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The impact of anaesthetic gases on mean pressure in patients with septic shock undergoing surgery

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ABSTRACT

Introduction: Septic shock is a critical condition that involves multi-organ dysfunction along with severe hypotension. Anesthesia, after all, frequently requires surgical intervention. When you do choose the right anesthetic gas, especially in cases involving mean arterial pressure (MAP), you can significantly impact the stability of hemodialysis. Tissue perfusion is maintained by hemodialysis stability, and it optimizes patient outcomes. This study aimed to examine how different anesthetic gases affect mean arterial pressure in patients with septic shock who are having surgery. The study looked at the average blood pressure (MAP) levels of patients with septic shock during and after surgery. These patients received different types of anaesthetic gases while they underwent surgical procedures. The findings demonstrated variable effects on hemodynamic parameters of the anesthetic agents. During intraoperative times, the probe showed some gases linked to greater drops in mean arterial pressure MAP. Some of the gases maintained relatively stable pressure levels. In septic shock cases, the choice of anesthetic agent can significantly impact circulatory dynamics, as indicated by data. Conclusions: The management of anaesthetics in septic shock patients must involve selecting agents that minimise adverse hemodynamic effects. The need for further research is urgent to establish anaesthetic protocols that balance adequate anaesthesia and haemodynamic support in this vulnerable population.

Keywords: anaesthetic gases, septic shock, intraoperative hemodynamics, surgical patients

1. INTRODUCTION

Septic shock is a critical and complex syndrome arising from a dysregulated host response to infection, and can result in life-threatening organ dysfunction. Despite advances in critical care, septic shock remains a significant cause of morbidity and mortality worldwide. Understanding this pathophysiology is crucial for improving clinical management and enhancing outcomes. Detailed interactions between infectious agents, the host immune system, neurohormonal regulation, microcirculatory abnormalities, along with cellular metabolic failure are factors in the process (Bughrara et al., 2020; Petitjeans et al., 2018). Septic shock is defined as persistent hypotension. Adequate fluid resuscitation is not resolving the

hypotension. It is therefore essential to maintain mean arterial pressure (MAP) at a level of 65 mmHg or above through the use of vasopressors. Serum lactate levels are commonly elevated in this state. These levels are indicative of impaired cellular oxygen utilisation. Severity is indicated for prognostic reasons by this key (Bughrara et al., 2020). The varied symptoms and multiple causes of septic shock make it challenging to provide the best perioperative care, notably in cases that require general anaesthesia.

Inhalational anaesthetic gases are vital, as they maintain unconsciousness and analgesia during surgery. However, these agents have large cardiovascular effects instead. Septic shock patients may experience hemodynamic instability. The evidence is indeed clear: they significantly impact systemic vascular resistance, in addition to contracting the myocardium, and regulate autonomic functions. Hemodynamic disturbances from septic shock are somewhat unpredictable. The situation may worsen in that context, or it may improve. A subtle and careful comprehension of the mechanisms is important in making clinical decisions due to this variability (Bara & Janczak, 2023; Horochowska et al., 2019).

Interestingly, septic shock is also believed to cause significant changes in how blood vessels respond, as well as in the function of the autonomic nervous system. These changes, in turn, can affect all the body's reactions to anesthetic gases. Because neurohormonal systems become dysregulated, incorporating aberrations in sympathetic nervous system tone as well as catecholamine sensitivity, that complex interaction affects mean arterial pressure throughout anesthesia (Petitjeans et al., 2021). Administering anesthetic gas requires selecting precise dosages with thoroughly monitoring to ensure safety and effectiveness. Because these factors ensure both safety as well as effectiveness, the process is smoother and more reliable for everyone involved. These factors can significantly affect outcomes during the surgery period. Sepsis, along with septic shock, has become more common among patients undergoing surgical procedures, as a notably higher risk of complications during the perioperative period comes with that. It seems helpful to examine more closely how anesthetic gases affect cardiovascular parameters, particularly mean arterial pressure, during surgery.

On the molecular level, septic shock is initially triggered by pathogen-associated molecular patterns (PAMPs), like lipopolysaccharide (LPS) from Gram-negative bacteria, along with damage-associated molecular patterns (DAMPs) released from injured host cells. These molecules trigger a cascade that leads to the release of various pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6), as well as other bioactive substances, including nitric oxide (NO) and prostaglandins (Bughrara et al., 2020).

The inflammatory response is a crucial factor in the development of septic shock. Endothelial integrity becomes disrupted, and vascular permeability increases, allowing plasma to extravasate into the interstitial spaces. Thus, inflammation gets deregulated, and hypovolemia occurs relatively speaking. The coagulation cascade can be abnormally activated simultaneously, resulting in disseminated intravascular coagulation (DIC) and microvascular thrombosis, which can exacerbate tissue hypoxia (Bughrara et al., 2020).

Vascular tone, as well as barrier function, along with hemostasis, are all maintained by the vascular endothelium. This lining is a crucial component. Inflammatory mediators have an impact on the process of endothelial activation. This impact results in subsequent injury during instances of septic shock. Disruption substantially influences the equilibrium between forces that vasodilate and vasoconstrict, as pro- and anticoagulant pathways also affect it. The result of this could be microcirculatory abnormalities, such as heterogeneous perfusion and the formation of non-prefaced capillary zones. Cardiac output can increase, but tissue hypoxia can persist due to poor blood flow distribution, worsening metabolic distress (Bughrara et al., 2020).

Recent studies have looked into the role of the sympathetic nervous system (SNS) in septic shock. The current research suggests that SNS activation acts as a way to keep blood pressure stable. However, Petitjeans et al., (2021) believe that the ongoing activity of the sympathetic nervous system may be harmful. Existing literature has shown a link between high catecholamine levels and cases of tachycardia, along with increased myocardial oxygen demand, which can cause heart problems. Long-term adrenergic stress has been shown to harm organs through increased inflammation. It may seem a bit unusual, but one can approach adjusting SNS activity from afar. For example, one can go on to use antihypertensive drugs such as alpha-2 adrenergic agonists. While maintaining blood flow, this strategy aims to mitigate the harmful effects.

Fever is a frequent clinical feature in septic shock, reflecting the inflammatory milieu but also imposing metabolic stress. Petitjeans et al., (2018) hypothesize that alpha-2 adrenergic agonists not only attenuate sympathetic hyperactivity but also play a role in modulating fever and the cytokine storm associated with sepsis and severe acute respiratory distress syndrome (ARDS). By dampening excessive neuroimmune activation, these agents may improve hemodynamic stability and organ function, representing a promising adjunct therapy in septic shock management.

A characteristic of septic shock pathophysiology is cellular metabolism dysfunction, notably impairment of mitochondria. Inflammatory mediators act to inflame, oxidative stresses then oxidize, microcirculatory insufficiencies work to reduce mitochondrial oxidative phosphorylation, ATP acts to diminish production, and apoptotic pathways start to activate. "Sepsis-induced mitochondrial dysfunction" compromises the cellular energy supply. This does exacerbate organ dysfunction as well as shock progression (Bughrara et al., 2020).

The process of septic shock starts with a strong activation of the innate immune system. This leads to the significant release of pro-inflammatory cytokines, chemokines, and other substances. This generates a systemic inflammatory response syndrome (SIRS), which, in septic shock, becomes dysregulated and perpetuates a vicious cycle of cellular and tissue injury (Bughrara et al., 2020). Localized infection quickly worsens into general damage, since activated neutrophils as well as monocytes impair microvascular integrity, also endothelial cells are damaged, and tissue factor is released, which exacerbates inflammation and then coagulation. Septic shock features an imbalance. Responses that are pro-inflammatory as well as anti-inflammatory are not balanced within this condition. Anti-inflammatory mediators such as interleukin-10 and transforming growth factor-beta increase as a feedback mechanism. However, in many patients, the initial hyper-inflammatory phase is followed by, or overlaps with, a state of immunosuppression or "immunoparalysis," increasing the risk of secondary infections and impaired pathogen clearance (Bughrara et al., 2020).

It has been suggested that widespread endothelial activation may be a hallmark of septic shock. It is thought that endothelial cells control vascular tone, leukocyte trafficking, and blood coagulation. It has been informed that the inflammatory milieu associated with sepsis may increase endothelial permeability and disrupt the balance between vasodilators (e.g., nitric oxide, prostacyclin) and vasoconstrictors (e.g., endothelin-1, angiotensin II). This may cause capillary leakage, profound hypotension, and peripheral vasodilation. Symptoms of microvascular dysfunction can persist even when normal blood pressure appears within range. The condition has perfused and non-perfused capillaries, in addition to tissue hypoxia. It also impairs the extraction of oxygen. It has been suggested that "microvascular shunting" may potentially contribute to multiple organ failure.

Inflammation and coagulation are closely linked during septic shock. Pro-coagulant changes, triggered by tissue factor expression and decreased activity of natural anticoagulants like protein C and antithrombin, can cause widespread fibrin deposition and platelet aggregation. Suppression of fibrinolysis may lead to microthrombi formation, worsening tissue ischemia. In severe cases, it can result in disseminated intravascular coagulation (DIC), increasing the risk of complications and mortality.

Recent studies, notably Petitjeans et al., (2021), challenge this old view, providing fresh perceptions. Alpha- and beta-adrenergic receptors may undergo desensitization under prolonged adrenergic stimulation, some researchers suggest. This desensitization may induce "vasoplegia." Vascular tone then fails to respond to high doses of vasopressors, including norepinephrine (Petitjeans et al., 2021; Yuan & Chen, 2024).

Research has explored the use of adrenergic modulators, notably alpha-2 agonists (e.g., clonidine, dexmedetomidine), to "reset" the sympathetic system. These agents have been observed to decrease central sympathetic outflow, reduce noradrenaline requirements, and may improve vascular reactivity by permitting receptor resensitization (Petitjeans et al., 2021; Petitjeans et al., 2018; Fathy et al., 2021). Experimental and early clinical data suggest that alpha-2 agonists may facilitate more effective control of blood pressure, microcirculation, and inflammation, as well as modulate fever and metabolic expenditure in severe sepsis and ARDS (Petitjeans et al., 2018; Saruhan et al., 2021).

Septic shock may impair mitochondrial function since it reduces ATP generation as well as increases oxidative/nitrosative stress, and favours apoptosis or necrosis. Adequate oxygen delivery will not prevent even this "cytopathic hypoxia," so mitochondrial-targeted therapies may be valuable ones for the future (Bughrara et al., 2020).

These pathophysiological processes can cause failure of multiple organ systems. It has been suggested that the interplay between inflammation, coagulopathy, and impaired autoregulation may have an impact on prognosis and treatment response (Bughrara et al., 2020; Petitjeans et al., 2018, 2021; Zuccari et al., 2020).

It is important to try to understand that volatile inhalational anesthetics are like anesthetic gases, which can be key for modern anesthesia methods. The function of the heart can affect it greatly. Blood vessels also share this possibility. It is their function. Various factors at the molecular, cellular, and systemic levels cause these effects on the heart and circulatory system. This is truly important for risk managers so they manage risk prior to, during, as well as after surgery, notably in patients with heart disease (Bara & Janczak, 2023; Horochowska et al., 2019).

Inhalational anesthetics such as sevoflurane, isoflurane, desflurane, and halothane have been observed to modulate cardiovascular physiology via several mechanisms: Cell Membrane Interactions: These agents integrate into lipid bilayers, which may in turn result in

alterations to membrane fluidity and the influence of embedded proteins, such as ion channels and receptors. Horochowska et al., (2019) demonstrated some disruptions in model biological membranes, which impacted cardiomyocyte excitability and vascular cell signalling.

Ion Channel Modulation: It is thought that volatile anesthetics regulate ion flux by modulating sodium, potassium, and calcium channels, which are critical for cardiac action potentials and vascular smooth muscle contractility. It has been informed that the inhibition of L-type calcium channels may contribute to adverse inotropic and vasodilatory effects (Horochowska et al., 2019).

Receptor Modulation: It is believed that these anesthetics influence adrenergic and cholinergic receptor activity, thereby tempering autonomic cardiovascular reflexes and altering heart rate and vascular tone (Horochowska et al., 2019; Lesar et al., 2022). Volatile anaesthetics limit calcium availability and depress mitochondrial function in cardiomyocytes. It has been demonstrated that halothane is notably potent in this regard, while newer agents such as sevoflurane and desflurane have attenuated myocardial depressive properties (Gelb & Vreede, 2024; Horochowska et al., 2019).

The heart rate responses of the participants varied according to their respective agents. Desflurane, along with isoflurane, can cause a fast heart rate, while halothane can cause a slow heart rate. Sevoflurane typically has neutral effects or a slight increase in heart rate at higher doses. Especially in patients with altered electrophysiological substrates, consideration of potential proarrhythmic effects is needed (Lesar et al., 2022).

Researchers found that volatile anaesthetics expand coronary vessels, a process that possibly steals blood from weakened coronary beds (Gelb & Vreede, 2024). The effects on the lungs are usually modest in nature. However, in some people, the right ventricle may be affected as well (Horochowska et al., 2019).

Cardiovascular profiles as well as specific anaesthetic agents are investigated by the present study. Halothane, being a vasodilator along with a potent myocardial depressant, can induce bradycardia, so that its use remains limited to regional administration (Gelb & Vreede, 2024).

The following substances are to be examined: Desflurane and Isoflurane. The substance vasodilates, and it also rarely depresses the myocardium. Reflex tachycardia often happens because of this (Wesołowski et al., 2023).

Sevoflurane: Subject profile was balanced, we found, since myocardium was mildly depressed and heart rate was stable (Horochowska et al., 2019).

Enflurane: Because of the fact that it can cause hypotension as well as arrhythmia, its use is restricted (Wesołowski et al., 2023).

Various patient factors, including age, comorbidities, and pharmacogenetics influence the cardiovascular effects of anesthetic agents. Administer anaesthetic agents with caution in patients who are elderly, those with mast cell disorders, and pediatric patients. A risk involves altered haemodynamic responses plus allergic reactions. Concerns exist that chronic occupational exposure to anaesthetic gases causes subclinical cardiovascular toxicity (Bara & Janczak, 2023). It has been proven that anaesthetic gases do significantly impact cardiovascular stability in septic shock patients who are undergoing surgery because they exacerbate hypotension. Volatile anaesthetics relax smooth muscle and impair calcium channel function to decrease systemic vascular resistance and cause negative inotropy (Bara & Janczak, 2023; Horochowska et al., 2019). It is clear that autonomic dysregulation accompanies septic shock. Additionally, this dysregulation weakens regulatory systems. This is characterized by excessive sympathetic nervous system activity and decreased receptor sensitivity. It is well-documented that this results in regulatory systems such as baroreflex-mediated adjustments to heart rate and vascular tone being undermined. This worsens haemodynamic instability during anaesthesia (Petitjeans et al., 2021).

Total intravenous anesthesia is what ensures hemodynamic stability in just a few clinical contexts. Norepinephrine provides timely vasopressor support afterward. Adequate perfusion is maintained, therefore (Petitjeans et al., 2021). Through continuous, invasive hemodynamic monitoring, hypotensive episodes can be detected as early as possible. For the management of all of these episodes, it relies on this detection. This method, which may involve catheterizing arteries as well as measuring cardiac output, most effectively addresses the issue (Bughrara et al., 2020).

2. REVIEW METHODS

This review article was conducted under the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure a transparent and reproducible literature synthesis process.

Search Strategy

A comprehensive literature search was conducted using multiple scientific databases, including PubMed, Scopus, Web of Science, and Google Scholar, encompassing publications from their inception through January 2025. The database queries combined keywords along with MeSH terms related to the impact of anaesthetic gases on mean arterial pressure in patients with septic shock when they underwent surgery (e.g., "anaesthetic gases," "mean arterial pressure," "septic shock," "perioperative management," "hemodynamics," "surgery"). In order to maximize the retrieval of relevant studies, Boolean operators such as "AND" and "OR" were applied.

Studies were included if it was found that they did meet the following criteria: (1) original research articles, reviews, clinical trials, or case series addressed at least one cardiovascular or hemodynamic effect of inhalational anesthetics in adult patients or in pediatric patients diagnosed with septic shock; (2) focus was given mainly to perioperative settings or to intraoperative settings involving surgical interventions. Studies also needed to meet these criteria: (3) data and discussions related to mean arterial pressure alterations or hemodynamic stability were reported, and (4) they were available in English or Polish.

The exclusion criteria were as follows: (1) studies did not relate to septic shock or anesthesia (e.g., non-surgical ICU sepsis management had no anesthetic context); (2) articles focused exclusively on local or regional anesthesia with no systemic effects; (3) publications did not have full text available; also (4) publications were non-peer-reviewed literature like abstracts, letters, or opinion pieces that had no substantive data.

Screening

A total of 1,134 references were identified through systematic database searches and relevant grey literature sources. After the removal of duplicates both automatically and manually, 872 unique records remained. Two reviewers independently screened titles and abstracts of these records for relevance to the impact of anaesthetic gases on mean arterial pressure in patients with septic shock undergoing surgery. Following this initial screening, 203 articles were selected for full-text review. These full texts were examined in detail against predefined inclusion and exclusion criteria, including the study population, exposure to volatile anesthetics, reporting of hemodynamic outcomes (especially mean arterial pressure), and the rigor of the study design. Disagreements were resolved by consensus or the involvement of a third reviewer. Ultimately, 46 studies met all eligibility criteria and were included in the qualitative synthesis for this review.

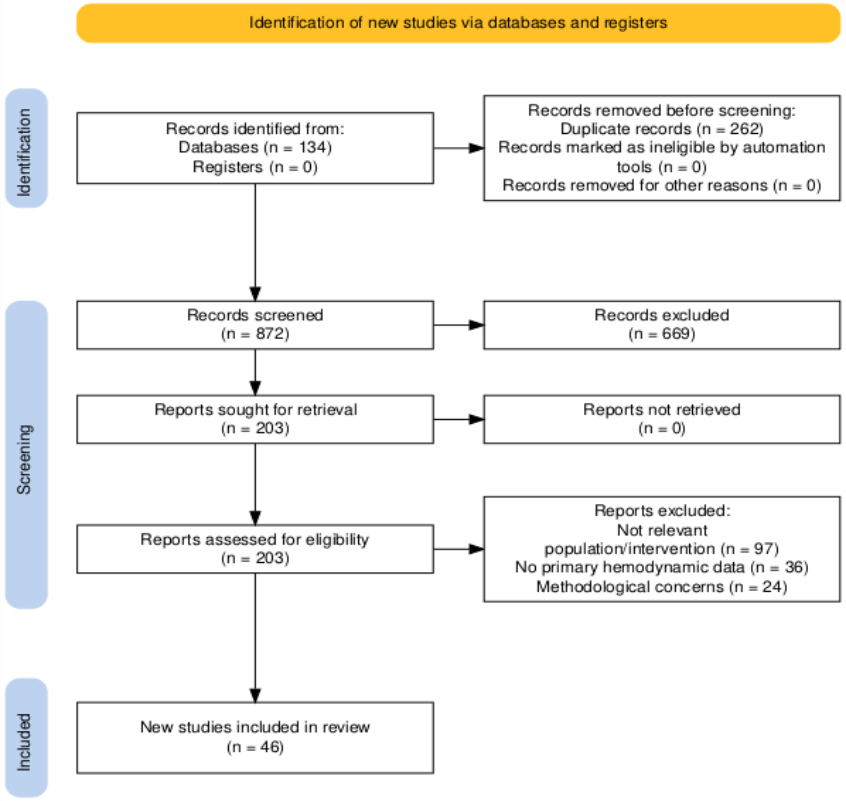

Figure 1. PRISMA diagram

As shown in Figure 1, the PRISMA flow diagram provides a clear and step-by-step overview of how the studies were selected for this review, explaining each stage from the initial identification of studies to their inclusion. The identification phase found 1,134 records from different scientific databases and literature. After removing 262 copies of records, 872 non-copy records moved on to the screening phase. Two different people looked at the articles. They used a list of rules about article inclusion decisions. Because of the fact that they were irrelevant and failed to meet the requirements, 669 records in the end were excluded. They had to pass a qualification test. Reading 203 articles in full was needed for the test. The study intended to establish the relevance as well as the reliability of this procedure. Many reasons explain why 157 reports were cut from the study. These included the lack of specific hemodynamic data, inappropriate patient populations, or inadequate study design. The conclusion, which is quite strong, is based on 46 solid studies.

3. RESULTS & DISCUSSION

Since 2018, much research has shown how anaesthetic gases can affect the heart in patients with septic shock who are having surgery. These studies demonstrate our understanding of diseases (Bara & Janczak, 2023; Bughrara et al., 2020; Carsetti et al., 2023). Blood vessels are affected deeply by inhalational anesthetics like desflurane, isoflurane, and sevoflurane. This action is designed to target tissue and blood vessel muscle cells. These agents have control of various types of ion channels. The channels particularly ease the flow of calcium into the cells. Blood vessel constriction relies on each of these channels. They prevent calcium from entering cells, thereby reducing the amount of calcium inside them. This relaxes the blood vessels, making them wider (Horochowska et al., 2019). Additionally, volatile anaesthetics make the activity of endothelial nitric oxide synthase (eNOS) increase, so they increase the production of nitric oxide (NO) throughout. NO is a key chemical for relaxing blood vessels. This occurs because NO spreads into the muscle cells for the activation of guanylate cyclase. Because of how blood vessels relax with more ease, cyclic GMP levels increase (Bara & Janczak, 2023). Table 1 gives an overview of the key characteristics and main findings for this systematic review.

Table 1. Key characteristics and findings of studies included in the systematic review.

Parameter	Outcome/Description
Number of studies included	46
Databases searched	PubMed, Scopus, Web of Science, Google Scholar
Timeframe	From inception to January 2025
Focus	Impact of anaesthetic gases on mean arterial pressure in septic shock during surgery
Study types included	Original research, systematic reviews, clinical trials, case series
Main agents analyzed	Sevoflurane, Isoflurane, Desflurane, Halothane
Main findings	Volatile anesthetics often worsen hypotension; sevoflurane generally most stable
Hemodynamic outcomes observed	MAP changes, need for vasopressors/inotropes, intraoperative hypotension
Recommendations	Use the most hemodynamically stable agent, individualized monitoring is essential

No synthesis triggered by inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-α) and interleukins, may cause widespread systemic vasodilation in septic shock (Bughrara et al., 2020). Using inhalational anesthetics in this situation may worsen low blood pressure by making the blood vessels more relaxed. This can significantly lower systemic vascular resistance (SVR) and decrease mean arterial pressure (MAP). As a result, it may create challenges for maintaining stable blood flow (Petitjeans et al., 2021).

Volatile anesthetics may have the potential for worsening hypotension in preclinical animal models of sepsis through specific mechanisms, which may require that clinicians augment vasopressor support to maintain perfusion (Petitjeans et al., 2021). There is also some evidence from clinical case series that intraoperative hypotension can occur in septic shock patients receiving volatile agents, with episodes of MAP falling below the critical threshold of 65 mmHg. It has been informed that this value may be associated with an increased risk of organ hypoperfusion and failure (Bughrara et al., 2020; Carsetti et al., 2023). It should be noted that vasodilation can

occur depending on the concentration of the anesthetic, as well as the patient's sensitivity. Precise titration matters greatly. Additionally, it is essential to continuously monitor patients' hemodynamic status.

Volatile anesthetics do possess negative inotropic properties and also these properties greatly affect cardiovascular function during septic shock. These molecular investigations did show some results. The results indicate that these agents disrupt mainly myocardial excitation-contraction coupling since they inhibit L-type voltage-dependent calcium channels. This leads to reduced calcium entry when there is a cardiac action potential as well as diminished calcium-induced calcium release from the sarcoplasmic reticulum (Horochowska et al., 2019). It is believed that this calcium deficit may impair the activation of contractile proteins, potentially weakening myocardial contraction strength. Volatile anesthetics might disrupt components inside the electron transport chain, specifically complexes I as well as III, impacting mitochondrial function. Adenosine triphosphate production is suggested to be subsequently reduced due to this (Lesar et al., 2022). People think the bioenergetic deficit exists. Cardiac function is further compromised by its presence it. A form of cardiomyopathy that is distinct may be contributed to by septic shock independently. Cytokines depress the myocardium with mitochondria malfunction, and autonomic nerves dysregulate, characterizing this cardiomyopathy form (Bughrara et al., 2020). When volatile anesthetics are added to this impaired cardiac milieu, their intrinsic myocardial depressant effects can potentially make contractile dysfunction worse. This can result in a decline in cardiac output, which in turn can lead to further reductions in MAP and potentially compromise vital organ perfusion.

Clinical observational studies support these mechanistic insights by showing that patients with septic shock who undergo surgery using volatile anesthetics often need higher doses of inotropic agents, such as dobutamine or epinephrine. This is necessary to counteract the myocardial depression caused by the anesthetics and to maintain adequate perfusion pressure. Septic shock patients undergoing surgery means for these patients that volatile anesthetics will often require higher doses for inotropic agents, such as dobutamine or epinephrine. Because anesthetics depress the myocardium, addressing this problem is necessary. Sustaining sufficient perfusion pressure is furthermore required (Carsetti et al., 2023; Bughrara et al., 2020). It is also worth noting that these patients may experience increased risk for myocardial ischemia.

It is thought that septic shock is marked by profound disturbances in autonomic nervous system function, involving multiple overlapping mechanisms that impair cardiovascular homeostasis. One central feature that has been observed is of baroreceptor reflexes reducing in sensitivity, as they typically detect blood pressure fluctuations then initiate compensatory sympathetic activation in order to restore vascular tone and heart rate (Petitjeans et al., 2021). In cases of septic shock, this reflex arc is less effective to some degree. Inflammation may induce dysfunction of afferent signalling pathways along with autonomic fibres possibly becoming efferent due to this.

Excessive sympathetic nervous system activity, initially compensatory so it seems, can also cause catecholamine receptor desensitization and downregulation, which may lower tissue responsiveness to vasopressors given endogenously and exogenously. This desensitization may cause vessels to constrict, vessels to be paralyzed, also blood pressure to become low (Petitjeans et al., 2021; Carsetti et al., 2023). Volatile anesthetics have been known to worsen these dysregulations by sympatholytic effects both central also peripheral. Brainstem regions that control sympathetic outflow might have activity of their nerve cells lowered by them. They might help control the release of neurotransmitters. They may also assist in signaling receptors in tissues that affect the heart and blood vessels (Bara & Janczak, 2023). These effects may have a bearing on critical compensatory responses such as reflex tachycardia and vasoconstriction, which are essential to counteract drops in MAP during surgery.

Studies show that this problem makes it hard to keep the proper blood pressure during anaesthesia for patients with sepsis. It is clear that the effects of different amounts of volatile anaesthetics vary; sevoflurane tends to have relatively stable effects on the nervous system, with less effect on the sympathetic nervous system, whereas isoflurane and desflurane tend to cause more pronounced sympathetic inhibition (Bara & Janczak, 2023; Petitjeans et al., 2018). This highlights the importance of selecting the appropriate treatment for each patient, tailored to their unique health and nervous system needs.

Much research has looked at how patients with septic shock who have been exposed to volatile anaesthetics during surgery have changes to their blood flow. This group is more likely to have low blood pressure during surgery, often requiring more intensive treatment with medicines to raise blood pressure (Bughrara et al., 2020; Carsetti et al., 2023). Research has shown that prolonged or repeated intraoperative hypotension (low blood pressure during surgery) is linked to negative results after surgery, including higher rates of organ failure, longer stays in intensive care, and higher mortality (Bughrara et al., 2020). These findings show how important it is to keep blood pressure steady during septic shock surgery and the adverse effects that can happen if blood pressure support is not given.

On the other hand, planned studies involving medical procedures demonstrate the advantages of carefully adjusting the anesthetic dose and continuously monitoring blood pressure. Techniques such as putting a tube into an artery for measuring blood pressure as well as checking the amount of blood pumped from the heart each minute allow detection in addition to treatment of low blood pressure that means medicines plus fluids can be used correctly (Bara & Janczak, 2023; Petitjeans et al., 2021). Adopting these rules has been associated with improved blood flow in organs, a reduced risk of acute kidney injury, and better overall surgical outcomes in patients with septic shock.

Research comparing different types of anaesthetics shows that they can have very different effects on the heart and blood vessels. Halothane, which is not something used all that often now in high-resource settings, can still have a real effect upon the heart since it slows that heart down, making the heart less fit for use for those cases of septic shock (Gelb & Vreede, 2024). Isoflurane and desflurane cause the blood vessels to dilate significantly. This may sometimes trigger that natural stress response of the body, and this response may cause the heart to beat faster, but also help to keep the heart functioning well (Wesołowski et al., 2023). Sevoflurane remains a type of anesthetic gas. It is used to induce sleep for surgery. Anesthesia is needed for unstable patients having sepsis, a nasty infection (Horochowska et al., 2019). To reduce blood pressure problems, exploration of new drugs and methods, including total intravenous anesthesia (TIVA), has occurred; however, the evidence that supports this approach is currently limited (Carsetti et al., 2023).

Recent studies suggest that using alpha-2 adrenergic agonists, such as dexmedetomidine or clonidine, in combination with other treatments may help improve blood pressure control and reduce sympathetic overactivity in patients with septic shock. Early trials suggest that these medications reduce the need for additional medications to manage blood pressure and inflammation when used both during and after surgery. However, more studies are needed to confirm this (Petitjeans et al., 2018; Mokhlesian et al., 2025).

All these findings show that anaesthetic gases have a significant effect on blood pressure and the body's overall response to surgery in patients with septic shock (Table 2). The combination of blood vessel widening, heart muscle weakening, as well as the body's natural regulatory system not working so well creates problems requiring anaesthetic management that is extraordinary, careful selection of drugs, continuous blood pressure monitoring, together with medicines used proactively for the purpose of improving patient outcomes, and reducing problems during and after surgery (Bara & Janczak, 2023; Bughrara et al., 2020; Petitjeans et al., 2021; Carsetti et al., 2023).

Table 2. Hemodynamic Effects by anaesthetic gas in septic shock.

Anaesthetic Gas	Hemodynamic Effects / Mechanisms	Clinical Implications	Key References
Sevoflurane	Moderate vasodilation; increases NO via eNOS; less pronounced sympathetic inhibition; inhibits L-type Ca2+ channels	Relatively stable autonomic effects; risk of hypotension; requires monitoring and titration; may need inotropes	Bara & Janczak 2023; Horochowska et al., 2019; Bughrara et al., 2020
Isoflurane	Pronounced vasodilation; significant sympathetic inhibition; inhibits L-type Ca2+ channels	Higher risk of hypotension; may require increased vasopressors; possible compensatory tachycardia	Petitjeans et al., 2021; Wesołowski et al., 2023
Desflurane	Strong vasodilation; marked sympathetic inhibition; can cause compensatory tachycardia	Higher risk of MAP drop; increased vasopressor and inotrope needs	Petitjeans et al., 2021; Wesołowski et al., 2023
Halothane	Negative inotropic; slows heart rate; vasodilation; mitochondrial depression	Not preferred in septic shock due to cardiac depression; risk of bradycardia and hypotension	Horochowska et al., 2019; Gelb & Vreede 2024

This review thoroughly explains how anaesthetic gases affect blood pressure in patients with septic shock who are having surgery. It shows how anesthetics work and how they interact with the body's natural responses to infection. These gases worsen heart problems for people who have sepsis, as studies show from patients in hospitals and from animals. Studies about how the drugs work also show this finding. The blood vessels, heart, and nervous system all simultaneously decrease in response to the gases (Bara & Janczak, 2023; Bughrara et al., 2020; Petitjeans et al., 2018, 2021).

The anesthetic gases cause widening within the body's blood vessels. An accumulation of nitric oxide in the body can lead to blood vessel issues, making it hard to maintain stable blood pressure (Horochowska et al., 2019; Bara & Janczak, 2023). Animal and human studies indicate that this can lead to low blood pressure during surgery, resulting in a higher need for vasopressor drugs such as norepinephrine (Bughrara et al., 2020; Petitjeans et al., 2021).

What is more, the effects of volatile substances on the heart can make sepsis-induced heart problems even worse. These medicines reduce the strength of the heart's contractions and the amount of blood it can pump, which can make heart failure worse (Lesar et al., 2022; Bughrara et al., 2020). A number of observational studies have shown that patients with septic shock require more support in order to increase their heart rate during surgery. Volatile anesthetics still remain important for use in clinical settings each day (Carsetti et al., 2023).

The body's natural stress response, known as the autonomic nervous system, can be impaired in septic shock, making it more challenging to control blood pressure during surgery. The body's natural stress response is less effective in patients with low blood pressure because the body's stress response system is less sensitive, and the body's stress hormones are less effective (Petitjeans et al., 2021; Carsetti et al., 2023). Volatile anaesthetics also reduce the activity of the sympathetic nervous system. This varies depending on the anesthetic, but generally causes the heart to beat more slowly and increases the likelihood of low blood pressure (Bara & Janczak, 2023).

The review explains the main differences between anaesthetics. Halothane has powerful effects on the heart and can cause it to beat more slowly, which makes it not ideal for use in cases of septic shock (Gelb & Vreede, 2024; Wesołowski et al., 2023). Sevoflurane is a good choice because it has a balanced effect on the heart and is less likely to cause problems with the autonomic nervous system (Horochowska et al., 2019). There is also growing interest in other strategies, such as alpha-2 adrenergic agonists, which may help alleviate problems with the body's natural stress response and reduce the need for medications that increase blood pressure (Petitjeans et al., 2018; Mokhlesian et al., 2025). However, more research is needed to confirm these results.

Although there are already some important studies on the effects of anaesthetic gases on the heart in cases of septic shock, significant gaps in our knowledge remain. Many studies have limitations, including small sample sizes, patients with diverse conditions, and unclear reporting of heart rate and blood pressure, particularly regarding patient management during surgery (Carsetti et al., 2023; Bughrara et al., 2020).

Another issue is that evolving definitions and treatment guidelines for septic shock complicate comparisons between studies. This review emphasizes the importance of early support, close patient monitoring, and careful anesthetic selection and dosing for preoperative teams and anesthesiologists.

Long-term trials for anaesthetic gases, along with other therapies, should be key areas for research. Research on dosing thresholds must also be a focus. Patient factors, such as genetics, should also be examined. The integration of molecular biomarkers and advanced hemodynamic monitoring may further enable personalized anesthesia care to optimize safety in this fragile population.

4. CONCLUSION

This review focuses on how volatile anaesthetics interact with the body's natural responses to infection. Gases used for anaesthesia have been shown to cause blood vessels to widen and the heart to beat more slowly. This results in a significant increase in the body's blood pressure, a key component of septic shock. Furthermore, these drugs can prevent the body's natural compensatory mechanisms from adjusting to changes (autonomic compensatory mechanisms) during anesthesia. Among volatile anesthetics, sevoflurane is better for the heart and blood vessels than halothane, isoflurane, and desflurane. This makes it a good choice for safer anesthetic management in cases of septic shock. Some treatments, such as alpha-2 adrenergic agonists, have shown promise in improving issues with the body's natural functions and reducing the need for medications that increase blood pressure. Managing patients before, during, and after surgery requires a variety of approaches, including selecting the appropriate anaesthetics and carefully adjusting the dose. This should be done in conjunction with continuous monitoring of blood pressure and the prompt administration of medications that increase blood flow, if necessary. These measures are crucial to ensure that blood reaches the organs, reduce low blood pressure during surgery, and improve patient outcomes afterwards. Although we have made progress, we still do not fully understand or effectively improve anaesthetic strategies for septic shock.

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Author contributions

Bartosz Zieliński - Conceptualization; writing - rough preparation; supervision
Karol Mateusz Wojnarowski - Writing - rough preparation
Justyna Gręda - Writing - rough preparation

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Conflict of interest

The authors declare that there is no conflict of interest.

Data and materials availability

All data associated with this study are present in the paper.

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