

The answer is dopamine or norepinephrine, but what is the question?

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Post-cardiac surgery circulatory shock is a unique perioperative clinical challenge that requires a multidisciplinary approach, including intensive monitoring and close communication among involved care teams. The causes for circulatory shock can include, solely or in combination, cardiogenic, hypovolemic, obstructive, and distributive shock.¹ Treatment strategies should be tailored to optimize tissue oxygenation and end-organ perfusion. Current treatment algorithms utilize intravenous fluids, vasopressors, and/or inotropes to address these issues. However, the ideal fluid, vasopressor, or inotrope in each clinical scenario is still to be elucidated. This clinical equipoise is reflected in significant institutional practice variation.

The authors (Lim et al., 2021) examine this question of ideal agent in the treatment of initial “circulatory shock,” norepinephrine or dopamine.² In this randomized controlled trial, norepinephrine or dopamine are chosen as initial agents as the common causes to hypotension after adequate fluid resuscitation after cardiac surgery is cardiogenic and vasodilatory shock. Norepinephrine is a potent alpha-1 agonist with some beta-adrenergic activity, with a net effect of increased SVR and minimal changes in contractility. Dopamine is a weak beta-1 agonist, with high affinity for dopamine receptors and trace amine-associated receptor 1, on the other hand, has a varied dose-related effect. At low dose (2-5 mcg/kg/min), it causes mild vasodilation, chronotropy, and mild increase in cardiac contractility. At higher doses (10-20 mcg/kg/min), the alpha-1 agonist predominates and there is greater vasoconstriction without any further increases in cardiac contractility.¹

In this study, after a set amount of fluid resuscitation and presumed euvolemia, the screened in patients were assigned to the norepinephrine or dopamine group to improve systemic blood pressure. Patients with preexisting atrial or ventricular dysrhythmias were excluded from the study.

It is difficult to conduct a true randomized controlled trial in a post-surgical population. The study had significant crossover, specifically in the dopamine group, where 77.1% of patients also received norepinephrine. Patients refractory to norepinephrine or dopamine received, additionally, vasopressin and epinephrine. This suggests that the population studied had both cardiogenic and vasodilatory shock. The study populations are not further subdivided by cause of circulatory shock by objective parameters, such as: filling pressures, echocardiography, or physical exam. It is conceivable that patients who received dopamine when in a state of mild vasoplegic and cardiogenic shock may have benefitted. Patients who initially received only norepinephrine while in a state of cardiogenic shock may have benefitted from initiation of an inotrope. The conclusions of this study do not yet elucidate the best “first-line” agent.

In this age of ever increasing non-invasive and invasive monitors, bedside echocardiography, and mechanical circulatory support, it may be warranted to diagnose the underlying cause of shock and use specific agents to personalize therapy.³ These issues also highlight the importance of postoperative care in high-intensity units with specifically-trained physicians.

Randomized controlled trials are challenging to complete in the heterogenous post-cardiac surgical population, and the authors are commended for their attempt to answer this difficult question. This trial, however, continues to leave clinical equipoise, as 29% of patients did well with simply dopamine, suggesting that dopamine as a sole initial agent is a possibly adequate treatment for a specific subset of patients.

References

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