Remarks

ON

THE CAUSATION OF THE INCREASED INTRACRANIAL PRESSURE ASSOCIATED WITH TUMOURS WITHIN THE CRANIUM.

(Preliminary Note.)

BY

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MANY theories have from time to time been suggested to explain the etiology of the increased intracranial pressure which is so frequently associated with tumours within the skull. Nevertheless the matter is still largely shrouded in mystery, although careful observations made upon large series of cases by a number of investigators offer some significant information. Although clinical evidence of raised intracranial pressure as manifested by headache, vomiting, and choked disc is not an absolutely constant accompaniment of tumours within the cranium, it is generally agreed that it occurs at some time in the course of the disease in upwards of 80 per cent. From researches which have been made with regard to the presence or absence of increased intracranial pressure it is clear that the anatomical site of the tumour is of first importance. The rate of growth and size of the tumour also have some influence, but they appear to be of much less significance than the anatomical situation. The histological character of the growth apparently bears no relation to the occurrence or absence of increased intracranial pressure.

Since the site of the tumour is the factor of fundamental importance, it would seem that the causation of the increased pressure might probably be of mechanical origin. The influence of rate of growth and size of the tumour is also compatible with a mechanical origin, since a slow-growing or small tumour would often permit some compensation for the mechanical disturbance, which is less possible in the case of growths rapidly increasing in size. When we examine the situation of tumours which are most frequently associated with manifestations of raised intracranial pressure further light is shed upon the problem. It is well known that tumours of the mid-brain and subtentorial growths practically always cause intracranial tension, whereas tumours of the pons do not as a rule, unless they invade neighbouring regions. Furthermore, on studying records of patients it is found that signs of increased tension are frequently absent in the case of growths of the frontal region, and less frequently absent in the parietal and occipital regions. The constancy of the occurrence of increased intracranial tension with subtentorial growths might at first sight suggest pressure upon the lateral sinuses, leading to interference with the absorption of cerebro-spinal fluid and its accumulation, with resultant rise of pressure within the skull; further reflection, however, makes such a contention untenable, and it is quite unnecessary in this paper to discuss the overwhelming evidence against such a theory.

Reflecting upon the information cited with reference to the situation of the tumour, compression of the great vein of Galen appears, on anatomical and physiological grounds, to be a much more likely cause for the increased pressure.

With this in mind I recently re-investigated the exact course and relations of this vein, and found them strikingly different from those figured in most anatomical textbooks. By the usual anatomical approach the falx cerebri and the tentorium are disturbed before the vein is examined, and its precise relations and direction are consequently grossly modified. Removing a segment of the calvarium by means of a horizontal saw-cut above the level of the tentorium and a sagittal one about half an inch lateral to the superior longitudinal sinus, it is possible to dissect away the cerebral hemisphere and display the origin, full course, and termination of the great vein of Galen as it really exists with the parts in its immediate vicinity intact. Such a dissection is shown in the accompanying drawing, and it will be seen that the vein passes at first backwards and upwards, and then forwards and upwards, to reach the hinder end of the inferior longitudinal sinus, curving round and in contact with the splenium of the corpus callosum. The relation with the splenium is always found to be most intimate, and the vein opens into the sinus as the meningeal tributaries open into the superior longitudinal sinus—that is to say, against the flow of the current in the sinus. Taking this course, which is much more vertical than horizontal, it will at once be noticed that anything which elevates the

Drawing of dissection described in text, to show the relation of the great vein of Galen to the splenium. Part of the inferior longitudinal and straight sinuses have been incised to display the termination of the vein. S.=splenium.

tentorium is certain to exert tension upon the vein and cause it to be compressed against the splenium. Lateral deflection of the falx cerebri, owing to its attachment to the tentorium, will similarly raise the tentorium and cause the same effect, although not so readily as direct elevation of the tentorium from below. The more rapidly the tension on the vein is applied, the greater must be the risk of compression.

Generally speaking, subtentorial growths and tumours in the parietal and occipital parts of the cerebral hemisphere are obviously more likely to produce this tension, and the suggested consequent compression of the vein, than those occurring in

the frontal region, which will only deflect the narrower apical part of the falx cerebri. Reference to the position and form of the falx cerebri and its attachment to the tentorium will show that very considerable lateral deflection would be required in the frontal region, and much less in the parietal and occipital regions, to produce elevation of the tentorium. Mid-brain tumours must be so near to the vein at its origin that direct pressure upon it, or compression of it against the splenium, is exceedingly probable in practically all cases. Tumours of the pons, on the other hand, are not likely to cause any interference with the vein for anatomical reasons, unless they invade neighbouring regions.

The question still remains to be answered as to how compression of the great vein of Galen can cause increased intracranial pressure. Compression of this vein near its origin will produce obstruction to the venous drainage from the choroid plexuses of the lateral and third ventricles, since the veins from these plexuses are tributaries of the two veins of Galen which lie in the velum interpositum and fuse to form the great vein of Galen. Such obstruction must produce an increased capillary pressure in the choroid plexuses, which is likely to lead to an increased production of cerebro-spinal fluid. Fortunately there is convincing experimental evidence to prove the latter statement. Dandy¹ has recently proved that occlusion of the great vein of Galen in dogs, provided it occurs at the origin of the vein and the development of a collateral circulation is thus

prevented, causes internal hydrocephalus on account of the overproduction of cerebro-spinal fluid. As would be anticipated, Dandy found that the hydrocephalus developed more slowly than if caused by occlusion of the aqueduct of Sylvius. In addition to this experimental evidence, a few cases have been reported of internal hydrocephalus caused by thrombosis of the great vein of Galen or pressure upon the straight sinus. These suggestions may be criticized, owing to the fact that obvious internal hydrocephalus is not an invariable or essential accompaniment of increased intracranial pressure from tumour. With reference to this it must be remembered that in Dandy's experiments, and in most of the reported cases, the venous obstruction was complete, and the cerebro-spinal fluid was produced in such large quantities that internal hydrocephalus was induced. Less complete obstruction is likely to cause less overproduction of fluid, and slight or negligible dilatation of the ventricular system, but sufficient to produce an increased intracranial pressure.

SUMMARY.

1. It is shown that the great vein of Galen is situated in such a position that it may be compressed against the

splenium of the corpus callosum indirectly or directly by tumours occupying sites which are known to give rise most constantly to increased intracranial pressure.

2. Such compression is not so likely in the case of tumours occupying situations which are known to cause less frequently increased intracranial pressure.

3. Experimental and clinical evidence has been submitted by Dandy and others that occlusion of the great vein of Galen leads to internal hydrocephalus from overproduction of cerebro-spinal fluid.

4. It is suggested that moderate compression without complete occlusion of the vein is likely to give rise to increased intracranial pressure, accompanied by slight or negligible dilatation of the ventricles.

5. If the views put forward in this paper are correct, the increased pressure caused by intracranial tumours is due to an excessive production of cerebro-spinal fluid, the fluid being produced in such quantities that absorption cannot keep pace with production, and the intracranial pressure being consequently raised.

REFERENCE.

¹ Dandy, W. E.: Experimental Hydrocephalus, *Annals of Surgery* vol. 70, 1919, pp. 129-142.

British Medical Association.

PROCEEDINGS OF SECTIONS AT THE ANNUAL MEETING, NOTTINGHAM, 1926.

SECTIONS OF ORTHOPAEDICS AND OF DISEASES OF CHILDREN.

E. MUIRHEAD LITTLE, F.R.C.S., in the Chair.

DISCUSSION ON SPASTIC PARALYSIS.

OPENING PAPERS.

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I ASSUME that the organizers of this discussion, when they chose the title of "spastic paralysis," intended that we should accept the word "spastic" in a strict sense and confine our attention to conditions in which stiffness and weakness of the muscles result from disease of the pyramidal system. Rigidity, paralysis, contractures, and deformities from extrapyramidal motor disease and from disease of the lower motor neurone or of the bones and joints, therefore, are outside the limits of our subject.

Further, we are concerned in this Section with spastic paralysis in children; hence the inflammations, degenerations, and compressions that account for most of the cases of spastic paralysis in adults, though many of them occur in children, may well be excluded from the medical part of our discussion, so that we may devote our time to some special morbid processes that produce their effects before or during birth or in early life—the cerebral diplegias, infantile hemiplegias, diffuse cerebral sclerosis, and certain familial diseases.

The commonest and therefore the most important of these is the form of cerebral diplegia generally known as Little's disease. Strictly speaking, Little's disease is a spastic diplegia associated with abnormal birth, characterized by a preponderance of rigidity over paralysis, the legs being most affected, and a tendency to recovery, but it is customary now to apply the name to all cases, whether birth is normal or abnormal.

To the accurate clinical description given by Little, an orthopaedic surgeon, more than eighty years ago, nothing material has been added, but controversy still rages concerning the pathogenesis of his disease. According to many, injury to the brain at birth is the most important etiological factor; according to some, the cause, whatever it is, acts during intrauterine life. In his presidential address delivered before the Neurological Section of the

Royal Society of Medicine Dr. James Collier gave a memorable exposition of his personal views on the pathogenesis of cerebral diplegia, and set forth the following conclusions: hereditary and familial influences are extremely rare; maternal ill health during pregnancy has been placed in an important position by many writers, but without justification; abnormalities of birth are common but by no means constant associates of diplegia, and they may be excluded as causal factors; the essential anatomical lesion is a primary degeneration of cerebral neurones from causes at present unknown; the evidence demands the verdict that meningeal haemorrhage should be deleted as a causal factor of any infantile spastic state.

Dr. Collier has shepherded or cudgelled most of us into what he considers to be the way of truth in this matter, but the last word has not been said and the subject is still open for discussion. Those who oppose his view are likely to marshal an array of facts which are not easily set aside; they would strengthen their position materially if they took their stand upon "birth injury," rather than the more particular "meningeal haemorrhage," as the most important etiological factor.

It has been said that such dissimilar factors as premature precipitate birth and prolonged and difficult labour could not produce the same effects. But cerebral lesions are most frequent proportionately in premature infants, while it cannot be denied that injury is likely to occur readily during a protracted labour. It is said, too, with truth, that in many cases of diplegia the labour was normal in every respect. But the brain may have been injured nevertheless, for one of the most important factors in producing birth injuries is one to which little consideration has been given in discussions on this subject, for the good reason that its existence was unknown. I refer to the mechanism described by Schwartz, who has shown that injury frequently occurs in the brain under the presenting part of the head, due to differences in intrauterine and atmospheric pressures.

This factor may be very important when labour is normal in its time of onset and in its duration. The remarkable researches upon the mechanism of birth injuries and the pathological changes produced thereby that are being carried out in Frankfurt by Schwartz and his associates demand the careful attention of obstetricians, paediatrists, and neurologists alike. Many hundreds of brains, from children who have died during the first seven months of life have already been examined; in 65 per cent. of them pathological changes due to birth injury were visible to the naked eye; by comparing the appearances in brains from children who lived a few days only with those who died after weeks, months, or years, it has been possible to follow the changes that occur in and around birth injuries and to recognize them at all ages.

Perhaps the most important part of Schwartz's work is