EXPERIMENTS ON THROMBOSIS OF THE SUPERIOR LONGITUDINAL SINUS

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(Received for publication February 27, 1946)

The experiments to be described were planned to throw light upon the problem of "otic hydrocephalus": a term originally introduced for a syndrome in which raised intracranial pressure and papilloedema are assumed to be due to sinus-thrombosis following otitis media, but are unaccompanied by evidence of leptomenigitis or of cerebral abscess. The cause and mechanism of this increased pressure are still controversial. Symonds (1937)\textsuperscript{13} believes the syndrome to be dependent upon thrombosis: the lumen of the lateral sinus is blocked and the blockage extends across the torcular Herophili, thence ascending in some instances into the superior longitudinal sinus. Alternatively, in those cases where jugular compression proves both lateral sinuses to be patent, he suggests that the endothelium of the sinuses may be plastered with a thin layer of clot by retrograde extension from the lateral sinus to the superior longitudinal sinus, thus obstructing the arachnoid villi.

While the existence of the syndrome is generally accepted, the presence of an accompanying hydrocephalus is debated. According to some (Gardner, 1939\textsuperscript{9}), the ventricles in these cases are of normal size or may even be reduced. Again, raised intracranial pressure with papilloedema may occur without clinical evidence of thrombosis or of antecedent infection either in the middle ear or elsewhere in the body—the so-called "pseudotumor cerebri" (Davidoff and Dyke, 1936;\textsuperscript{7} Dandy, 1937;\textsuperscript{9} McAlpine, 1937\textsuperscript{11}). To such cases the term "toxic hydrocephalus" has also been given (McAlpine). No satisfactory explanation of the increased intracranial pressure has been offered for these. Yet there can be little doubt from certain records (Ellis, 1937;\textsuperscript{9} Bailey and Hass, 1937;\textsuperscript{1} Russell, 1944\textsuperscript{12}) that thrombosis of the longitudinal sinus may be followed clinically by the syndrome and, in the cases quoted, ventriculography or postmortem examination has secured evidence of internal hydrocephalus.

In view of the existence of such cases it was felt that, could a spreading thrombosis from the longitudinal sinus be induced experimentally, the clinical and pathological study of such animals would throw further light upon the syndrome. So far as we are aware, only one attempt has been made in this direction. In one dog, Bize (1931)\textsuperscript{3} occluded the torcular Herophili and hind end of the longitudinal sinus with a tampon of iodoform gauze, and in

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a second dog he injected 1 cc. of quinine urethane solution at the same site; in neither experiment was there any significant consecutive clinical or pathological change.

Since the middle segment of the longitudinal sinus appears to be the site of election in the initiation of spontaneous thrombosis (Welch, 1909; Bailey and Hass, 1937), this region was selected for our experiments. These fall into groups as follows:

A. Rabbits

1. Inducement of Stasis. The exposed sinus was occluded by a silver clip placed just anterior to the torcular Herophili (4 animals), or by two clips placed from 1.0 to 1.8 cm. apart (3 animals). The animals remained in normal health and were killed at periods from 2 days to 3 weeks later. None showed evidence of thrombosis either macroscopically or microscopically. The brains were normal.

2. Stasis with Introduction of Coagulants. The above procedure was combined with the injection of coagulants into the lumen of the sinus. (a) A physiological coagulant, thrombin, was used in 5 animals. When the greater part of the sinus had been exposed an occluding silk ligature was tied immediately anterior to the torcular, and about 0.1 c. cm. of a mixture in equal parts of a 10 per cent dilution of the thrombin solution and thorotrast was slowly (30 sec.) injected into the lumen anterior to the ligature. The mixture used was tested for us by Dr. R. G. MacFarlane and was found to clot rabbit's blood within 30 seconds in vitro. By adding thorotrast we hoped to gain radiographic information concerning the site of the expected thrombosis. The animals, however, remained normal and no trace of thrombosis was found on examination of the tissues from 5 to 22 days later.

(b) A chemical coagulant, ethamolin (an aqueous solution of 5 per cent ethanolamine oleate with 2 per cent benzyl alcohol), which is used clinically for the injection of varicose veins, was tried in 9 animals. In these experiments the ligature was omitted, and the lumen of the sinus was compressed in two places with artery-forceps while the injection was made into the segment between them. Both the forceps and the needle of the syringe were held in situ for 2 minutes after the injection had been completed. Most of the animals in this group appeared unduly excitable on the following day but, with two exceptions, they returned to normal and remained so until killed from 7 to 40 days later. The latter then showed neither thrombosis nor any other abnormality of the brain. Of the two exceptions one animal was found dead on the following day with extensive intraventricular haemorrhage, apparently due to accidental operative trauma. The other showed retraction of the head and convulsions following operation, and was killed on the second day, as it appeared moribund. The sinus at the site of injection and the great vein of Galen were greatly engorged, and there was moderate congestion of the superficial cerebral veins. The middle lobe of the cerebellum was swollen and protruded for a distance of about 0.6 cm. through the foramen magnum. On section the cortex forming the postero-medial angles of the occipital poles appeared softened and the occipital horns were dilated. On microscopic examination the torcular was partly occluded by recent thrombus but none was found in the longitudinal sinus. There was slight diffuse subarachnoid haemorrhage, more marked at the base of the brain than elsewhere. The occipital poles showed gross oedema of both grey and white matter, with occasional perivascular haemorrhages about the subependymal blood-vessels. The ependyma of the tips of the horns, which normally are not patent in the rabbit, was interrupted in many places by the blowing out of the cavity.

Comment. It was not expected that the degree of stasis induced by ligatures alone would effect thrombosis. But it seemed desirable to perform these initial experiments in view of the suggestion that the longitudinal sinus is particularly susceptible to thrombosis. But our failure when this measure