of tongue movements and the presence of spasticity or athetosis. If speech is forthcoming its extent and quality can be assessed. This initial clinical screening will generally need several interviews, and the first one at least is better carried out in the child's home. This simple clinical approach can give useful if limited guidance in many children. Suspicion of mental defect will be aroused by excessive restlessness, lack of concentration, inability to design and sustain individual play for any length of time, destructiveness, and capricious emotional behaviour. However, the exact diagnosis of moderate mental retardation demands expert assessment before suspicion is allowed to harden into certainty.

With severe deafness for all frequencies, the child fails to respond to some or all of the everyday noises as well as to speech. What is more, he may be so frustrated by his failure to understand and be understood that he has violent tantrums and may be first brought to the doctor as a behaviour problem. On the other hand, in the child with high-frequency deafness, the beginnings of speech are not greatly delayed, and so the condition is often missed, sometimes well into school life. Such a child may be treated for defective articulation unless inquiry has recorded that he responds well to sounds and music, but hardly at all to speech and stories unless he can watch the speaker's face. The most difficult children to distinguish from these with developmental aphasia, however, are children with varying degrees of partial deafness, particularly with loss in the middle frequency range. These children are not silent, even though speech is delayed, and sensible parents have often failed to recognize the true state of affairs. In any child with delayed speech, especially where comprehension is defective, hearing must be thoroughly investigated by an otologist expert in screening tests and audiometric methods suitable for children of different ages. Only in this way will these children with partial deafness be discovered and proper training instituted, with the use of hearing-aids where necessary. The distinction between partial deafness and receptive aphasia can be particularly difficult and may require further observation or even a period of residence in a special school. Moor House School is the only school in the country at present which caters for the more complex disorders of speech.

Where the delayed speech is due to a mainly expressive aphasia there is good understanding for speech, and if the child's intelligence is normal there should be little difficulty in its recognition. The psychotic child with delayed speech, though uncommon, may go unrecognized for a long time under the label of mental defect or behaviour difficulties. Gradually, however, a more bizarre pattern of behaviour becomes evident and psychiatric help is sought.

Treatment

As we believe the delayed development of the use of language is the manifestation of delayed neurological development, attempts to teach a child to speak will produce little result. It is necessary, however, that nothing should be done which would hinder the child's spontaneous development. Overanxiety on the part of the parents, repeated attempts to make the child say words, undue attention paid to any words he may say, producing self-consciousness, will tend to delay rather than assist the development of speech. It is, however, necessary to ensure that there is sufficient environmental stimulus. The natural urge to speak is usually so strong that the child will speak if he can; but contact with children of his own age may be helpful, and, without producing any undue sense of frustration in the child, the mother should not always respond immediately to gestures, but should so manage the situation that the child realizes the usefulness of words rather than signs.

An interest in naming objects and pictures may be stimulated if the child is asked to point to an object named rather than asked, until he is ready, to say the word himself. A collection of pictures in a scrapbook of his own may also help. If the child is more than usually silent and has done

little or no babbling, it may be possible to encourage imitation of oral sounds such as consonants or vowels, or animal noises, and in one boy of 6½ years the ability to pick out and make the appropriate sound for a single letter is developing before he is able to use words. If the child of 5 years understands speech he can attend the normal school and can learn even if he cannot speak. If, however, use of expressive speech is delayed beyond the age of 6 years his educational progress will be seriously handicapped.

Summary

We have presented our experience of the delayed development of speech. The main causes are mental deficiency, severe and partial deafness, developmental aphasia, cerebral palsy, and, rarely, psychotic illness.

High-frequency deafness is not associated with any great delay in speaking. In spite of traditional objections we favour the term developmental aphasia for children who are mentally normal, have good hearing and no evidence of cerebral palsy or disease, but show a considerable delay in the development of speech. We have no idea of the true incidence of this condition, but the fact that in the past six years we have seen 15 striking examples and 49 children with a similar though less prolonged delay makes developmental aphasia a more important cause of delayed speech than we expected.

The help which can reasonably be expected from speech therapy has been described. It is important to remember that in many of these children there is a complicating alexia, and that in some there are difficulties with articulation as well.

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SIGNS OF OBSTRUCTION OF THE SUPERIOR LONGITUDINAL SINUS FOLLOWING CLOSED HEAD INJURIES (TRAUMATIC HYDROCEPHALUS)

BY

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The *British Medical Journal* has published some of the most important contributions to the knowledge of the clinical effects of non-infective thrombosis of the intracranial venous structures and the conditions in which such thrombosis is likely to occur. Among the more comprehensive papers we may recall those of Collier (1891), Holmes and Sargent (1915), Symonds (1940), Martin and Sheehan (1941), Martin (1941). Full notice is taken of these contributions in the recent monograph of Garcin and Pestel (1949), in which the whole subject is admirably reviewed.

There is another small group of cases which I believe to be of a similar nature, and which has been slow to receive general recognition. It is a group of cases of head injury in which signs of raised intracranial pressure

develop—usually at an interval after the injury—without signs of any severe intracranial bleeding, and in which ultimate recovery occurs without operation. The cases (or most of them) have many points of similarity to those of "otitic hydrocephalus" and similar conditions (McAlpine, 1937), and I believe the symptoms are attributable to the same disturbance—namely, complete or partial obstruction of the superior longitudinal sinus.

Obstruction of the superior longitudinal sinus is usually incomplete and gives rise to two syndromes, which may occur separately or in combination. Since this sinus is the channel into which the cerebrospinal fluid is absorbed and in which it mixes with the blood stream, obstruction of the sinus causes a stoppage of the absorption of the fluid and a consequent rise of intracranial pressure. In such cases the increased pressure of the intracranial fluid is the same outside and inside the brain, and no dilatation of the ventricles results from it (though some may be present from other causes). Headache and papilloedema, however, occur and are often severe. The obstruction may be in the superior sagittal sinus itself, but not infrequently it seems to be in the transverse sinus into which the superior sagittal sinus turns (therefore usually the right one).

The other syndrome is a paralytic one resulting from obstruction and consequent thrombosis of cortical veins which drain into the sinus. Thrombosis of these veins gives rise to extensive bleeding because of rupture of their tributary venules, both on the surface of the brain and, which is more important, within its substance. The syndrome was described by Holmes and Sargent during the first world war in a classical paper based on a number of cases in which the superior sagittal sinus was injured or grazed in gunshot wounds of the skull. As the affected veins are often those which drain the superior portions of the motor gyri, the resulting syndrome, as is now well known, consists of paralysis of the legs and (partial) paralysis of the trunk and upper arms, with sparing of the hands and face. Convulsions, sometimes of great severity and duration, may accompany the onset.

Both of these syndromes are met with occasionally after closed head injuries, the paralytic syndrome being fortunately much rarer than the other.

Case 1

A bus-conductor aged about 40 was riding his bicycle along a dark avenue leading from the garage when he ran into an obstruction: he was thrown over the handlebars and evidently fell on his head, and was subsequently found lying unconscious on the roadway. He was taken to a local hospital, where he was found to have severe abrasions of the scalp over the vertex, but x-ray examination revealed no fracture of the skull. He soon regained consciousness, and for the next twenty-four hours he seemed to be making normal progress. On the second day he had a number of severe convulsions, and these continued into the following day, and thereafter he slowly regained consciousness in the course of a week or so. It was then observed that he had partial paralysis of both sides of his body, and it was discovered that he had severe papilloedema. After some further observation and delay—about three weeks after his injury—he was transferred to the National Hospital, Queen Square, as a supposed case of cerebral tumour. The means of investigation in vogue at that time failed to reveal any convincing indications for operation, and as the patient seemed to be improving none was undertaken. The patient

gradually recovered; his headaches and papilloedema subsided, but he was left with a spastic weakness of his legs, trunk, and upper arms, and some slight impairment of intelligence and of emotional control.

Comment.—In the light of the outcome of this case, thrombosis of the superior sagittal sinus seems much the most probable diagnosis. The bilateral paralysis, with its characteristic distribution, and the onset with convulsions are all indicative of thrombosis of superior cortical veins in relation to the sagittal sinus, and the presence of papilloedema completes the syndrome of sagittal sinus obstruction.

Case 2

A bus-conductress aged 39 was transferred to the National Hospital, Queen Square, fourteen days after an accident in which she had fallen in the street and injured her head and been rendered unconscious. Immediately after the accident she had been admitted to another hospital; she had a small laceration of her scalp at the back of her head and an underlying haematoma. On recovering consciousness she was irritable, but presented no signs of gross cerebral injury. Six days after the accident she was examined by a neurologist and had then no papilloedema. X-ray examination showed a linear fracture on the right side in the squamous temporal bone. A day or two before being transferred to the National Hospital she became lethargic and disorientated, and showed slight rigidity of her neck; bilateral papilloedema was present, and she had also bilateral weakness of the external rectus muscle of the eye, and weakness of the right side of the face.

By the time of her admission to the National Hospital she was again correctly orientated and fairly alert. Her scalp injury had healed completely. She complained of severe headaches and movements of her neck were painful. She had bilateral papilloedema with haemorrhages. The left external ocular muscle was paralysed and the right one partly so: upward movement of the eyes was inadequate. She was tender over the right temporo-mandibular joint, and the right jaw and right side of the face moved less well than the left. She moved all her limbs normally. Her tendon reflexes were all weak or absent, but both plantar reflexes were extensor. The pressure of the cerebrospinal fluid was 280 mm. of fluid; the effect of bilateral jugular compression was to produce a brisk rise in the pressure, but unfortunately the effects of unilateral jugular compression were not tested. The fluid contained only one cell per c.mm. and 65 mg. of protein per 100 ml. The Wassermann reaction was negative. The E.E.G. showed much slow activity on both sides of the brain, but more on the left than on the right. The fracture on the right side of the skull extended obliquely across the squamous temporal bone and down into the greater wing of the sphenoid. (It therefore did not cross the line of the right lateral sinus.)

A fortnight after her admission the patient was much better. Her headache was minimal, flexion of her neck was full and painless, her papilloedema was subsiding, and her ocular movements showed great improvement. She was discharged after a month, fully recovered except for a residual haemorrhage in her left retina.

Case 3

The patient was a girl of 11 years of age, whom I saw at the National Hospital, Queen Square, through the kindness of my colleague Dr. J. St. C. Elkington. Three weeks previous to her admission to the hospital she fell while running, and struck the right side of her head on the pavement. A passer-by helped her to rise and she went on to school. About ten days after the fall she began to complain of headaches, situated over the right parietal region, occurring in the morning and lasting about half an hour. Also, a few days after the fall she developed double vision, and later slight unsteadiness in walking and running.

When examined at the hospital she had bilateral papilloedema, with a few haemorrhages and some exudate round the edge of each disk. There was great weakness of both external ocular recti muscles and a suggestion of weakness of the left half of the face. She showed very slight ataxia in movements of the left arm and leg. All her tendon jerks were weak or absent, and the plantar reflexes were flexor.

X-ray examination of the skull revealed a long vertical linear fracture on the right side extending from the anterior part of the parietal bone down to the mastoid region and across the line of the transverse sinus. The E.E.G. was normal. Ventriculography showed no abnormality of the ventricular system. The fluid withdrawn from the right lateral ventricle was faintly blood-stained and contained 4,100 red blood cells per c.mm., with normal protein. Ten days later the C.S.F. pressure measured at lumbar puncture was 240 mm. of fluid; the fluid contained no cells, but the protein amounted to 80 mg. per 100 ml. The Wassermann reaction was negative. Within three weeks the child made a complete recovery clinically and before discharge her C.S.F. pressure was normal.

Case 4

A woman aged 44 was transferred from another hospital on September 20, 1950. She had had a bicycle accident on August 25 and had been admitted to a local hospital unconscious, and was slow in recovering: the only external wound was a small laceration of her scalp on the right side near the back, which required one stitch: this subsequently healed perfectly without any sign of infection. After her recovery of consciousness headache was continuous, and in the first few days she vomited a number of times. X-ray examination showed a fracture in the right occipital bone.

She was kept in bed for three weeks, headaches being almost continuous during that time, and she experienced some blurring of vision. At the end of three weeks she was allowed to sit up in a chair for an hour or two, and while doing so she had a fit. It was then found that she had marked swelling of both optic disks, with oedema of the surrounding retina. Her visual fields were full and movements of the eye were normal. Power and sensation and the reflexes were normal in her limbs with the exception of the left plantar reflex, which was extensor. The C.S.F. pressure four weeks after her accident was 160 mm. of fluid; the fluid contained 214 red blood cells per c.mm. and 45 mg. of protein per 100 ml. The Wassermann reaction was negative. The fracture of the skull extended vertically throughout the length of the right occipital bone, crossing the line of the transverse sinus about one inch (2.5 cm.) from the middle line of the head. The E.E.G. was normal.

The patient made a complete recovery without active treatment, and left hospital two months from the date of her accident with no abnormal physical signs except slight residual blurring of the margins of her optic disks.

Case 5

In this instance the symptoms and signs of obstruction of the superior sagittal sinus developed while the patient was actually under my observation, and it was the first case in which I recognized this condition, which I take to be post-traumatic sinus thrombosis. The patient was an active middle-aged woman whom I saw with her doctor in April, 1940. She had had a fall from her bicycle on the previous day and was not seriously injured: she had grazed the right side of her face and scalp, but in the course of the afternoon and night she had developed a very large haematoma under the scalp, chiefly on the right side. Twenty-four hours after the injury she had little headache and no abnormal signs referable to her central nervous system. X-ray examination of the skull showed no fracture. In the succeeding week she made good progress and the haematoma under the scalp was aspirated under aseptic conditions: at no time was there any suggestion of infection in it and her abrasions

healed quickly. Ten days after the accident she complained of severe headache, small ecchymoses appeared in her upper eyelids, and her optic disks became blurred and within twenty-four hours considerably swollen. No other abnormal physical signs developed in her central nervous system or elsewhere. She was kept completely at rest with mild dehydration, and her symptoms and signs disappeared in the course of three weeks.

Comment

In all of these cases, with the possible exception of the first, the accidents were relatively slight—falls when walking or cycling—and it may be that the absence of more severe injuries allowed the complication we are concerned with to be recognized more easily.

The clinical features in all the cases are those of obstruction of the superior longitudinal sinus, with admittedly some additional features in Cases 2 and 3—that is, unilateral or bilateral paralysis of the external rectus muscle of the eye. The occurrence of these ocular palsies makes the diagnosis of sinus thrombosis still more probable, because it is exactly similar to that which occurs in another condition in which the superior longitudinal sinus is affected—namely, otitic hydrocephalus with Gradenigo's syndrome. The paralysis of the abducens nerve has been attributed by Symonds (1944) in the otitic cases to thrombosis of the inferior petrosal sinus, and it is fully probable that in these traumatic cases, similarly, the inferior petrosal sinus, on one side in Case 2 and on both sides in Case 3, was thrombosed.

In all of the cases the course was similar to that which we are familiar with in cases of sinus thrombosis due to other causes.

It seems likely that in these cases of closed head injury there is as a rule an interval between the injury and the onset of the signs of sinus obstruction. In my Case 5 the interval was ten days, in Case 2 it was more than six days, in Case 1 it was possibly less than 24 hours, and in the other cases the history does not enable us to determine it.

The diagnosis of this particular variety of sinus thrombosis is not at all difficult once the examiner is aware that it may happen. The position is analogous to that of otitic hydrocephalus, but this traumatic hydrocephalus is more likely to be overlooked because the headaches may be attributed to other effects of the trauma. The rapid onset of papilloedema after a head injury, without any localized signs to suggest a space-occupying lesion and in the presence of a clear cerebrospinal fluid without inflammatory cells, is almost sufficient for the diagnosis, but in cases of doubt a ventriculogram is required to confirm that the ventricles show no deformity. Syphilitic hydrocephalus is now uncommon, but it is a simple precaution to have the Wassermann reaction done in all cases.

My later cases have been treated with magnesium sulphate, given at first by rectum and afterwards by mouth, and all have recovered without impairment of sight. In a case characterized by convulsions—for example, Case 1—an anticoagulant such as ethyl biscoumacetate ("tromexan") might be prescribed in the hope of preventing further venous thrombosis, provided there was no longer any reason to fear haemorrhage from other effects of the accident and provided adequate control of prothrombin or of clotting-time could be exercised.

The factors responsible for the thrombosis in these cases call for some brief discussion. In two of the cases I have cited there were fractures which crossed the line of the right transverse sinus, and in a third case there was a fracture on the right side extending across the squamous temporal bone into the greater wing of the sphenoid. If thrombosis occurred in the two former cases in the transverse sinus it was probably a consequence of injury of the dura forming the wall of the sinus. But for the dura to be damaged it is not necessary that the bone against which it lies should be fractured. The damage to the dura may be a result of the deformation of the skull at the time of the injury, and, in

fact, I have once seen a small longitudinal tear in the lateral sinus in a case of head injury without any fracture of the skull. In Case 2 there was a fracture on the right side of the skull, showing that severe stress must have occurred in the neighbourhood of the lateral sinus, and it seems to me probable that the dura forming the wall of the sinus was damaged by the same deformation as caused the fracture in the squamous bone.

In some, and probably all, of these cases there were abrasions or small lacerations of the scalp, and in one case a very large haematoma underlying it. In no case was there obvious infection. Thrombosis may have extended inwards from damaged small veins passing into the diploë, and thence the thrombus may have extended into the sagittal or transverse sinus. In some of the cases of Holmes and Sargent (1915) the outer table only of the skull was damaged, a gutter being left with diploë in its base, and it was evident that such wounds were sufficient to cause thrombosis in the sagittal sinus by spread from the vessels of the diploë. In Case 5 of this series the haematoma under the scalp probably arose from torn emissary veins, and the damaged vessels would afterwards become thrombosed. Such extension of thrombosis must therefore be taken into consideration. Moreover, we know that when infection is present this mode of extension of thrombosis is frequent and provides a potent vehicle for the transference of infection (Turner and Reynolds, 1931). In this way infection is conveyed from the mastoid cells into the substance of the brain, to give rise to a cerebral or cerebellar abscess, and in this way infection is conveyed from a pustule on the face, to cause thrombosis and infection of the cavernous sinus. If there had been any evidence of septic infection in our cases we would have had to admit this mode of spread, and possibly it may have obtained even though there was no sepsis. Non-septic infection may have been present, or the clot may have spread without any infection. If, however, extension of thrombosis by way of diploic vessels were an important factor in causing sinus thrombosis after trauma one would expect this complication of head injuries to be much more frequent than it is, since abrasions or lacerations of the scalp are seldom lacking.

On the whole, therefore, it seems likely that injury of the dura is the more important of these factors.

Summary

Five cases are described in which signs and symptoms attributed to obstruction of the superior longitudinal sinus occurred after closed-head injuries. In four of the cases the manifestations were closely comparable to those of otitic hydrocephalus: in the remaining case the full syndrome described by Holmes and Sargent developed.

Damage to the dura in the wall of a sinus and extension of thrombosis inwards from abrasions of the scalp or damaged emissary veins are probably the most important factors causing sinus thrombosis in such cases.

Since the superior longitudinal sinus usually turns into the right transverse sinus, a major importance, so far as this complication is concerned, attaches to injuries in the neighbourhood of the right transverse sinus.

ADDENDUM.—Foley, J. (*Brain*, 1955, **78**, 1) has described similar cases of benign hydrocephalus following head injury.

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CHLORPROMAZINE AND OPERATIVE STRESS IN A TROPICAL HOSPITAL

BY

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In Jamaican patients of the poorer classes the reaction to surgical intervention is excessive and therefore dangerous. Ill-judged or badly administered anaesthesia or inefficient resuscitation quickly leads to circulatory collapse; violent fluctuations in blood pressure are the inevitable result of moderate traction on abdominal viscera; and even the biochemical effects of anaesthesia and surgery are more pronounced than in the European patient.

Sudden circulatory failure may follow a long or short period of bad anaesthetic management, but it is more often due to a combination of slow haemorrhage and autonomic reflexes from the operation site. These reflexes may stall the heart by direct action through its control mechanism, or the main effect may be on the peripheral vascular bed, which becomes so expanded that the heart receives too little blood to carry on its own circulation. Prolonged anoxia due to undetected or uncontrolled bronchospasm, in an otherwise healthy patient, may cause death towards the end of a long operation. In none of these cases does the pathologist find a plausible cause of death. Malnourished elderly patients suffering from slow haemorrhage cannot withstand sudden peripheral vascular dilatation, nor can the effects of sixty years of undernourishment be corrected by two weeks in hospital and two pints (a litre) of blood.

Anaesthetic and resuscitation methods in this hospital do not differ materially from those in Great Britain except that low spinal or extradural blocks are more often employed in superficial operations.

For the past three years efforts have been made to develop anaesthetic methods which would not worsen the poor risk presented by the patient of 60 suffering from nutritional anaemia or nutritional cirrhosis of the liver, with alteration in the serum protein ratio and a systolic blood pressure fluctuating, even under resting conditions in the ward, by 40 mm. Hg. Spinal analgesia was tried in combination with light general anaesthesia, but it was not found effective in stabilizing the circulation—even in pelvic operations. The post-operative condition of patients given deep ether or cyclopropane anaesthesia would not be regarded as satisfactory by an anaesthetist, and in many cases their use during operation is precluded by the diathermy. The nearest approach to a solution of the problem was found in the employment of what is now a familiar technique: induction of anaesthesia with thiopentone and maintenance with nitrous oxide and pethidine usually combined with a relaxant. This has reduced but not abolished the incidence of dangerous vasomotor reactions. The appearance of a group of drugs which were claimed to abolish or at least damp excessive autonomic reaction to operative stress was welcomed.

Racial Reactions to Anaesthesia

It is unfortunate that little work has been published on the reactions of a population such as the Jamaican to surgery