Vasodilatory shock occurs in 5% to 25% of patients after cardiac surgery and results in poor outcomes. It is treated with vasopressor that leads to vasoconstriction of the splanchnic and skeletal circulation, resulting in end-organ hypoperfusion. Although catecholamine vasopressor therapy, its duration, and number of vasopressors used are associated with an increased risk of adverse cardiac and noncardiac events, vasopressin appears to have a more effective and safer profile in the treatment of vasodilatory shock after cardiac surgery.

Vasopressin, a naturally occurring peptide, produces vasoconstriction by activating V1a receptor, blocking the opening of potassium adenosine triphosphate channels in vascular smooth muscle, and reducing nitric oxide production. Vasopressin depletion and decreased circulating levels of vasopressin are fundamental mechanisms responsible for vasodilatory shock after cardiac surgery. As such, vasopressin is commonly used to treat and prevent vasodilatory shock after cardiac surgery. In a prospective randomized controlled trial, Hajjar and colleagues showed that the use of vasopressin after cardiac surgery is associated with a reduction in 30-day mortality, postoperative renal failure, and postoperative atrial fibrillation compared with norepinephrine. Other investigators previously demonstrated that vasopressin is effective in treating vasodilatory shock after cardiac surgery, sepsis, and other vasodilatory conditions, decreasing catecholamine use with no increase in adverse events.

Low ejection fraction, prolonged cardiopulmonary bypass (CPB), left ventricular assist device insertion, and preoperative use of angiotensin-converting enzyme inhibitors and beta-blockers are predictors of vasodilatory shock after cardiac surgery. Despite the profound microvascular dysfunction seen in diabetes, diabetes has not been identified clinically as a risk factor for vasoplegia. However, diabetes is associated with poor outcomes after cardiac surgery. Some of those adverse outcomes can be explained by the microvascular and macrovascular dysfunction seen in diabetic patients, which are exacerbated by CPB.

In the current issue of the Journal, Sellke and colleagues explore the in vitro response of human atrial arterioles to vasopressin obtained before and after CPB in patients with poorly controlled diabetes and in nondiabetic controls. The in vitro vasoconstrictor response to vasopressin was increased in both diabetic and nondiabetic patients after CPB. The vasoconstrictor response was further amplified in diabetic patients, mediated by an increased expression of the vasopressin V1 receptor in vascular smooth muscle. This study adds to the seminal contributions on the topic of vascular reactivity in diabetic patients by the authors. The authors previously demonstrated that the contractile responses of skeletal muscle arterioles to phenylephrine and vasopressin were decreased after CPB in humans, while mesenteric vessels showed an increased response to norepinephrine and vasoconstriction with vasopressin remained unchanged. Khan and colleagues suggested that the use of vasopressin as a vasopressor after CPB may reduce the risk of mesenteric ischemia. The current study suggests that vasopressin use after CPB in patients with poorly controlled diabetes and in nondiabetic patients, but to a lesser degree, may be associated with atrial microvascular vasoconstriction and cardiac ischemia. The clinical significance of these findings is unknown, but if the effect of vasopressin is demonstrated in ventricular arterioles, it would raise more concern for adverse clinical implications. However, before changing your selection of vasopressors for the management of patients after cardiac surgery, a few cautionary points should be made.
1. This was a small study that examined only atrial tissue arterioles, with no ventricular arterioles examined.
2. Other vasoconstrictors different than vasopressin were not evaluated. As such, it is possible that catecholamine vasopressor may have a similar or worse effect than vasopressin on atrial arterioles.
3. The increased response to vasopressin was not correlated with adverse outcomes in the present study.
4. Vasopressin has been extensively used in diabetic patients after cardiac surgery.
5. Diabetes has not been identified as a risk factor for adverse outcomes in the trials that examined the use of vasoconstrictors after vasodilatory shock.
6. Additional studies are necessary to confirm these findings and to elucidate their clinical implications.

References