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 cerebral 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.

### Cerebral Fat Embolism Following Cardiac Surgery

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**SUMMARY** We describe 3 cases of fatal 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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**References**

Post-operatively, the patient remained unresponsive and became anuric with blood pressure maintained with dopamine. Additionally, he developed gastrointestinal bleeding and abnormal liver enzyme studies. An exploratory laparotomy was done on the following day for possible ischemic bowel. The abdominal exploration was unrevealing. The patient developed intractable seizures and died on the fourth post-operative day without ever becoming conscious after the operation.

Autopsy revealed recent wounds of thoractomy and laparotomy. Examination of the heart revealed a pericardiotomy with fibrinous pericarditis. The coronary artery bypass grafts were intact. The margin of resection of the ventricular aneurysm was also intact. The atrial septum showed contusions and a roughly circular defect measuring 1.0 cm in diameter. The mitral valve implant was in place.

The brain weighed 1450 gms with an unremarkable external surface. Coronal sections showed widespread petechial hemorrhages in the white matter characteristically seen in fat embolism (fig. 1). Microscopic examination revealed numerous hemorrhagic and non-hemorrhagic microinfarcts. In paraffin sections empty-looking vacuoles presumably representing fat emboli were frequently seen in such lesions (fig. 2). Oil red O stains were used to confirm the intravascular fat globules in frozen sections.

Grossly normal kidneys showed ballooning of glomerular capillaries shown by special stains to be fat emboli. The lungs showed congestion and intraalveolar hemorrhage, but no fat emboli. Other findings included patchy areas of ischemic necrosis in the small and large bowel and centrilobular necrosis of the liver parenchyma.

Case 2

A 36 year old male was admitted for heart transplantation due to end stage heart failure resulting from idiopathic hypertrophic subaortic stenosis. The transplantation was carried out with the patient on cardiac bypass for 2½ hours. During the procedure generalized oozing at the left atrial suture line necessitated transfusion of fourteen units of whole blood and seven units of fresh frozen plasma.

Post-operatively the patient never regained consciousness. Cardiac output was borderline and ventricular pacing and pressor agents were required to maintain blood pressure. On the second post-operative day, myocardial performance continued to decline. Additionally, purpuric skin hemorrhages developed with a platelet count of 35,000/mm. On the third post-operative day the patient lost all electrical activity in the heart and was pronounced dead.

Autopsy revealed widespread petechial and purpuric skin hemorrhages. There was 600 ml of blood in the left pleural cavity and 150 ml of blood in the right pleural cavity.

Further examination revealed 250 ml of fresh and clotted blood in the pericardial space. The anastomoses of the aortic and pulmonary vasculature were intact. The anastomosis of the right atrium showed focal edema and at the left atrium showed marked edema and contusion. Most notably, there was dehiscence along the left atrial suture line with epicardial fat protruding into the atrial chamber. The ventricles and all valves of the donor heart were unremarkable.

The brain weighed 1440 grams. An old cystic infarct of the left superior temporal gyrus was present. Coronal sections revealed multiple punctate hemorrhages scattered in the white matter throughout the brain including the brain stem and occasionally in the gray matter of the basal ganglia and cerebellum. Microscopically fat globules were seen in small cerebral vessels often accompanied by microinfarct with ball and ring hemorrhages.

Microscopic examination of the kidney revealed fat emboli in the glomerular capillaries. The lungs showed acute and chronic passive congestion; however, no fat emboli were seen.

Case 3

A 65-year-old male underwent coronary artery bypass, left ventricular aneurysmectomy and mitral valve replacement. During the 5½ hour procedure the patient lost 850 ml of blood and became hypotensive.
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Discussion

Despite extensive investigation, the pathophysiological mechanism of posttraumatic fat embolism remains controversial.1,4,6 It is now generally held that the fat globules originate from adipose tissue at the site of trauma, enter the venous circulation and become trapped in the pulmonary capillary bed. After a variable interval, these globules somehow pass through the lungs and cause systemic embolic lesions, thus explaining the delayed onset of neurologic deficits characteristically seen in this condition.7 The possibility of fat embolism is often considered among various complications of cardiac surgery. Although the clinical and certain other aspects of fat embolism associated with cardiopulmonary bypass have been discussed2,3,8 reports on detailed pathologic study of this phenomenon are surprisingly rare. Therefore, the present findings are of interest particularly, since they suggest that the pathogenetic mechanisms of cerebral fat embolism following cardiac surgery may significantly differ from that underlying post traumatic embolism.

In case 2, protrusion of epicardial fat into the left atrial cavity probably provided the source of embolic fat introduced directly into the systemic circulation. In case 1, the creation of an atrial septal defect during surgery might have predisposed the passage of fat globules into the systemic circulation resulting in paradoxical fat embolism as has been described previously.9 The fat globules in this case might have been derived from adipose tissue at various sites of surgical incisions including sternotomy. Thus, in both cases at least some of the fat globules could have entered the systemic circulation bypassing the pulmonary capillary bed. This might be a possible explanation why these patients remained comatose following surgery and did not show the latent interval between the surgical procedures and the onset of neurologic deficits usually seen in posttraumatic fat embolism. In contrast, the neurologic manifestations in case 2 developed two days after the surgical procedure and there was no anatomical evidence of autopsy that might suggest a direct access of fat globules to the systemic circulation. Thus the evolution of clinical manifestations in this case seemed to conform to that seen in posttraumatic cases.

A clinical diagnosis of cerebral fat embolism is often difficult and usually presumptive. It is probably even more difficult in patients undergoing cardiac surgery since various other factors such as circulatory failure, hypoxia, air embolism, etc. may produce similar clinical manifestations.2,30 Thus autopsy study seems to be essential to establish the true incidence of fat embolism following cardiac surgery. More important, only such studies can uncover the source of fat and the routes by which these reach the brain. Such information is essential for prevention of this serious complication.

References

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Stroke. 1983;14:619-621
doi: 10.1161/01.STR.14.4.619

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