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CEREBRO-SPINAL FEVER AND THE SPHENOIDAL SINUS.

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PRELIMINARY ARTICLE.

We bring forward the following cases to show that empyema of the sphenoidal sinus has a relation, and probably a causal relation, to the infection of cerebro-spinal fever. The spread of epidemic cerebro-spinal meningitis was originally considered to be different from that of other bacterial infections such as cholera and plague. It did not appear to start from a definite focus, and no traceable series of links could be discovered between the cases. It has now been shown that the bacterial infection spreads from person to person, giving rise to a rhinitis, similar to that produced by other "influenza cold" organisms. Of the infected people a very small percentage subsequently develop meningitis. What determines the onset of meningitis, or the resolution of the rhinitis, has not been ascertained. It has been regarded as being due to a variation in the virulence of the organisms or difference in the resistance of the individual. It seems important in this connexion to establish the path by which the meningococcus gains access to the meninges from the nasopharynx. There are two possible routes: (1) By the blood-stream, and (2) by the lymphatics.

With regard to the blood-stream route, it seems probable that the meningococcus very early gains access to the blood, as do all microorganisms in bacterial infections. That the organism has been demonstrated in the blood and urine of people suffering from the disease is not important in this connexion, since the organism could as readily have obtained access to the blood from the cerebro-spinal fluid as from the nasal mucosa. What is of importance is that the organism has been demonstrated in the urine of people carrying the micro-organism in the nasopharynx and yet having no symptoms of meningitis. The organism, to appear in the urine, must be circulating in the blood-stream. So it is possible that the organisms may gain access to the meninges via the blood-stream.

With respect to the route by the lymphatics direct from the naso-
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pharynx, we would bring forward the following points which we believe support this path of infection.

Post-mortem examinations of three fatal cases of cerebro-spinal meningitis:

Case 1.—Nasopharyngeal mucous membrane very swollen and rugous. Sphenoidal mucous membrane injected; cavity full of glairy pus; ostia impervious to a probe. Osteitis of bone surrounding sinus; numerous pus cells present. No organism seen or cultivated from the bone. Deep congestion of the inner table of the skull; in the region of the sinus the dura mater injected; thick purulent lymph lying on the surface of the dura mater.

Case 2.—The same findings, but the meningococcus was readily demonstrated in both the pus cells in the sphenoidal sinus and in the bone.

Case 3.—The same findings, but in addition the organism was demonstrated in the cancellous bone, in which the inflammation was much more extensive and reached to the apex of the petrous bone.

In none of these cases was there any inflammation occurring in the cribriform plate, the ethmoidal, frontal sinus or the middle ears. The findings suggest that the meningococcus infection spreads from the nasopharyngeal mucous membrane into the sphenoidalsinus. The opening of the sinus is then obstructed by the swelling of the mucous membrane, which is very liable to excessive swelling in children and adolescents in the course of inflammation. The occlusion would be favoured by any abnormal contraction of the ostia or pathological change.

Subsequent to empyema of the sinus infection of the dura mater may supervene. So it might be suggested that among the contacts who have a mucosa infection, it is those who develop an empyema of the sphenoidal sinus who develop cerebro-spinal meningitis. It is accepted that a drop or two of pus in the sphenoidal sinus may cause death (Sir StClair Thomson), while the operation establishing drainage is not a serious procedure.

Fatal cases of sphenoidal sinus empyema usually develop cavernous thrombosis, clinically evident by proptosis of the eyes. In these cases the organisms present cause thrombosis. On the other hand, when the meningococcus infects an empyema in the sinus, thrombosis does not occur, and the meningococcus infects the meninges. The headache of cerebro-spinal fever is frequently referred to the occipital region to which the pain of sphenoidal empyema on manipulation is also referred.

Clinically, the carriers fall into two classes, typical examples of which are given: (1) Acute, lasting about three weeks, and (2) chronic.

Class 1.—Private ——, a Canadian, who yielded practically a pure cultivation of meningococci from his nasopharynx. The mucous membrane of the nose and nasopharynx was swollen and red, and exuded a clear profuse discharge. The ostia of the sphenoidal sinus were
not located by a probe. Ung. hyd. nit. dil. ʒss., menthol gr. v, ol. olivae ad. ʒi, was painted into the nostrils, and the tonsils were swabbed with lot. hydrogen peroxide. In three weeks a swab proved negative. The acute carrier may pass on to Class 2.

Class 2.—The chronic carrier does not exhibit the acute congestion of the nasal mucous membrane, and the organism is not always found, suggesting the presence of a reservoir which leaks from time to time. Case 1 is an instance of this condition. Though the patient was actually suffering from the disease, the same local signs appeared on rhinoscopic examination.

Three healed cases of cerebro-spinal fever were admitted from Tréport Stationary Hospital, and one of us (E. A. P.) examined them. The mucous membrane of the nose and nasopharynx was pale, dry and shrunken. Nasal obstruction in one case prevented location of the sphenoidal ostia, but in the other two the ostia were unusually patent, so that an empyema was almost impossible. It is credible that these men owed their recovery to this patency.

With these data in hand, one of us (E. A. P.) proceeded to drain the sphenoidal sinus in Case 1 (referred to above).

An extract from the notes of Lieutenant Rowlands, of the Welsh Hospital:

—, aged 17, North Irish Horse, landed at Havre, August 21, 1914. December, bad cold, slight haemorrhage from the lung. January 17, Rouen: tubercle diagnosed. February 14, admitted to Welsh Hospital, Netley, with “rheumatic pains”; apparent recovery. 23rd, when up and about, temperature rose to 104°F. (a cerebro-spinal carrier was subsequently discovered in the patient who occupied the opposite bed). 24th, severe frontal headache; erythematous rash; pain at back of neck. 25th, definite meningeal symptoms; increased reflexes; Kernig’s sign present. Lumbar puncture revealed cerebro-spinal fluid under pressure and contained pus cells. No organisms discovered (Dr. Klein’s report). Twenty-five cubic centimetres of serum were injected 28th. Meningococci grown from cerebro-spinal fluid. March 6th, patient has had in all eight injections of serum after lumbar puncture. Head still retracted.

7th, no growth from cerebro-spinal fluid. 9th, relapse; nasopharyngeal swab negative. 10th, autogenous vaccine (five millions) prepared by Dr. Klein was injected by Lieutenant Rowlands, hypodermically. 12th, soamin, five grains, injected intramuscularly. 13th, cyanosis. 17th, twitching; dusky hue; incontinence of urine; no growth from cerebro-spinal fluid. 20th, improvement, and lower temperature. 21st, another relapse; lumbar puncture, without serum injection. 25th, meningococci in nasopharynx. April 2, meningococci not found in nasopharynx. 3rd, pulse 110-120; generally better; occasional delirium. 4th, worse; lumbar puncture and serum. 7th, meningococci not found. 14th, meningococci were found in the nasopharynx; nasal treatment
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with ung. hyd. nit. dil. 5ss., menthol gr. v, ol. olivæ ad. ʒi, painted into the nose. 16th, urotropine given; pulse 110. 30th, transferred to Royal Victoria Hospital on concentration of cerebro-spinal cases. Up to this time seventeen injections of serum have been given, but relapses still occur. May 3rd, chloroform was given by Lieutenant Adeney, and the sphenoidal sinus opened from the nose by one of us (E. A. P.); a dram of pus escaped from the left sinus. Neither ostium could be found with the probe, so the thin bone was broken down with Heath’s small mastoid burr. Meningococci were found in the glairy pus. Subsequent history: The patient was rather better from the first day. There is now no retraction of the head, and Kernig’s sign is only slightly present, but the pulse keeps up to 115-125. May 7, temperature up to 101° F.

Conclusions.

(1) Sphenoidal empyema is associated with cerebro-spinal fever in a causal manner.

(2) Cerebro-spinal fever is a meningitis due to organisms entering the meninges from the sphenoidal sinus by way of the lymphatics.

(3) Adults are less susceptible, owing to a diminished tendency to sphenoidal empyema, as they are not prone to excessive swelling of the mucous membrane and so to closure of the ostia as is seen in adolescents.

It may be advisable to open the sphenoidal sinus in all cases of cerebro-spinal fever. It is certainly advisable to treat the nasopharynx on the lines used for the acute carrier (Class 1, ante). We would register this paper as a plea for nasal hygiene in the case of recruits.

A CASE OF HYSTERICAL PARAPLEGIA.

BY LIEUTENANT ADOLPHE ABRAHAMS.

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C. G., a private of the 1st East Lancs., was admitted to the Connaught Hospital, Aldershot, in September, 1914, suffering from paraplegia.

His history was that on September 8, 1914, whilst he was carrying food from a wagon to the firing line, a shell burst close to him and a companion who was close to him. He remembered the shell bursting and striking the wagon, but a period of unconsciousness of four or five days' duration then elapsed, on recovery from which he was suffering from a small wound in the left buttock and complete paralysis of both legs, with pain in the back in the region of the fourth lumbar vertebra, due in his opinion to the fall of a spare wheel of the wagon upon him.
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