Circulatory effects

It is almost trivial to state that the cardiovascular manifestations of shock are many and varied. Even in the attempts that have been made to find the locus of the primary change that might initiate all the others, attention has constantly shifted from one part of the cardiovascular system to another.

For a long time the capillaries were held to be the site of the primary lesion; the heart has been implicated in some instances, as having vessels on the arterial and venous sides of the circulation between them. The original cause of the primary disturbance has been sought in toxins or in some aberration of the nervous control of the peripheral vasculature. Indeed the bewildered reader of all the accounts of the many investigations that have been carried out in this field might wonder if it will ever be possible to bring order to it. Dr Stalker's account of the microcirculation in shock shows a neat and simple way in which this can be done. His lucid introduction justifies his definition of the process as essentially one of loss of microcirculatory control, with hypoperfusion. It is established that prolonged hypoxia leads to failure of contraction of the smooth muscle of arterioles and precapillary sphincters from which they may never recover. The smooth muscle of venules is more resistant to a prolonged lack of oxygen; hence capillaries dilate, flow is sluggish, and outward filtration of fluid predominates. Dr Stalker emphasizes the aggregation of red cells that may occur under these conditions and the obvious worsening of the situation which this phenomenon induces. This brings the rheological changes to the fore, and certain it is that they add a new dimension to the analysis. In particular the potential significance of fibrin microthrombi is being currently emphasized, and platelet aggregation is a further phenomenon of real relevance. This last though not part of Dr Stalker's account, might come to dominate future investigations, as a primary factor in haemorrhagic shock at least.

The paper which follows, by Dr Walters, emphasizes the low cardiac output of established shock, and the control of treatment designed to remedy this defect. He points to the inadequacies of attempts to measure blood volume and the difficulty of estimating what the normal blood volume of an individual patient should be. He recommends reliance on central venous pressure as an indication of the adequacy of the volume of circulating blood. It may be a pedantic point that the measurement actually required is that of central filling pressure rather than central venous pressure, but the latter is a very simple measurement to make and may be adequate for clinical purposes. It is clear, however, from Dr Walters' account that central venous pressure is not sufficient on its own to assess the adequacy of a transfusion, though it does seem to enable a good measure of control to be achieved.

In his paper entitled 'Electronic and computer monitoring in shock' Dr Raison takes us to the limits of what is now possible in the measurement of a wide variety of parameters together with on-line computation of derived functions of possible importance. Quite apart from the cost effectiveness of the set-ups envisaged, or even the possibility of accommodating such large amounts of apparatus around a patient still allowing access to a nurse, one cannot help asking whether more time should not be spent investigating the useful application of these techniques as research tools before they are applied extensively on the ward.

Finally, Dr Fleat takes us into rather different territory. He accepts that tissue hypoperfusion is the common feature of shock from a variety of causes, and then proceeds to examine the resulting changes that may occur at the cellular level, in particular the altered movement

of sodium and potassium across the cell membrane, and their implications for shifts of water between the intracellular and extracellular compartments. His thesis is that these are the primary cellular events which are responsible for the changes in body water and other electrolytes which occur after trauma, and that they may have a valuable energy-sparing function.

The contributions in this section of the symposium thus cover a variety of topics relevant to the field, hypoperfusion of tissues perhaps providing the main idea common to them all. It only remains to ask whether hypoperfusion of the central nervous system might not add a further complication, quite apart from its possible significance for the recovery of adequate cerebral activity, by setting a limit on its own account to the recovery of cardiovascular function.

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