The finding of emboli of haemopoietic tissue in the lungs is not uncommon in experimental animals, but is extremely rare in man. Most of the reported cases have been associated with fractures, and in the remainder convulsions preceded death. Bone-marrow embolism was first demonstrated by Lubarsch (1898) and Lengemann (1897) in rabbits following the injection of liver, kidney, or placental cells into the jugular vein. Lubarsch was also the first to observe bone-marrow emboli in human beings, in four women who had died from eclampsia. In all these cases haemopoietic tissue was transported without any destruction of bone, a fact which Lubarsch emphasized in opposition to Maximow (1898), who, in his experiments on rabbits, saw such emboli only after extensive fragmentation of bone. Lubarsch laid great stress on local hyperaemia and softening of the bone-marrow as predisposing factors, though he admitted that in human beings some shock to the bone-marrow might be needed as well. Lengemann (1901), while agreeing that some alteration of the bone-marrow was a predisposing factor, attached greater importance to concussion of bone in both man and experimental animals, and considered that the violent movements of the latter during manipulation played some part. The reports of these early German workers, and some experimental work on the ultimate fate of these emboli, were reviewed by Ceelen (1931), who added that no sequels of any importance followed bone-marrow embolism, for even if small or medium sized arteries were blocked a collateral circulation developed.

Since that time only a few reports have appeared in the literature. Sotti (1910), reporting a necropsy on a woman of 78 who died of senile dementia in a mental hospital, described the finding of a bone-marrow embolus in the lung; although he gave no further clinical history it is probably reasonable to assume that convulsions preceded death. Karlén (1942) found fat and bone-marrow emboli in the lungs of a patient who died in convulsions following intradural "perabrodil" myelography. The same observations were made by Schenken and Coleman (1943) in a woman of 86 who died after the operative reduction of a fractured neck of femur. Lindsay and Moon (1946) reported three cases following multiple fractures. Warren (1946) observed marrow particles in the pulmonary arteries in three out of 100 cases of fat embolism. Recently Rappaport, Raum, and Horrell (1951), in a study based upon cases in the files of the Armed Forces Institute of Pathology, Washington, reported 27 new cases; of these two were personally observed by the authors, another five were found in the files with the diagnosis of bone-marrow embolism, and the remainder were brought to light by a systematic survey of cases in which death followed trauma with multiple fractures, accidental electrocution, and generalized
convulsions, and in which specimens and sections were available for study. Of the 27 cases, 18 were instances of post-traumatic death, two of accidental electrocution, and seven of death following severe generalized convulsions. These cases bring the total number recorded in the literature to 40.

The following case of fat and bone-marrow embolism following crush injuries of the ankles has recently been encountered.

**Case Report**

A healthy man, aged 44 years, was engaged in operating an overhead crane and grab, moving coal from a large heap and depositing it over a conveyor belt. While he was making some adjustments the brake slipped, the grab descended to the ground, and he was trapped by his legs between two steel rollers which carry the cables operating the grab. These rollers are only 2½ in. apart, and the securing bolts had to be cut by oxy-acetylene flame to free him. He was admitted to hospital one hour after the accident. Respirations were then found to be absent, but the radial pulse was perceptible for about ten minutes.

**Necropsy.**—This was performed for the coroner by the casualty officer. He found severe crush injuries of both ankles, involving both soft tissues and bones. There was also widespread laceration of the soft tissues in the lumbar region. No other bony injuries were found. Nothing abnormal was found in the brain, heart, liver, kidneys, spleen, or alimentary tract. There were some old pleural adhesions at the right base, and a small piece of the right lung was submitted to the laboratory for histological examination. There is unfortunately no record of the naked-eye appearance of the lungs. Death was attributed to primary shock.

**Histology.**—Paraffin sections of the lung stained with haematoxylin and eosin showed intense capillary congestion with widespread intra-alveolar haemorrhages, and alter-
nating zones of oedema and emphysema. Apart from many dust-laden phagocytes there was no cellular reaction. The most striking feature, however, was the presence of fragments of haemopoietic tissue in branches of the pulmonary artery; some of these were sufficiently large to make their nature obvious and included several fat cells (Fig. 1), but careful search revealed many smaller fragments, and individual marrow cells were scattered widely throughout the pulmonary arterioles and capillaries. Frozen sections stained with Sudan IV confirmed a gross degree of fat embolism.

Discussion

It is evident that bone-marrow embolism may occur in two distinct types of injury. In the first place convulsions may impart such a shock to the skeleton as to dislodge fragments of haemopoietic tissue. Secondly it may follow fracture of bone, as in the present case and most of those reported, the marrow fragments being forced into the veins by the increased pressure resulting from local haemorrhage and oedema. Most of the traumatic cases have been road, railway, or aeroplane accidents in which fractures were multiple, and in these circumstances the occurrence of bone-marrow embolism is not really surprising. In trauma of lesser degree, however, the very nature of the injury may be a predisposing factor. Schenken and Coleman, for example, whose patient died four days after the operative reduction of a fractured neck of femur, postulated that fragments of marrow may have been forced into the veins mechanically by the wooden bone screws which were used; and in the present case it is tempting to suppose that a similar mechanical action resulted from the squeezing of the ankles between the steel rollers.

It is noteworthy that bone-marrow embolism is by no means invariably accompanied by fat embolism, that is to say embolism by extracellular fat. Rappaport et al. found no evidence of fat embolism in nine out of 21 of their cases in which suitable material was available for study, nor did they observe any direct relationship between the severity of fat and bone-marrow embolism. These workers also present some interesting figures on the incidence of bone-marrow embolism; they found 13 instances (6%) in a survey of 203 consecutive cases with multiple fractures and none in a control series of 240 necropsies. Thus it seems likely that a greater awareness of this condition among pathologists will lead to its detection more frequently than hitherto. Their main thesis, however, concerns the mechanism of bone-marrow embolism in deaths following generalized convulsions. They consider that detachment of marrow fragments by concussion alone is unlikely, and suggest that in those cases of their series in which convulsions preceded death bone-marrow embolism was due to undetected vertebral fractures, a suggestion which Karlén had also made in his case. This is an intriguing hypothesis, for indeed a crush fracture of a vertebra might well predispose to bone-marrow embolism by its very nature. Anatomical proof is not easy to obtain, even if looked for, at necropsy. This problem can be solved only in a post-mortem room where radiographic facilities are available, and it is proposed to make it the basis of a future study.

Summary

A case of fat and bone-marrow embolism following crush injuries of the ankles is described.

The literature on bone-marrow embolism is reviewed.

The pathogenesis of this condition is discussed.
I am indebted to Mr. T. H. Church, H.M. Coroner for Gravesend, for allowing me access to his records.

REFERENCES
Maximow, A. (1898). Ibid., 151, 297.