# Clinical consequences of shock

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Unresuscitatable shock Patients who are in profound shock for a prolonged period of time become ‘unresuscitatable’. Cell death follows from cellu lar ischaemia and the ability of the body to compensate is lost. In the heart there is myocardial cell death from poor coronary perfusion and myocardial depression from severe acidaemia and hyperkalaemia. This leads to poor cardiac output and limited response to fluids or inotropic therapy . Peripherally there may also be loss of the ability to maintain systemic vascu lar resistance and further hypotension ensues. The peripheries no longer respond appropriately to vasopressor agents. Once patients enter this stage of systemic ischaemic injury , death is inevitable. Ischaemia–reperfusion and the systemic inflammatory response syndrome (SIRS) During the period of systemic hypoperfusion, cellular and organ damage progresses owing to the direct e ff ects of tissue hypoxia and local activation of inflammation. Further injury occurs once normal circulation is restored to these tissues. The acid and potassium load that has built up can lead to direct myocardial depression, vascular dilatation and further hypotension. Molecules released from the interior of cells are released into the circulation. These are sensed by and activate leukocytes. These, together with cellular and humoral elements activated by the hypoxia (complement, neutrophils, micro vascular thrombi), overwhelm the local anti-inflammatory response and are flushed back into the systemic circulation, where they cause injury to distant organs such as the lungs and the kidneys. This leads to acute lung injury , acute renal injury , cerebral oedema, multiple organ failure and death. Reperfu sion injury can currently only be attenuated by reducing the extent and duration of tissue hypoperfusion. Multiple organ failure As techniques of resuscitation have improved, more and more patients are surviving shock. Where intervention is timely and the period of shock is limited, patients may make a rapid, uncomplicated recovery . However, the result of prolonged systemic ischaemia and reperfusion injury is end-organ damage and multiple organ failure. Multiple organ failure is defined as two or more failed organ systems. There is no specific treatment for multiple organ fail ure. Management is support of organ systems, with v entilation, cardiovascular support and haemofiltration/dialysis until there is recovery of organ function. Multiple organ failure currently carries a mortality of 60%; thus, prevention is vital by early aggressive identification and reversal of shock. Thomas Addison , 1799–1860, physician, Guy’s Hospital, London, UK, described the e ff ects of disease of the suprarenal capsules in 1849. Effects of organ failure /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF -

Cardiac: Cardiovascular failure Lung: Acute respiratory distress syndrome Kidney: Acute renal insuf /f_i ciency Liver: Liver failure and coagulopathy Brain: Cerebral swelling and dysfunction

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