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VERTEBRAL ARTERY INSUFFICIENCY SECONDARY TO TRAUMA AND OSTEOARTHRITIS OF THE CERVICAL SPINE‡

Obstruction of the vertebral arteries can lead to sudden death, extensive spinal cord destruction, insufficiency of the brain stem, impaired cerebellar function, damage to the occipital cortex, and may well be responsible for certain forms of cerebral palsy.

We have recently treated a case of post-traumatic vertebral artery insufficiency by debridement of an osteophyte obstructing the intraspinal portion of the vessel. Reviewing this problem we have found that relatively little attention has been focused on osteophytes obstructing the vertebral arteries and that these vessels are frequently not studied in the management of both traumatic and degenerative diseases of the cervical spine. This is in contrast to those osteophytic growths which cause spinal nerve root compression. Greater consideration for preservation of vertebral artery function may improve the prognosis of certain cervical spine fractures, cervical osteoarthritis (especially in patients with arteriosclerosis of large extracranial vessels), and congenital anomalies of the neck. It is the purpose of this report to emphasize the need for the consideration of vertebral artery circulation in cervical spine lesions, and to present some of the problems encountered in the diagnosis and treatment of such a case.

CASE HISTORY

The patient was a 51 year-old male in normal good health until he was involved in an automobile accident on August 24, 1962. On admission to the local hospital he was found to have a partial quadriplegia with a "pin-prick" level to C6, intact proprioception in all four extremities, and more severe motor dysfunction in the upper than in the lower extremities. X-rays showed a 3 mm. anterior subluxation of C4 on C5 with minimal

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osteoarthritic changes of the C5-C6 interspace. No other injuries or abnormalities were noted. The past history, the review of systems, and all laboratory studies including a lumbar puncture were within normal limits.

Clinical course

The patient was placed in skeletal traction with Crutchfield tongs and his C4-5 dislocation reduced without difficulty. Within 24 hours his neurologic status started to improve. On October 1, 1962 he was transferred to the West Haven Veterans Administration Hospital for rehabilitation. Examination at that time with the patient in skeletal head and neck traction showed an equivocal horizontal nystagmus on right lateral gaze, but no isolated cranial nerve deficits. Motor function was very limited. Bilateral weak hand grasp was present and the left biceps and deltoid showed partial function. The left leg and right thigh were quite weak. The right leg had normal strength. Vibratory sense was absent below the neck and pain and cold sensibility were decreased over the right body below C6. Position and light touch sensation were intact.

Skeletal traction was maintained until October 22, 1962, when rehabilitation was begun with the patient in a cervical collar. A satisfactory return of motor function occurred and in two months the patient was able to ascend stairs. Further therapy resulted in continuous improvement until July 1963, at which time there was an increase in extremity spasticity and the patient began to complain of "dizzy spells." He fainted while erect with his head in slight extension. Repeat neurologic examination at this time showed intact sensory function throughout. There was residual quadriceps and gastrocnemius weakness, more on the left than right, and 4+ hyperactive reflexes with sustained ankle clonus and extensor plantar reflexes. His neurologic status was considered improved and the abnormalities were attributed to his known history of spinal cord contusion. However, because of the patient's syncopal episodes and sudden deterioration in motor performance further studies were carried out.

Routine bending and extension films of the cervical spine showed residual (recurrent) subluxation of C4 on C5, minimal motion at the C4-C5 level and some degenerative changes of the C5-C6 interspace (Figs. 1A and 1B). A cervical myelogram with cineroentgenography revealed normal flow of dye without cord compression. An EEG in a routine supine position was normal, but an EEG with the patient in a semi-erect position (70° tilt) and the head in hyperextension produced a tracing compatible with decreased cerebral blood flow (Figs. 2A and B). There were no significant blood pressure changes during this procedure.

On the basis of the above findings it was decided to perform an arteriogram. Dye was injected in the ascending aorta via percutaneous retrograde femoral artery puncture. Serial injections were made to show the patient's cervical vessels with the neck in neutral and hyperextension. This study showed compression of the left vertebral artery by an osteophyte at C5-C6 (one level below the dislocation), with almost complete obstruction of the vessel on neck extension. The right vertebral artery was very narrowed from its origin to the level of the fracture-dislocation (C4-C5) without any filling beyond that point. Although congenital narrowing or absence of the right vertebral was considered, we postulated a post-traumatic thrombosis because of the appearance of the right artery and the relatively normal size of the left (Fig. 3).

Treatment

After the diagnosis of vertebral insufficiency had been made and the exact site of partial occlusion established, surgical treatment was carried out on October 8, 1963, through an anterior approach previously described.[1] By meticulous subperiosteal reflection of the longus colli muscle the left lateral masses from C4 to C7 were exposed. It was necessary to remove the entire anterior portion of the transverse process of C6 to visualize the point of vertebral artery obstruction. The artery was compressed by osteophytes that seemed to originate from the region of the joint of Luschka. These osteophytes were removed with simple curettes and small bone rongeurs. (Fig. 4). After the arterial debridement had been completed a two level anterior interbody fusion was performed (C4-C5 and C5-C6).

Postoperative course

There were no operative complications. The patient's post-operative course was uneventful. He remained in bed for approximately six days. During this time sand bags along both sides of the neck were used for head immobilization. The sutures were removed on the sixth postoperative day and the patient ambulated with his neck in a simple felt and plaster collar.

During the six months following surgery the patient's motor performance improved, his upper extremities and hands became stronger, he reported a general sense of well-being and had no further syncopal episodes.

Postoperative studies have shown that his spine is fused (C4-5-6) and his EEG has been very interesting as well as gratifying to observe. It was possible to hyperextend the patient's neck approximately 25-30 degrees beyond the pre-operative range and to mantain it in this position for indefinite periods of time without producing EEG changes or syncope. It was not deemed justifiable to repeat the arteriograms.

DISCUSSION

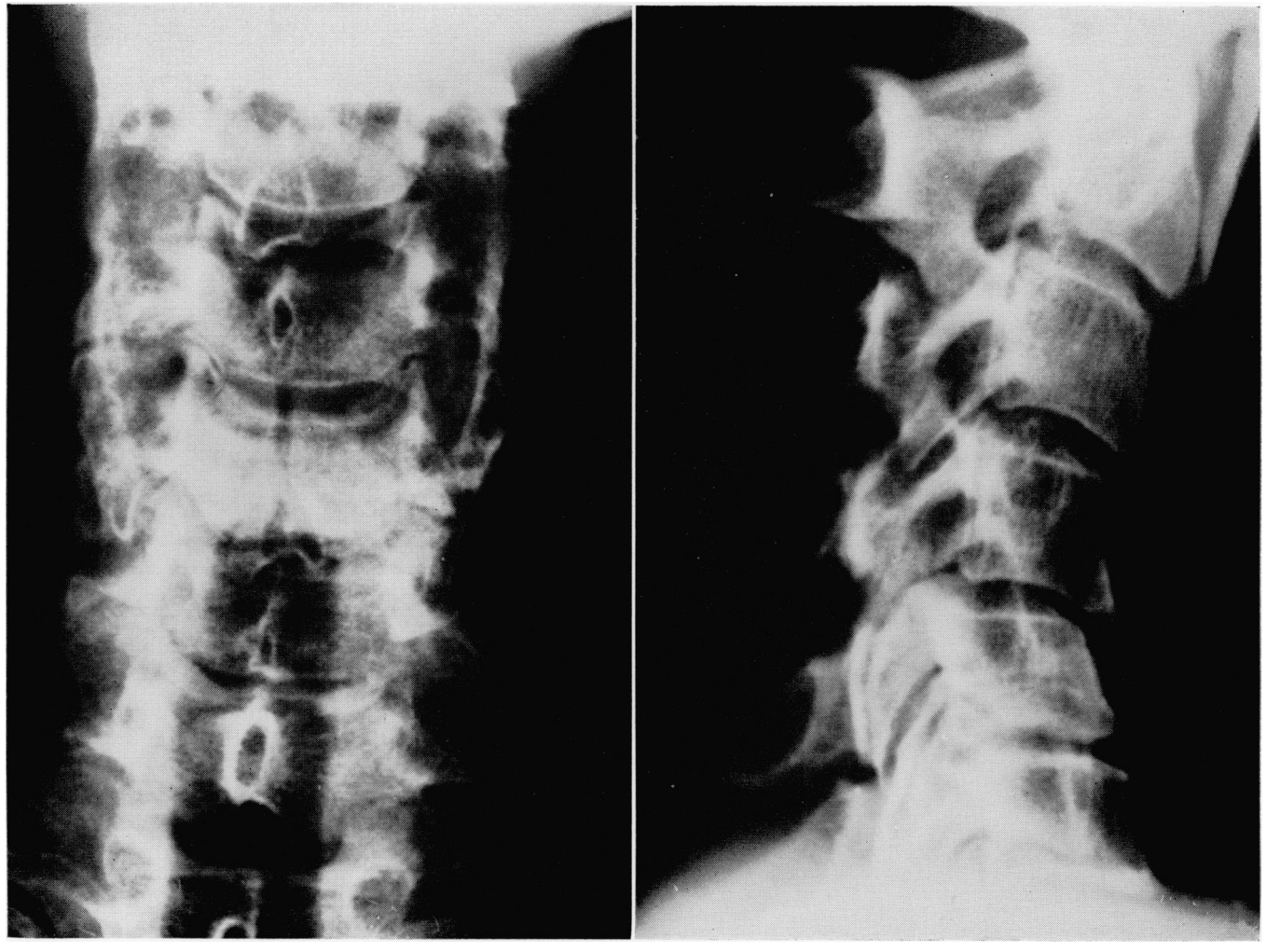
The vertebral arteries have received only minimal attention in the treatment of cervical spine disease and trauma. This case has shown some of the serious complications associated with injury and obstruction of these arteries. It is also an illustration of the studies leading to a definite diagnosis and of the surgical treatment undertaken to relieve the arterial extraluminal obstruction. It serves to emphasize the role of the vertebral arteries in the explanation of some of the unusual symptoms associated with minor and major cervical problems.

The signs and symptoms of vertebral artery insufficiency are clinically manifested by neurologic sequellae of reduced basilar artery blood flow. Since the initial modern description of basilar artery insufficiency by Kubik and Adams in 1946, the resulting syndromes have received widespread attention in the literature. Among recent major reviews are those by Sheehan, Bauer, and Meyer[3] and Williams.[8] Anatomically, involvement of the cervical cord,[4,5] medulla, pons, cerebellum and diencephalon as well as the posterior cerebral cortical regions result in numerous varying clinical syndromes reviewed thoroughly by Duffy.[6] Most commonly the signs and symptoms are of disturbances of coordination and equilibrium, visual complaints, headache, vertigo, nausea, vomiting, tinnitus, nystagmus, unilateral deafness, dysphagia, dysarthria and ataxia. If loss of consciousness is rapid, as in our patient, the more localizing signs of "posterior" circulatory insufficiency may not be apparent. Interestingly enough, some of these symptoms in various gradations are noted in relatively minor neck injuries or diseases.

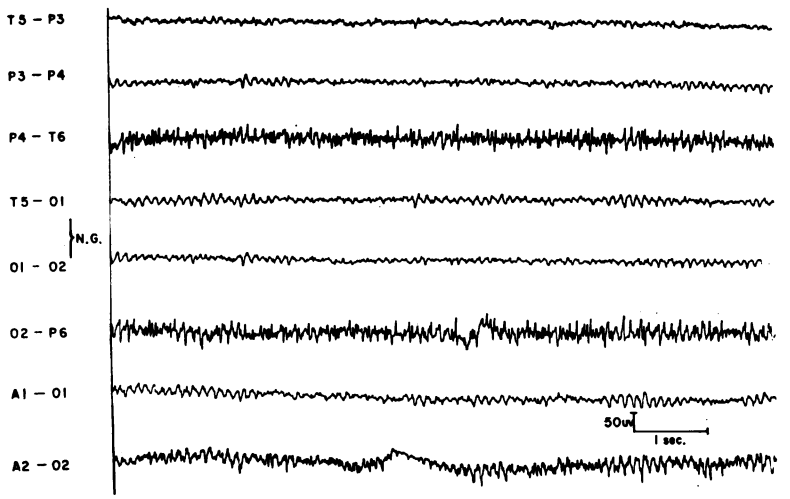
Clinically the common cause of the vertebral-basilar syndrome is degenerative vascular involvement of the basilar artery itself, but any disease process involving either the extracranial or intracranial portion of these vessels can cause identical neurologic complications.

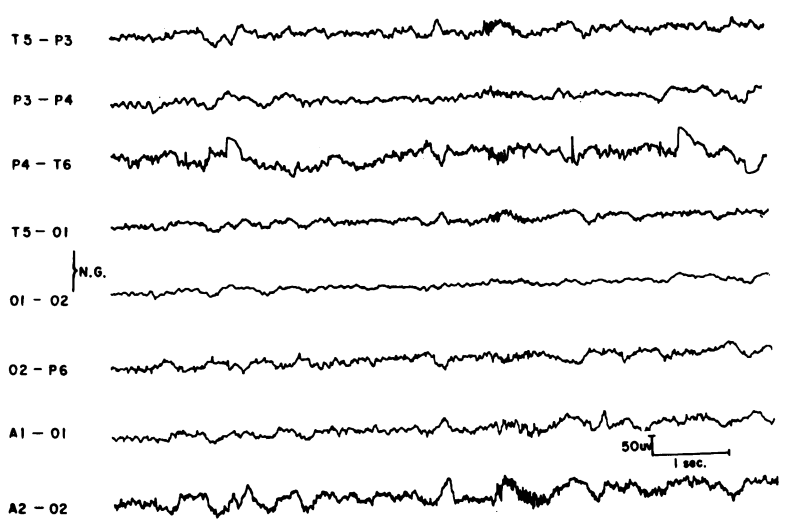
A list of such causes includes:

1. Arteriosclerosis[3,6–14]
2. Osteoarthritis of the cervical spine[2,7,15–17]
3. Trauma (severe fractures and dislocations, but also lesser injuries such as "whiplash" of the neck may account for vertebral artery injury)[8,9,18–27]
   a. Contusion (with such complicating factors as thrombosis, extension of thrombosis into the cerebellar and/or basilar systems, intra- or extra-mural hematomas, arteriospasm, etc.)



FIGS. 1A, 1B. These roentgenograms show the residual subluxation of C4 C5 and the degenerative changes at C5 C6.





FIGS. 2A, 2B. The first sample tracing displays the patient's resting EEG pattern when supine. The second sample shows the same montage within 10 seconds after tilting to 70°; no significant change in blood pressure was observed. The total disappearance of normal occipital alpha rhythm and substitution of medium to high voltage slow waves can be readily appreciated.

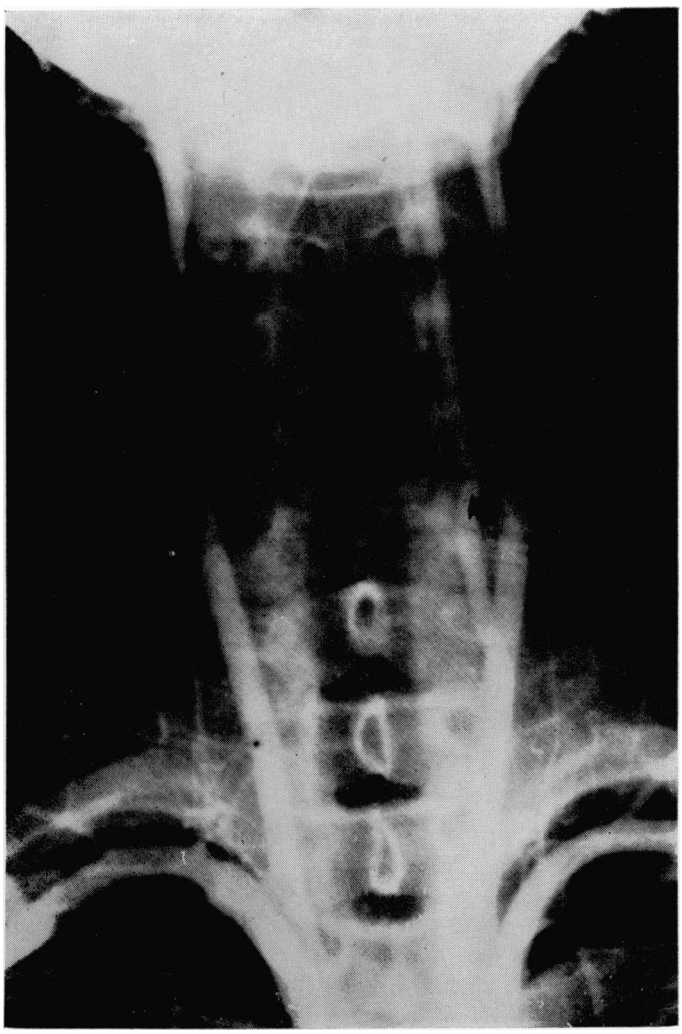


FIG. 3. The arteriogram shows the compression of the left vertebral artery at the C5 C6 level and the narrowed right vertebral terminating at the level of the dislocation, C4 C5.

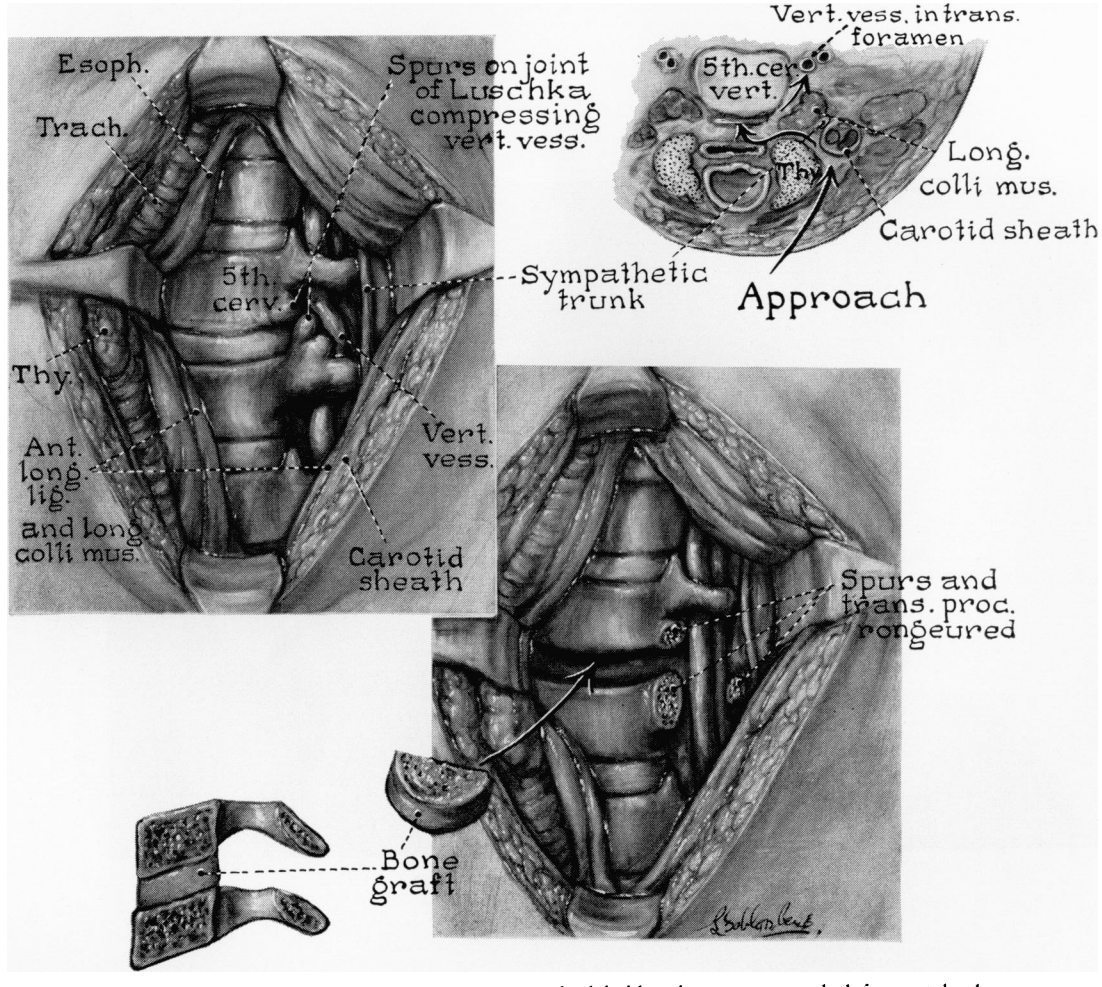


FIG. 4. The surgical approach used to expose and debride the compressed left vertebral artery.

   b. Direct compression, distortion or stretching by abnormal vertebral body position
   c. Laceration
   d. Avulsion
4. Birth trauma (may lead to perinatal death or severe cerebral palsy)[28]
5. Position of the head (not in all individuals)[29, 30]
6. Muscle spasm in the suboccipital regions causing compression at the C1 level[9]
7. Muscle spasm in the scalenus anticus region causing compression proximal to the transverse process of C6[31, 32]
8. Spontaneous subluxation (or chronic instability) of the facet joints[33]
9. Soft tissue bands[32, 34]
10. Tortuosity of the arteries (congenital or arteriosclerotic)[34]
11. Kinks of the artery[34]
12. Aneurysms of the arteries[11, 35, 36]
13. Congenital anomalies of the cervical spine (syncope)[37–39]
14. Tumors[10]

In a discussion of vertebral artery insufficiency it is important to note the free connection of the vertebrals with each other through their spinal branches and of the vertebrals with the carotids through the circle of Willis.[40–43] In a normal individual this is a very flexible system of collateral and compensatory blood flow. It has been shown that the normal direction of blood flow through the vertebrals can be completely reversed. This means that a simple compression of one vertebral within its spinal portion can go undetected because the other vertebral and the carotids will increase their flow to compensate for the incompetent vertebral. Unless a complicating thrombosis occurs, blood will also flow down from the basilar artery to the level of obstruction. Experiments and observations during neck surgery have confirmed this phenomenon and in some cases the entire upper extremity depends on the reversed vertebral artery flow for its blood supply. This happens in cases of arteriosclerotic obstruction of the subclavian artery proximal to the origin of the vertebral and is known as the "subclavian steal" syndrome.[44–46]

In the normal individual this interconnected carotid-basilar-vertebral system can adjust to many injuries. In some cases of arteriosclerosis where one of the major vessels has already been compromised, the patient may be completely asymptomatic until obstruction of one of the other three main cervical arteries produces neurological damage and symptoms. Thus in our case a single osteoarthritic spur caused severe cerebrovascular insufficiency

because the other vertebral had been previously injured and thrombosed (injury rather than congenital narrowing or absence of the right vertebral was postulated on the basis of the radiographic appearance of the vessel and its termination in the region of the fracture dislocation). In a similar manner it is obvious that arteriosclerotic obstruction of the carotids is likely to be more symptomatic in an individual with osteoarthritis of the cervical spine and partial or complete obstruction of the vertebrals. In addition to arteriosclerosis, such entities as trauma, osteoarthritis, congenital anomalies of the cervical spine and variation in the cervical arteries may be aggravating factors in the evolution of extra-cranial cerebrovascular syndromes. These factors are all related and should all be considered when any one of them is noted. Cervical spine trauma should not be evaluated in terms of skeletal and nerve injury alone—the status of the vertebrals and the carotids must also be considered. Osteoarthritis of the cervical spine must be evaluated with special attention to the status of the neck vessels. Conversely the symptoms of carotid or vertebral arteriosclerosis are affected by the patency of the bony vertebral canals.

Perhaps the most important thing learned in this case lies in the effect upon vertebral arteries of treatment of cervical spine fractures and injuries. The preservation and protection of the vertebral arteries depends on anatomical alignment and immobilization by spine fusion. An absolute statement cannot be made, but it is possible to speculate that in our case the right vertebral artery would not have thrombosed and the left compressing osteophyte would not have developed if an anatomical reduction and fusian had been accomplished.

The other area where the vertebral arteries may play an underestimated role is in cerebral palsy. Yates[36] has found that in the necks of 60 infants dying in the perinatal period there was evidence of injury to the vertebral arteries in 24 (40%). We cannot help but wonder about this type of injury as a contributing factor in cerebral palsy. It is an area which deserves further study, not only by arteriography, but by careful anatomical dissection.

SUMMARY

A case of documented vertebral artery insufficiency secondary to cervical spine dislocation has been presented along with its surgical treatment at the Yale-New Haven Medical Center.

The importance and inter-dependence of the carotids and the vertebrals with cervical trauma, osteoarthritis, congenital anomalies, cerebrovascular insufficiency and cerebral palsy has been discussed.

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