CAVERNOUS SINUS THROMBOSIS.*

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THE cavernous sinus, extending from the sphenoidal fissure backwards to the apex of the petrous portion of the temporal bone, fed mainly by the ophthalmic veins in front, and drained behind by the superior and inferior petrosal sinuses, is not frequently the seat of thrombosis, but when this does occur the symptoms are so striking, as a rule, as not to be readily mistakable. The bulging, immobile eyeball, with œdema and probably discoloration of the surrounding skin, the extreme chemosis of the conjunctiva and rapid depreciation of vision, combined with marked febrile symptoms, are characteristic of no other lesion. In cases complicating suppuration of the middle ear, however, the onset of these symptoms may be quite gradual, and I offer below an explanation of this fact. The following four cases occurred, as it happened, in one year, and each illustrates a different origin.

In the first case I was able to demonstrate, by a somewhat heterodox line of treatment, that the superior petrosal sinus had been *primarily* infected from the middle or the inner ear without previous involvement of the sigmoid or lateral sinuses, and as far as I have been able to find in the literature of the subject, this has not previously been observed.

The second case had a more common origin in a carbuncle on the nose. The third was of the more usual middle-ear type, where the sigmoid sinus was first thrombosed, and the process extended to the cavernous, presumably by either or both petrosals. The fourth was an example of infection following a frontal sinusitis, of which one case is included in the collected statistics of Dwight and Germain,[1] and one has been reported by Dighton.[2]

It is not necessary here to detail further the anatomy of the cavernous sinus, but there are two points I would emphasise. First, the anastomosis is so free between the right and left sinuses by means of the circular sinus, that thrombosis of both is probably the rule. In those cases of Dwight and Germain's

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series, where *post-mortem* results were obtainable, thrombosis on both sides was definitely stated in two-thirds. Of my cases, I obtained consent for *post-mortem* examination in two only, Cases II. and IV., and I found both sides involved in II., while in IV. the only demonstrable septic clot was on the distal side. In the other cases, where *sectio* was not allowed, the proptosis and chemosis involved both eyes, and almost synchronously. The importance of this is obvious. It is our greatest obstacle to successful direct attack on the sinus, and doubtless the explanation of most of our failures. Second, the petrosals are not merely channels for emptying the cavernous sinuses into the sigmoid sinus and the jugular bulb, but they receive blood also from small tributary veins in their course, helping to drain the triangular area of petrous bone, bounded by them and by the sigmoid. The superior petrosal (I quote from Macewen[3]) receives blood from the inferior cerebral and superior cerebellar veins and small branches from the tympanum, many of which issue through the petro-squamosal suture, the remainder passing through the bone directly into the sinus. The inferior petrosal receives blood from the inferior cerebellar veins, and from those of the aqueduct of the cochlea, the vestibule, and the fossa subarcuata.

The sigmoid sinus, because of its abutting directly on the mastoid cells, is the most common site of septic thrombosis, and it is undoubtedly true that most cases of cavernous sinus thrombosis of otitic origin are extensions from the sigmoid, but it is obvious that the petrosals may be directly involved by extension along the small veins referred to, and my first case is an illustration of this possibility.

Case I.—J. D., aged 12, was first seen on 13th December 1918, suffering from sickness and giddiness of forty-eight hours' duration, vomiting irrespective of the taking of food. He was too giddy to stand and always tended to fall forwards. There was a history of discharge from the left ear two months earlier, with recurrence for one week before admission. There was thick pus in the meatus, but the perforation could not be detected after mopping. Tuning-fork, B C > A C. Conversational voice seemed to be heard at 1 foot. There was marked spontaneous nystagmus to the left; ataxia was present and swaying on doing the Romberg test. Kernig and Babinski tests negative; no headache; temperature normal; pulse 60. Cold caloric test to the left ear did not reverse the nystagmus. Dr Matheson Mackay reported both fundi normal. During twelve

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days' stay in hospital, pulse and temperature were normal, the nystagmus gradually disappeared, as also the giddiness and ataxia, so that after a week he was able to be up and about the ward. The discharge also gradually diminished until at the end of a week the ear was practically dry. It seemed that the labyrinthitis had subsided, and when on 24th December he asked to get home for Christmas he was allowed to do so, to return in five days.

He was readmitted, however, on the 26th with partial facial paralysis on the same side (the left): he had vomited once since leaving hospital; neither nystagmus nor giddiness had recurred. The radical mastoid operation was performed at once, pus under pressure, and granulations being found in the antrum. The dura of the middle fossa was exposed and appeared healthy. The dura of the posterior fossa was not exposed as the symptoms did not suggest sinus thrombosis, and the sinus plate was healthy. On removing the bridge there seemed to be granulations covering the adital portion of the facial canal and the wall of the external semicircular canal, but in neither situation could a definite gap be determined. I may state here that during the whole subsequent course of the case nystagmus and giddiness remained absent and the facial paralysis continued. Three days after operation the temperature rose to 104° with a rigor, and the same happened the following day, so that I exposed the sigmoid sinus which, however, appeared healthy and was left untouched. Lumbar puncture yielded a clear cerebro-spinal fluid under normal pressure. During the next two days the same rise of temperature with rigors occurred, and I explored the sigmoid sinus with a syringe. Fluid blood was drawn off and retained for examination. The jugular vein was ligated. Next day there was no rise of temperature and no rigor, and on the following day a slight temperature was recorded, but neither rigor nor headache; by this time the pathologist's report on the blood had arrived and he had found pneumococci.

I presumed then that there was a mural septic clot in the sigmoid sinus, not extensive enough to occlude the sinus completely, and that my needle had acquired some septic material when passing through this to the blood-stream beyond, and I proceeded to open and clear this out. The blood in the sinus was now, of course, thrombosed from ligation of the jugular a few days before, but the clot was clean and no septic part adhering to the wall could be found. When this clean red clot had been scooped away, however, and the gush of blood from above had been established, just before we succeeded in stemming the latter, two small, worm-like pieces of soft blackish grey clot were washed out, just of the calibre of the superior petrosal sinus. After this the temperature still continued to rise daily to 101° and

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102°, but without rigors. Three days later, *i.e.*, fourteen days after the radical mastoid operation, œdema of the face and both eyelids was noticeable. Gradually the eyes became more and more chemotic and proptosed, with limitation of the movements of the orbs. The septic thrombosis of the superior petrosal had extended to the cavernous sinus. Further operation was not suggested. Towards the end, there was incontinence of urine and fæces, but none of the cardinal signs of meningitis ever appeared. Post-mortem examination was not obtained.

Case II.—J. G., aged 31, was admitted on 16th February 1919 with a history of carbuncle on the right side of the nose a week earlier. There were marks of several small incisions on the right side and on the bridge of the nose. There was marked proptosis and chemosis of the left eye with discoloration of the lids and of the forehead. The eyeball did not move and the sight was quite gone, even the light of a strong electric torch not being distinguished. Dr Matheson Mackay reported the retinal veins very large and dark-coloured. The right eye showed neither chemosis nor limitation of movement and had good vision. Examination of nose and throat was negative. Ears normal. Temperature 101°, pulse 78. Reflexes did not point to meningitis. Three hours later the temperature reached 104°. When seen again twelve hours after admission, the forehead and the left eyelids were quite blue and the veins stood out prominently. There was some bulging and œdema of the right eye. Babinski's test gave extensor responses on both sides. Kernig still negative. Death occurred fifteen hours after admission.

Post-mortem.—The whole of the left cavernous sinus was full of septic clot; the communicating sinus exuded liquid pus; the anterior part of the right cavernous sinus contained septic clot and the posterior part uninfected clot. The sigmoid sinuses were normal. There was no pus in the orbital cavities. In death, the œdema of the face and eyelids, as well as the proptosis, had quite disappeared. In this last detail, the case differed from one I saw in the wards of Dr Maitland Ramsay,[4] of Glasgow, ten years ago. This case was described by Ramsay in a lecture to the Ophthalmological Congress at Oxford in 1911, and details will be found in the *Glasgow Medical Journal* of that year. The picture presented was very similar to what I have just described, but Professor Teacher's *post-mortem* report shows proptosis persisting after death, and the explanation is apparently to be found in the fact that he noted abscess in each orbit.

Case III.—G. B., aged 12, was admitted on 6th August 1919 with temperature 105°. History of left otorrhœa of long duration; distinct tenderness over left mastoid and down the neck; no signs of

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meningitis. Radical mastoid operation immediately performed. The sigmoid sinus was thrombosed and gangrenous in appearance. It was opened and evacuated and the jugular ligated. At the operation the question was raised as to whether there was not slight protrusion of the left eye. Two days later there was distinct bulging and chemosis of both eyes with a slight diminution of movement of the left. Dr Mackay reported both fundi normal. I suggested operation on the cavernous sinus, but did not press much against the parents' objections in view of the fact that both sinuses were evidently involved. The symptoms became quickly more pronounced and death took place five days after admission. *Post-mortem* was not allowed.

CASE IV.—C. V., aged 14, was admitted on 10th September 1919. He had been seen ten days earlier by Dr Legge Roe at an Ophthalmic Dispensary with swelling over the left eye, redness of the conjunctiva, and paresis of the external rectus. Three days later there was distinct swelling of the right eyelids, and a tentative diagnosis of cavernous sinus thrombosis was made: patient was advised to seek admission to the Infirmary. This advice was not followed till a week later, when he was admitted under my care moribund. Temperature was 103°, lips blue, face a dusky, livid colour, slight swelling of left eyelids, but no chemosis of either eye. An incision had been made over the left eye and a little pus came from it. Retraction of the neck was well marked; Kernig well marked; Babinski extensor. Examination of nose, throat, and ears negative. A diagnosis of lepto-meningitis was made and this was confirmed by drawing off turbid cerebro-spinal fluid. Patient died half an hour later.

Post-mortem.—A purulent exudate covered the whole of the left side of the brain and several ounces of pus drained into the basin. The lateral, sigmoid and longitudinal sinuses were normal; the left cavernous sinus showed no pus, but the circular sinus and the anterior part of the right cavernous sinus showed pus and soft thrombi. On stripping the dura from the cerebral wall of the left frontal sinus, it was found to be adherent and the bone dark-coloured. This dark area of bone was removed and exposed the frontal sinus full of thick pus and gangrenous mucous membrane. The left ethmoid cells and left antrum of Highmore were in the same condition; the sphenoidal sinus, which was a single cavity with an incomplete septum in its posterior third, contained pus, but the mucous membrane appeared healthy. The sinuses on the right side were normal.

When I examined this case I thought the previous diagnosis of cavernous sinus thrombosis was wrong, as the patient was obviously dying of a septic lepto-meningitis, and the cardinal signs of the former condition had already disappeared, as they do in death if no orbital

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abscess is present to maintain the proptosis. The early diagnosis was, however, justified by the actual demonstration of the thrombosis *post-mortem.*

Treatment.—I do not wish to do more than merely mention the various methods of direct attack on an infected cavernous sinus, having no new suggestion to make. Dwight and others have approached it by turning down a bone-flap in the temporal region as in the Krause approach to the Gasserian ganglion. Grünert has suggested reaching the infected area by way of the nose and the sphenoidal sinus, and a modification of this has been proposed, using Moure's para-nasal route for more direct access to the sphenoidal. Luc has shown on the cadaver that the best line for treatment *viâ* the sphenoidal is to reach the latter from the antrum of the opposite side. Krönlein proposes in cases of frontal origin to exenterate the orbit, and reach the anterior part of the cavernous sinus through the sphenoidal fissure. Results have been most discouraging, and it seems to me that, especially in cases of otitic origin, conservative treatment deserves further consideration.

It is obvious that there is an essential difference in cases arising from infection in front as compared with those originating *â tergo.* The blood current is from the ophthalmic vein and its anastomoses backwards to the cavernous sinus, thence by the superior and inferior petrosals to the sigmoid and the jugular bulb. A septic clot forming in one of the small tributary veins in the region of the orbit or the nose may be transplanted thence to the cavernous sinus, and, in the slower current of the latter, set up septic thrombosis with rapid onset of symptoms. In middle-ear cases, however, the septic extension is against the direction of the blood-stream, and must necessarily occur forwards along a preformed aseptic clot. In other words, when cavernous sinus thrombosis occurs in connection with septic clotting in the sigmoid or petrosal sinuses, the cavernous sinus condition is at first, and for some time, one of venous stasis from damming back of its blood current by a septic clot situated in either of these other sinuses, and the cardinal signs, startling and outstanding as they always are, may be merely signs of blocking, not of sepsis, in the cavernous sinus. If this be so, is it not reasonable to suppose that some cases might be saved, and is it not probable that some of the cases of spontaneous recovery, that is, recovery without direct attack, should be so explained? Spontaneous recovery occurred in 14 of Dwight and Germain's

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182 collected cases. J. S. Fraser[5] and Dan M'Kenzie[5a] have each reported cases, and in the *Journal of Laryngology* of October 1919, a review appeared of a case reported by Torrini.[6] Dighton[7] was doubtless right in attributing the recovery of his otitic case to the suction action of lavage of the jugular bulb, the septic block being aspirated from the inferior petrosal. The elaborate arrangements made by Nature to safeguard the sinuses from interference by the respiratory movements, as outlined by Macewen[8] in his chapter on the Dynamics of the Sinuses, no doubt reduces the force of such aspiration, but it is probably quite efficient.

Similar lavage of the posterior end of the superior petrosal is not, of course, feasible, but I have demonstrated how it was effected, in Case I., by the blood current from the lateral sinus. If we have reason to think there is septic blocking of the superior petrosal, say in a case with sigmoid sinus thrombosis and early cavernous sinus symptoms, where on clearing out the sigmoid, bleeding from below demonstrates a patent inferior petrosal, would it be possible to attempt to apply suction to the superior petrosal by removing the upper sigmoid plug daily for a few seconds, instead of leaving it in position for four or five days till clotting has occurred behind it?

In conclusion, I would submit that although thrombosis of the cavernous sinus is one of the most fatal conditions met with, a sufficient number of recoveries have been recorded, without direct attack, to encourage us to use every available measure to attain that end. When dealing with a sigmoid sinus thrombosis, the most scrupulous cleansing of the mastoid cavity must be secured before opening the vein, and the greatest care observed in inserting the upper plug, so that the clot forming behind this shall remain aseptic. Every possible aid should be supplied to the patient's natural resistance—sera or vaccines, and suitable feeding. Equally important is fresh air. The physicians claim, in recent years, to have reduced the death rate in pneumonia and broncho-pneumonia by treating their cases on the balcony: the general surgeons report similar results from the same treatment of prolonged suppuration in bones. Might we not get better results in all forms of intracranial suppuration if we could adopt this simple procedure? In many of our unsuccessful cases of intracranial complications, after drainage of cerebral or cerebellar abscess, or of the meninges, or of a sinus thrombosis, the fight is so prolonged as to give one

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the impression that some little advantage at some point or other of the course might have tipped the balance on the side of success. Is it not possible that treatment in the open-air might provide that advantage?

REFERENCES.—[1] Dwight and Germain, *Boston Medical Journal*, 1902. [2] Dighton, *Practitioner*, March 1913. [3] Macewen, *Pyogenic Diseases of the Brain and Spinal Cord*, p. 30. [4] Ramsay, *Glasgow Medical Journal*, 1911. [5] Fraser, *Journal of Laryng. Rhin. and Otology*, March 1912. [5a] M'Kenzie, *Journal of Laryng. Rhin. and Otology*, February 1913. [6] Torrini, *Arch. Ital. di Otol.*, vol. xxx. No. 1. [7] Dighton, *Ibid.* [8] Macewen, see under 3, p. 33.

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