Arteries that enter bony tunnels tend to be protected from trauma until the bone is fractured, in which case the fixation of the artery in its foramen together with the propinquity of mobile bone is hazardous. The vertebral arteries, unique in passing through a series of bony rings, appear particularly vulnerable in this respect. Reports of vertebral artery thrombosis following trauma are, however, few. The following report is of a case in which the site of vascular damage in the lower part of the neck was verified at autopsy and found paradoxically to be contralateral to the main vertebral fracture.

CASE REPORT

A 42-year-old man, previously healthy, fell 10 feet to the ground from a tree. He was unconscious for 2 minutes and afterwards was unable to move his legs. At a nearby hospital he was put into a Glissou sling with 5 lbs. of traction in mild hyperextension. He was admitted to the Montreal Neurological Institute 6 hours after his fall.

Examination. On admission his pulse rate was 80 per minute, respirations 24 per minute, blood pressure 86/60 and temperature 101°F. Bruises were present on the left side of his face. There was tenderness over the spinous processes of the lower cervical vertebrae. His abdomen was distended. A Foley catheter was in place. He seemed slightly drowsy but was fully cooperative. Cranial-nerve functions were normal. There was weakness of the left triceps and flexors of the left hand. The left triceps jerk was absent. Perception of pin prick was impaired over the ulnar side of the left arm, forearm, and hand. All sensation was lost below the level of the 2nd rib on the left and the 3rd rib on the right. There was flaccid paralysis of all muscles of the legs and abdomen.

Soon after the initial examination, the patient complained of sudden intense vertigo and vomited. He now displayed horizontal nystagmus, accentuated on right lateral gaze.

Roentgenograms showed a compression fracture of C7 with splaying out of the vertebral body and increase in the interpeduncular distance (Figs. 1 and 2). The C6-C7 disc had ruptured the superior surface of the body of C7; the posterior two-thirds of the superior surface of C7 were displaced downwards and posteriorly, so that a portion projected 4–5 mm. into the spinal canal in the midline. A fracture of the right lamina of C7 was also visible. Soft-tissue swelling anterior to C7 was prominent.

Course. Cone-Barton tongs were applied with 20 lbs. of traction. Lumbar puncture yielded bloody fluid at 210 mm pressure; manometry indicated a complete block. On the following morning the patient’s left-hand grip was weaker, and he complained of difficulty in breathing. Treatment included oxygen, a Levine tube,
Prostigmin, and Chloromycetin. In the evening his respirations were shallow; he showed cyanosis. While preparations for a tracheostomy were being made, his breathing stopped, and 3 minutes later cardiac arrest occurred. The time of death was 29 hours after his injury.

Autopsy was limited to the head and back. Bruises were present over the left eye and on the left cheek. The scalp, skull, and cranial dura mater were unremarkable. During removal of the brain, the lower part of the brain stem was found to be displaced to the right by the enlarged left cerebellar hemisphere. The brain weighed 1460 gm. Gross abnormality was limited to the cerebellum, the left hemisphere of which was soft and swollen over its entire inferior surface (Fig. 3). The left vertebral artery was distended with firm thrombus almost up to the origin of the anterior spinal artery. The thrombus extended into the left posterior inferior cerebellar artery, which on this side was the sole major vessel supplying the inferior surface of the cerebellum. The vertebral arteries were equal in size.

The left vertebral artery in the neck did not pass through any part of the 7th cervical vertebra. Proximal to its entry into the transverse foramen of C6 the artery was thrombosed and its wall had a pale greenish tint. The left lateral mass of C7 was intact. The fractures corresponded to their roentgen-ray description: collapse of the body of C7 with protrusion of the posterior superior surface into the spinal canal slightly to right of midline; fracture of the right lamina, and in addition, a fracture through the right pedicle. The cord was flattened and bruised in its 1st and 2nd thoracic segments. On section the whole thickness of the cord at T1 was pulped.

Histological Examination. Step serial sections of medulla and lower pons showed an early infarct extending from the sensory decussation to just below the acoustic striae. This infarct, which was limited to the left lateral medullary region, involved the spinocerebellar tracts, the spinal tract and nucleus of the trigeminal nerve, the subtrigeminal nucleus, the lateral cuneate nucleus, and (rostrally) the medial vestibular nucleus and the nucleus of the tractus solitarius. The inferior folia of the left cerebellar hemisphere were freshly infarcted; there was early polymorphonuclear infiltration of their meninges.

The 1st and 2nd thoracic segments of the spinal cord were lacerated and necrotic. The anterior spinal artery was uninjured. No abnormality was seen in segments above the 8th cervical or below the 3rd thoracic.

The wall of the left vertebral artery at the level of the C6-C7 interspace had been disrupted by hemorrhage. The muscularis was destroyed in much of its circumference (Figs. 4 and 5). The lumen was filled with ante-mortem thrombus. Numerous polymorphonuclear leucocytes had invaded the adventitia. Immediately proximal and distal to this level the artery was much less damaged, although blood had dissected beneath the intima, and a small intimal cushion of atheroma was present.

DISCUSSION

The closest parallel to the present case is Murray's account of a 16-year-old boy whose right arm was drawn into a band saw. It was later surmised that his head had been forced strongly to the left. The arm had to be amputated. Several hours later the patient became comatose and Babinski's signs developed. Eventually he had respiratory arrest. At autopsy there was massive softening of the right lobe of the cerebellum, with a small hemorrhagic area in the left lobe. The right vertebral artery was thrombosed from close to the basilar artery down to its entrance through the dura mater. The artery in the neck could not be examined. No dislocation or fracture was seen in the cervical spine.
It is uncertain whether actual thrombosis of a vertebral artery contributed to the clinical picture in Suechting and French's case of a 20-year-old man who fractured the right lateral mass of C5 in a diving accident and dislocated C5 on C6. Four days after being put in traction there developed sudden bilateral facial pain and numbness of the right side of the body. These symptoms were relieved by cessation of traction; traction was resumed a few hours later. After 15 days the patient had signs of right lateral medullary damage. Removal of traction was again followed by improvement. The reversibility of signs and their different pattern on the two occasions suggest that insufficiency of the vertebral artery had occurred without thrombosis. Such a mechanism has been invoked by Schneider and Crosby to explain the clinical picture in 2 cases of cervical dislocation in which the vertebral contribution to the anterior spinal artery was compromised. Ford's case of paroxysmal syncope and vertigo in a boy with deficient odontoid may also be cited. The contribution of arterial spasm in such cases has not yet been elucidated.

Autopsy findings have been published in 4 cases in which thrombosis followed manipulative rotation of the head. Pratt-Thomas and Berger in 1947 presented 2 cases of patients, aged 32 and 35, who had lost consciousness during chiropractic treatment. Thrombus was found in the basilar artery and in one posterior inferior cerebellar artery in both cases; thrombus apparently was absent in the vertebral arteries below their junction. The arteries in the neck were not examined. In 1956 Ford and Clark reported the case of a 37-year-old man who died 60 hours after his wife...
gave his neck a therapeutic twist. His immediate symptoms were vertigo, tinnitus, and right hemianopia. Thrombi were found in the left posterior cerebral, the basilar, and the left posterior inferior cerebellar artery. The arteries in the neck could not be examined. Holzer's patient, aged 38, had his head maintained in right rotation during a left stellate ganglionectomy. Post-operatively he complained of left facial pain before lapsing into coma. The basilar artery was thrombosed. The lower end of the clot began in the left vertebral artery at its entrance through the dura mater, while the arteries in the neck were healthy and patent.

Similar cases which followed manipulation but in which the patient survived were reported by Kunkle et al., Schwarz et al., Ford and Clark, and Green and Joynt. Only 2 of these 5 patients were aged over 33. The first case of Green and Joynt must be accepted with reservation in view of her almost complete recovery and the concomitant diagnosis of multiple sclerosis. Her signs (weakness of the left limbs, left Babinski's sign, and loss of all sensation on the left in body and face) were not particularly those of a medullary lesion, whereas the other patients, with some variation, all had a lateral medullary syndrome.

The primary site of thrombosis in these cases of rotation of the head presumably is in the region of the atlas and the atlanto-occipital membrane. In this region a vertebral artery normally can be narrowed or occluded when the head is rotated sufficiently far to the opposite side. This has been demonstrated by the perfusion studies of de Kleyn and Versteegh and of Holzer and by the postmortem angiography of Tatlow and Bammer. Toole and Tucker obtained different results from perfusion in that they were unable to predict from the direction of head turning which vertebral artery would be occluded. These results probably were related to the presence of widespread atheroma and cervical osteophytes in some cadavers of their series. Narrowing and displacement of vertebral arteries by osteophytes have been demonstrated angiographically by Schechan et al.

Yates dissected the necks of 60 infants dying in the perinatal period and in 24 found hemorrhages into the adventitia of one or both vertebral arteries. Often the hemorrhage was large enough to narrow the lumen. In 1 infant dying 12 days after birth, vertebral thrombosis was present. On histological grounds this was judged to be 3 to 4 days old; thus it could not confidently be ascribed to birth trauma.

The results of unilateral vertebral thrombosis are as variable as those of carotid occlusion. Perhaps this is why reports of traumatic cases are so few. Surgical ligation of one vertebral artery occasionally has been performed with impunity, a fact that has been reviewed by French and Haines who reported a case in which vertebral ligation was followed by basilar thrombosis and death. Certainly propagation of thrombus is of ominous importance. Such propagation has been present in many but not all of the fatal cases of traumatic carotid thrombosis. Anticoagulants did not prevent it in Murray's first (carotid) case.

The state of health of collateral vessels is also important in the outcome. Atheroma of the carotid and atheroma of the vertebral artery occur in frequent association. Occlusion of one vertebral artery may lead to insufficiency in the territory of the basilar or carotid artery. Most cases of traumatic vertebral thrombosis have, however, occurred in young adults whose vessels in all probability were patent.

The anatomical distribution of vessels may vary decisively. The blood supply of rostral paramedian medullary structures may come directly from the vertebral artery or from the anterior spinal or from the basilar. The lateral medulla may be supplied by a branch of the basilar, by the posterior inferior cerebellar artery, or by the vertebral. More important, one vertebral artery may be hypoplastic. Stopford, in a study of 150 brains, found 22 in which one vertebral was half the size (or less) of the other. The anterior inferior (middle) cerebellar artery arises normally as a discrete branch of the basilar. This artery was absent bilaterally in 1 of Stopford's 150 brains and absent unilaterally in 2 more. Such an anomaly, by which the territory of the middle cerebellar artery becomes dependent on the posterior cerebellar artery, was found in the present case.

Damage to the intima is probably necessary for traumatic thrombosis. Carotid arteries have proved more accessible to pathologists than vertebral arteries. The case of closed carotid thrombosis of Clarke et al. showed a small intimal tear, while in the cases of Northcroft and Morgan and of Murray traumatic dissecting aneurysms were present. Caldwell and Hadden found fragmentation of the wall of the vessel with areas of hemorrhage and infiltration by many polymorphonuclear leucocytes. Atheroma, when present, was not considered to be a significant factor. Cystic medial necrosis was not found in these cases or the present case, although it can occur in a carotid or in a vertebral artery.

In the present case the wall of the vertebral artery was necrotic. Blood had dissected subintimally for a few millimeters proximally and distally. The fractured pedicle paradoxically was contralateral to the thrombosed artery. Between its subclavian origin and the transverse foramen of C6 the vertebral artery normally is bound down tightly by fascia. From the left-sided distribution
of bruises on the patient's face, his neck must have been bent violently to the right, causing an abrupt stretching of the left vertebral artery. We envisage that thrombosis followed the intimal damage, and propagation of thrombus ensued until the posterior inferior cerebellar artery was occluded. This artery was the sole major vessel supplying the inferior surface of the cerebellum on the left; therefore, while the lateral medullary infarct was small, the cerebellar infarct was massive. The swelling of the cerebellum was sufficient to displace the brain stem and cause the fatal outcome in this case.

SUMMARY

An autopsied case of traumatic vertebral artery thrombosis is presented, associated with a fracture of C7. The fracture was more extensive on the side opposite the thrombosis. Death resulted from swelling of the infarcted cerebellum. The literature of traumatic vertebral thrombosis is reviewed, and factors influencing the outcome are discussed.

I wish to express my thanks for the interest and advice of Dr. Gilles Bertrand, Dr. D. L. McRae and Dr. Gordon Mathieson.

REFERENCES