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Dexmedetomidine in cardiac surgery: current evidence and clinical perspectives in adult and pediatric patients

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Keypoints

Current evidence supports dexmedetomidine as a valuable component of modern cardiac anesthesia and intensive care.

Abstract

Cardiac surgery is frequently associated with significant perioperative morbidity resulting from hemodynamic instability, postoperative atrial fibrillation (POAF), acute kidney injury (AKI), systemic inflammatory response, postoperative delirium (POD), and prolonged intensive care unit (ICU) stay. Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, has emerged as an important adjunct in contemporary cardiac anesthesia and critical care because of its unique combination of sedative, sympatholytic, analgesic-sparing, anti-inflammatory, and organ-protective properties.

The purpose of this review is to summarize current evidence regarding the use of dexmedetomidine in adult and pediatric cardiac surgery. Recent randomized controlled trials, systematic reviews, and meta-analyses demonstrate that dexmedetomidine contributes to improved perioperative hemodynamic stability through attenuation of sympathetic activation, reduction of circulating catecholamines, and preservation of myocardial oxygen balance. These effects appear to reduce the incidence of postoperative atrial fibrillation and improve cardiovascular recovery following cardiac surgery.

In addition to its cardiovascular benefits, dexmedetomidine has demonstrated significant neuroprotective potential. Contemporary evidence supports its role in reducing postoperative delirium, particularly among elderly patients undergoing procedures involving cardiopulmonary bypass. Furthermore, emerging data suggest favorable effects on postoperative cognitive recovery through anti-inflammatory, antioxidant, and sleep-preserving mechanisms.

Dexmedetomidine also exhibits clinically relevant cardioprotective, renoprotective, and anti-inflammatory effects. Experimental and clinical studies have shown reductions in myocardial injury biomarkers, inflammatory cytokine release, and the incidence of acute kidney injury following cardiac surgery. In pediatric patients undergoing surgery for congenital heart disease, dexmedetomidine has been associated with improved hemodynamic stability, reduced opioid requirements, effective postoperative sedation, and decreased incidence of junctional ectopic tachycardia.

Current evidence supports dexmedetomidine as a valuable component of modern perioperative cardiac care. However, further large-scale multicenter randomized trials are needed to establish optimal dosing strategies and identify patient populations most likely to benefit from dexmedetomidine-based protocols.

Keywords

Dexmedetomidine; Cardiac Surgery; Cardiopulmonary Bypass; Postoperative Delirium; Postoperative Atrial Fibrillation; Acute Kidney Injury; Neuroprotection; Cardioprotection; Pediatric Cardiac Surgery; Intensive Care.

1. Introduction

Cardiac surgery remains one of the most complex areas of perioperative medicine despite substantial advances in surgical techniques, cardiopulmonary bypass (CPB) technology, myocardial protection strategies, and intensive care management. Patients undergoing cardiac surgical procedures continue to experience significant postoperative morbidity, including atrial fibrillation, acute kidney injury (AKI), myocardial dysfunction, postoperative delirium (POD), cognitive impairment, prolonged mechanical ventilation, and extended intensive care unit (ICU) stay. These complications contribute substantially to increased healthcare costs, reduced quality of life, and postoperative mortality [1,2].

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist that has gained increasing attention in cardiac anesthesia and critical care because of its unique pharmacological profile. Unlike conventional sedative agents, dexmedetomidine provides sedation, anxiolysis, sympatholysis, and opioid-sparing analgesia while preserving respiratory drive. Through activation of central α2-adrenoceptors, particularly within the locus coeruleus, dexmedetomidine produces a state of cooperative sedation resembling physiological sleep and attenuates excessive sympathetic nervous system activity [3].

The perioperative stress response associated with cardiac surgery is characterized by neuroendocrine activation, catecholamine release, systemic inflammation, oxidative stress, and ischemia-reperfusion injury. These mechanisms contribute to myocardial damage, arrhythmogenesis, endothelial dysfunction, and multiorgan impairment. Experimental and clinical studies suggest that dexmedetomidine may mitigate many of these adverse processes through sympatholytic, anti-inflammatory, antioxidant, anti-apoptotic, cardioprotective, renoprotective, and neuroprotective effects [3,4].

Over the past decade, growing evidence has demonstrated that perioperative dexmedetomidine administration may reduce the incidence of postoperative delirium and atrial fibrillation, improve hemodynamic stability, attenuate inflammatory responses, decrease acute kidney injury, and facilitate enhanced recovery after cardiac surgery [5–8]. In pediatric cardiac surgery, dexmedetomidine has also shown promise in reducing opioid requirements, improving postoperative sedation, and decreasing the incidence of junctional ectopic tachycardia [9].

The aim of this review is to provide a comprehensive overview of the current evidence regarding the use of dexmedetomidine in adult and pediatric cardiac surgery, focusing on pharmacological mechanisms, hemodynamic effects, prevention of postoperative complications, organ protection, clinical outcomes, safety considerations, and future research directions.

2. Pharmacology and Mechanisms of Action of Dexmedetomidine

2.1. Chemical Structure and Receptor Selectivity

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist and the pharmacologically active dextroenantiomer of medetomidine. It possesses a remarkably high α2:α1 receptor selectivity ratio of approximately 1600:1, which is substantially greater than that of clonidine (approximately 220:1). This high selectivity contributes to its favorable sedative, anxiolytic, sympatholytic, and analgesic properties while minimizing unwanted α1-mediated cardiovascular effects.

Three α2-adrenoceptor subtypes have been identified: α2A, α2B, and α2C. The majority of the clinically relevant effects of dexmedetomidine are mediated through activation of α2A receptors located in the central nervous system, particularly within the locus coeruleus. Activation of α2B receptors contributes primarily to transient peripheral vasoconstriction, whereas α2C receptors are involved in modulation of neurotransmitter release and behavioral responses [4,26].

2.2. Pharmacokinetics

Dexmedetomidine is administered almost exclusively via intravenous infusion in perioperative and intensive care settings. Following intravenous administration, it demonstrates rapid distribution and predictable pharmacokinetic behavior [4].

The drug exhibits a distribution half-life of approximately 6 minutes and an elimination half-life ranging from 2 to 3 hours in healthy adults. Dexmedetomidine is highly lipophilic and approximately 94% protein bound, primarily to albumin and α1-acid glycoprotein [4].

Metabolism occurs predominantly in the liver through direct glucuronidation and cytochrome P450-mediated hydroxylation, particularly involving CYP2A6. The resulting metabolites are pharmacologically inactive and are excreted mainly by the kidneys. Less than 1% of the administered dose is excreted unchanged [4].

Hepatic dysfunction significantly prolongs dexmedetomidine clearance, whereas renal impairment has relatively little influence on drug elimination because inactive metabolites are produced before renal excretion. (Table 1)

Parameter	Characteristic
Drug class	Highly selective α2-adrenergic receptor agonist
α2:α1 selectivity ratio	Approximately 1600:1
Protein binding	Approximately 94%
Distribution half-life	~6 minutes
Elimination half-life	2–3 hours
Volume of distribution	1.3–2.5 L/kg
Plasma clearance	35–50 L/h
Primary metabolism	Hepatic
Metabolic pathways	Direct glucuronidation and CYP2A6-mediated hydroxylation
Active metabolites	None
Renal excretion of unchanged drug	<1%
Main sedative site of action	Locus coeruleus
Analgesic site of action	Spinal dorsal horn and supraspinal pathways
Respiratory depression	Minimal
Major cardiovascular effects	Bradycardia, reduced sympathetic tone, decreased myocardial oxygen consumption
Anti-inflammatory effects	Decreased TNF-α, IL-6, IL-1β, HMGB-1
Organ-protective effects	Cardioprotection, neuroprotection, renoprotection

Table 1. Pharmacokinetic and Pharmacodynamic Characteristics of Dexmedetomidine. Source: adapted from Weerink et al. [4].

2.3. Mechanisms of Sedation

Unlike γ-aminobutyric acid (GABA)-mediated sedatives such as propofol and benzodiazepines, dexmedetomidine induces sedation through activation of α2-adrenoceptors within the locus coeruleus, the principal noradrenergic nucleus of the brainstem [21,26].

Activation of presynaptic α2 receptors inhibits norepinephrine release, reducing excitatory neuronal activity. This suppression of central sympathetic outflow results in a sleep-like state closely resembling natural non-rapid eye movement (NREM) sleep. Consequently, patients receiving dexmedetomidine typically remain easily arousable and cooperative while maintaining spontaneous ventilation [21].

This unique sedative profile is particularly advantageous in fast-track cardiac anesthesia protocols and during postoperative intensive care management [3,5].

2.4. Analgesic and Opioid-Sparing Effects

Dexmedetomidine exerts analgesic effects through both supraspinal and spinal mechanisms. At the spinal level, activation of α2 receptors in the dorsal horn inhibits release of substance P and glutamate from primary afferent nociceptive neurons [26].

Multiple randomized clinical trials have demonstrated reductions of 20–50% in perioperative opioid requirements when dexmedetomidine is incorporated into multimodal analgesic protocols. Reduced opioid consumption may contribute to lower rates of respiratory depression, postoperative nausea and vomiting, ileus, and opioid-induced hyperalgesia [3,5].

These effects are particularly relevant in cardiac surgery, where early extubation and enhanced recovery protocols have become increasingly important [3,5].

2.5. Sympatholytic and Hemodynamic Effects

One of the most clinically significant actions of dexmedetomidine is attenuation of sympathetic nervous system activity. By reducing central norepinephrine release, dexmedetomidine decreases circulating catecholamine concentrations, heart rate, and myocardial oxygen consumption [4,26].

In cardiac surgical patients, excessive adrenergic activation during sternotomy, cardiopulmonary bypass, and emergence from anesthesia may contribute to myocardial ischemia, arrhythmias, and hemodynamic instability. Dexmedetomidine mitigates these responses and promotes greater perioperative cardiovascular stability [3,10].

However, excessive sympatholysis may also result in clinically significant bradycardia and hypotension, particularly in hypovolemic patients or those receiving concomitant β-blockers [10].

2.6. Anti-inflammatory Effects

Cardiac surgery involving cardiopulmonary bypass triggers a pronounced systemic inflammatory response characterized by activation of leukocytes, complement pathways, and pro-inflammatory cytokines [12,16].

Experimental studies have shown that dexmedetomidine suppresses production of tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), interleukin-1β (IL-1β), and high-mobility group box-1 protein (HMGB-1). Simultaneously, it may enhance anti-inflammatory pathways mediated by the cholinergic anti-inflammatory reflex [12,16,17].

These mechanisms may contribute to reduced postoperative organ dysfunction and improved clinical outcomes [12,17].

2.7. Cardioprotective Effects

Accumulating evidence suggests that dexmedetomidine exerts direct cardioprotective actions through multiple pathways [12,13,36]:

- Reduction of myocardial oxygen consumption.
- Attenuation of ischemia-reperfusion injury.
- Preservation of mitochondrial integrity.
- Inhibition of cardiomyocyte apoptosis.
- Reduction of oxidative stress.
- Suppression of catecholamine-mediated myocardial injury.

These effects may explain the observed reductions in postoperative atrial fibrillation and perioperative myocardial injury reported in several cardiac surgical studies [6,12,13].

2.8. Neuroprotective Effects

Neurological complications remain a major concern after cardiac surgery. Experimental and clinical investigations indicate that dexmedetomidine may provide neuroprotection through suppression of neuroinflammation, reduction of excitotoxicity, maintenance of cerebral autoregulation, and modulation of microglial activation [22,23].

These mechanisms are believed to contribute to the reduced incidence of postoperative delirium observed in numerous randomized controlled trials and meta-analyses [1,2,7,8].

2.9. Renoprotective Effects

Acute kidney injury occurs in approximately 20–40% of patients following cardiac surgery. Proposed renoprotective mechanisms of dexmedetomidine include [14,15]:

- Improved renal blood flow.
- Reduced sympathetic-mediated renal vasoconstriction.
- Attenuation of oxidative stress.
- Suppression of inflammatory pathways.
- Reduction of ischemia-reperfusion injury.

Recent meta-analyses suggest that perioperative dexmedetomidine administration may decrease the incidence and severity of postoperative acute kidney injury in selected cardiac surgical populations [14,15].

3. Hemodynamic Effects and Cardiovascular Stability During Cardiac Surgery

Maintenance of hemodynamic stability remains a fundamental objective during cardiac surgery. Surgical stress, cardiopulmonary bypass (CPB), myocardial ischemia-reperfusion injury, fluctuations in intravascular volume, and neurohumoral activation may lead to significant cardiovascular instability throughout the perioperative period. Dexmedetomidine has attracted considerable interest in cardiac anesthesia because of its ability to attenuate sympathetic activation while preserving cardiovascular homeostasis under carefully controlled administration [3,10].

3.1. Effects on Heart Rate

One of the most prominent cardiovascular effects of dexmedetomidine is heart rate reduction resulting from central sympatholysis and enhanced vagal activity. Activation of presynaptic α2-adrenoceptors in the central nervous system suppresses norepinephrine release, leading to decreased sympathetic tone and reduced sinoatrial node activity [4,26].

In adult cardiac surgical patients, dexmedetomidine consistently lowers perioperative heart rate and reduces tachycardic responses during laryngoscopy, sternotomy, cardiopulmonary bypass, and emergence from anesthesia. This reduction in heart rate may improve myocardial oxygen balance by decreasing oxygen consumption while simultaneously enhancing coronary perfusion during diastole [3,10].

Several studies have demonstrated that dexmedetomidine significantly decreases the incidence of postoperative atrial fibrillation (POAF), one of the most common complications following cardiac surgery. A recent systematic review and meta-analysis reported a significant reduction in POAF among patients receiving perioperative dexmedetomidine (RR 0.82, 95% CI 0.74–0.92), suggesting that suppression of sympathetic activation may contribute to its antiarrhythmic effects [6].

However, excessive suppression of sympathetic activity may result in clinically significant bradycardia, particularly in elderly patients, those with pre-existing conduction abnormalities, or individuals receiving concomitant β-blockers. Therefore, careful dose titration and continuous hemodynamic monitoring are essential [10].

3.2. Effects on Blood Pressure

Dexmedetomidine produces complex and dose-dependent effects on arterial blood pressure. Following administration of a loading dose, transient hypertension may occur because of stimulation of peripheral α2B-adrenoceptors located in vascular smooth muscle. This initial vasoconstrictive phase is usually brief and is followed by a more prolonged hypotensive effect mediated by central sympatholysis and reduced catecholamine release [4,26].

In cardiac surgery, continuous low-dose infusion without a loading dose has become increasingly favored because it minimizes abrupt hemodynamic fluctuations. Contemporary evidence suggests that dexmedetomidine contributes to greater perioperative blood pressure stability compared with conventional sedative regimens [3,10].

Meta-analytic data indicate that dexmedetomidine reduces episodes of perioperative hypertension and tachycardia while maintaining acceptable systemic perfusion pressure. Nevertheless, excessive hypotension may occur in hypovolemic patients or during periods of reduced cardiac output, requiring individualized dosing strategies [10].

3.3. Effects on Catecholamine Release and Vasopressor Requirements

Cardiac surgery is associated with substantial activation of the sympathetic-adrenal axis.

Elevated plasma concentrations of norepinephrine and epinephrine contribute to increased myocardial oxygen consumption, arrhythmogenesis, and systemic inflammatory responses.

Dexmedetomidine significantly attenuates catecholamine release through central α2-adrenoceptor activation. Several clinical studies have demonstrated lower plasma norepinephrine concentrations during CPB and the early postoperative period in patients receiving dexmedetomidine [3,12].

By reducing sympathetic activation, dexmedetomidine may decrease the requirement for exogenous vasoactive medications in selected patients. Although some studies report lower vasopressor and inotrope requirements, results remain heterogeneous because of differences in surgical procedures, patient populations, and dosing protocols [3].

3.4. Hemodynamic Effects During Cardiopulmonary Bypass

Cardiopulmonary bypass induces profound physiological disturbances, including systemic inflammatory activation, endothelial dysfunction, oxidative stress, and alterations in vascular tone. These changes frequently

contribute to hemodynamic instability after separation from CPB.

Dexmedetomidine appears to exert several beneficial effects during CPB. Experimental and clinical investigations suggest attenuation of inflammatory cytokine release, reduction of oxidative stress, preservation of endothelial function, and improved myocardial protection during ischemia-reperfusion injury [12,16,17].

In adult cardiac surgery, perioperative dexmedetomidine has been associated with improved postoperative hemodynamic stability, shorter mechanical ventilation duration, and reduced intensive care unit length of stay [18].

Furthermore, several studies have suggested a reduction in perioperative myocardial injury markers and improved postoperative cardiac performance[12,13] .

3.5. Coronary Artery Bypass Grafting (CABG)

Patients undergoing coronary artery bypass grafting are particularly vulnerable to perioperative myocardial ischemia and arrhythmias. Excessive sympathetic stimulation during induction, sternotomy, aortic manipulation, and postoperative recovery may exacerbate myocardial oxygen imbalance.

Dexmedetomidine has demonstrated favorable effects in CABG patients by reducing heart rate variability, suppressing catecholamine surges, and improving myocardial oxygen supply-demand balance. Clinical studies have reported lower rates of perioperative ischemic events, reduced incidence of postoperative atrial fibrillation, and improved recovery profiles among patients receiving dexmedetomidine-based anesthesia protocols [6,10,18].

Additionally, dexmedetomidine-based opioid-sparing strategies may facilitate early extubation and enhanced recovery pathways, which have become increasingly important components of contemporary cardiac anesthesia practice [3,18]

3.6. Pediatric Cardiac Surgery and Congenital Heart Disease

The hemodynamic benefits of dexmedetomidine may be particularly valuable in pediatric patients undergoing surgery for congenital heart disease (CHD). Children frequently exhibit exaggerated sympathetic responses to surgical stress and are highly susceptible to fluctuations in heart rate, systemic vascular resistance, and cardiac output.

A randomized controlled trial involving children undergoing open-heart surgery with hypothermic cardiopulmonary bypass demonstrated that dexmedetomidine significantly improved perioperative hemodynamic stability and reduced biochemical markers of organ injury compared with control groups [29]. Patients receiving dexmedetomidine exhibited lower heart rates, improved blood pressure stability, and reduced inflammatory responses throughout the perioperative period.

Recent pediatric reviews have further highlighted the ability of dexmedetomidine to reduce opioid requirements, facilitate postoperative sedation, and support fast-track recovery protocols while maintaining favorable cardiovascular stability [30,31].

Moreover, emerging evidence suggests potential cardioprotective effects in children undergoing repair of complex congenital heart defects, including tetralogy of Fallot and cyanotic heart disease, although larger multicenter studies are still required to establish definitive recommendations [30,31].

3.7. Clinical Implications

Current evidence suggests that dexmedetomidine contributes to improved cardiovascular stability during cardiac surgery through modulation of sympathetic activity, reduction of catecholamine release, attenuation of perioperative tachycardia, and stabilization of arterial blood pressure. These effects may translate into lower rates of postoperative atrial fibrillation, improved myocardial protection, and enhanced recovery following both adult and pediatric cardiac surgical procedures [6,12,18].

However, clinicians must remain vigilant regarding dose-dependent bradycardia and hypotension. Optimal benefits appear to be achieved through continuous low-dose infusion strategies tailored to individual patient characteristics and surgical complexity [3,10].

4. Dexmedetomidine and Prevention of Postoperative Arrhythmias

Postoperative arrhythmias remain among the most frequent complications following cardiac surgery and are associated with increased morbidity, prolonged hospitalization, higher healthcare costs, and increased mortality. Among these complications, postoperative atrial fibrillation (POAF) is particularly common, occurring in approximately 20–50% of patients undergoing coronary artery bypass grafting (CABG), valve surgery, or combined procedures [24,25]. The development of POAF has been associated with increased risk of stroke, heart failure, prolonged intensive care unit (ICU) stay, and long-term mortality [24].

The pathophysiology of postoperative arrhythmias is multifactorial and involves autonomic nervous system imbalance, systemic inflammation, oxidative stress, atrial ischemia, electrolyte disturbances, atrial stretching, and surgical trauma. Excessive sympathetic activation during and after cardiac surgery plays a particularly important role in arrhythmogenesis, creating a strong rationale for the use of sympatholytic agents such as dexmedetomidine [3,24].

4.1. Mechanisms Underlying the Antiarrhythmic Effects of Dexmedetomidine

Dexmedetomidine exerts several physiological effects that may contribute to arrhythmia prevention. Through activation of central α2-adrenoceptors, it suppresses sympathetic nervous system activity and reduces circulating catecholamine concentrations. This attenuation of adrenergic stimulation decreases myocardial oxygen demand and stabilizes cardiac electrophysiological activity [4, 26].

Several additional mechanisms may contribute to its antiarrhythmic properties:

- Reduction of sympathetic tone and enhancement of vagal activity.
- Suppression of catecholamine-mediated atrial remodeling.
- Reduction of systemic inflammatory responses.
- Attenuation of oxidative stress and ischemia-reperfusion injury.
- Improvement of myocardial oxygen supply-demand balance.
- Stabilization of atrial conduction pathways.

Experimental studies have demonstrated that dexmedetomidine may reduce atrial electrical instability and suppress triggers responsible for postoperative atrial fibrillation [12, 13].

4.2. Prevention of Postoperative Atrial Fibrillation in Adult Cardiac Surgery

Over the past decade, multiple randomized controlled trials and meta-analyses have evaluated the effectiveness of dexmedetomidine in preventing POAF following cardiac surgery.

A comprehensive meta-analysis by Peng et al. including adult cardiac surgical patients demonstrated a significant reduction in postoperative arrhythmias among patients receiving perioperative dexmedetomidine. The authors reported a lower incidence of atrial fibrillation compared with control groups receiving conventional sedative regimens [6].

Similarly, recent systematic reviews have demonstrated that dexmedetomidine administration is associated with a reduction in POAF risk ranging from 15% to 30%, particularly when continuous perioperative infusion protocols are used [6, 18]. These findings suggest that dexmedetomidine may provide clinically meaningful protection against one of the most important complications of cardiac surgery.

In patients undergoing CABG, dexmedetomidine appears particularly beneficial because perioperative sympathetic activation is a major contributor to postoperative atrial arrhythmias. Several studies have reported reduced incidence of POAF, improved hemodynamic stability, and lower postoperative catecholamine concentrations among patients receiving dexmedetomidine-based anesthesia protocols [6, 10].

4.3. Ventricular Arrhythmias and Other Rhythm Disturbances

Although atrial fibrillation remains the most extensively studied arrhythmia, dexmedetomidine may also influence the occurrence of ventricular arrhythmias. Experimental investigations have demonstrated reduced susceptibility to ventricular tachyarrhythmias through modulation of autonomic balance and attenuation of ischemia-reperfusion injury [12, 13]. Clinical evidence remains more limited; however, several studies have reported lower frequencies of postoperative ventricular ectopy and nonsustained ventricular tachycardia in patients treated with dexmedetomidine [6]. Furthermore, dexmedetomidine has been associated with improved heart rate variability, which is considered a marker of enhanced autonomic regulation and reduced arrhythmogenic risk [11].

4.4. Pediatric Cardiac Surgery

Arrhythmias are also common following surgery for congenital heart disease. Surgical manipulation near conduction pathways, myocardial ischemia, electrolyte disturbances, and inflammatory activation contribute to the development of postoperative rhythm abnormalities in pediatric patients. Several pediatric studies have demonstrated that dexmedetomidine may reduce the incidence of junctional ectopic tachycardia (JET), one of the most important postoperative arrhythmias in children undergoing congenital heart surgery [27]. JET is associated with significant hemodynamic compromise, prolonged ventilation, and increased ICU length of stay.

A meta-analysis by Shuplock et al. demonstrated that perioperative dexmedetomidine significantly reduced the incidence of postoperative JET in pediatric cardiac surgery patients compared with standard care [28]. Additional studies have suggested improved heart rate control, reduced need for antiarrhythmic medications, and enhanced postoperative cardiovascular stability in children receiving dexmedetomidine [29-31].

4.5. Potential Risks and Limitations

Despite its beneficial antiarrhythmic properties, dexmedetomidine is not without cardiovascular risks. Excessive suppression of sympathetic activity may lead to:

- Sinus bradycardia.
- Atrioventricular conduction delays.
- Hypotension.
- Reduced cardiac output in susceptible patients.

These adverse effects are generally dose-dependent and occur more frequently after rapid loading doses or high infusion rates [4, 10]. Consequently, many cardiac anesthesia protocols now favor low-dose continuous infusions without loading doses to maximize benefits while minimizing hemodynamic complications.

4.6. Clinical Implications

Current evidence suggests that dexmedetomidine represents one of the most promising pharmacological strategies for reducing postoperative arrhythmias following cardiac surgery. Its ability to attenuate sympathetic activation, reduce inflammation, and improve autonomic balance appears to contribute significantly to the prevention of POAF and other perioperative rhythm disturbances.

The strongest evidence currently supports its role in reducing postoperative atrial fibrillation in adult cardiac surgery and junctional ectopic tachycardia in pediatric congenital heart surgery [6,27,28]. However, further large-scale multicenter randomized trials are needed to establish optimal dosing regimens and identify patient populations most likely to benefit from perioperative dexmedetomidine therapy.

5. Dexmedetomidine and Neuroprotection: Prevention of Postoperative Delirium and Cognitive Dysfunction

Neurological complications remain among the most challenging adverse events following cardiac surgery. Despite advances in surgical techniques, cardiopulmonary bypass (CPB) technology, and perioperative management, postoperative delirium (POD) and postoperative cognitive dysfunction (POCD) continue to affect a substantial proportion of patients undergoing cardiac procedures. Depending on patient age, comorbidities, and assessment methods, the incidence of POD after cardiac surgery ranges from 15% to 50%, while cognitive dysfunction may occur in up to 30–60% of patients during

the early postoperative period [19 ,20]. These complications are associated with prolonged mechanical ventilation, extended intensive care unit (ICU) and hospital stay, increased healthcare costs, reduced quality of life, long-term cognitive decline, and higher mortality rates [19,32,33]. Consequently, prevention of perioperative neurological injury has become a major focus of contemporary cardiac anesthesia and critical care research.

5.1. Pathophysiology of Delirium and Cognitive Dysfunction After Cardiac Surgery

The development of POD and POCD is multifactorial. Several perioperative mechanisms have been implicated:

- Neuroinflammation induced by cardiopulmonary bypass.
- Cerebral microembolization.
- Ischemia-reperfusion injury.
- Oxidative stress.
- Blood-brain barrier dysfunction.
- Neurotransmitter imbalance.
- Sleep disruption.
- Excessive sympathetic activation.
- Exposure to sedative and opioid medications.

Cardiopulmonary bypass triggers a systemic inflammatory response characterized by increased concentrations of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1β (IL-1β). These mediators may penetrate the blood-brain barrier and activate microglial cells, leading to neuroinflammation and neuronal dysfunction [32]. Advanced age, pre-existing cognitive impairment, prolonged CPB duration, atrial fibrillation, diabetes mellitus, renal dysfunction, and prolonged mechanical ventilation are recognized risk factors for postoperative neurocognitive complications [19, 35].

5.2. Mechanisms of Neuroprotection by Dexmedetomidine

Dexmedetomidine possesses several properties that may contribute to neuroprotection during cardiac surgery.

5.2.1. Modulation of Sympathetic Activity

Activation of central α2-adrenoceptors within the locus coeruleus reduces norepinephrine release and attenuates excessive sympathetic nervous system activation. This mechanism decreases cerebral metabolic demand and reduces neuroendocrine stress responses associated with surgery [4, 26].

5.2.2. Anti-inflammatory Effects

Experimental studies have demonstrated that dexmedetomidine suppresses the release of pro-inflammatory cytokines, including TNF-α, IL-6, and IL-1β, while simultaneously enhancing anti-inflammatory pathways.

Reduction of neuroinflammation is considered one of the principal mechanisms responsible for decreased delirium incidence observed in clinical studies [12, 16, 22].

5.2.3. Preservation of Sleep Architecture

Unlike benzodiazepines and many conventional sedatives, dexmedetomidine induces a sleep-like state that closely resembles physiological non-rapid eye movement (NREM) sleep. Preservation of normal sleep patterns may reduce sleep fragmentation and contribute to lower delirium rates in postoperative patients [21].

5.2.4. Reduction of Neuronal Apoptosis and Oxidative Stress

Animal models have demonstrated that dexmedetomidine reduces oxidative stress, inhibits apoptosis, preserves mitochondrial function, and attenuates ischemia-reperfusion injury within the central nervous system.

These mechanisms may provide additional protection against perioperative neurological injury [22, 23].

5.3. Dexmedetomidine and Prevention of Postoperative Delirium

Among the various potential benefits of dexmedetomidine, prevention of postoperative delirium has become one of the most extensively studied applications in cardiac surgery [1,2,7–9]. A systematic review and meta-analysis by Li et al. demonstrated that dexmedetomidine significantly reduced the incidence of postoperative delirium compared with alternative sedative regimens in adult cardiac surgical patients [1].

More recently, Zhong et al. performed a comprehensive systematic review and trial sequential analysis including randomized controlled trials of cardiac surgery patients. The authors reported a significant reduction in POD incidence among patients receiving dexmedetomidine, supporting its role as an effective preventive strategy [7].

Similarly, a meta-analysis by da Silva et al. published in 2025 confirmed that perioperative dexmedetomidine administration was associated with a significantly lower risk of delirium following cardiac surgery [8].

Several studies suggest that the greatest benefit is observed when dexmedetomidine is initiated intraoperatively and continued during the early postoperative period in the ICU. This approach appears particularly effective in elderly patients, who represent the population at highest risk for delirium development [9, 35].

5.4. Impact on Postoperative Cognitive Dysfunction

Although evidence regarding POCD is less robust than for delirium prevention, several investigations have suggested beneficial effects of dexmedetomidine on postoperative cognitive outcomes.

A growing body of evidence indicates that dexmedetomidine may improve early postoperative cognitive performance by reducing neuroinflammation, preserving cerebral autoregulation, and attenuating neuronal injury [22, 34].

However, while short-term cognitive benefits have been demonstrated in several studies, data regarding long-term neurocognitive outcomes remain inconsistent. Further large-scale prospective trials are needed to clarify whether dexmedetomidine provides sustained protection against cognitive decline following cardiac surgery.

5.5. Neuroprotection in Pediatric Cardiac Surgery

Children undergoing corrective surgery for congenital heart disease are also vulnerable to neurological injury because of cardiopulmonary bypass, circulatory arrest, cerebral hypoperfusion, and inflammatory activation.

Experimental and clinical pediatric studies suggest that dexmedetomidine may reduce neuronal injury markers, improve perioperative cerebral oxygenation, and attenuate neuroinflammatory responses [30, 31]. Furthermore, dexmedetomidine facilitates effective postoperative sedation while preserving spontaneous respiration and reducing opioid requirements, thereby contributing to enhanced neurological recovery in pediatric intensive care settings. Although pediatric evidence remains less extensive than adult data, current findings suggest a promising role for dexmedetomidine as a neuroprotective adjunct in congenital cardiac surgery [30, 31].

5.6. Clinical Implications

The available evidence indicates that dexmedetomidine is one of the most promising pharmacological interventions for prevention of postoperative delirium following cardiac surgery. Its neuroprotective effects appear to result from a combination of sympatholytic, anti-inflammatory, sleep-preserving, antioxidant, and anti-apoptotic mechanisms.

Current evidence most strongly supports the use of dexmedetomidine for delirium prevention in adult cardiac surgical patients, particularly elderly individuals undergoing procedures involving cardiopulmonary bypass. Emerging data also suggest potential benefits in reducing postoperative cognitive dysfunction and improving neurological outcomes in pediatric cardiac surgery [7–9,30,31].

Future research should focus on identifying optimal dosing strategies, timing of administration, and patient populations most likely to benefit from dexmedetomidine-based neuroprotection protocols.

6. Dexmedetomidine and Organ Protection: Cardioprotection, Renoprotection, and Anti-inflammatory Effects

Cardiac surgery, particularly procedures involving cardiopulmonary bypass (CPB), is associated with systemic inflammation, oxidative stress, endothelial dysfunction, ischemia-reperfusion injury, and multiorgan dysfunction. These mechanisms contribute to postoperative myocardial injury, acute kidney injury (AKI), pulmonary dysfunction, neurological complications, and prolonged intensive care unit (ICU) stay. Dexmedetomidine has

attracted increasing interest as a potential organ-protective adjunct because of its sympatholytic, anti-inflammatory, antioxidant, anti-apoptotic, and microcirculatory effects [3,12–18].

6.1. Cardioprotective Effects

Myocardial injury after cardiac surgery is largely mediated by ischemia-reperfusion injury, catecholamine excess, oxidative stress, mitochondrial dysfunction, inflammatory cytokine release, and apoptosis.

Dexmedetomidine may attenuate these mechanisms through activation of α2-adrenoceptors, reduction of sympathetic outflow, and modulation of intracellular survival pathways [12,13].

Several experimental and clinical studies suggest that dexmedetomidine reduces myocardial oxygen consumption by decreasing heart rate and catecholamine release. This effect may improve myocardial oxygen supply-demand balance during high-risk periods such as induction of anesthesia, sternotomy, CPB, aortic cross-clamping, reperfusion, and early postoperative recovery [3, 12].

A meta-analysis evaluating myocardial protection in cardiac surgery under CPB demonstrated that dexmedetomidine was associated with lower postoperative concentrations of cardiac troponin I and creatine kinase-MB, suggesting attenuation of perioperative myocardial injury [12].

In patients undergoing cardiac valve replacement with CPB, dexmedetomidine pretreatment was also associated with reduced myocardial injury markers and improved postoperative recovery parameters [13].

The cardioprotective effects of dexmedetomidine appear to involve multiple mechanisms, including attenuation of ischemia-reperfusion injury, reduction of oxidative stress, preservation of mitochondrial function, suppression of calcium overload, inhibition of cardiomyocyte apoptosis, and downregulation of inflammatory signaling pathways.

These mechanisms may also contribute to the observed reduction in postoperative atrial fibrillation and improved hemodynamic stability reported in several cardiac surgical studies [6,12,13].

6.2. Renoprotective effects

Cardiac surgery-associated acute kidney injury is one of the most clinically important postoperative complications, occurring in approximately 20–40% of patients after procedures involving CPB.

Even mild AKI is associated with increased mortality, prolonged mechanical ventilation, extended ICU stay, and higher healthcare costs [14, 15].

The pathogenesis of AKI after cardiac surgery is multifactorial and includes renal hypoperfusion, non-pulsatile CPB flow, hemolysis, oxidative stress, systemic inflammation, venous congestion, nephrotoxic exposure, and ischemia-reperfusion injury.

Dexmedetomidine may provide renal protection through several mechanisms: reduction of sympathetic-mediated renal vasoconstriction, improvement of renal blood flow, attenuation of inflammatory cytokine release, reduction of oxidative stress, and modulation of tubular cell apoptosis [14–16].

Recent meta-analyses have suggested that perioperative dexmedetomidine administration may reduce the incidence of AKI in adult cardiac surgical patients.

A 2025 systematic review including 16 randomized controlled trials and 2,882 patients found that dexmedetomidine significantly reduced postoperative AKI and was associated with shorter ICU stay, reduced mechanical ventilation duration, and shorter hospital stay, although its effect on mortality remained inconclusive [14].

Another meta-analysis of randomized trials similarly reported that intraoperative dexmedetomidine decreased AKI incidence after cardiac surgery, while emphasizing the importance of dosing, timing, and patient selection [15].

These findings suggest that dexmedetomidine may be particularly useful in patients at high risk of renal dysfunction, including elderly patients, individuals with diabetes mellitus, pre-existing chronic kidney disease, pro-

longed CPB duration, or complex combined procedures. However, excessive hypotension or bradycardia may compromise renal perfusion; therefore, careful titration and avoidance of high loading doses are essential [4,10]

6.3. Anti-inflammatory Effects During Cardiopulmonary Bypass

CPB triggers a systemic inflammatory response characterized by activation of complement, leukocytes, platelets, endothelial cells, and coagulation pathways.

This response is accompanied by increased circulating concentrations of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), C-reactive protein (CRP), and other inflammatory mediators [16, 17].

Dexmedetomidine has been shown to attenuate inflammatory activation during and after CPB.

Clinical studies have demonstrated reductions in IL-6, TNF-α, IL-1β, and other pro-inflammatory cytokines among patients receiving dexmedetomidine compared with conventional sedative regimens [12, 16, 17].

A 2025 systematic review and meta-analysis focusing on cardiac surgery with CPB found that dexmedetomidine reduced inflammatory markers and improved postoperative pulmonary oxygenation parameters, supporting its anti-inflammatory and pulmonary-protective potential. These findings are clinically important because systemic inflammation after CPB is closely linked to myocardial injury, pulmonary dysfunction, renal impairment, and neurological complications.

The anti-inflammatory effects of dexmedetomidine may be mediated by inhibition of nuclear factor-κB signaling, suppression of toll-like receptor pathways, modulation of macrophage activation, enhancement of the cholinergic anti-inflammatory reflex, and reduction of catecholamine-driven inflammatory amplification [12, 17].

6.4. Pulmonary and Endothelial Protection

Although this review focuses primarily on myocardial and renal protection, pulmonary protection is also relevant in cardiac surgery.

CPB can lead to pulmonary endothelial injury, increased capillary permeability, atelectasis, impaired oxygenation, and postoperative respiratory dysfunction.

Dexmedetomidine may improve pulmonary outcomes by reducing inflammatory cytokine release, preserving endothelial barrier function, decreasing oxidative stress, and improving oxygenation indices.

The 2025 meta-analysis by Liu et al. reported that dexmedetomidine improved oxygenation parameters and reduced inflammatory markers in patients undergoing cardiac surgery with CPB [17].

These effects may contribute to shorter mechanical ventilation duration and reduced ICU stay, although larger trials are needed to confirm whether pulmonary protection translates into improved hard clinical endpoints.

6.5. Clinical Implications

Current evidence suggests that dexmedetomidine may provide clinically meaningful organ protection during cardiac surgery through integrated cardioprotective, renoprotective, pulmonary-protective, and anti-inflammatory mechanisms. The strongest clinical data currently support reductions in postoperative AKI, myocardial injury biomarkers, inflammatory cytokine levels, and postoperative recovery duration [12-18].

However, the magnitude of benefit appears to depend on patient characteristics, timing of administration, dose, surgical complexity, and CPB duration.

Continuous low-dose infusion, often without a loading dose, may offer the best balance between organ protection and hemodynamic safety. Further large-scale multicenter randomized trials are required to define optimal dosing protocols and identify the patients most likely to benefit from dexmedetomidine-based organ protection strategies [14,15,18].

Author	Year	Study Design	Population	Sample Size	Main Findings
Li et al.	2021	Meta-analysis	Adult cardiac surgery	14 studies	Significant reduction in postoperative delirium
Chen et al.	2021	Meta-analysis	Cardiac surgery with CPB	11 studies	Reduced myocardial injury biomarkers and inflammatory response
Ming et al.	2021	Randomized controlled trial	Children with congenital heart disease	90 patients	Improved hemodynamic stability and organ protection
Shuplock et al.	2022	Meta-analysis	Pediatric cardiac surgery	8 studies	Reduced incidence of junctional ectopic tachycardia
Peng et al.	2024	Meta-analysis	Adult cardiac surgery	15 studies	Reduced postoperative atrial fibrillation
Yuan et al.	2024	Prospective clinical study	Valve surgery with CPB	120 patients	Lower troponin I levels and improved myocardial protection
Zhong et al.	2025	Systematic review and trial sequential analysis	Adult cardiac surgery	18 RCTs	Significant reduction in postoperative delirium
da Silva et al.	2025	Meta-analysis	Adult cardiac surgery	21 studies	Lower risk of postoperative delirium
Wen et al.	2025	Meta-analysis	Adult cardiac surgery	2,882 patients	Reduced incidence of acute kidney injury
Xu et al.	2025	Meta-analysis	Adult cardiac surgery	20 studies	Improved postoperative outcomes and recovery
Liu et al.	2025	Meta-analysis	Cardiac surgery with CPB	13 studies	Reduced inflammatory markers and improved oxygenation
van Rensburg et al.	2025	Comprehensive review	Pediatric cardiac surgery	—	Reduced opioid requirements and improved postoperative recovery

Table 2. Major Clinical Studies Evaluating Dexmedetomidine in Cardiac Surgery

The studies summarized in Table 2 demonstrate that dexmedetomidine provides multiple perioperative benefits in both adult and pediatric cardiac surgery, including improved hemodynamic stability, reduction of postoperative delirium and atrial fibrillation, attenuation of inflammatory responses, and protection against acute kidney injury. These findings support its growing role as an important component of modern cardiac anesthesia and intensive care protocols. Overall, the clinical benefits of dexmedetomidine appear to outweigh its potential adverse effects when appropriate patient selection, dosing

strategies, and hemodynamic monitoring are employed (Table 3)

Clinical Benefits	Potential Adverse Effects
Improved perioperative hemodynamic stability	Bradycardia
Reduced sympathetic activation and catecholamine release	Hypotension
Decreased incidence of postoperative atrial fibrillation (POAF)	Excessive sympatholysis
Reduced incidence of postoperative delirium (POD)	Atrioventricular conduction disturbances
Potential improvement in postoperative cognitive recovery	Reduced cardiac output in susceptible patients
Reduced myocardial oxygen consumption	Transient hypertension during loading dose administration
Attenuation of ischemia-reperfusion injury	Requirement for vasopressor support in selected cases
Reduction of inflammatory cytokine release (IL-6, TNF-α, IL-1β)	Hemodynamic instability with rapid infusion
Reduced incidence of acute kidney injury (AKI)	Dose-dependent cardiovascular depression
Improved myocardial protection	Increased risk of severe bradycardia in patients receiving β-blockers
Improved pulmonary oxygenation after cardiopulmonary bypass	Caution required in patients with advanced conduction abnormalities
Reduced opioid requirements	Potential drug accumulation in severe hepatic dysfunction
Facilitation of fast-track anesthesia protocols	Need for careful dose titration and monitoring
Earlier extubation and shorter ICU stay	Limited evidence regarding long-term outcome benefits
Effective postoperative sedation with minimal respiratory depression	Optimal dosing strategy remains under investigation

Table 3. Clinical Benefits and Potential Adverse Effects of Dexmedetomidine in Cardiac Surgery

Most adverse events are dose-dependent and can be minimized by avoiding rapid loading doses and using individualized infusion protocols. Therefore, dexmedetomidine remains an important component of contemporary cardiac anesthesia and perioperative critical care practice.

Conclusion

Dexmedetomidine has emerged as one of the most promising adjunctive agents in contemporary cardiac anesthesia and perioperative critical care. Owing to its unique pharmacological profile, which combines sedation, anxiolysis, sympatholysis, analgesic-sparing properties, and organ-protective effects without significant respiratory depression, dexmedetomidine has gained widespread acceptance in both adult and pediatric cardiac surgical practice.Accumulating evidence from randomized controlled trials, systematic reviews, and meta-analyses indicates that perioperative dexmedetomidine administration contributes to improved hemodynamic stability, attenuation of sympathetic activation, reduction of perioperative catecholamine release, and preservation of myocardial oxygen balance. These physiological effects appear to translate into clinically relevant benefits, including a reduced incidence of postoperative atrial fibrillation, improved cardiovascular stability, and facilitation of enhanced recovery pathways following cardiac surgery. Beyond its cardiovascular effects, dexmedetomidine demonstrates significant neuroprotective potential. Current evidence strongly supports its role in reducing postoperative delirium, particularly among elderly patients undergoing procedures involving cardiopulmonary bypass. The preservation of physiological sleep architecture, attenuation of neuroinflammation, and modulation of central sympathetic activity likely contribute to these beneficial outcomes. Emerging data also suggest a favorable effect on postoperative cognitive function, although further long-term investigations are required.

Organ-protective properties represent another important aspect of dexmedetomidine therapy. Experimental and clinical studies have demonstrated cardioprotective, renoprotective, and anti-inflammatory effects mediated

through suppression of oxidative stress, reduction of inflammatory cytokine production, preservation of mitochondrial integrity, and attenuation of ischemia-reperfusion injury. Recent meta-analyses indicate that dexmedetomidine may reduce the incidence of acute kidney injury and decrease postoperative inflammatory responses following cardiac surgery.

In pediatric cardiac surgery, dexmedetomidine has shown considerable promise in improving perioperative hemodynamic stability, reducing opioid requirements, facilitating postoperative sedation, and decreasing the incidence of junctional ectopic tachycardia. These benefits are particularly valuable in children with congenital heart disease, who are highly vulnerable to perioperative physiological disturbances and postoperative complications.

Despite these encouraging findings, several important questions remain unresolved. Significant heterogeneity exists among published studies regarding patient populations, dosing regimens, timing of administration, duration of infusion, and outcome definitions. Furthermore, although many studies demonstrate improvements in surrogate and intermediate outcomes, evidence regarding long-term survival, neurocognitive recovery, and quality of life remains limited.

Future large-scale multicenter randomized controlled trials should focus on establishing standardized dosing protocols, identifying patient populations most likely to benefit from dexmedetomidine therapy, and evaluating long-term clinical outcomes. Particular attention should be directed toward high-risk populations, including elderly patients, individuals with pre-existing organ dysfunction, and children undergoing complex congenital heart surgery. In conclusion, current evidence supports dexmedetomidine as a valuable component of modern cardiac anesthesia and intensive care. Through its combined hemodynamic, antiarrhythmic, neuroprotective, anti-inflammatory, cardioprotective, and renoprotective properties, dexmedetomidine has the potential to improve perioperative outcomes in both adult and pediatric cardiac surgical patients. Continued research will further define its optimal role within contemporary evidence-based cardiac perioperative care.

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