INFARCTION IN THE VERTEBRO-BASILAR artery distribution has been noted in a variety of circumstances associated with head turning and hyperextension of the neck, but the interval between the trauma and the cerebrovascular stroke is usually short.

This paper describes a patient who developed a cerebellar infarction five weeks following a car accident, an unusually long delay which deserves mention for its pathophysiological and medico-legal implications.

Case Report
A 38-year-old man, while driving his car, was suddenly run into by a car coming at high speed from behind. There was no head support on the driver's seat back and he had forgotten to fasten his seat belt. After the accident he fell unconscious for a few minutes and was admitted to a general hospital where he stayed four days. A week later an electroencephalogram was found normal. The patient complained of a stiff neck and occipital headache for about ten days, but afterwards he was able to return to work.

Thirty-seven days after the accident, on awakening, he complained of the abrupt onset of dizziness and vomiting. Admitted to our wards, the patient was confined to bed and complained of intense exacerbation of dizziness from any attempt to change his position. His medical history before the accident had been completely uneventful. There was no family history of hypertension or diabetes and he never smoked. His alcohol consumption was moderate. On examination the visual fields were intact. The pupils were slightly miotic and reactive to light. Extraocular movements were full, but there was a coarse horizontal nystagmus on the leftward gaze. The face moved symmetrically. The tongue protruded slowly and ataxia was evident at the finger-to-nose test. Sensation was normal for touch, joint-position, vibration, temperature and double tactile stimulation. The blood pressure was 130/80 mmHg. After 24 hours the patient became confused and drowsy. At this time the plantar response was extensor on both sides, but the neurological picture was otherwise unchanged.

The computed tomography (fig. 1) showed a low density area occupying the whole cerebellar territory of the right posteroinferior cerebellar artery partially extending to the territory of distribution of the superior cerebellar artery on the same side. The fourth ventricle was shifted to the left.

The patient was referred to the neurosurgical unit for a possible posterior fossa decompression. However, since a slow but persistent improvement took place in the following week, after dexamethasone 8 mg intramuscular every 6 hours and glycerol 50 g/day by intravenous infusion, surgical intervention was given up. No other drugs, in particular anti-coagulants and antiplatelet medications were given. Twelve days after admission a control CT scan showed a marked reduction of the low density area in the right cerebellar hemisphere. At this time there were no longer signs of ventricular displacement. Flexion and extension radiograms of the cervical spine were normal. No other vascular abnormality was found either in the territory of the right carotid artery or in that of the left vertebral artery which was explored by means of a left brachial angiography. Repeated screening by laboratory tests excluded any general condition favouring to early occlusive events, such as diabetes, hyperlipidemia, collagen and other obvious hematological diseases. Electrocardiogram and echocardiography were normal. Doppler-ultrasonography of the carotid system was also normal.

During the five weeks of his hospitalization the patient regained his ability to walk, although his gait remained ataxic. Three months after the onset of his illness, he was able to walk without aid, but continued to have difficulty in writing and performing fine movements with his right hand. Neurological examination at that time showed only a slight dysmetria on the right side at the finger-to-nose test.

Discussion
Extreme degrees of neck flexion, extension and rotation are potentially hazardous to the vertebral arteries. Vertebro-basilar distribution infarctions have been described following chiropractic manipulations,1,2 yoga exercises,3 atlanto-axial subluxation,4 hyperextension of the neck associated with athletic activities or work5,7 and even after spontaneous head turning.8 It has been suggested9 that vertebral arteries are es-
especially vulnerable in the atlanto-axial dislocation at the C₁ intervertebral foramina and at the occipital-atlantal junction where the occipital condyles can slide forward over the articular facets of C₁.

Angiographic abnormalities have been repeatedly reported in the atlanto-axial region following neck trauma, and in the present case too angiography revealed a vertebral artery narrowing at C₁ level. The collision was described as sudden and absolutely unexpected, a fact which is most likely to have caused extreme degrees of neck movement and the consequent compression of the vertebral artery. Furthermore our patient had neither preexisting cerebrovascular diseases nor any risk factor for them.

Figure 1. CT scan showing right cerebellar infarction.

Figure 2. Antero-posterior (A) and lateral (B) views of the right brachial angiogram showing a remarkable narrowing of the right vertebral artery at the C₁ level (arrows).
Delays as long as three weeks have been described in a recent series of cerebral arterial occlusions following blunt trauma of the head and neck. To the best of our knowledge, a delay of five weeks has never been reported before.

In spite of this long interval, we believe that the neck trauma is the most plausible explanation of the occurrence of a cerebellar softening in our patient, considering his relatively young age, his negative history for previous vascular disease and the absence of risk factors favouring their appearance. Furthermore the site of vertebral artery stenosis in this patient is relatively uncommon following a vascular disease from atherosclerosis, but it is the commonest site following cervical trauma.

Trauma to the vertebral artery may have compressed and stretched the vessel with resulting tearing of the intima and thrombus formation.

Either embolism or haemodynamic failure may have subsequently caused the right cerebellar infarction.

The proximity of the trauma to the onset of the symptoms has been so far considered a most important criterion in proving before the law that the stroke was caused by the initial trauma, but our case suggests that this criterion should not be applied too rigidly and that the site of the damage to the vertebral artery as well as the overall dynamics of the accident ought to be carefully considered.

References

Cerebral Fat Embolism Following Cardiac Surgery

NITYA R. GHATAK, M.D., ROBERT J. SINNENBERG, M.D., AND GEORGEAN G. DEBLOIS, M.D.

SUMMARY We describe 3 cases of cerebral fat embolism associated with cardiac surgery. The autopsy findings suggested that in 2 cases, the embolic fat globules entered the systemic circulation directly as a result of protrusion of epicardial fat into the left atrial chamber along the suture line in one case, and through an atrial septal defect in the other. The characteristic latent interval between surgical trauma and onset of neurologic manifestations was lacking in both cases. The evolution of neurologic manifestations and the pathologic findings in the third case were similar to those usually seen in posttraumatic fat embolism. This study suggests that the pathogenetic mechanisms of cerebral fat embolism following cardiac surgery may vary in different cases. Also, it emphasizes the importance of autopsy in the investigation of this serious but preventable complication.

CEREBRAL FAT EMBOLISM is most often seen in severe trauma. Although it is known to occur following cardiac surgery, reports on pathologic study of this complication are rare. Moreover the pathogenesis of fat embolism following cardiac surgery is not clear and is generally assumed to be similar to that in skeletal injury. We describe three cases of fatal cerebral fat embolism associated with cardiac surgery. Our findings suggest that different pathogenetic mechanisms may be involved in post-operative fat embolism.

Case 1

A 72 year old man underwent surgery consisting of coronary artery bypass and mitral valve replacement and excision of a ventricular aneurysm. The procedure lasted for 6 hours during which there were brief periods of hypotension and an estimated blood loss of 400 ml. At the time of attempted access to the left atrium for mitral valve repair the atrial septum was incised and then closed before entering the left atrium. The procedure was then completed without further complication.
Delayed cerebellar infarction following a car accident.
P Nichelli, M Gibertoni and C Guerzoni

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