Fat embolism

The topic of fat embolism forms a clearly defined area of discussion within the field of the pathology of trauma and the three papers which follow summarize some of our present knowledge and views and give an idea of certain unresolved problems which remain after a century's work.

It is more than a hundred years since fat embolism was first described. Lower of Oxford is said to have been concerned with experimental fat embolism some three hundred years ago although modern investigations began with Magendie who, in the middle of the last century, described the effects of the condition but in connexion with experiments which were directed to other ends. Natural fat embolism was first described by Zenker in 1862 and a connexion between pulmonary fat embolism and injury involving bone marrow was demonstrated by Busch shortly afterwards. Systemic, as distinct from pulmonary, fat embolism was recognized at about the same time and the clinical diagnosis during life was first made by von Bergmann in 1873. He also reported that, following the intravenous injection of fat into cats, some of it entered the systemic circulation with the formation of emboli in liver and kidneys and some of it was excreted in the urine. Thus, fundamental observations were made quite early in the history of pathology in its modern sense.

In spite of the fact that the condition has been recognized and has been the subject of enquiry for a long time, there are still important matters about which controversy continues. In his book on the subject, Dr Sevitt states that the main differences of opinion have been about the origin and nature of the fat emboli, their mode of action, the significance of pulmonary emboli in the production of clinical effects, and the nature of the illness which the emboli produce. In addition there is the latent period between injury and the development of the clinical picture and the possible role of pulmonary fat embolism in the production of cerebral effects as a consequence of hypoxia. This second point is considered in two of the papers which follow.

Two other points may be mentioned. The first concerns the way in which fat emboli are formed. The classical, and most natural, view is that they arise through the ingress of local fat into the blood stream at the site of injury. There is much evidence to support this view. It is possible to produce fat emboli by mechanical disturbance of bone marrow without gross fracturing; fat which has been dyed *in situ* in the bone marrow can be recognized subsequently in fat emboli following fracture, and after fracture the development of the condition can be prevented by the application of a tourniquet. A more recent view of the origin of the fat, which continues to receive some support, is that the fat originates elsewhere than in the bone marrow or other injured tissue and plasma fat is the suggested source. This view is supported by the fact that fat emboli sometimes occur in the lungs of uninjured subjects thus excluding physical trauma as a cause. It has also been suggested, though the point is debated, that the fat content of a long bone may not be sufficient to account for all the embolic fat found in the lungs. This theory postulates that altered conditions in the blood following injury may cause flocculation or agglutination of the chylomicrons to produce fat in the form seen as emboli. Yet another view is that fat may be mobilized from tissues as a consequence of a rise in serum lipase after injury.

The mode of action of the emboli is also in dispute. The older view, again the more natural and obvious one, is that the pathological effects are haemorrhagic or ischaemic consequences

of the temporary occlusion of small vessels by fluid emboli. This is supported by the fact that most emboli probably produce no clinical effects and that, histologically, they show no evidence of an inflammatory response or of a toxic necrotizing effect on vascular endothelium. The other view suggests that it is the breakdown of the emboli, with the formation of free fatty acids, that causes the damage, but again there are objections. Histochemical methods have been unhelpful in demonstrating fat splitting, and the fact that fat embolism of the kidney has not been shown to be accompanied by the effects that follow the injection of free fatty acids is difficult to explain. Furthermore, the increase in split fat in the blood which has been shown to follow haemorrhage and trauma has not been shown to produce toxic effects. It seems that any free fatty acids are rapidly removed, probably by plasma proteins.

Other unresolved issues could be cited and the papers which follow are concerned with some of them. The purpose of this brief introduction is simply to emphasize that this well known subject is still far from fully explored and that different views continue to be held even about some of its fundamental features. Active work continues and the three papers in this section are contributed by people whose own work in the field has been important. Dr Szabo has undertaken a great deal of experimental work on the subject, Dr Watson has been closely concerned with morbid anatomical aspects, and Dr Prys-Roberts's investigations have been clinical, based on pulmonary function studies in injured patients. When due regard is paid to their work and to the other controversial aspects, the pathologist with no specialized knowledge of the field may be tempted to wonder if fat embolism is always produced by the same set of factors or, if so, if these factors perhaps vary in importance from one case to another.

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