International Journal of Research in Medical Sciences
Bhise PP et al. Int J Res Med Sci. 2025 Mar;13(3):1335-1343
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20250711

Review Article

A comparative study of inotropes and vasopressin in critical care unit

Pranjali P. Bhise*, Pravin F. Gawali, Dhakne G. Sugriv, Akashay S. Bhambare

Department of Pharmacy Practice, Shivlingeshwar College of Pharmacy, Almala, Ausa, Latur, Maharashtra, India

Received: 15 December 2024
Revised: 16 January 2025
Accepted: 01 February 2025

***Correspondence:**
Dr. Pranjali P. Bhise,
E-mail: bhise.pranjali10@gmail.com

ABSTRACT

In critical care, inotropes and vasopressin are key agents for addressing hemodynamic instability. This article evaluates their effectiveness, safety, and clinical outcomes in conditions like septic shock, cardiogenic shock, and post-cardiac arrest syndrome. Inotropes such as dobutamine and epinephrine enhance cardiac performance but may pose risks like arrhythmias and increased myocardial oxygen consumption. Vasopressin, acting as a non-catecholamine vasopressor, promotes vasoconstriction without significantly impacting heart rate or myocardial contractility, often complementing catecholamine therapy to reduce their dosage requirements. Studies indicate vasopressin can lower the reliance on high-dose catecholamines and may decrease cardiovascular complications, though further investigation is needed in specific groups, such as patients with kidney impairment. The discussion underscores the importance of tailoring treatment to individual patient needs and stresses the necessity for more research to refine dosing strategies and identify response predictors. This review supports clinicians in improving the management of critically ill patients with circulatory challenges.

Keywords: Inotropes, Vasopressin, Critical care, Septic shock, Cardiogenic shock, Vasopressors, Hemodynamic management, Catecholamines, Myocardial contractility, Tissue perfusion

INTRODUCTION

Hemodynamic instability poses a significant risk in critically ill patients, requiring prompt and effective pharmacological management to restore circulation and ensure adequate organ perfusion. Among the available therapeutic options, inotropes and vasopressin play critical roles. Inotropes, including agents such as dobutamine, dopamine, and epinephrine, enhance cardiac contractility and boost cardiac output, while vasopressin, through V1 receptor activation, exerts its effects by inducing vasoconstriction. This unique mechanism makes vasopressin a valuable alternative or complementary agent to catecholamines in specific scenarios.

Selecting the appropriate agent involves consideration of the underlying clinical condition, patient-specific factors, and treatment goals. Each class of drug offers distinct advantages and potential drawbacks, underscoring the need for a balanced evaluation of their relative efficacy and safety. Vasopressin, in particular, has gained prominence for its ability to reduce reliance on high doses of catecholamines and mitigate associated risks, especially in managing conditions like septic and cardiogenic shock. Despite this, uncertainties remain regarding its optimal dosing and application in specific patient populations.

Aim of the study

This review aims to critically assess the roles of inotropes and vasopressin in critical care settings by exploring their pharmacological properties, clinical utilities, and outcomes from recent research.

By providing insights into their comparative strengths and limitations, this article seeks to inform clinical decision-making and identify areas for further investigation.

MECHANISM OF ACTION

Inotropes

Inotropes enhance myocardial contractility and cardiac output. Dobutamine stimulates β1-adrenergic receptors, increasing cAMP and calcium influx.

Dopamine acts dose-dependently: stimulating dopaminergic receptors at low doses, β1-adrenergic receptors at moderate doses, and α-adrenergic receptors at high doses.

Epinephrine activates β1, β2, and α1 receptors, improving cardiac output and vascular resistance.

Vasopressin

Vasopressin induces vasoconstriction via V1 receptors and promotes water reabsorption via V2 receptors, increasing blood volume. It bypasses adrenergic pathways, making it effective in catecholamine-resistant states.

CLINICAL INDICATIONS

Inotropes

It is used to improve cardiac output in: cardiogenic shock after myocardial infarction or heart failure exacerbation, septic shock with persistent hypoperfusion despite fluids, and post-cardiac surgery to support cardiac function.

Vasopressin

It is indicated for: septic shock as adjunctive therapy to catecholamines, vasodilatory shock unresponsive to catecholamines, and catecholamine-resistant conditions to stabilize blood pressure.

COMPARATIVE STUDIES AND LIMITATIONS

Septic shock (VASST trial)

Vasopressin reduced catecholamine needs but did not significantly improve survival. Subgroup analysis was limited.

Cardiogenic shock

Inotropes are better studied; vasopressin data is sparse and largely observational.

Adverse effects

Inotropes may cause arrhythmias and high oxygen demand, while vasopressin at higher doses risks ischemia due to excessive vasoconstriction. Long-term effects need more research.

PHARMACOLOGICAL OVERVIEW

Inotropes

Inotropes are drugs that increase myocardial contractility, leading to improved cardiac output.

They primarily work by stimulating adrenergic receptors, enhancing heart function and vascular tone.

Mechanism of action

Beta-adrenergic stimulation

Medications like dobutamine and epinephrine act on β1-adrenergic receptors, increasing intracellular cAMP and calcium influx into myocardial cells, thereby boosting contractility.

Epinephrine also stimulates β2 receptors at lower doses, causing vasodilation, and α1 receptors at higher doses, leading to vasoconstriction and increased vascular resistance.

Dopamine receptor stimulation

Dopamine has a dose-dependent effect: at low doses, it activates dopaminergic receptors, causing vasodilation; at moderate doses, it stimulates β1 receptors to enhance cardiac output; and at high doses, it activates α1 receptors, leading to vasoconstriction.

Norepinephrine

Norepinephrine acts on α1 receptors to cause vasoconstriction and β1 receptors to improve cardiac output.

Common drugs

Dopamine

It is used in shock states such as cardiogenic and septic shock, offering dose-dependent effects on both vasodilation and vasoconstriction.

Dobutamine

A synthetic catecholamine typically used in cardiogenic shock to improve cardiac output without significantly increasing vascular resistance.

Epinephrine

Used in emergencies like anaphylactic shock and cardiac arrest, providing both vasoconstriction and enhanced myocardial performance.

Norepinephrine

The first-line vasopressor in septic shock, providing vasoconstriction and mild inotropic effects to maintain blood pressure and organ perfusion.

Vasopressin

Mechanism of action

Vasopressin is a non-catecholamine vasopressor that binds to V1 receptors in smooth muscle to induce vasoconstriction, increasing systemic vascular resistance. It also acts on V2 receptors in the kidneys to promote water reabsorption, helping to expand blood volume.

Key role

Vasopressin plays a critical role in raising blood pressure, especially in septic shock. Its ability to promote water retention supports blood volume expansion, which can reduce the need for high-dose catecholamines while maintaining perfusion.

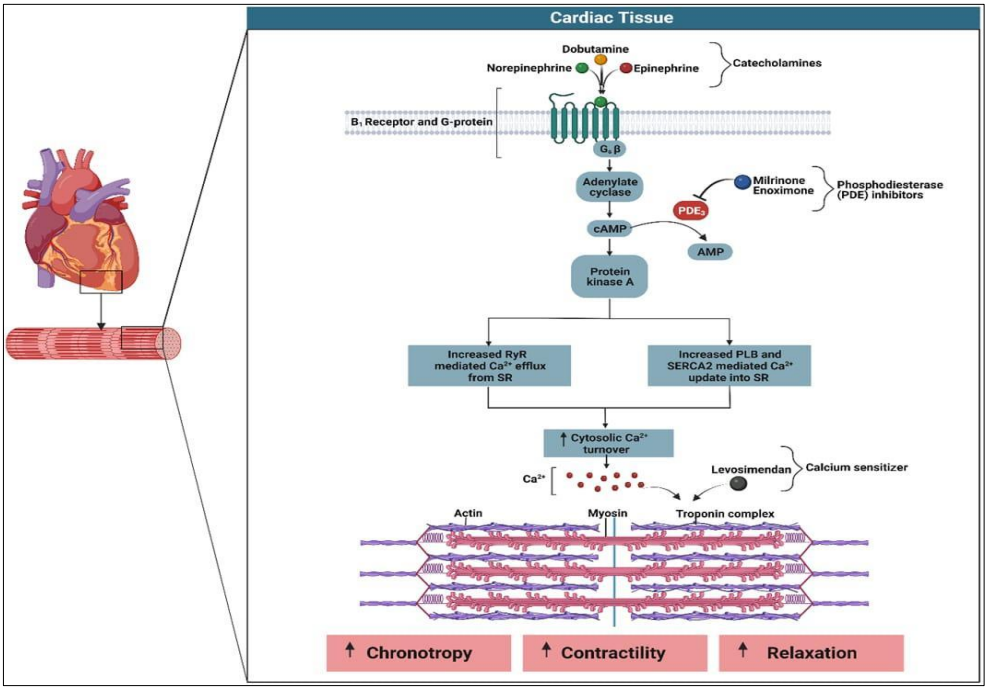

Figure 1: MOA of inotropes.[36]

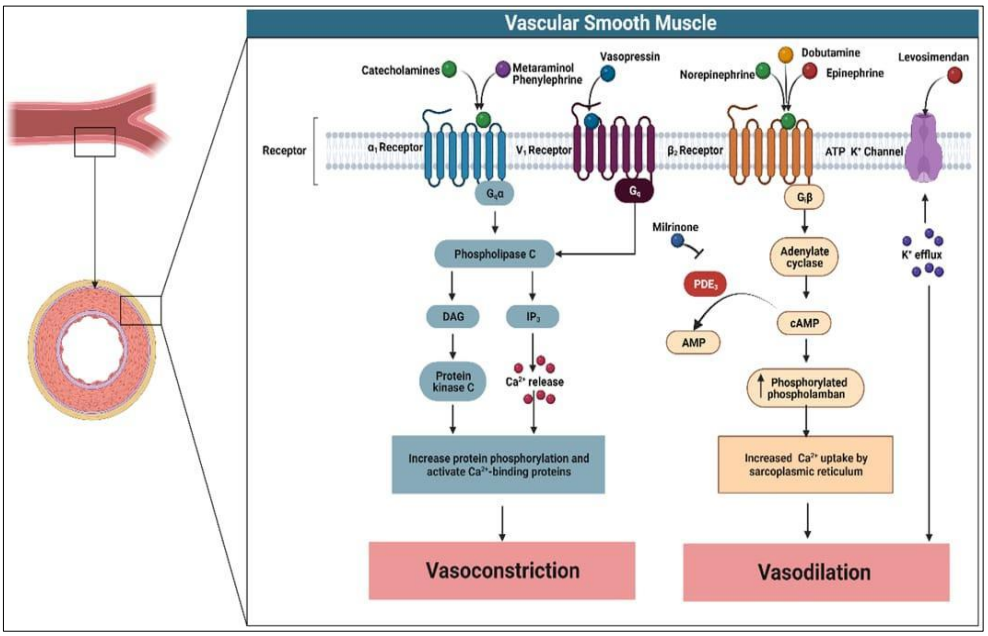

Figure 2: MOA of vassopresin.[37]

INDICATIONS AND CLINICAL APPLICATIONS

Inotropes

Inotropes are crucial in conditions where enhancing cardiac output is essential to ensure tissue perfusion and survival.

Cardiogenic shock

In this state, the heart's inability to pump blood effectively results in poor tissue perfusion. Inotropes such as dobutamine and dopamine are used to increase myocardial contractility, improving cardiac output and hemodynamics.

Acute heart failure

In patients with acute heart failure, where fluid management and diuretics alone are insufficient, inotropes help increase cardiac output, particularly dobutamine, which is commonly used for heart support.

Post-cardiac surgery recovery

After significant cardiac surgery, inotropes like dobutamine and epinephrine are used to stabilize cardiac function and maintain blood pressure, supporting recovery during the early postoperative period.

Vasopressin

Vasopressin is effective in shock conditions where catecholamines are either ineffective or contraindicated.

Septic shock refractory to catecholamines

In septic shock, when patients do not respond to first-line vasopressors like norepinephrine, vasopressin is used as an adjunctive therapy. It promotes vasoconstriction and enhances blood pressure without significantly increasing myocardial oxygen consumption.

Vasodilatory shock in critical care

In conditions such as neurogenic shock or drug-induced hypotension, where vasodilation causes severe hypotension, vasopressin acts as a second-line agent to restore vascular tone and improve hemodynamics.

CLINICAL EVIDENCE: EFFICACY

Inotropes

Clinical evidence supports the use of inotropes in improving cardiac output and organ perfusion, particularly in conditions like cardiogenic shock and acute heart failure.

Cardiogenic shock

In this condition, where the heart struggles to pump blood effectively, inotropes such as dobutamine and dopamine help by increasing myocardial contractility, thereby improving cardiac output. Dobutamine has been found to significantly enhance hemodynamics and tissue perfusion, especially in patients recovering from acute myocardial infarction or heart failure exacerbations.

Acute heart failure

In patients with acute decompensated heart failure, dobutamine and other inotropes increase cardiac output when fluid management and diuretics are insufficient. Short-term studies support their use for symptom alleviation and improving hemodynamics. However, the potential long-term risk of arrhythmias and myocardial oxygen demand limits their prolonged use. Inotropes are particularly effective when myocardial contractility is compromised, but they come with risks such as arrhythmias and elevated myocardial oxygen consumption, especially at higher doses.

Vasopressin

Vasopressin is primarily used for stabilizing blood pressure in conditions like septic shock, especially when patients are resistant to catecholamine therapy.

Septic shock

The VASST trial revealed that adding vasopressin to norepinephrine in septic shock patients reduced the need for high-dose catecholamines and helped maintain blood pressure. It has also been linked to lower mortality rates in catecholamine-resistant patients, with the added benefit of minimizing complications like arrhythmias, which are common with high-dose catecholamines.

Vasodilatory shock

In conditions like neurogenic shock or drug-induced hypotension, vasopressin helps restore vascular tone, providing an effective alternative when adrenergic agents are insufficient or contraindicated.

Vasopressin excels in conditions where vasodilation is the primary issue, such as in septic shock or neurogenic shock, and is particularly useful in cases of catecholamine resistance, where inotropes may fail to achieve sufficient hemodynamic control.

COMPARATIVE SETTINGS

Cardiogenic shock

Inotropes are preferred due to their ability to improve myocardial contractility and cardiac output. Vasopressin is not effective in enhancing cardiac output in this scenario.

Septic shock

While inotropes can support cardiac output, vasopressin is often more effective in addressing hypotension, particularly in cases of catecholamine resistance or adverse effects from high-dose catecholamines.

Post-cardiac surgery

Inotropes are typically used to enhance cardiac output and maintain perfusion, while vasopressin may be added if vasodilation-related hypotension persists.

SAFETY PROFILES

Adverse effects of inotropes

Inotropes play a crucial role in enhancing cardiac output, but they come with notable risks, especially in critically ill patients.

Arrhythmias

One of the primary concerns with inotropes such as dobutamine and epinephrine is the potential to induce arrhythmias. The increase in myocardial workload and elevated intracellular calcium levels can predispose patients to atrial and ventricular arrhythmias, which can complicate the clinical management and worsen outcomes.

Myocardial ischemia

Agents like dopamine and epinephrine can significantly raise myocardial oxygen demand. In patients with preexisting coronary artery disease, this increased demand can lead to myocardial ischemia, potentially resulting in further cardiac injury or infarction, particularly in cases where coronary circulation is compromised.

Tachyphylaxis

Dobutamine and other inotropes can lose their effectiveness over time due to tachyphylaxis, a condition where continued use leads to a diminished response. This phenomenon may require dose escalation, which increases the risk of exacerbating other adverse effects like arrhythmias or myocardial ischemia.

Adverse effects of vasopressin

Although vasopressin is effective in stabilizing blood pressure, it also has its own set of risks.

Ischemic complications

Due to its vasoconstrictive properties, vasopressin can cause ischemic complications, particularly in peripheral regions such as the fingers or toes. In some cases, excessive vasoconstriction may even lead to necrosis in these microvascular areas, especially with higher doses.

Hyponatremia

Vasopressin induces water retention via activation of V2 receptors in the kidneys. This can lead to hyponatremia, especially in patients receiving large doses, as excessive water retention dilutes the sodium concentration in the bloodstream, causing potentially dangerous electrolyte imbalances.

COMPARATIVE RISK-BENEFIT ANALYSIS

When evaluating the use of inotropes and vasopressin in critically ill patients, a careful assessment of their risk profiles is necessary.

Inotropes are most beneficial in conditions where the primary issue is impaired myocardial contractility, such as cardiogenic shock or acute heart failure. However, their use is limited in patients with preexisting coronary artery disease or those at high risk for arrhythmias, as they may exacerbate these issues.

Vasopressin is particularly useful in septic shock or vasodilatory shock, especially in patients who do not respond to or tolerate catecholamines. Despite its advantages in these settings, it comes with risks such as ischemic complications and hyponatremia, which should be carefully monitored in patients with renal dysfunction or peripheral vascular disease.

MECHANISTIC INSIGHTS AND SYNERGISTIC USE

The combination of vasopressin and inotropes is often used in critical care, particularly in conditions like septic shock and cardiogenic shock, to enhance hemodynamic stability. By leveraging the unique mechanisms of both agents, this approach offers comprehensive support for patients with complex circulatory failure.

Rationale for combining vasopressin with inotropes

Reduced catecholamine requirements

High-dose catecholamines, such as norepinephrine or dopamine, are frequently used in shock management but come with significant risks, including arrhythmias, myocardial ischemia, and increased oxygen demand.

Vasopressin, acting through V1 receptors in vascular smooth muscle, induces vasoconstriction to help stabilize blood pressure without causing a significant increase in heart rate or myocardial oxygen consumption. This allows for a reduction in the need for high-dose catecholamines, minimizing their adverse effects.

Complementary mechanisms

Inotropes like dobutamine or epinephrine enhance myocardial contractility, increasing cardiac output and improving perfusion. In contrast, vasopressin works primarily by increasing vascular tone, elevating systemic vascular resistance, and supporting blood pressure. These complementary actions help address both impaired myocardial performance and vascular dysfunction, which are often present together in critical conditions like septic shock or post-cardiac surgery.

Enhanced tissue perfusion

The combination of vasopressin's vasoconstrictive effects and inotropes' cardiac output improvement helps to ensure adequate perfusion to vital organs such as the kidneys, brain, and liver. This is crucial in critically ill patients; as inadequate tissue perfusion can lead to multi-organ failure.

Underlying mechanisms influencing patient outcomes

Vasopressin's vasoconstrictive effects

By activating V1 receptors, vasopressin induces vasoconstriction, raising mean arterial pressure (MAP) and systemic vascular resistance. This helps stabilize blood pressure in shock states, allowing for reduced reliance on catecholamines. Vasopressin also does not increase heart rate or myocardial oxygen demand, making it beneficial in situations where catecholamine-induced tachycardia or arrhythmias could be harmful.

Inotropic support

Inotropes stimulate adrenergic receptors to increase intracellular calcium, improving myocardial contraction and enhancing cardiac output. This supports organ perfusion by ensuring that oxygenated blood reaches critical tissues. Dobutamine, for example, can be particularly effective when low cardiac output contributes to shock, aiding in restoring circulatory function.

Impact on patient outcomes

Combining vasopressin with inotropes can improve hemodynamic stability and organ perfusion in critically ill patients. This approach has been shown to reduce the need for high-dose catecholamines, lower the risk of arrhythmias, and potentially enhance survival rates, especially in septic shock. However, careful monitoring is required to avoid the risk of ischemic complications (from vasopressin) and arrhythmias (from inotropes), ensuring the benefits of therapy outweigh the risks.

Cost-effectiveness

The cost-effectiveness of vasopressin and inotropes in critically ill patients is a crucial factor in treatment decisions, particularly in resource-limited settings. Beyond the direct cost of the drugs, the healthcare burden associated with these therapies, including extended ICU stays, monitoring, and managing adverse effects, significantly influences the overall cost.

DRUG COSTS

Inotropes like dobutamine, dopamine, epinephrine, and norepinephrine are commonly used in critical care and are generally less expensive than vasopressin. Drugs like dopamine and norepinephrine are first-line treatments for septic and cardiogenic shock due to their availability and lower cost. However, their use is associated with higher potential for adverse effects such as arrhythmias and myocardial ischemia, which can lead to longer hospital stays and the need for additional interventions, thus increasing treatment costs.

Vasopressin, a non-catecholamine vasopressor, is costlier than traditional inotropes. However, its role in reducing catecholamine requirements, particularly in septic shock and catecholamine-resistant shock, can result in cost savings by lowering the need for high-dose adrenergic agents and minimizing related complications. As a second-line agent, vasopressin can help reduce overall treatment costs when used alongside inotropes.

Table 2: Inotropes and vasoactive agents.

Agent	Physiologic response	End result	Examples
Inotrope	↑ Cardiac contraction	↑ CO, BP unchanged or ↑	Dopamine, dobutamine, milrinone, adrenaline
Chronotrope	↑ HR	↑ CO, ↑ HR	Isoprenaline, dopamine, adrenaline, dobutamine (higher dose)
Vasopressor	↑ Vascular tone, ↑ SVR and PVR	BP unchanged or ↑, CO unchanged or ↓	Adrenaline, noradrenaline, vasopressin, dopamine (higher dose)
Vasodilator	↓ Arterial and venous Tone, ↓ SVR and PVR	BP unchanged or ↓, CO ↑	Sodium nitroprusside (SNP), nitroglycerin (NTG), milrinone
Inodilator	↑ Cardiac contraction, ↓ SVR and PVR	↑ CO, BP unchanged or ↑	Milrinone, dobutamine, levosimendan
Lusitrope	↑ Diastolic relaxation of ventricles	↑ CO (if diastolic dysfunction present)	Milrinone

HEALTHCARE BURDEN

Both inotropes and vasopressin come with the risk of complications, such as arrhythmias and ischemic events, which can prolong ICU stays and increase healthcare expenditures. ICU resources, such as nursing care, equipment, and laboratory tests, contribute to the overall cost of therapy. When vasopressin is combined with inotropes, it can reduce the duration of vasopressor therapy and minimize the need for intensive monitoring, which can mitigate healthcare costs, especially in high-resource settings.

RESOURCE AVAILABILITY AND TREATMENT CHOICES

In regions with more advanced healthcare infrastructure, vasopressin can be used in combination with inotropes, as it may optimize hemodynamics and reduce the need for high doses of catecholamines. This combination, despite its higher upfront cost, can improve organ perfusion and reduce arrhythmia risks, making it a cost-effective strategy in managing critically ill patients.

In contrast, low-resource regions may prioritize inotropes like dopamine and norepinephrine, which are more affordable but may increase the risk of complications, leading to higher costs in the long run. The higher cost and limited availability of vasopressin may limit its use in these settings, despite its potential benefits in managing severe shock conditions.

LIMITATIONS IN CURRENT KNOWLEDGE

To further optimize the use of inotropes and vasopressin in critical care, several research gaps must be addressed.

Long-term outcomes

While current studies on inotropes and vasopressin mainly focus on short-term efficacy (such as improving hemodynamic and stabilizing blood pressure), there is a notable lack of data on long-term outcomes. Research is needed to assess the impact of these therapies on survival rates, post-ICU recovery, and the prevention of long-term organ damage. This would help determine their broader benefits, including the prevention of long-term complications like organ dysfunction, neurological impairment, and quality of life after critical illness.

Effects in specific populations

Most clinical trials on inotropes and vasopressin have predominantly involved adult populations, leaving out important subgroups such as pediatric and geriatric patients. These populations may exhibit unique responses due to differences in pharmacokinetics, pharmacodynamics, and comorbid conditions. Pediatric patients may metabolize these drugs differently, and geriatric patients, with age-related declines in organ function, may face distinct risks and benefits. More targeted research is needed to understand the safety and effectiveness of these therapies in these specific groups.

Need for large-scale head-to-head trials

Head-to-head trials comparing the efficacy and safety of inotropes and vasopressin in septic shock, cardiogenic shock, and vasodilatory shock are limited. Large-scale studies are needed to compare the effectiveness of combining vasopressin with inotropes versus using either class independently. This would help clarify which approach offers the best outcomes for various shock states and guide clinicians in selecting the optimal therapy. These studies could also address cost-effectiveness, offering insights into the economic impact of using these agents in critical care. By addressing these gaps, we can enhance the understanding and clinical use of inotropes and vasopressin, potentially improving outcomes for critically ill patients across diverse populations.

CONCLUSION

Inotropes and vasopressin are essential in managing critically ill patients, particularly in shock states where maintaining hemodynamic stability is crucial. Inotropes, such as dobutamine, dopamine, and epinephrine, improve myocardial contractility and cardiac output, making them valuable in conditions like cardiogenic shock, acute heart failure, and post-cardiac surgery recovery. These agents are most effective when the primary issue is impaired cardiac contractility, thereby enhancing blood flow and oxygen delivery to vital organs. However, their use carries the risk of adverse effects, including arrhythmias, myocardial ischemia, and tachyphylaxis. On the other hand, vasopressin primarily works as a vasoconstrictor, increasing systemic vascular resistance and mean arterial pressure. It is particularly helpful in septic shock, especially when catecholamines are ineffective or contraindicated. Vasopressin can reduce the need for high-dose catecholamines, thus minimizing complications associated with these agents. However, it carries risks such as ischemic complications and hyponatremia, particularly at higher doses. Both agents serve distinct functions but can be used synergistically to improve patient outcomes. Combining vasopressin with inotropes can help reduce the requirement for high-dose catecholamines, enhance tissue perfusion, and improve hemodynamic stability, particularly in complex shock states.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Meyer MA. Inotropes and Vasopressors in Sepsis: A Comprehensive Review. Critical Care Medicine, 2019;47(3):e175-e184.

2. Perner A. Hydrocortisone Therapy in Sepsis: A Systematic Review and Meta-Analysis of Randomized Trials. Critical Care. 2017;21(1):121.
3. Russell JA. Management of Sepsis. N Engl J Med. 2010;362(11):1055-65.
4. Tessier PA. Vasopressin in Septic Shock: Mechanisms of Action and Clinical Evidence." Frontiers in Medicine. 2020;7:345.
5. Benedict CR. Vasopressin: A Review of its Role in Critical Illness. Journal of Intensive Care Medicine. 2003;18(2):49-61.
6. Krause SA. Vasopressin vs. Norepinephrine for Shock: An Overview of Mechanisms and Outcomes." Critical Care Clinics. 2015;31(3):357-0.
7. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, et al; Early Goal-Directed Therapy Collaborative Group. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345(19):1368-77.
8. Fayad A. Inotropes in Acute Heart Failure: Mechanisms, Clinical Use, and Evidence. Current Heart Failure Reports. 2017;14(5):170-81.
9. Mackenzie IS. Dobutamine and Dopamine in the Treatment of Cardiogenic Shock: A Meta-Analysis. European Journal of Heart Failure. 2014;16(4):363-70.
10. Frey AW. Septic Shock and Catecholamine Use: A Review of Pharmacologic Mechanisms and Impact on Organ Perfusion." American Journal of Physiology-Heart and Circulatory Physiology. 2017;313(6), H1202-H1211.
11. Schneider A. Vasopressin and Inotropes in Shock States: A Pharmacologic and Therapeutic Review. Journal of Clinical Pharmacology. 2020;60(3):399-409.
12. Goldstein DE. The Use of Inotropes and Vasopressors in Critical Care Medicine: A Review of Clinical and Mechanistic Approaches. Journal of Critical Care. 2002;17(4):134-42.
13. Hollenberg SM. Current Concepts in the Use of Vasopressors in Sepsis. Clinical Therapeutics. 2019;41(7):1343-57.
14. Khardori R. Comparative Effectiveness of Inotropes in Shock: A Meta-Analysis of Randomized Controlled Trials. JAMA. 2015;313(14):1460-7.
15. Pratt SL. Inotropes, Vasopressors, and Antiseptic Therapy: A Critical Care Pharmacology Review. Pharmacotherapy. 2016;36(11):1226-37.
16. Abraham E, Kaneko DJ, Shenkar R. Effects of endogenous and exogenous catecholamines on LPS-induced neutrophil trafficking and activation. Am J Physiol. 1999;276:L1-8.
17. Adamson RH, Liu B, Fry GN, Rubin LL, Curry FE. Microvascular permeability and number of tight junctions are modulated by cAMP. Am J Physiol. 1998;274:H1885-94.
18. Albanese J, Leone M, Garnier F, Bourgoin A, Antonini F, Martin C. Renal effects of norepinephrine in septic and nonseptic patients. Chest. 2004;126:534-9.
19. Albert M, Losser MR, Hayon D, Faivre V, Payen D. Systemic and renal macro- and microcirculatory responses to arginine vasopressin in endotoxic rabbits. Crit Care Med. 2004;32:1891-8.
20. Alexander SPH, Mathie A, Peters JA. Guide to Receptors and Channels (GRAC), 5th Edition. Br J Pharmacol. 2011;164(1):S1-324.
21. Alia I, Esteban A, Gordo F, Lorente JA, Diaz C, Rodriguez JA, et al. A randomized and controlled trial of the effect of treatment aimed at maximizing oxygen delivery in patients with severe sepsis or septic shock. Chest. 1999;115:453-61.
22. Alousi AA, Johnson DC. Pharmacology of the bipyridines: amrinone and milrinone. Circulation. 1986;73:III10-24.
23. Altenburg SP, Martins MA, Silva AR, Cordeiro RS, Castro-Faria-Neto HC. LPS-induced blood neutrophilia is inhibited by alpha 1-adrenoceptor antagonists: a role for catecholamines. J Leukoc Biol. 1997;61:689-94.
24. Annane D, Vignon P, Renault A, Bollaert PE, Charpentier C, Martin C, et al. Norepinephrine plus dobutamine versus epinephrine alone for management of septic shock: a randomised trial. Lancet. 2007;370:676-84.
25. Arcaroli J, Yang KY, Yum HK, Kupfner J, Pitts TM, Park JS, et al. Effects of catecholamines on kinase activation in lung neutrophils after hemorrhage or endotoxemia. J Leukoc Biol. 2002;72:571-9.
26. Aviado DM, Jr, Schmidt CF. Effects of sympathomimetic drugs on pulmonary circulation: with special reference to a new pulmonary vasodilator. J Pharmacol Exp Ther. 1957;120:512-27.
27. Azoui R, Cuche JL, Renaud JF, Safar M, Dagher G. A dopamine transporter in human erythrocytes: modulation by insulin. Exp Physiol. 1996;81:421-34.
28. Bailey AR, Burchett KR. Effect of low-dose dopamine on serum concentrations of prolactin in critically ill patients. Br J Anaesth. 1997;78:97-9.
29. Ballou LM, Tian PY, Lin HY, Jiang YP, Lin RZ. Dual regulation of glycogen synthase kinase-3beta by the alpha1A-adrenergic receptor. J Biol Chem. 2001;276:40910-6.
30. Barrett LK, Singer M, Clapp LH. Vasopressin: mechanisms of action on the vasculature in health and in septic shock. Crit Care Med. 2007;35:33-40.
31. Barth E, Albuszies G, Baumgart K, Matejovic M, Wachter U, Vogt J, et al. Glucose metabolism and catecholamines. Crit Care Med. 2007;35:S508-18.
32. Bass AS, Kohli JD, Lubbers N, Goldberg LI. Mechanisms mediating the positive inotropic and chronotropic changes induced by dopexamine in the anesthetized dog. J Pharmacol Exp Ther. 1987;242:940-4.
33. Beale RJ, Hollenberg SM, Vincent JL, Parrillo JE. Vasopressor and inotropic support in septic shock: an evidence-based review. Crit Care Med. 2004;32:S455-65.
34. Beani L, Bianchi C, Crema A. The effect of catecholamines and sympathetic stimulation on the

release of acetylcholine from the guinea-pig colon. Br J Pharmacol. 1969;36:1-17.
35. Bearn AG, Billing B, Sherlock S. The effect of adrenaline and noradrenaline on hepatic blood flow and splanchnic carbohydrate metabolism in man. J Physiol. 1951;115:430-41.
36. MOA of inotropes. Available at: https://images.app.goo.gl/6gNLa24brta7rV8QA. Assessed on 17 November 2024.
37. MOA of vassopresin. Available at: https://images.app.goo.gl/rT1tRgwW2XniZfpz5. Assessed on 17 November 2024.

Cite this article as: Bhise PP, Gawali PF, Sugriv DG, Bhambare AS. A comparative study of inotropes and vasopressin in critical care unit. Int J Res Med Sci 2025;13:1335-43.