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 corneal reflexes were equal. Hearing was normal. There was mild scanning of speech, but neither paretic dysarthria, nor dysphagia. The finger-to-nose and the heel-to-knee tests disclosed a gross dysmetria on the right side at the finger-to-nose test.

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, yoga exercises, atlanto-axial subluxation, hyperextension of the neck associated with athletic activities or work and even after spontaneous head turning.

It has been suggested 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.
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.

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Received December 3, 1981; revision accepted January 6, 1983.

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References


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 trauma. 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.
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Stroke. 1983;14:617-619
doi: 10.1161/01.STR.14.4.617
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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