Agent	Mechanism of Action	Initial Dose	Max dose	Contractility	HR	SVR	PVR	Examples of Indications
Dobutamine	Primarily β_1-adrenergic activity	2.5-5 mcg/kg/min	10	↑↑	↑↑↑	↔/↓	↔/↓	HIE- low systolic BP, RV dysfunction
Dopamine	Primarily α-adrenergic effects. Relative effects at different doses are uncertain because of developmental differences in endogenous norepinephrine stores, α-adrenergic, β-adrenergic, and dopamine receptor functions, and ability of neonatal heart to increase stroke volume	5 mcg/kg/min	10	↑	↑↑	↑↑	↑↑↑	Sepsis, no pulmonary hypertension
Epinephrine	Potent stimulator of both α- and β-adrenergic receptors	0.03-0.05 mcg/kg/min	0.3	↑↑↑	↑	↑↑↑	↑↑	Severe heart dysfunction, low BP, HIE
Milrinone	Selective phosphodiesterase III inhibitor, leads to increased intracellular cyclic AMP, increased myocardial intracellular calcium, and increased reuptake of calcium after systole. Vasodilatation is related to increased levels of cyclic GMP in vascular smooth muscle.	0.25-0.33 mcg/kg/min	1	↑↑	↔	↓↓	↓↓	Pulmonary hypertension, normal or high BP, post-ligation cardiac syndrome
Norepinephrine	Primarily α-adrenergic activity resulting in peripheral vasoconstriction, and some β-adrenergic activity leading to inotropic stimulation of the heart and coronary artery vasodilation	0.02 mcg/kg/min	0.3	↑	↑↑↑	↑↑↑	↑↑	Sepsis, low diastolic BP
Vasopressin	Contracts vascular smooth muscle via V1 receptors coupled to phospholipase C, which stimulate the release of vasoconstrictive calcium. In coronary, mesenteric, and cerebral circulations the V1 receptor may lead to vasodilation via increased cyclic AMP leading to increased intracellular nitric oxide.	0.3 milli-units/kg/min	2.5	↔	↔/↓	↑↑↑	↓	Pulmonary hypertension, low BP