

parasites which can cause this type of infection are: (a) horse botfly (*Gasterophilus intestinalis*), (b) cattle warble fly (*Hypoderma bovis* and *Hypoderma lineatum* (de Villiers)); and (c) sheep nostril fly (*Oestrus ovis*).

There are certain differences between the infections caused by the British botflies and those due to the African tumbu flies. As opposed to botflies, the tumbu fly produces a fixed cutaneous myiasis: fixed, because the larva stays in the skin until it either dies or is ready to re-emerge; cutaneous, because it remains confined to the skin and immediate subjacent hypodermal tissue. For infection with horse and cattle botflies, direct contact with the animals is necessary to allow transference of the larvae from skin to skin—for example, in riding and grooming animals. No such intimate contact with animals is required for infection with tumbu fly: the eggs are not laid directly on the host, but in some suitable part of his habitat. In the cases of the two children, the sand was probably the site chosen by the fly to deposit her eggs.

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HUGH CALVERT, M.B., M.R.C.P.,
Consultant Dermatologist to the Reading Group
of Hospitals.

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Traumatic Thrombosis of the Aorta

For a number of years it has been realized that closed injuries may lead to thrombosis of peripheral arteries; Edwards and Lyons (1954) described an example of thrombosis of the external iliac and common femoral arteries in a man of 32 after a closed injury, and in this case claudication was first noticed when he tried to return to work 26 days after his accident. A search of the literature has failed to reveal any case of thrombosis of the aorta in which trauma was regarded as the major factor in its occurrence. Parmley *et al.* (1958) studied at the Armed Forces Institute of Pathology 296 cases of aortic rupture due to closed injury—most often rapid acceleration or deceleration, and all but 38 were rapidly fatal: all but two subsequently died, the two being saved only by excision of their aneurysms and insertion of grafts. In only 13 of these cases was injury to the abdominal aorta the sole lesion.

Since Leriche (1940) first described the syndrome of thrombosis of the terminal part of the aorta, many cases have been reported; it is usually assumed that they are due to atherosclerosis, and operations for its relief, either by grafting or by disobliteration, are now routine procedures in surgical departments treating vascular disease. Morel (1943) reported a case in a man of 29 when symptoms were of 18 months' duration, but there was no history of injury.

In view of the importance of closed injuries of major arteries, the following case is put on record.

CASE REPORT

A motor-coach driver of 50 was first seen in March, 1960. He gave a history that on January 10, 1958, while driving his coach, he was involved in a head-on collision. He was dazed and had scalp injuries, for which he was treated at a hospital but not detained, but he rested in bed at home because of pain in his lumbar region. Three weeks later, when he became more active, he felt pain in his calves after walking about 80 yards, and also had cramp in his buttocks and "pins-and-needles" in his feet. These symptoms gradually became worse, claudication occurring after 30 yards, and he could not manage driving light vans because of the cramps.

He had a blood-pressure of 180/100, but the only other abnormality was the absence of femoral and all distal pulses in both legs. The feet were warm and sensation was normal. He was rather stout and no abdominal aortic pulsation could be felt. On May 3 the abdominal aorta was exposed through a right paramedian incision. The upper part of the vessel pulsated normally, but no pulsation could be felt in the iliac arteries. About 3 cm. below the renal vessels the aorta was blocked by a plug of partly calcified thrombus for a length of 3 cm. Below this the vessel walls were healthy, except for an atheromatous plaque in the right side of the aortic wall just above the bifurcation, and another in the left common iliac artery. The aorta above this obstruction and the common iliac arteries were clamped and most of the plug was removed piecemeal through an incision just below it. All his peripheral pulses were present after operation.

Part of the wound in the abdominal wall gave way on the ninth post-operative day and had to be resutured, and he had a small pulmonary embolus on the nineteenth day; he was treated by anticoagulants and left hospital 30 days after his endarterectomy.

Phenindione was continued until six months after his operation. At that time he had no claudication in the calves, all pulses were present in the legs, but he still complained of slight cramps in his buttocks. He could walk 200 yards without pain and was working as a motor driver.

COMMENT

Although this patient had slight atheroma, there seems to be no doubt that the injury precipitated the occlusion of the abdominal aorta, though this cannot have been complete from the onset, for in such a case symptoms in the lower limbs would have been present soon after the accident; there must have been time for the collateral circulation to open up. It seems probable that he was struck in the abdomen by the steering-wheel as he was thrown forward, and that this accounts for the position of the lesion in a fairly healthy aorta. Whether thrombosis can ever occur in a perfectly healthy artery is not known, and in this case it seems likely that the injury caused damage to the intima of the vessel at a site where an atheromatous lesion already existed. However, except for the small plaques in the lower aorta and common iliac arteries, there was no other evidence of arterial disease.

We are grateful to Dr. Mark Bradford, of Weymouth, who referred the patient and supervised his anticoagulant therapy after his return home.

ANNA G. WALKER, M.B., Ch.B.,
R. MILNES WALKER, M.S., F.R.C.S.,
Department of Surgery, University of Bristol.

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