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"Some observations on Cerebro-Spinal Fever with
special reference to 50 Vaccine treated cases."

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Some observations on Cerebro Spinal Fever with special reference
to 50 Vaccine treated cases.

During the last four years it has been my privilege to see, and to treat, about 100 cases of Cerebro Spinal Fever, and to make observations which substantiate certain factors which are supposed to play a part in the Etiology, Infection, and Dissemination of the disease.

I have also gone carefully into the Bacteriology, Pathology as far as post mortem examinations are concerned, and Treatment. From the clinical aspect I have carefully observed the Symptomatology with a view to being able to give a Prognosis.

In this thesis while I wish to give my observations on these points, I wish to pay special attention to Vaccine treatment in 50 cases, and compare it with other forms of treatment which have been carried out by myself and which have been extensively written about by other writers.

Etiology.

The first factor in the Etiology which took up my attention was the prevalence of the disease during cold weather.

I have only seen two cases of Cerebro Spinal Fever which occurred during really warm weather. Apart from these I have not seen a case during the late Spring, Summer, or early Autumn.

50% of my cases occurred when the weather was bitterly cold. During the Winter months it was also very marked how with a short spell of mild weather there was a fall in the number of cases, which broke out again as soon as the weather conditions became more severe. 16% of my cases were seen during a bitterly cold March when there was almost a constant East wind blowing and the thermometer rarely stood much above Zero.

The living conditions of the patient, have, too, in my opinion a marked influence on the incidence of the disease.

Wright does not think that these play a part in the Etiology, but Ker on the other hand states that cases are most prevalent in small houses, and crowded areas. This has been my experience, and I can say from my observations that overcrowding, bad ventilation, and a dry atmosphere laden with CO_2 and particulate organic matter are very marked factors in the Etiology, as can frequently be proved by the disease breaking out in overcrowded, badly ventilated huts where stoves are kept burning, and windows closed, more or less all night. This is well seen in military camps where the percentage of cases is much higher in camps where the men are sleeping in huts, than in camps where the men are under canvas.

Fatigue, exposure and age may well be considered together I think the young adult is very susceptible to the disease, especially where he is overworked and exposed to hardships and weather conditions of all kinds. This was well seen in my series of cases, where 80% were lads of 18, who were being trained rapidly, and consequently were exposed for long periods and got practically no time for recreation. This exhaustive training was altered, and even with the other etiological factors at work there was a marked falling off in the number of cases.

In no case was I able to say that injury played any part in the Etiology.

Infection & Dissemination

In all the cases which I have seen, and where I have examined the contacts I have not failed to find a "carrier" and in 45% of my cases the "carrier", and the "case" were closely associated with one another. In no case was I able to trace a source of infection from patient to patient, and I never found that any nurses or attendants took the disease. Robertson in the Lethbridge epidemic was able to trace direct transmission in twenty cases but most other observers seem to have found that this mode of transmission is not likely.

I think that a "carrier" is necessary before the disease can become epidemic, and that the etiological factors before mentioned must be present.

The etiological factors lead to catarrhal conditions in the nose and throat, and the presence of a "carrier" amongst a number of people so affected is all that is necessary to make the disease epidemic.

I found that 5% of the people I swabbed who had been in contact with the cases gave positive results when examined for the Meningococcus, and that in 80% the same Type of organism was found in the "carrier" as was found in the case.

This was very well exemplified in two of my cases where a Gram. negative diphtheroid diplococcus was found in the cerebro-spinal fluid of ~~the~~ ^{the} cases and was also found in the ~~the~~ ^{the} naso-pharyngeal swabs of contacts who were closely associated with the cases.

Ostermann successfully cultivated the Meningococcus from 70% of a series of contacts and Couric & Faser from 14%. I think these findings are high and probably inaccurate as they were done before the agglutination reactions had been practised + in all probability in these percentages a number of Gram negative diplococci are included which are not the Meningococci.

Bacteriology.

In all my cases I found that it was difficult to obtain a growth directly from the cerebro-spinal fluid on Assetic agar, and that growth was best obtained on Trypsin legumen agar with blood added as recommended by Gordon. On this medium the cocci were easily grown and subcultured on Starch agar, and further subcultured on Trypsin legumen agar with a view to obtaining a pure culture for autogenous vaccines.

I did not attempt to grow the Meningococcus on any of the media experimented with by Gordon, and described by him in the British Medical Journal of 18.11.16, but I found

that the following prescription gave good results and might be improved on by experiment.

Human or Sheep's brain minced
add 3 lbs. saline & bring to $40^{\circ} - 50^{\circ} \text{C}$.

make slightly alkaline to litmus

add 1% liquid Trypsin Co. (A & H)

Incubate 12 hrs.

Strain and boil 10 mins.

Alkalinize

add 30 grms. sugar to 1 lb. of above

10 grms. starch

50 cc. 10% solⁿ. of slightly alkaline plasma.

I think both these media superior to ascitic agar although that is the medium recommended by Muir (Muir & Ritchie) and Her (Infectious diseases). The latter states that on subculture the Meningococcus is often difficult to decolorize by Gram's method but even after several subcultures had been made I found that there was no difficulty in decolorizing. The differentiation of the types of organisms in my 50 Vaccine treated cases was carried out by agglutination, and the following table gives the Type found and the percentage of cases.

<u>Type of coccus</u>	<u>Number of cases</u>	<u>Page.</u>
Type I	26	52
" II	6	12
" III	14	28
" IV	2	4
Diplobacillus	2	4

In 90% of my cases the diplococcus was found on direct examination and in all cases on culture with the exception of the two cases where a Gram negative diplobacillus was found both on direct examination & on culture.

In 80% of cases the meningococcus was obtained from the naso-pharyngeal swabs and in these cases the type of coccus was the same as was found in the Cerebro-spinal fluid.

While engaged in the bacteriological work I had two cases that clinically presented the appearance of C.S.F. but a Gram negative diphtheroid diplobacillus was found both on direct examination of the fluid and on culture. The colonies were difficult to differentiate from meningococcus but were slightly more opaque, somewhat larger, and of a sticky consistency. This organism, as I have stated before was also cultured from the naso-pharynx of two contacts who had been closely associated with the patient.

A similar organism is mentioned by Horder (Cerebro Spinal Fever) and by Donaldson (The Practitioner May 1917 + Lancet 26/6/15). I can make no definite statement about this organism further than that it was the only organism found + the fluid was examined in each case on three separate days and that the clinical condition justified a diagnosis of C.S.F. and both patients recovered under treatment by an autogenous vaccine prepared from the diplo-bacillus.

Another interesting culture which I got from the Cerebro-Spinal fluid was a pure culture of Gonococcus. A similar result was got by Short (An Index of prognosis and end results of treatment.) when investigating cases of Cerebro Spinal Fever.

As well as being able to cultivate the meningococcus from the Cerebro-spinal fluid I was able, in ten cases out of twenty which I tried, to cultivate it from the blood. This was got on the third day of illness when the patient's condition could be said to be one of acute septicaemia. In two other cases I was able to cultivate the meningococcus from purulent conjunctivitis. The agglutination reactions in these cases shewed the type of coccus to be the same as in the cerebro spinal fluid.

The fact that in the first instance the organism is present in the throat and can be recovered from the blood on the third day led me to begin vaccine treatment at the onset and not only in the chronic stage as practiced by Garrod, Horder + others (Horder - Cerebro Spinal Fever)

Colebrook is of the opinion that vaccine treatment should be employed early and repeated every third day but Horder does not think this desirable (Horder. Cerebro-Spinal Fever)

Mode of Entrance.

I am of opinion that the blood stream is the source of infection because in 48% of my cases treated on the first day of illness the symptoms were more septicaemic than meningeal and the cerebro-spinal fluid in 60% was only slightly turbid although the patients were profoundly ill. The mode of infection along nerve roots appears to me to be without foundation for it is rare that one finds any implication of the nerves in the early stages and post-mortem ~~there~~ I have not found any signs or appearances in any of the nerve roots to substantiate this finding. The fact too, that the organism has been recovered from the lungs, nose, bronchial and cervical glands (Cland 1891 on Infectious diseases) leads one to believe that the blood is the primary source of the general infection.

Morbid Anatomy.

I held post-mortem examinations on 30% of my deaths and with a few exceptions they presented the same phenomena.

There was turbid fluid in the subarachnoid space and along the sulci the fluid tended to be pus-tular unless in cases which died during the first few days. In these cases the meningeal condition was more inflammatory than septic. As a rule the pons and cerebellum were most affected.

In two cases the lateral, and fourth ventricles contained thick pus and in these cases there was occlusion of the foramen of Magendie, and much matting at the base of the brain so that on lumbar puncture no fluid could be obtained after puncture had been performed twice. In both these cases glycosuria was present as a symptom.

In two cases out of the ten which gave a positive blood culture I found a condition of acute ulcerative endocarditis post mortem. In the other cases the heart was more or less normal. The meninges were little affected in the two cases which showed ulcerative endocarditis.

• In all cases which came to post-mortem patches of broncho-pneumonia were found in the lungs.

Symptomatology.

The symptomatology of the different stages I found to be much the same as described in text books but there was one case in which I observed a phenomenon described by Osler and referred to by Ket and that was, the presence on the extremities of blood filled vesicles. This was seen in one of my chronic cases which had a purpuric rash and a purpuric herpes labialis. The rash tended to disappear but the vesicles, some of which burst & ulcerated persisted until death, which took place on the 40th day of illness.

Complications

Severe neuritis with atrophy of muscles of upper arm & shoulder	1%	Recovered.
Otitis Media which ultimately got well	1%	Recovered.
Transient deafness	2%	Recovered
Cervical adenitis in convalescence	1%	Recovered.
Orchitis	1%	Recovered
Hydrocephalus (external)	1%	Recovered
" (internal)	2%	Recovered
Arthritis	10%	Recovered
Severe arthritis with swelling of joints	1%	Recovered
Permanent internal strabismus	1%	Recovered
Hemiplegia (died 3 rd day)	1%	Died.
Facial paralysis	1%	Died.
Broncho pneumonia & Bronchitis	50%	10% Died.
Temporary paresis of lower limbs	1%	Recovered

Relapses.

Ket in his series of cases had 15.20% who had relapses

In my series I had 10% of my total cases and of that 10% two died. One died in his second relapse and one in his first. The relapses took place a fortnight after convalescence had set in and the patients were apparently getting better. One of these cases after the second relapse developed the chronic type of the disease and died after a prolonged illness of two months. The other died from acute symptoms. In all cases of relapse the patients were treated by autogenous vaccine and the effects in these cases were very encouraging and distinct improvement ensued, the temperature becoming normal, and the symptoms disappearing within a couple to three days. Vomiting was a common symptom in relapse. Brownlee of Glasgow had a case where 13 relapses were noted and the patient ultimately recovered. One patient of mine had four relapses and recovered, but the second and third relapses were mild, and only lasted about 36 hours.

Diagnosis

My chief difficulty in differential diagnosis were encountered with

I Apical Pneumonia.

I was asked to see two cases of this nature when there was little to be made out on physical examination of the chest, and in practically all respects they presented typical clinical pictures of Cerebro-spinal Fever, the diagnosis only being cleared up by lumbar puncture and the subsequent development of definite pneumonic signs in the chest.

II Typhoid Fever.

I had two such cases to see as suspected Cerebro Spinal Fever, and on clinical grounds one could not be dogmatic as to a diagnosis until lumbar puncture had been performed and Widal's reaction taken.

III

Gonorrhoeal Meningitis

The case that presented this condition was the most difficult of all to diagnose even after lumbar puncture



had been performed. It was only after cultural and agglutination tests had been carried out that the case could be differentiated from Cerebro Spinal Fever. It is so interesting that a short history of the case may be given.

Man, aged 26, was admitted to hospital complaining of severe headache and pain in the back accompanied by vertigo & sickness. He had severe vomiting, and Kernig's sign was positive. I was asked to see him two days after admission and found him semi-comatose. His head was retracted, and there was a marked positive Kernig's sign. He had strabismus, and was passing urine and faeces in bed. From his clinical condition I made a diagnosis of Cerebro Spinal Fever and performed lumbar puncture. The fluid obtained was small in amount and very purulent. On examination it contained numerous Gram negative intra and extra cellular diplococci. No culture was made of this fluid as the case seemed so positive. Two days later I noticed a discharge from the urethra and the following day patient had retention of urine and I was forced to pass a catheter. I found that there was a stricture. After this I again lumbar punctured and cultivated the fluid obtained. Cultural examination showed colonies of *Gonococcus* and there was no agglutination of the organism with any of the recognised types of meningococcal sera.

This must be exceptional but I have already stated that a similar case is recorded by Short (An Index of prognosis & end results of treatment) Lumbar puncture & Cerebro Spinal Fluid.

During my investigations of Cerebro Spinal Fever I can say that I punctured at least 50 cases which were negative on culture & where nothing but clear fluid was obtained. Sometimes large quantities up to 50 or 60 cc. were removed on lumbar puncture in cases which were negative, but where nevertheless the patients had headache and slight temperatures, and in some cases no temperature at all and presented no clinical symptoms of Cerebro Spinal Fever.

These cases seemed greatly relieved by lumbar puncture and

the relief of tension. In my opinion delirium, and conditions of semi-consciousness so often met with in febrile conditions, could in many cases be relieved by lumbar puncture. From my experience I think that these symptoms are often caused by increased pressure in the spinal canal and ventricles of the brain rather than by toxæmia. I experimented in cases of pneumonia and uræmia and found that lumbar puncture was beneficial to the comfort of the patient and in all cases there was increased pressure and the fluid clear. The symptoms these patients suffer from is really akin to the Meningism of Dupré.

The chemical and microscopical examination of these fluids showed the consistence to be normal.

Leucocyte count.

The leucocyte count was found to be increased in all cases and very little help was got from it as regards diagnosis and still less as regards prognosis. Patients very frequently had a good leucocyte count a few hours before death.

Prognosis.

To begin with one may say that no patient is too ill to get better and no patient is too well to die.

First of all considering the mortality - of my cases the death percentage was 35% and considering my vaccine treated cases 28%.

During the epidemics which I have seen the prognosis was most unfavourable in the cases occurring at the height of the epidemics. The early cases were mild and the last cases more or less mild. This is unlike the findings of Hordet (Cerebro-spinal fever) who states that the initial cases are the most severe.

In acute fulminating cases the prognosis is invariably bad, death taking place in almost every case. I had one astonishing case of this type which is worthy of note.

A lad, aged 18 years, was seen by me at 4 p.m. He was very ill, with a severe headache, and he was cyanosed. At that time he had been ill only one hour. He had a dry mouth and tongue with sores on the lips. His skin & mucous membranes were cyanosed and he was roused with difficulty but was conscious. At 4:30 p.m. he was unconscious and lumbar puncture was performed & 35 cc. slightly turbid fluid removed. Before finishing the operation the lad was in convulsions. 50,000,000 cocci of a polyvalent vaccine were given. I waited for half an hour expecting death with every convulsion but although his condition did not improve he did not get worse.

I saw the boy the following morning and the convulsions had ceased, though he was still unconscious. He was passing urine and faeces in bed, and was markedly hyperaesthetic. He was lumbar punctured without being given a vaccine, and the following day lumbar puncture was again performed and 100,000,000 cocci of a polyvalent vaccine given. Twelve hours later he was conscious. Further treatment by lumbar puncture and autogenous vaccine was carried out as will be described later but no serum was used and an excellent recovery without complications was the result.

Horder (Cerebro-Spinal Fever) states that early loss of consciousness is of grave significance but I can't say that that is my experience. 90% of my cases were totally unconscious, or semi-comatose when I first saw them, and yet the prognosis was not always bad unless other symptoms of bad prognostic outlook were present.

Of the early symptoms I think cyanosis, and an extensive purpuric rash are the most unfavourable. Only 5% of my cases with marked cyanosis recovered, and only 1% of those with extensive purpuric rashes.

The respiratory rhythm and the type of respiration afford a good deal of information regarding prognosis. If the respirations remain normal in rhythm, even although hurried, there is nothing to be feared from that source, but if they are irregular e.g. Cheyne Stokes, or if broken by spells

of sighing respirations then the prognosis is serious.

The pulse gives little or no indication as to the patient's present, or future condition.

The cerebro-spinal fluid gives some little indication to prognosis. If small in amount, and purulent the prognosis is bad or if there are a large number of extra-cellular meningococci. A "dry tap" is almost always fatal, as it points to exudate, and adhesions at the base of the brain and along the cord. Horder in an article in the Lancet about the last week in April 1914 states, that a true "dry tap" is not met with, and says that if no fluid is obtained, it is due to the fact that the canal has not been pierced, and that the needle has only gone into a walled off cavity, and that manipulation of the point will ultimately get into the cerebro-spinal fluid.

I do not agree with this. I have found cases where little or no fluid was obtained at first, and where manipulation of the needle resulted in obtaining fluid under pressure but there are also cases where no fluid can be obtained, and these cases rapidly die and post mortem one finds that the cord and membranes are adherent & there are adhesions at the base of the brain and within the membranes of the cord there is no fluid, or at least very thick sticky fluid. Like Netter, Debré and Short I believe that the greatest factor of all in prognosis is the early treatment of the case whether this is by vaccine or by serum.

The wasting of the frame is no indication as the patient may be only skin and bone and yet recover.

Persistent vomiting in the later stages is bad as it indicates internal hydrocephalus.

Hyperpyrexia is of bad omen if combined with other symptoms which give a bad prognosis

Treatment

I am not going to discuss here the general symptomatic treatment of Cerebro-spinal fever not yet do I wish to say much about the ordinary Serum treatment but I

wish to give my observations on the Vaccine treatment of 50 cases and discuss how I think from my experience treatment could be improved upon.

Considering that the majority of cases show a septicæmic tendency, I cannot see why intra-thecal injection of serum should be the only means employed to treat the disease, and I think that either early intravenous injection of Serum, as suggested by Fairly & Stewart of Australia (Monograph on Cerebro-Spinal Fever) or early vaccine treatment should be employed. Many authorities have used vaccines late in the disease, but although good results have been recorded it seems to me to be the wrong time to use the vaccine, as I have never been able to cultivate the organism from the blood in chronic or advanced cases and in the early cases I have.

Taylor in a paper on "The antibody content of the cerebro-spinal fluid in Meningococcal infections" puts forward the theory that Vaccine treatment is useless because no antibodies are present in the cerebro spinal fluid and he substantiates this by five experiments as under.

<u>Experiment</u>	<u>Type of coccus</u>	<u>Agglutination titre of Serum</u>	<u>Agglutination titre of cerebro spinal fluid</u>
1	I	1 - 320	Nil
2	III	1 - 320	Nil
3	iii	1 - 256	Nil
4	ii	1 - 256	Nil
5	Control	Nil	Nil

I don't think this substantiates his findings at all unless he is going to give the vaccine intrathecally, for surely when the serum gives agglutination results which are positive in such high dilutions, there is an indication that a vaccine may help the general infection, especially when one considers that the meningococcus can be isolated from lymphoid tissue, pneumonic patches in the lung, & the blood before meningeal symptoms are marked. I am of the opinion that the fact of the serum being able to agglutinate

is an indication for subcutaneous or intravenous injection of vaccine. He might as well state that Serum treatment was of no use intrathecally because the Cerebro spinal fluid is deficient in complement and so also is Antmeningococcal Serum (Fairly + Stewart)

In using vaccine in treatment I always combined it with lumbar puncture to relieve the tension and quieten the patient. I also always kept a stock polyvalent vaccine in order to begin treatment as early as possible, and not have to wait on the autogenous vaccine which I prepared. In preparing my vaccine I grew the cocci and subcultured it as described before. I then washed the colonies off with Normal Saline 5ccs. after 24 hours growth, and put the bacterial emulsion in a sterile test tube which I sealed off and shook to break up the colonies. The sealed tube was then put into a water bath at $55^{\circ} - 60^{\circ} \text{C}$ for an hour & again shaken. The emulsion was then tested for sterility. The organisms were then counted against red blood cells and standardised, or else counted by the haemocytometer. As soon as a patient was admitted I performed lumbar puncture and withdrew fluid until the cerebro-spinal fluid was dropping at the pulse rate. I then gave 50,000,000 cocci of a polyvalent vaccine. I lumbar punctured on the second day but gave no vaccine. On the third day I gave 100,000,000 cocci of the polyvalent vaccine & again lumbar punctured. The fourth day I again lumbar punctured and gave no vaccine. On the 5th day my autogenous vaccine was prepared and I began with doses of 5,000,000 cocci combined with lumbar puncture and continued every alternate day with double the amount of cocci each time. I performed daily lumbar puncture until the fluid escaping was clear and under no tension. In no case did I give a vaccine exceeding 640,000,000 cocci.

In cases where the temperature became normal before this dose had been given I suspended vaccine injection.

after the temperature had been normal one week and only renewed treatment when the temperature showed a tendency to rise.

after an injection of vaccine the patient had a reaction with slight rise of temperature and quickening of the pulse. The local reaction was slight. In some cases redness of the skin, and pain in the seat of inoculation were present but no severe local symptoms were observed. The day following the injection the patient as a rule felt improved and the temperature was lower.

The turbidity of the cerebro spinal fluid took longer to disappear in vaccine treated cases than it does in Serum treated cases and I have no doubt that this is due to the fact that antibodies do not pass over at least in any quantity into the cerebro spinal fluid as proved by Taylor's experiments before mentioned.

Table giving Type of organism, No. of times inoculated, Maximum dose, Results.

No. of case	Type of Cocci	No. of times inoculated including two doses polyvalent	Maximum dose given of autogenous vaccine	Result.
1	I	4	10,000,000	<u>Death</u>
2	II	10	640,000,000	Recovery
3	Diphtheria	5	20,000,000	Recovery
4	T	8	160,000,000	Recovery
5	III	12	640,000,000	Recovery
6	T	10	640,000,000	Recovery
7	IV	3	5,000,000	Recovery
8	T	12	640,000,000	<u>Death</u>
9	III	6	40,000,000	Recovery
10	II	14	640,000,000	Recovery
11	T	16	640,000,000	Recovery
12	T	14	640,000,000	Recovery
13	II	15	640,000,000	<u>Death</u>
14	II	4	10,000,000	Recovery
15	T	6	40,000,000	<u>Death</u>

No of case	Type of cocci	No of times inoculated including two doses polyvalent	Maximum dose given of Autogenous Vaccine	Result
16	I	4	80,000,000	<u>Death</u>
17	IV	11	640,000,000	Recovery
18	I	8	160,000,000	Recovery
19	III	9	320,000,000	Recovery
20	I	10	640,000,000	<u>Death</u>
21	II	4	10,000,000	Recovery
22	I	9	320,000,000	Recovery
23	I	4	80,000,000	Recovery
24	I	4	80,000,000	<u>Death</u>
25	I	14	640,000,000	Recovery
26	I	10	640,000,000	Recovery
27	II	12	640,000,000	<u>Death</u>
28	III	11	640,000,000	Recovery
29	III	11	640,000,000	Recovery
30	I	13	640,000,000	<u>Death</u>
31	I	9	320,000,000	Recovery
32	I	8	160,000,000	Recovery
33	III	5	20,000,000	Recovery
34	III	14	640,000,000	Recovery
35	I	12	640,000,000	Recovery
36	III	20	640,000,000	Recovery
37	III	18	640,000,000	Recovery
38	I	12	640,000,000	<u>Death</u>
39	I	10	640,000,000	Recovery
40	I	5	20,000,000	<u>Death</u>
41	III	11	640,000,000	Recovery
42	III	4	80,000,000	Recovery
43	I	9	320,000,000	<u>Death</u>
44	III	10	640,000,000	Recovery
45	Diplobacillus	5	20,000,000	Recovery
46	III	11	640,000,000	Recovery
47	I	15	640,000,000	Recovery
48	I	12	640,000,000	<u>Death</u>
49	I	20	640,000,000	Recovery
50	III	18	640,000,000	<u>Death</u>

From my results in these cases I conclude that vaccine treatment has not to be regarded as useless although I cannot say that it is of greater value than Serum treatment.

I am of the opinion that combined treatment is more satisfactory than either used separately. My reasons for this statement are, that whereas Vaccine has a marked influence on the general infection it does not appear to affect the actual meningeal condition so quickly as intrathecal injection of Serum, and injection of Serum does not have any marked effect on the general infection although the meningeal condition appears to respond readily when treatment is begun early.

By combined treatment one then attacks simultaneously the general infection, and the meningeal condition, and so brings about a more rapid reaction, and increases the patient's chances of recovery.

Going still further I believe it would be useful to employ a re-inforced serum as suggested by Fairly + Stewart (Monograph on Cerebro Spinal Fever) i. e. ordinary antimeningococcic serum + serum from convalescent patients or from inoculated animals. By so doing one would introduce complement as explained previously.

In the B. M. J. 19:2:17 Drew suggests that simple puncture is as useful in treatment as puncture + injection of serum, but I doubt if this is the case. I have seen cases treated in this manner and I am of opinion that the mortality is heavier, and in the cases which recover convalescence is slower + complications more frequent. The statistics of naval cases published by Surgeon General Rolleston also negate this statement + distinctly show that early treatment is more beneficial when serum is used than when simple drainage is resorted to.

There is one type of case which appears to evade any medical lines of treatment and that is the case where there is matting at the base of the brain, and where the foramina of the ventricles become occluded.

In my opinion these cases are more or less hopeless if treated either by Vaccine or Serum treatment & Surgical measures would have to be resorted to before the patient had any chance of recovery. In these cases the ventricles become filled with fluid which contains numerous organisms & becomes very purulent.

In this type of case surgical measures in the nature of trephining & puncturing the lateral ventricles would require to be carried out. In the B.M.J. 4.4.17 Dickson deals with cases of this kind and Surgical measures on the above lines are being carried out but no statistics of the results obtained have yet been published.

There is also the case where the fluid is too sticky to flow through a needle or where adhesions prevent the complete drainage. In these cases Drew suggests laminectomy and thereby establishing complete drainage B.M.J. 14.2.17.

I have seen none of these surgical measures carried out but it is my opinion that when one has to consider such lines of treatment the prognosis is hopeless and it becomes a question if the operative measures are justifiable.

From the cases which I have seen I am of the opinion that to successfully combat the disease we must perfect our Serum or our Vaccine and work to prevent the disease as well as cure it.

David Smith M.B., Ch.B.
