CIRCULATORY FAILURE IN THE POISONED PATIENT – USE OF VASOPRESSORS OR INOTROPES.

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Circulatory failure may be due to myocardial failure, extreme vasodilatation, or both. In the poisoned patient, many drugs and toxins depress myocardial contractility through various mechanisms, a large number of them are also vasodilatators. Reduced myocardial contractility, diminished preload and low systemic arteriolar resistance (SVR) decrease cardiac output (CO) and tissue perfusion pressures; if this progress to failure of the O_2 supply to maintain tissue aerobic metabolism, the patient is in circulatory shock. A similar type of shock may develop during massive inflammatory reactions, like sepsis (septic shock) and after post-ischemic reperfusion (e.g. post-resuscitation shock). Since circulatory failure in the poisoned patient is induced by more temporary factors than endogenous inflammatory reactions, the prognosis is often better regarding restitution of both myocardial and vascular function.

The goal of circulatory interventions in shock, regardless of etiology, is to re-establish blood flow, and thus O_2 supply, to all organs. Interventions may broadly be classified as *i*) preload optimalization (usually iv. infusions), *ii*) drugs with positive inotropic effect on the myocardium (inotropes) and *iii*) agents that contracts vascular muscles (vasopressors). To reduce the potential negative side effects of such interventions, their use and the amount given must be tailored to the pathophysiological changes in each particular poisoned patient. Their effect on arterial blood pressure (ABP) is a poor indicator of whether the therapeutic goal has been reached. ABP is a function of CO and SVR; for bedside purposes, the relationship between mean arterial pressure (MAP), CO and SVR can be written MAP ≈ CO x SVR. This illustrates that a critical reduction of CO to 67% of normal is compatible with an unchanged ABP if the corresponding SVR increase by 50% above normal. Inversely, a 50% SVR decrease will not change MAP if CO increases by 100%. Other parameters, like increased O_2 saturation in central venous blood and reduction in blood lactate levels, are probably more reliable indicators of therapeutic success – but also these may under certain conditions (mitochondrial dysfunction, increased glycolysis) be misleading.

For optimal results, the amount of fluid infused plus the balance between inotropy and vasopressor must be based on analysis of the contribution of cardiac function and vascular tone to the circulatory failure. To achieve this, advanced hemodynamic monitoring including measurements of CO, estimation of preload and calculation of SVR, should be instituted rapidly in poisoned patients if the response to initial therapy is poor or dubious.