as a potential cause of sterility amongst males cannot be exaggerated. All authorities recognize the frequency with which the posterior urethra becomes implicated in gonorrhoea, and it is with posterior infections that sterility is usually associated.

The frequency with which gonorrhoea, and especially gonorrhoea affecting the posterior urethra, produces sterility is borne out by many observers. In this connexion figures published by Benzler (*Arch. f. Derm. u. Syph.*, 1898, xlv) are especially interesting. This observer followed the history of German soldiers who had suffered from gonorrhoea and had been subsequently married for three years or over. Especial attention was paid to a previous history of epididymitis.

Of those who had escaped epididymitis ...	10.5% were childless.
Of those who had had unilateral epididymitis ...	23.4% were childless.
Of those with previous bilateral epididymitis ...	41.7% were childless.

Clinical observations on the husbands of childless women with normal genitalia support these figures. The majority of these, when sterile, will be found to have suffered from gonococcal prostatitis, with or without epididymitis. It is often the prostatitis rather than the epididymitis that is the determining factor, and, when the proximity of the ejaculatory ducts and their liability to occlusion is remembered, this is not surprising. Moreover, even when no mechanical obstruction has been produced, pathological changes in the prostatic fluid resulting from inflammation are alone capable of producing necrospermia or oligospermia. Even healthy spermatozoa lose their vigour in the presence of pus or blood, and when, in addition to this, the activating secretions of the prostate are deficient, necrospermia and asthenospermia are extremely probable.

The second step, therefore, in the investigation of a case of male sterility is a careful and complete examination of the genitalia and urethra for evidence of past or present gonococcal infection. This must invariably conclude with a urethroscopy of the posterior urethra, for it is in the presence of a prostatitis, a vesiculitis, or a colliculitis, that the explanation of sterility will often be found.

The final step in the examination includes a microscopic examination of the male secretions. As a preliminary the secretions expressed as a result of prostatic massage are subjected to examination. Attention is paid to the presence of pus cells, and the appearance of such spermatozoa as may be present is noted. This examination is, however, but a preliminary to the more detailed investigation of the seminal fluid obtained after coitus with a condom. This examination, although it gives valuable information, is unfortunately open to error. The condition of spermatozoa some hours after evacuation into a condom does not necessarily reflect their condition when deposited in the vagina. For this reason Wolbarst urges the advisability of examining the vaginal and cervical secretions at various intervals after coitus has taken place. Such an examination is not always obtainable, but when suitable arrangements can be made the results are invaluable. Details of the examination are beyond the scope of this article, but as a result of observation of the spermatozoa, with reference to their number, freedom from imperfect or degenerate forms, mobility, and vigour, the question of the fertility or non-fertility of the male can in the great majority of cases be put beyond doubt.

In the above summary no attempt has been made at completeness. The subject of sterility in the male is far beyond the limits of a brief note. What has been attempted is merely to emphasize two facts which, although they may be known, are apparently too often forgotten. The first of these is the relative frequency with which sterility occurs amongst males; the second is the lack of justification in subjecting any woman to operation for sterility unless her husband has undergone a careful preliminary examination.

BIBLIOGRAPHY.

- Barney: *Boston Medical and Surgical Journal*, 1914.
Belfield: *Journal of the American Medical Association*, 1912.
Benzler: *Arch. f. Derm. u. Syph.*, 1898, xlv.
Janet: *L'Assoc. Franco. d'Urolog.*, 1910.
Lier and Ascher: *Arch. f. Derm. u. Syph.*, 1910.
A. L. Wolbarst: *New York Medical Journal*, 1917.