THROMBOSIS OF INTERNAL CAROTID ARTERY IN CHILDHOOD AFTER INJURIES IN REGION OF SOFT PALATE

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Injuries in the region of the soft palate, though uncommon at all ages, occur more often in children. The particular liability of children to such injury is due to the common practice of holding pointed objects in the mouth and the likelihood of falling when doing so. The object may then be driven backwards and penetrate the soft palate or faucial pillars. Lacerations caused in this way are usually of minor degree and uncomplicated. However, recent experience in Edinburgh indicates that serious complications may occur. Three patients have been seen in whom such an injury was followed by hemiplegia. The nature of this serious complication, and the prevention and treatment of the responsible injury, seem to require attention.

Case 1

On June 28, 1941, a right-handed girl aged 4 years and 3 months was attempting to press a metal rod into the ground with her teeth, when it slipped and injured her throat in the region of the right tonsil. Bleeding was slight. A bland mouthwash was given, iodine was applied to the wound, and she was put to bed. She fell asleep almost immediately.

She was aroused for her evening meal without any difficulty about half an hour later and appeared to be quite normal. Nevertheless she was maintained at rest for a further two hours before being allowed up. Immediately after rising—that is, about three hours after the injury—she became very unsteady, turned pale, and slumped to the ground. In lifting her the mother noticed that the left side of her mouth was drooping and that her left arm and leg were hanging limply. She was replaced in bed and recovered consciousness within a few seconds.

An hour later the facial asymmetry was no longer obvious and her colour had improved considerably. She was bright and active, and was allowed up again. She was able to walk, though with slight difficulty. All essential movements were present in the left upper limb, but there remained a definite weakness of the hand grasp.

She was transferred to a hospital in Perth later that evening. During the journey the weakness of the left side of the body became more pronounced, but no symptoms developed. At no time did she complain of her vision, nor did she mention any sensory disturbance. Only slight improvement in her condition was observed in the next ten days. She was then removed to Edinburgh under the care of Mr. Norman Dott.

On examination a hemiplegia was noted on the left side, involving the face, arm, and leg, associated with muscular hypertonicity and hyperreflexia. The abdominal reflexes on the left side were absent and the plantar response was extensor. An accurate assessment of sensory function was not possible, but the impression of the examiner was that exteroceptive sensation was depressed on the left half of the body. The left corneal reflex was slightly depressed. Her fields of vision appeared normal and ophthalmoscopic examination was negative. She was alert and co-operative, and there was no evidence of a psychological disorder. Speech was normal. There was no great swelling in the throat or in the right side of the neck externally. X-ray examination of the skull showed no abnormality.

Power began to return to the affected limbs three weeks after the injury, and by the sixth week she was able to walk unassisted. When last seen, three months after her injury, she still had moderate hypo-aesthesia to touch and pin-prick on the left side and a mild hemiparesis.

Case 2

A right-handed girl aged 4 years and 10 months was climbing on to a sofa with a pencil in her mouth, when she lost her grip. As she fell to the floor her head struck an adjacent chair. She lay unconscious for two or three minutes with the pencil held firmly between her teeth. The pencil was removed. There was slight bleeding from the mouth which stopped spontaneously. She was at once brought to the Royal Hospital for Sick Children, Edinburgh (October 4, 1953).

On admission to the ward she was fully conscious, and, apart from the presence of bilateral extensor plantar responses, neurological examination was negative. Inspection of the throat revealed a laceration $\frac{1}{4}$ in. (6 mm.) long in the anterior pillar of the fauces on the left side. There was no swelling of the throat or in the left side of the neck. Local treatment was not required.

A diagnosis of concussion head injury was made and she was detained for observation. The following day she was a little drowsier, but there was no other evidence of cerebral dysfunction. Two days after admission a weakness of the right arm and a right extensor plantar response were discovered on routine examination. In the course of the next few hours she became progressively drowsier and the paralysis extended to involve the whole of the right side of the body, including the face. The limbs were flaccid. The light reflex on the left side was a little sluggish. There was no papilloedema. A more detailed assessment of nervous function was impossible because of her drowsiness.

Next day engorgement of the retinal veins and slight bilateral papilloedema developed. Her state of consciousness was unchanged and tone in the limbs was returning. A day later she was much more alert and co-operative, and a severe dysphasia was now apparent. She could say no more than "Yes" or "No," and often tended to confuse the two words. Consequently an accurate evaluation of sensory function was impossible.

She was unable to co-operate adequately until six days after the injury, when the confrontation test revealed the presence of a right homonymous inferior quadrantic hemianopia. The papilloedema receded and motor power and speech improved steadily until she was discharged 23 days after admission.

Her further progress has been supervised in the out-patient department in close co-operation with the orthopaedic, physiotherapy, and speech therapy departments. When last examined, nine months after the injury, she was cheerful and friendly but somewhat reticent of speech. Her speech appeared to be normal, but her mother had noticed that if she spoke for any length of time she developed a slight hesitancy. Her principal residual disability was an inability to perform fine finger movements and a marked weakness of the grasp on the right side. She had a barely perceptible hemiparetic limp, which was even less obvious when she ran. Muscle tone was increased on the right side of the body, but there was no impairment of sensation. The visual fields were normal. She had made excellent progress in a school for the physically handicapped and was about to enter an ordinary school.

Case 3

On April 12, 1955, a male child aged 1 year and 7 months was walking on the lawn at about 4.45 p.m. with a small garden stake in his mouth when he stumbled and fell. The blunt and ragged end of the stake was driven against the right side of the soft palate and adjacent faucial pillar, causing profuse bleeding, which stopped spontaneously. He was seen by his doctor shortly afterwards and was given prophylactic injections of penicillin and antitetanus serum.

During the night he appeared to be feverish and vomited twice. The following day he was fretful, irritable, and listless, but not drowsy. At 3.45 p.m. (23 hours after the accident) he developed twitching of the left side of the face lasting two or three minutes. In the evening his parents observed that he was unable to sustain weight on his left leg. He was sedated and put to bed. At 2.45 a.m. he woke up crying, and on attending to him his mother noticed a further twitching of the left side of the face for a brief period.

Next morning he was cheerful and alert, but his parents observed that he was moving neither his left arm nor his left leg. He was immediately sent to the Royal Hospital for Sick Children, Edinburgh.

On examination he was found to have a flaccid paralysis of the left arm and leg and a weakness of the left side of the face. The abdominal reflexes on the left side were absent; the left plantar response was extensor. He was irritable and uncooperative. His pupils were equal and reacted briskly to stimulation. There was no papilloedema; the retinal vessels were normal.

Inspection of the throat revealed an adherent exudate concealing a wound extending from just to the right of the uvula to the pillars of the fauces on the same side. There was no detectable difference between the vigour of the carotid pulsation on the two sides. He was afebrile.

Lumbar puncture produced clear cerebrospinal fluid under normal pressure. It contained 3 lymphocytes per c.mm. and 16 mg. of protein, 65 mg. of sugar, and 720 mg. of chlorides per 100 ml. His total white cell count was 6,600 per c.mm. and the erythrocyte sedimentation rate was 10 mm. in the first hour.

Penicillin was continued for ten days. He rapidly regained power in the limbs and was discharged with appropriate splintage three weeks after admission.

He was last seen four months after the injury, when he was able to walk unaided but had a slight tendency to rotate his affected lower limb internally. He was reluctant to use his left upper limb, but he had good power in all the muscle groups and there was only a minimal adduction contracture of the thumb. There was no obvious sensory disturbance. He was cheerful and his vocabulary was excellent for his age. He had had no further convulsions, and an electroencephalogram performed a month previously was normal.

Treatment

In all three cases the pattern of events was essentially similar. An injury in the region of the faucial pillars caused by a pointed object was followed after an interval of varying duration by a contralateral hemiplegia and other neurological manifestations. These sequelae were ascribed to cerebral ischaemia following traumatic thrombosis of the internal carotid artery on the side of the injury.

Anticoagulants.—Anticoagulant drugs were administered only in Case 2, in which symptoms were rapidly progressive. In Case 1 the patient was seen too late for similar treatment to have been of value, and in Case 3 a "waiting" policy was adopted. This patient was thought to be a little better by the time he reached hospital, so it was decided to withhold anticoagulants and to give them only if there was any sign of deterioration. The unfortunate progression of events in the case described by Caldwell (1936), in which thrombosis extended from the neck into the middle cerebral artery, suggests that the use of these drugs may well be advisable. Treatment should be instituted if thrombosis of the carotid artery is suspected following an injury of the type described.

Cerebral Vasodilators.—These may be given in conjunction with anticoagulants, although there is no proof that the cerebral blood flow will be significantly improved (Scheinberg and Jayne, 1952).

Sympathetic Block and Sympathectomy.—These may be undertaken if found to be of value in other instances of

carotid occlusion. The operation may be performed on the side of the lesion or on the normal side in an effort to improve the collateral circulation. The results of such procedures would always be difficult to assess, as these cases tend to improve spontaneously.

Other Measures.—As the extent of rehabilitation is greatly influenced by the intensity of the orthopaedic care and the physiotherapy and speech therapy which the patient receives, these measures should be instituted as soon as possible.

Discussion

The proximity of the internal carotid artery to the tonsillar bed is well known. Intimal tears of this vessel without penetration of its wall have been described by Verneuil and Greco (quoted by Schneider and Lemmen, 1952) and Caldwell and Hadden (1948). It is postulated that such a lesion occurred in our cases and served as a nidus for thrombus formation, which progressed until occlusion of the lumen was complete.

The manifestations that may follow occlusion of the internal carotid artery, whether arising spontaneously or as a result of surgical ligation, are well documented. These include contralateral hemiplegia, homonymous hemianopia, and aphasia if the dominant cerebral hemisphere is affected. These signs are due to ischaemia in the region of the brain supplied essentially by the middle cerebral artery. Homolateral blindness occasionally results from ischaemia in the field of the ophthalmic artery.

In Case 1 the hemiplegia was associated with impairment of sensation on the same side. In Case 2 it was associated with aphasia, right inferior homonymous quadrantic hemianopia, and transient papilloedema. Carotid artery occlusion appeared to be the cause of these symptoms. (The papilloedema was probably related to temporary brain swelling associated with ischaemic softening.) In Case 3 it is uncertain whether the cerebral lesion was caused by venous or by arterial occlusion. This case differed from the previous two cases in respect of the occurrence of premonitory unilateral twitching, a phenomenon indicative of cortical dysfunction, 12 hours before the onset of paralysis. This symptom suggests that the child may have suffered a cortical venous thrombosis. It is possible that a traumatic venous thrombosis had extended from the site of injury along the pharyngeal plexus of veins into the deep pterygoid plexus and through the foramina in the base of the middle cranial fossa and into the cortical venous system.

Localized epilepsy may, however, be provoked by arterial ischaemia. It remains doubtful whether arterial or venous occlusion was responsible for the cerebral lesion in Case 3. The former appears the more likely.

The diagnosis of internal carotid artery occlusion can be established definitely only by demonstrating an interruption of the blood flow through the vessel by means of carotid angiography. In our cases this method of investigation was not employed, as it was thought that it would give no clinical benefit.

Since the brain is abundantly supplied with blood by four great vessels and there are usually extensive anastomoses between them, the development of ischaemic changes in certain areas following the occlusion of one major vessel requires explanation. Schorstein (1940) has reviewed the more important possibilities that have been advanced to explain the occurrence of cerebral damage following carotid artery occlusion by ligation. Some of these are relevant to our cases and merit a brief consideration.

Anatomical deficiencies of the circle of Willis were probably not present in our patients, as Schorstein (1940) and Dorrance (1934) have indicated that such abnormalities are distinctly uncommon; moreover, when they occur there is usually ample compensation by other vessels.

Spasm of the arteries at the base of the brain, extending there from the injured portion of the carotid artery, is

unlikely to have been a significant factor, since it fails to explain the interval of normality which preceded the onset of symptoms in all our cases.

Embolism from the carotid artery into the middle cerebral artery or direct extension of the thrombosis to that vessel are two possibilities which account for this interval satisfactorily. The former is unlikely, since Schorstein (1940) has pointed out that such emboli are a rare finding at necropsy of patients who develop hemiplegia following ligation of the carotid artery. The latter is supported by the necropsy findings in the case recorded by Caldwell (1936). This case is the only one resembling ours which we encountered in the literature, and many of the details are strikingly similar to those reported above.

Caldwell's patient, who was 16 years old, received a penetrating wound of the soft palate following a fall on to a hedge. The immediate result was local pain and swelling but no loss of consciousness. Four hours after his injury he was seen by his doctor, who recommended hot gargles. A few hours later he became drowsy and could not talk, though he apparently understood what was said to him. He was admitted to hospital in this state 10 hours after the injury. The abnormal findings on examination were a right hemiplegia and a diminished corneal reflex on that side. There were twitching movements of both lips on the right side of the face. A jagged wound 2 cm. in diameter was seen in the roof of the mouth at the junction of the hard and soft palate slightly to the left of the midline. The findings on lumbar puncture were normal. While in hospital his stupor increased, and he died six days after admission. At necropsy the left internal carotid artery contained a well-formed thrombus which extended from the cervical portion of the artery into the cranial portion and on into the middle cerebral and Sylvian arteries. The left cerebral hemisphere was softer than the right, and on section a haemorrhagic infarct was found in the central portion.

The part played by failure of the systemic circulation in our cases is difficult to assess. Irreversible anoxic changes in the brain may result from a fall in the systemic blood pressure due to shock or as a result of an increase in pressure in the carotid sinus (due to distal occlusion of the internal carotid artery) acting reflexly (Rogers, 1947). In our cases there was no evidence of serious impairment of the general circulation, except in the initial episode in Case 1, which may well have been of this nature.

A word or two may be added on the subject of prevention. Similar catastrophes could be largely avoided by the application of simple common-sense measures. The rarity of the clinical picture described should not detract from the importance of enforcing these measures. Whenever possible, pointed objects should be kept out of the reach of young children, and they should be strongly discouraged from holding any such object in the mouth. In the case of older children the dangers of holding pointed objects in the mouth should be explained. Finally, it would seem a wise precaution for practitioners to keep under close surveillance for a period of two or three days any child who suffers a potentially penetrating injury in the region of the soft palate. In this way neurological symptoms will be detected early and treatment started promptly.

Summary

Three cases of penetrating injury in the region of the soft palate followed by hemiplegia believed to be due to traumatic thrombosis of the internal carotid artery are described.

The possible pathogenesis of the symptoms is discussed.

Recommendations regarding treatment and prevention are made.

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COAGULASE-POSITIVE STAPHYLOCOCCI

A SERIAL SURVEY FOR NASAL CARRIERS DURING THE FIRST SIX MONTHS OF NURSING TRAINING

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A feature of the epidemiology of staphylococcal disease acquired in hospital which is rather puzzling is the relative infrequency of outbreaks of manifest disease in wards staffed by nurses who show a high nasal carrier rate for coagulase-positive staphylococci. This puzzle prompts the question, Are there *within* the group of coagulase-positive staphylococci a few types which are responsible for outbreaks of disease? If this is so it is obviously desirable in the interests of prevention that we should be able to identify such types. Type differentiation of the genus *Staphylococcus* by both phage and serological methods at present lacks precision owing to our lack of knowledge of the fine antigenic structure of the organism. Nevertheless serological typing of staphylococci has proved to be of value in the study of epidemiological problems of staphylococcal disease (Hobbs, 1948; Oeding, 1954; Brodie *et al.*, 1955, 1956). Serological typing has the great advantage over phage typing in that it takes less time and is simpler to carry out.

The objects of the present investigation were to determine, by serial observations, the influence of nursing training on the nasal carrier rate of coagulase-positive staphylococci, to ascertain the prevalence of antibiotic resistant strains in nurses, and to study the serological types which might have colonized the hospital.

The analysis is based on a six-months serial survey for staphylococcal carriage of a group of 30 student nurses from the start of their training. The examinations of the nasal staphylococci of each nurse were made at weekly intervals. The survey began in October, 1954, and ended in March, 1955, during which time the student nurses spent three months in the preliminary training school of a general hospital followed by three months on duty in the wards of the hospital.