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EDITORIALS AND ANNOTATIONS

FAT EMBOLISM AND POST-TRAUMATIC HYPOXAEMIA

The fat embolism syndrome has been recognised for more than half a century, yet it has been viewed from so many different angles that, like the six wise men of Indostan who viewed the elephant but could not describe the whole entity, so do we still have difficulty in solving the enigma of fat embolism. In this issue three aspects of the condition are highlighted, and as in the past the role of hypoxaemia is emphasised. Gurd and Wilson have rightly drawn attention to the difficulty of distinguishing between the existence of embolic fat in the lungs of patients following injury (Sevitt 1962) and the true syndrome in which a chemical interstitial pneumonitis is a prominent feature associated with a variety of clinical presentations and signs. They confirm that the majority of patients survive the syndrome if proper and prompt attention is paid to the pulmonary disorder, which manifests itself in the form of arterial hypoxaemia (Wertzberger and Peltier 1968*a*).

Because of its varied presentation, the physiologist with an interest in the syndrome faces a number of difficulties, not the least of which is that the syndrome is uncommon and tends to present suddenly, at times and in places which do not readily conform to the ordered processes of a research protocol. Control values of pulmonary gas exchange are difficult to establish unless sequential complex measurements are made in the researcher's institution in all or many of the patients who sustain limb fractures and other major injuries. The logistics of this exercise alone have been sufficient to deter most workers. Nevertheless, it is important that such baseline information should become available, since at present we have little accurate and useful information concerning pulmonary gas exchange following various degrees of injury (Prys-Roberts 1973). We cannot at present exclude the possibility that the fat embolism syndrome represents the tip of an iceberg, where the majority of injured patients have lesser degrees of ventilation perfusion (V Q) disturbances in their lungs. Hypoxaemia alone is a crude index of pulmonary gas exchange, and has such a diverse etiology that the previous observations of the severity and incidence of hypoxaemia associated with fat embolism simply serve to identify a problem rather than to solve it. Based on observations of patients with the established fat embolism syndrome (Prys-Roberts, Greenbaum, Nunn and Kelman 1970), it appears that a biphasic disturbance of pulmonary gas exchange may occur. We can predict, and it has been observed, that when emboli of fat or any other material enter the pulmonary capillary circulation and occlude the flow of blood to discrete or diffuse groups of alveoli whose ventilation is maintained, then these areas behave functionally as increased alveolar