

Modelling Decision-making Strategies in Supporting the Shocked Patient

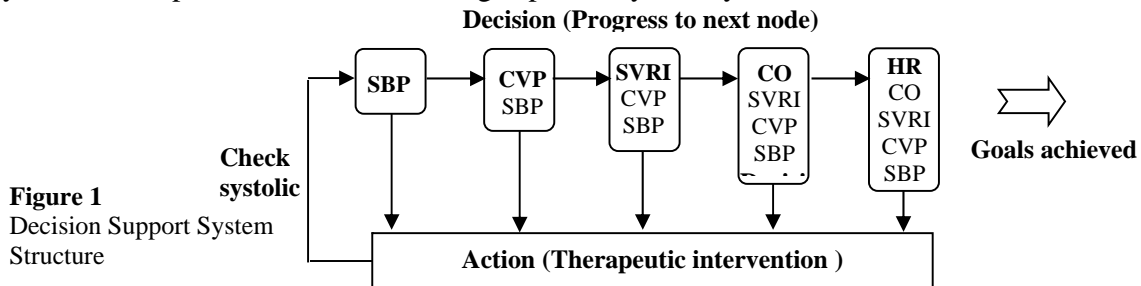
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Modelling the function of the cardiovascular system in disease has usually led to a disease-led construct with loose control parameters, where the diagnosis and decision-strategies have been merged at each stage. Use of this modelling technique implies that the clinical condition causing the picture of shock must be identified for the model to be implemented successfully. It means too that excursions from the model (eg pre-existing disease or superceding developments) may be poorly tolerated, or destabilize, the model.

We have developed a decision support system (DSS) (Fig 1) that defines each component of the cardiovascular system (preload, contractility, heart rate, systemic vascular resistance and cardiac output) in modular form. Each component is identified as a 'node' or 'module' and the order of these modules may be prioritised or re-ordered according to the pathogenesis of the shock. Every feature of these 'nodes' of the CVS can be normalized to a value pre-selected by the clinician. If the value cannot be normalized then it is optimized and the failure of the strategy to normalize the value is considered in the decision to move to the next node and incorporated in the decisions made regarding the choice of therapy downstream. For instance, a patient severely vasodilated may never achieve normotension by CVP normalisation (fluid administration) alone. 'Optimisation' means that the DSS can track to the next node, introducing a vasopressor, with a sub-normal CVP. Commencing this therapy leads to normalization and stabilization of the CVP, and therefore the systolic blood pressure. As almost all interventions in all nodes cause changes in the other components, our decision support model always refers decision outcomes back to the systolic blood pressure as the overriding supervisory 'safety shell'.



Our model is being tested in patients being weaned from cardiopulmonary bypass, where blood pressure control is of overriding importance, to avoid hypotension causing the myocardium to fail through hypoperfusion, or suture lines to fail through hypertension. Preload is prioritised next as hypovolaemia causes a brittle control environment and over-filling causes the myocardium to fail. The afterload is controlled next as persistent vasodilation renders control of the perfusion pressure difficult, and vasoconstriction tends towards brittle control surfaces. The cardiac output is last to be prioritized: a sub-optimal CO can be tolerated for short periods.

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