

Pulmonary Failure after Peripheral Injury

Despite the effectiveness of modern methods of resuscitation and treatment a patient may die from pulmonary failure after severe injury to a peripheral part of the body. The importance of delayed secondary effects on the lung has recently been emphasized by F. W. Blaisdell and colleagues.¹

Reviewing the cases of patients admitted with severe trauma during one year to the department of surgery at the San Francisco General Hospital, H. T. Cafferata and his associates² found that of the 14 who survived the first 24 hours seven ultimately died of respiratory failure. Prolonged ischaemia of the lower limbs would seem often to be followed by this complication, since respiratory failure was regarded as the main cause of death of eight of the 13 patients who had been successfully treated for injury to a lower limb. In other conditions in which systemic shock has been severe—for example, after ruptured aneurysm—pulmonary failure has also largely determined the final outcome.³

The pulmonary damage may show itself within the first 24 hours after injury or some days later, when even the initial response to treatment has clearly been satisfactory. Shortness of breath, restlessness, a rise in respiratory rate, or cyanosis may all be observed. The radiographic changes resemble those of pulmonary oedema. A change in lung compliance can be shown before there is arterial desaturation. An arterial PO_2 of less than 60 mm. Hg and a PCO_2 of more than 40 mm. Hg are the criteria by which these authors define the state of respiratory failure. Peripheral shock develops again as a terminal stage in pulmonary failure.

Blaisdell and his colleagues¹ report the findings in the lungs of 30 patients who died of pulmonary failure after severe soft-tissue injury caused by either vascular disease or trauma and who were in a state of shock on admission to hospital. Even during the first 18 hours after the onset of shock petechial haemorrhages, patchy congestions, and airlessness were seen. Microscopical examination showed, in addition, thromboemboli in small pulmonary blood vessels. These changes progressed to produce haemorrhagic consolidation so widespread that after a few days the lung sometimes resembled liver.

Infarcts, as such, are not commented upon, which makes it unlikely that a rise in pulmonary venous pressure, as might occur in heart failure, is of prime importance. The nature of the change suggests a widespread damage to small blood vessels.

The authors suggest that direct damage to the small blood vessels of the lung is caused by the arrival of damaged cells, particularly platelets, released from the peripheral circulation and by thrombi and clots dislodged from the damaged regions being caught in the filter of the pulmonary artery bed. Light microscopy shows changes in the human lung, but in experimental studies electron microscopy disclosed additional endothelial damage within 72 hours of the injury.⁴ The alveolar oedema and haemorrhage that follow produce hypoxia, which in its turn causes further capillary damage. Pulmonary vasoconstriction produced by the release of vasoactive amines,

such as serotonin and histamine, is a further disturbance contributing to what quickly becomes a vicious circle. This hypothesis is supported by the findings in studies on the dog.⁵ Though an understanding of the pathogenesis of pulmonary failure after soft-tissue injury is not yet precise, the mechanism is certainly complex. Recognition that it is so common and grave a complication should influence the routine management of patients with severe peripheral injuries. Blaisdell and colleagues recommend that tracheal intubation and mechanical ventilation be promptly initiated in the management of severe peripheral injuries associated with shock. They emphasize the importance of periodic hyperinflations of the patient's lungs during mechanically assisted ventilation. When assisted ventilation has not been started (or in doubtful cases) blood-gas analysis is probably the best guide to developing trouble. Any fall in oxygen tension should be regarded as calling for assisted ventilation.

Treatment of the original shock by careful control of the restoration of blood volume is of main importance. These authors recommend the administration of high-oxygen mixtures. Heparin and steroids are suggested to minimize the danger of intravascular coagulation and platelet aggregation. Once the condition has developed, low-molecular-weight dextran may be of use in addition to the above measures, and the diuretics frusemide and ethacrynic acid help to correct the effect of overhydration. Certainly prevention seems the wiser course, as treatment of the established condition is unlikely to be successful.

Some reservation must still be held about the prevalence—at least in Great Britain—of the syndrome described by this group of workers and about its pathogenesis, particularly as methods of treating shock may themselves contribute to pulmonary damage. Administration of gas mixtures with too high an oxygen content may cause serious alveolar damage, a result of treatment that is recognized as increasingly important. Overhydration and disturbance of electrolyte equilibrium in the early stage of shock may also lead later to impairment of lung function.

The Name of the Game

Many doctors have been unhappy about the proposal in the recent report¹ from the B.M.A. Planning Unit to call general practice "primary medical care." Misgivings about the new title were clearly apparent in the debate at the General Medical Services Committee's last meeting (see *Supplement* page 1). To some extent the division of opinion on nomenclature reflects a divergence of views on the future development of general practice. Should the present pattern continue, with occasional adjustments made to cope with outside changes, or should the profession, accepting change along lines that some regard as already identifiable, move deliberately along them?

While accepting their suggestion was not perfect, the authors of the report admit to changing the name as a means of promoting an uninhibited reassessment on the future of general practice. Defenders of the status quo naturally tend to resist any tinkering with a name that has stood the test of time. In their eyes the new title would contribute to the destruction of the family doctor as the only generalist doctor of first contact.

¹ Blaisdell, F. W., Lim, R. C., jun., and Stallone, R. J., *Surgery, Gynecology and Obstetrics*, 1970, 130, 15.

² Cafferata, H. T., Aggeler, P. M., Robinson, A. J., and Blaisdell, F. W., *American Journal of Surgery*, 1969, 118, 281.

³ Blaisdell, F. W., et al., *Archives of Surgery*, 1966, 93, 776.

⁴ Goodman, J. R., Lim, R. C., jun., Blaisdell, F. W., Hall, A. D., and Thomas, A. N., *American Journal of Pathology*, 1968, 52, 391.

⁵ Lim, R. C., jun., Blaisdell, F. W., Choy, S. H., Hall, A. D., and Thomas, A. N., *Surgical Forum*, 1966, 17, 13.