Pressors and Inotropes

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KEYWORDS

• Vasopressors • Inotropes • Chronotropes • Hemodynamic effect

KEY POINTS

- Although disease-specific guidelines provide a framework for vasopressor, inotrope, and chronotrope selection and usage, real-world applications demand therapy be tailored to the individual patient.
- Physiologic parameters, such as fluid status or cardiovascular reserve, may alter an individual patient's response to a particular agent.
- Patients with preexisting, poorly controlled hypertension may require higher than normal goal blood pressures to achieve treatment goals.

INTRODUCTION

To effectively treat an aging and increasingly complex patient population, emergency physicians and other acute-care providers must be comfortable with the use of vasopressors, inotropes, and chronotropes. These medicines are used to augment the cardiovascular function of critically ill patients.

Each class of medication produces a different hemodynamic effect. Vasopressors induce peripheral vasoconstriction, increasing systemic vascular resistance (SVR) and mean arterial pressure (MAP). Inotropes increase the force of cardiac contractility, increasing cardiac output and MAP. Chronotropes increase heart rate. Some agents produce only one of these actions, whereas others have multiple effects. For the emergency physician, these agents are used with the explicit goal of preserving vital organ perfusion during acute and severe illness. This article reviews the physiologic receptors targeted by such drugs, common agents used, and specific clinical indications for their use.

PHYSIOLOGY

Vasopressors, inotropes, and chronotropes act on various adrenergic receptors (**Table 1**). Ubiquitous within the smooth muscle of arterial walls, α_1 receptors induce

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Table 1
Commonly used vasopressors, inotropes, and chronotropes

Drug	Action	Common Doses	Adverse Effects
Norepinephrine	$\alpha_1 > \beta_1$	0.01–0.5 μg/kg/min	Tachyarrhythmias, increased myocardial oxygen consumption, myocardial banding necrosis with prolonged infusions
Vasopressin	α_1, V_1, V_2, V_3	0.04 U/min	Possible gastrointestinal hypoperfusion
Dopamine	α_1, β_1, dopa 1	0.5–25 μg/kg/min	Tachyarrhythmias, increased myocardial oxygen consumption
Epinephrine	α_1, β_1, β_2	0.01–0.75 μg/kg/min	Tachyarrhythmias, leukocytosis, increased myocardial oxygen consumption
Phenylephrine	α_1	0.15–0.75 μg/kg/min	Reflex bradycardia
Isoproterenol	$\beta_1 > \beta_2$	0.01–0.02 μg/kg/min	Tachyarrhythmias, flushing, increased myocardial oxygen consumption
Dobutamine	$\beta_1 > \beta_2$	2.0–20 μg/kg/min	Tachyarrhythmias, increased myocardial oxygen consumption, pharmacologic tolerance in prolonged infusions
Milrinone	Phosphodiesterase inhibition	0.3–0.8 μg/kg/min	Headache, hypotension, tachycardia
Levosimendan	Increased calcium-dependent binding of troponin C	0.05–0.2 μg/kg/min	Headache, hypotension, prolonged half-life of active metabolites

arterial vasoconstriction, increasing SVR. They are also present to a lesser extent in the heart and can increase systolic contraction without affecting chronotropy, although the clinical significance of this action is unclear.[1] β_1 receptors predominate in cardiac smooth muscle. They act on the sinoatrial node to produce positive chronotropy and on atrial and ventricular muscle to produce inotropy. Located throughout the body but notably in bronchial smooth muscle, β_2 receptors increase calcium uptake by the sarcoplasmic reticulum, resulting in mild vasodilation and, of particular importance, pulmonary bronchodilation.[2] They are also located in uterine muscle, and are targeted during tocolysis treatment. Dopamine receptors are located in renal, splanchnic, and coronary vasculature and the central nervous system; their actions are complicated and diverse. Vasopressin receptors are located in vascular smooth muscle, the anterior pituitary gland, and the renal collecting duct. Their activation results in vasoconstriction, adrenocorticotropic hormone and prolactin release, and renal water reabsorption.[3]

Although each drug carries its own side effect profile, some adverse effects can be seen with multiple agents. All catecholamines may cause myocardial contraction band