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Dexmedetomidine Versus Propofol for Early Postoperative Sedation After Adult Cardiac Surgery: A Narrative Review of Hemodynamic Safety and Recovery Outcomes

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Abstract

Early postoperative sedation after adult cardiac surgery requires balancing comfort and ventilator tolerance against evolving circulatory function and readiness for extubation. This narrative review compares dexmedetomidine and propofol during postoperative mechanical ventilation and progression toward extubation, emphasizing hemodynamic safety and recovery outcomes. The targeted literature search separated direct postoperative comparisons from perioperative adjunct trials and general intensive care evidence. Direct randomized comparisons yielded mixed findings for delirium, arrhythmia, and hemodynamic outcomes. Pooled findings showed differing patterns for hypotension, bradycardia, and vasopressor use, with interpretation dependent on the definitions used. Recovery findings were inconsistent; some randomized and pooled analyses favored shorter ventilation with dexmedetomidine, while a historical cohort associated it with longer adjusted time to extubation. Pooled delirium benefits were also sensitive to publication bias adjustment. Differences in treatment timing, sedation targets, analgesic co-interventions, and outcome definitions limit comparison across studies. Neither agent shows universal superiority for hemodynamic safety or recovery outcomes. Selection should reflect the sedation goal, administered regimen, and observed circulatory response. Future comparisons should standardize sedation and rescue protocols and distinguish transient adverse events from treatment-limiting compromise.

Categories: Pharmacology, Cardiac/Thoracic/Vascular Surgery, Anesthesiology
Keywords: bradycardia, cardiac surgery, dexmedetomidine, hemodynamics, postoperative sedation, propofol, vasopressors

Introduction And Background

During early recovery after adult cardiac surgery, sedation supports patient comfort and tolerance of mechanical ventilation while allowing assessment and progression toward extubation. Interpret sedative effects in the context of rapidly changing circulatory function. Following cardiopulmonary bypass (CPB), myocardial dysfunction, fluid shifts, and altered vascular tone may contribute to hemodynamic instability. Vasoplegia involves reduced systemic vascular resistance despite preserved or increased cardiac output, whereas low cardiac output reflects inadequate cardiac pumping and may compromise organ perfusion. These conditions require assessment and treatment in their own right; sedative selection is one component of postoperative management [1,2].

Propofol permits rapid titration across sedation depths but may reduce vascular tone, venous return, and cardiovascular performance [3,4]. Dexmedetomidine produces sedation through α_2-adrenoceptor activation and reduces central sympathetic activity, with relatively limited respiratory depression. However, sympatholysis can also cause bradycardia and hypotension, particularly when circulation depends on heart rate or adrenergic support [5]. These pharmacological differences do not establish either agent's comparative clinical superiority. A lower heart rate does not necessarily indicate improved perfusion, and propofol’s vasodilatory effects do not establish that dexmedetomidine causes less hypotension [3-6].

Existing systematic reviews and meta-analyses address ventilation duration, delirium, length of stay, and hemodynamic outcomes [6-8]. Contemporary reviews also discuss physiology-guided selection of cardiovascular anesthetics and sedatives [9,10]. Nevertheless, uncertainty remains about how to interpret comparative findings when studies differ in sedation targets, loading and maintenance regimens, analgesic co-interventions, and adverse-event definitions. A recorded episode of hypotension, a change in vasopressor use, and earlier extubation represent distinct outcomes and should not be treated as interchangeable measures of hemodynamic safety or recovery.

This narrative review critically evaluates dexmedetomidine versus propofol for early postoperative sedation after adult cardiac surgery, primarily during mechanical ventilation and progression toward extubation. Regimens initiated at chest or sternal closure are included when their intended role is postoperative sedation. Direct postoperative comparisons are considered separately from perioperative adjunct trials and

general intensive care evidence. Rather than providing a new quantitative synthesis, this review examines how treatment regimens and outcome definitions influence clinical interpretation and uses an educational physiological framework to organize reassessment. This framework complements existing approaches and is not a validated treatment algorithm.

Review

Scope and approach to the literature

This non-systematic narrative review used a targeted literature search in October 2026, drawing on PubMed-indexed records and journal websites. Search concepts included dexmedetomidine, propofol, cardiac surgery, postoperative sedation, hemodynamics, hypotension, bradycardia, vasopressors, extubation, recovery, and delirium. Relevant systematic reviews and their reference lists were used to identify additional comparisons. Key trials, observational studies, and guidance documents were cross-checked against original reports or indexed abstracts. The review considered evidence available through October 2026. Embase and the Cochrane Library were not independently searched.

The scope prioritized direct comparisons of intravenous dexmedetomidine and propofol for sedation during early recovery after adult cardiac surgery, principally during mechanical ventilation and progression toward extubation. Regimens initiated at chest or sternal closure were considered when their intended role was postoperative sedation. Perioperative adjunct trials, general ICU studies, and relevant guidance were included as separately identified contextual evidence. Pediatric surgery, non-intravenous administration, and non-surgical procedural sedation were excluded. Early postoperative care was defined by the treatment phase and sedation objective rather than by a fixed hourly cutoff.

Findings are organized by evidence type and clinical outcome. Direct postoperative comparisons are discussed separately from perioperative adjunct trials and general ICU evidence. Interpretation considers study design, population, comparator, treatment timing, sedation targets, analgesic co-interventions, outcome definitions, and rescue treatment, where reported. Hemodynamic events, vasopressor use, delirium, arrhythmias, and recovery milestones are considered distinct outcomes. Published estimates are attributed to their source studies; no new quantitative pooling was performed. Meta-analyses containing overlapping primary trials are treated as related syntheses rather than independent confirmation. Table *1* provides a selected evidence map rather than an exhaustive inventory.

Studies and design	Setting and comparator	Principal quantitative results	Interpretive limitations
Djaiani et al. [11]; randomized trial, n=183	Postoperative ICU sedation: dexmedetomidine versus propofol	Delirium: 17.5% versus 31.5%; OR: 0.46, 95% CI: 0.23-0.92; p=0.028.	Primary endpoint was delirium rather than comparative hemodynamic safety.
Liu et al. [12]; randomized trial, n=88	Postoperative sedation; dexmedetomidine versus propofol	Atrial fibrillation: 13.6% versus 36.4%; OR: 0.28, 95% CI: 0.10-0.80; p=0.025. Hypotension: 56.8% versus 29.5%; p=0.017; CI unavailable in the consulted abstract.	Small, open-label trial; rhythm benefit coexisted with more hypotension.
Subramaniam et al. [13]; factorial randomized trial, n=120	Acetaminophen versus placebo and dexmedetomidine versus propofol; treatment from chest closure	Delirium with dexmedetomidine versus propofol: 17% versus 21%; reported difference -4 percentage points, 95% CI: -18 to 10; p=0.54.	Analgesic co-intervention and short sedative exposure affect interpretation.
Turan et al. [14]; randomized placebo-controlled trial, n=794 analyzed	Perioperative dexmedetomidine versus saline	Atrial fibrillation: 30% versus 34%; RR: 0.90, 97.8% CI: 0.72-1.15; p=0.34. Delirium: 17% versus 12%; RR: 1.48, 97.8% CI: 0.99-2.23; p-value unavailable in the consulted abstract.	Adjunct intervention rather than replacement of propofol; preserve the original multiplicity-adjusted confidence level.
Lei et al. [15]; randomized placebo-controlled trial, n=1,073 completing	Perioperative dexmedetomidine versus saline	Major complications: 161/536 versus 169/537; reported RR: 0.93, 95% CI: 0.72-1.21; p=0.66.	Does not directly compare postoperative sedative strategies.

follow-up			
Heybati et al. [16]; systematic review and meta-analysis, 41 trials, n=3,948 overall	Mechanically ventilated ICU adults; dexmedetomidine versus propofol	Cardiac surgical subgroup: ventilation duration MD: -0.67 h, 95% CI: -1.31 to -0.03; p=0.041. Delirium RR: 0.49, 95% CI: 0.29-0.87; p=0.019.	Overall review size is not the denominator for these subgroup outcomes. Outcome-specific subgroup counts and heterogeneity were unavailable in the consulted abstract.
Peng et al. [17]; meta-analysis, seven trials, n=1,004 overall	Cardiac surgery; dexmedetomidine versus propofol	Ventricular arrhythmias: three trials, n=546; OR: 0.14, 95% CI: 0.03-0.66; p=0.01; I^2=0%. Bradycardia: four trials, n=570; OR: 2.88, 95% CI: 1.02-8.17; reported p=0.05; I^2=0%. Atrial fibrillation: seven trials, n=1,004; OR: 0.69, 95% CI: 0.36-1.29; p=0.24; I^2=51%.	Few ventricular events; the bradycardia p-value is reproduced at the source's reported precision.
Montaseri et al. [6]; systematic review and meta-analysis	Cardiac surgery; dexmedetomidine versus propofol	Hypotension: seven trials, n=807; OR: 1.76, 95% CI: 1.25-2.48; p<0.001. Bradycardia: six trials, n=718; OR: 2.89, 95% CI: 1.28-6.49; p=0.01. Vasopressor use: five trials, n=706; OR: 0.52, 95% CI: 0.32-0.84; p=0.007. The authors reported no observed heterogeneity for these outcomes.	Definitions, treatment timing, dosing, and rescue protocols varied. Lower odds of vasopressor use do not mean a lower vasopressor dose.
Herr et al. [18]; randomized trial, n=295	Post-CABG sedation; dexmedetomidine-based versus propofol-based care	The original abstract reports similar mean weaning and extubation times but provides no numerical estimates, CIs, or p-values for these comparisons.	Open-label design, supplemental propofol, and local comparator practice complicate attribution.
Preveden et al. [19]; randomized trial, n=120	Postoperative cardiac surgical sedation; dexmedetomidine versus propofol	Mechanical ventilation was 2.2 h shorter with dexmedetomidine; p<0.001; CI unavailable in the consulted abstract. Delirium: 11.7% versus 25%; p=0.059.	Shorter ventilation did not translate into shorter ICU or hospital stay.
Liu et al. [20]; randomized trial, n=61 analyzed	Postvalve surgery sedation; dexmedetomidine versus propofol	At 4 h, median change in perfused small-vessel density: 1.3 versus 0 mm/mm²; p=0.025. Median change in De Backer score: 0.9 versus -0.1 mm^{-1}; p=0.005. Between-group CIs unavailable in the consulted abstract.	Small, single-center study using surrogate microcirculatory outcomes. Values are within-group changes compared between groups, not treatment-effect estimates.
Sattar et al. [7]; meta-analysis, 14 trials, n=1,360 overall	Cardiac surgery; dexmedetomidine versus propofol	Ventilation duration: 10 trials; reported MD: 0.75 h, 95% CI: 0.06-1.44; p=0.03; I^2=94%, interpreted by the authors as shorter ventilation with dexmedetomidine.	The positive estimate reflects the source's comparison direction and should not be presented as dexmedetomidine minus propofol. Substantial heterogeneity limits interpretation.
Euteneuer et al. [21]; historical cohort	Postoperative cardiac surgical sedation; dexmedetomidine versus propofol	Adjusted time to extubation was 1.8 h longer with dexmedetomidine; 98.3% CI: 1.5-2.1 h; p<0.001.	Different practice periods and residual confounding limit causal interpretation; preserve the original confidence level.

TABLE 1: Selected comparative evidence on sedation after adult cardiac surgery.

Results compare dexmedetomidine with propofol unless otherwise specified. Comparison directions and confidence levels are retained as reported. Statistics unavailable in the consulted source were not calculated or inferred. Overall meta-analysis sample sizes must not be interpreted as outcome-specific denominators.

CABG: coronary artery bypass grafting; RR: risk ratio; MD: mean difference

Study selection was targeted and involved author judgment. No prospectively registered protocol, duplicate screening, formal study-level risk-of-bias assessment, or standardized certainty grading was undertaken. Discussion of individual study limitations does not substitute for formal risk-of-bias assessment. The search concepts reported above are not a complete executable search strategy, and the search and selection process cannot be fully replicated from this description. Reliance on one bibliographic database, supplemented by journal websites and reference checking, may have omitted relevant studies and introduced selection bias.

Accordingly, this review does not claim exhaustive coverage or PRISMA-compliant systematic-review methods.

Pharmacology in relation to treatment goals

Dexmedetomidine reduces central sympathetic activity and can provide arousable sedation with relatively limited respiratory depression. These properties may be useful when interaction and progression toward liberation from ventilation are priorities. The same reduction in sympathetic support can become undesirable when cardiac output depends on heart rate or adrenergic drive. Bradycardia and hypotension must therefore be assessed as potential treatment-limiting effects, rather than exceptions to an otherwise protective hemodynamic profile [5,16].

Propofol permits rapid adjustment of hypnotic depth. Its hemodynamic effects involve vascular tone, venous return, and concentration-dependent cardiovascular depression. In a physiological study of 17 adults after upper abdominal surgery, de Wit et al. examined several circulatory components, showing why blood pressure alone cannot describe the entire response [4]. Effects observed under controlled conditions should not be translated directly into predictions for unstable postcardiopulmonary bypass (CPB) patients [3,4].

The intended depth of sedation matters as much as the drug name. An intraoperative dexmedetomidine infusion used alongside general anesthesia is an adjunct intervention, whereas postoperative dexmedetomidine may be the main sedative. A propofol regimen targeting deep sedation cannot be assumed equivalent to a dexmedetomidine regimen targeting cooperative sedation. Opioid exposure, other anesthetics, and rescue sedatives influence both the apparent efficacy and hemodynamic cost of each strategy [5,16].

Direct postoperative comparative evidence

This section examines direct postoperative comparisons, focusing on study design, sedation regimens, and the distinction between hemodynamic and recovery outcomes. Interpretation requires attention to whether the interventions represent comparable sedation strategies, including their timing, intended depth, and permitted supplemental medications. Hemodynamic adverse events should be considered separately from delirium, arrhythmias, and recovery milestones, because improvement in one outcome does not establish benefit across the others. The following discussion therefore considers randomized and observational comparisons separately and highlights regimen-specific limitations.

Randomized comparisons

An early multicenter, open-label trial by Herr et al. randomized 295 coronary artery bypass graft patients to dexmedetomidine-based or propofol-based sedation [18]. Mean weaning and extubation times were similar, although morphine requirements were lower with dexmedetomidine. Importantly, the dexmedetomidine regimen began at sternal closure with a loading infusion and permitted supplemental propofol, whereas the comparator followed local practice. The findings therefore compare treatment regimens rather than isolated drug effects; differences in analgesia and cardiovascular medication use also complicate attribution of hemodynamic outcomes [18].

Djaiani et al. randomized 183 older cardiac surgical patients to postoperative dexmedetomidine or propofol [11]. Delirium occurred in 17.5% and 31.5%, respectively. This supports a possible postoperative benefit in the studied population, but delirium was the primary endpoint; the trial did not test drug selection according to vascular tone, ventricular dysfunction, or conduction phenotype. A favorable neurocognitive outcome should not be reinterpreted as proof of superior circulatory tolerance [11].

Liu et al. studied 88 postoperative cardiac surgical patients without pre-existing atrial fibrillation or flutter [12]. Dexmedetomidine reduced postoperative atrial fibrillation, but hypotension was more frequent than with propofol, occurring in 56.8% versus 29.5%. This finding is particularly relevant because it shows that an apparent rhythm benefit can coexist with a hemodynamic disadvantage in the same trial [12].

The 120-patient DEXACET factorial trial evaluated acetaminophen versus placebo and dexmedetomidine versus propofol in older cardiac surgical patients. Sedative treatment began at chest closure and continued for up to 6 h. Delirium did not differ significantly between sedative groups. Its principal positive finding concerned acetaminophen and should not be attributed to dexmedetomidine. The study shows how analgesic co-interventions and treatment duration affect interpreting a sedative comparison [13].

Preveden et al. randomized 120 cardiac surgical patients to postoperative dexmedetomidine or propofol [19]. Dexmedetomidine shortened mechanical ventilation by 2.2 h, but did not shorten ICU or hospital stay. Delirium was numerically less frequent, but not statistically significant. This illustrates why earlier extubation should be distinguished from broader recovery benefits and from evidence of superior circulatory tolerance [19].

Dose and administration strategy further affect interpretation. The Herr et al. trial used a dexmedetomidine

loading infusion, whereas the Liu et al. atrial fibrillation trial compared titrated maintenance infusions targeting the same sedation range [12,18]. Loading exposure, infusion rate, achieved sedation, concurrent analgesia, and supplemental sedatives should therefore be reported alongside adverse events. Blood pressure and vasoactive requirements describe the response to the entire regimen; neither a lower infusion rate nor earlier extubation alone establishes better hemodynamic tolerance [12,18].

A separate trial by Liu et al. evaluated sublingual microvascular perfusion after elective valve surgery, with 29 dexmedetomidine-treated and 32 propofol-treated patients analyzed [20]. Both infusions targeted a Richmond Agitation-Sedation Scale score of 0 to -3. Selected microvascular perfusion measures improved more with dexmedetomidine at 4 h. These findings support assessing perfusion beyond arterial pressure, but remain surrogate outcomes from a small, single-center study, and do not establish improved organ outcomes or a phenotype-specific treatment rule [20].

Observational recovery evidence

Euteneuer et al. compared dexmedetomidine and propofol during historical postoperative sedation periods [21]. After inverse probability of treatment weighting, dexmedetomidine was associated with 1.8 h longer time to extubation (98.3% confidence interval: 1.5-2.1 h). The confidence level accounted for multiple comparisons across three primary outcomes. The report gives inconsistent sample totals in its abstract and main results, so a single cohort count is not reproduced here. Its convenience sample and differing practice periods also limit causal interpretation. Nevertheless, the direction differs from several randomized and pooled estimates and argues against the promise of consistently earlier extubation with dexmedetomidine [21].

Key Point

Direct comparisons show differing effects on delirium, arrhythmias, hemodynamic events, and extubation. Interpretation requires considering achieved sedation, analgesia, and rescue treatment, not the primary sedative alone.

Context from perioperative adjunct trials

The Dexmedetomidine for Reduction of Atrial Fibrillation and Delirium after Cardiac Surgery (DECADE) trial evaluated perioperative dexmedetomidine against placebo, rather than substituting dexmedetomidine for propofol. Infusion began before incision and continued into the postoperative period. It did not demonstrate a reduction in postoperative atrial arrhythmias or delirium. Its results challenge a general assumption that adding sympatholysis improves recovery, but cannot establish which sedative is preferable in a postoperative head-to-head comparison [14].

The multicenter Dexmedetomidine and Outcomes of Cardiac Surgery (DOCS) trial similarly compared perioperative dexmedetomidine with saline in adults undergoing CPB-assisted surgery. Among 1,073 participants who completed the study after randomization, dexmedetomidine did not reduce death or major complications. This is relevant to claims of overall perioperative benefit, while remaining a different question from sedation selection after ICU admission [15]. These adjunct trials constrain claims of general perioperative protection. They do not determine the effect of replacing propofol during postoperative ventilation and should not be treated as direct evidence for that decision.

Comparative syntheses and their applicability

Abowali et al. reviewed 11 postoperative cardiac surgical trials comprising 1,184 patients [8]. Their synthesis suggested advantages in time to extubation and ICU stay, without a clear reduction in hospital stay. This supports considering recovery outcomes, but does not establish a hemodynamic treatment rule for particular patient categories [8].

Sattar et al. pooled 14 randomized trials comprising 1,360 patients and reported shorter mechanical ventilation, ICU, and hospital stays, and less delirium with dexmedetomidine [7]. Their recovery-focused synthesis complements the hemodynamic reviews, but overlapping trials mean these meta-analyses do not independently confirm the findings. Variations in populations and sedation protocols also limit translating pooled recovery benefits into a preferred drug for a particular circulatory pattern [7].

Heybati et al. included 41 trials involving 3,948 mechanically ventilated adults [16]. In the cardiac surgical subgroup, dexmedetomidine was associated with less delirium and a small reduction in ventilation duration of about two-thirds of an hour, without a significant difference in ICU stay. The clinical importance of a modest time benefit depends on local extubation practice and whether comparable sedation targets were achieved [16].

Peng et al. analyzed seven cardiac surgical trials involving 1,004 patients [17]. Dexmedetomidine was associated with fewer ventricular arrhythmias and more bradycardia, without a statistically significant reduction in atrial fibrillation. Although the ventricular signal is clinically interesting, only three trials

supported the endpoint, and few events were reported. It should not be converted into a recommendation to use dexmedetomidine as antiarrhythmic treatment [17].

A 2026 hemodynamic meta-analysis by Montaseri et al. reported greater odds of hypotension and bradycardia with dexmedetomidine than propofol, alongside lower odds of reported vasopressor use [6]. The pooled odds ratios were 1.76 for hypotension, 2.89 for bradycardia, and 0.52 for vasopressor requirement. Differences in protocols and endpoint definitions limit patient-level application [6].

Li et al.'s 2026 review of 15 trials in older cardiac surgical populations found a pooled reduction in delirium and increased bradycardia [22]. Comparators included placebo and alternative sedative or analgesic regimens, with a less clear delirium effect in placebo-controlled studies. This broader synthesis informs the balance of potential benefit and harm but is not a pure dexmedetomidine-versus-propofol comparison [22].

Hunt et al. analyzed 12 randomized trials involving 3,539 participants using Bayesian methods [23]. The pooled delirium estimate favored dexmedetomidine before adjustment for publication bias (odds ratio: 0.67; 95% credible interval: 0.45-0.92), but a model accounting for publication bias did not establish benefit (odds ratio: 1.15; 95% credible interval: 0.93-2.51). Their reanalysis of DECADE was also sensitive to assumptions about prior evidence. These findings qualify pooled delirium benefits and should not be interpreted as proof of harm or as a direct postoperative dexmedetomidine-versus-propofol comparison [23]. Hunt et al. also examined comparator and administration timing [23]. Their analysis estimated a 93% posterior probability that the delirium benefit was greater in trials comparing dexmedetomidine with propofol than in those using normal saline. Postoperative administration had an 85% probability of greater benefit than intraoperative administration alone and an 83% probability of greater benefit than combined intraoperative and postoperative administration. These between-study comparisons suggest that comparator and timing may influence pooled findings, but do not establish the superiority of a particular regimen, given differences between trials and the sensitivity of the overall findings to publication bias [23].

Chen et al. pooled 22 randomized trials involving 4,063 cardiac surgical patients and found a lower overall risk of postoperative atrial fibrillation with perioperative dexmedetomidine [24]. The reduction was evident in placebo-controlled trials, while comparisons with active sedatives did not show a clear difference. This comparator-dependent result supports distinguishing adjunct prophylaxis from replacement of propofol; it does not establish dexmedetomidine as the preferred sedative for patients with a tachycardic circulatory pattern [24].

Key Point

These syntheses contain overlapping trials and differ in comparator, treatment timing, and sedation objective. Their findings should therefore be interpreted within the regimens studied rather than counted as independent confirmation of benefit.

Interpreting safety and recovery outcomes

Heart Rate and Rhythm

Heart rate reduction can be desirable when sympathetic activation is excessive, but can reduce output when stroke volume cannot increase. Conduction disease, concurrent rate-limiting treatment, and postoperative pacing status affect interpretation. The recurring bradycardia signal supports explicit review of these factors; it does not validate a specific heart rate cutoff for switching sedatives. Propofol also requires rhythm surveillance, particularly during hypotension or combined drug administration [3,17].

Blood Pressure and Vasoactive Support

Reported hypotension and vasopressor use measure different aspects of care. A pressure threshold may identify a brief episode, whereas treatment depends on duration, severity, presumed mechanism, and local practice. The discordant outcomes in the 2026 synthesis therefore require examining event definitions and rescue protocols before concluding overall stability. Its inclusion of intraoperative and immediate postoperative studies further limits application to a single recovery phase [6].

In practice, arterial pressure should be interpreted alongside perfusion, ventricular function, and changes in support. Escalating vasopressors post-CPB may reflect vasoplegia, low output, or a mixed state. Reducing sedative exposure can be considered during reassessment, but a drug change should not distract from identifying bleeding, ventricular dysfunction, or other causes of deterioration [1,2,25].

Cardiac Output and Ventricular Reserve

A low ejection fraction does not by itself specify whether vasodilation, reduced venous return, or slowing of heart rate will be least tolerated. The direction of a sedative's physiological effect must be interpreted against the current circulation. Available echocardiographic findings and cardiac output measurements can

help, but should not be presented as validated predictors of comparative drug response. No trial discussed here establishes phenotype-specific efficacy for left- or right-ventricular failure [1,4,25].

Recovery Outcomes and Treatment Context

Time to extubation reflects readiness assessment, sedation interruption, analgesia, and local workflow, as well as drug effects. Herr et al. found similar mean extubation times; Preveden et al. reported shorter ventilation without shorter ICU or hospital stay; and the historical cohort found longer extubation time with dexmedetomidine [18,19,21]. These findings should be presented with their designs and regimens rather than averaged informally. The clinically useful question is whether a strategy achieves adequate comfort and timely liberation without treatment-limiting circulatory compromise.

Supporting Evidence From General ICU Care

Prolonged general ICU sedation provides supporting evidence, not direct estimates, for early cardiac surgical recovery. PRODEX compared dexmedetomidine with propofol, while SPICE III compared early dexmedetomidine-based sedation with usual care and frequently required additional sedatives. SPICE III showed no mortality advantage. Their illness trajectories and exposure durations differ from short postoperative ventilation [26,27].

MENDS2 found no important dexmedetomidine advantage over propofol for days alive without delirium or coma in ventilated adults with sepsis. A2B did not demonstrate shorter successful extubation time with dexmedetomidine-based sedation and reported more severe bradycardia. These findings support attention to treatment adequacy and safety but do not validate selection in post-CPB vasoplegia [28,29].

The 2018 PADIS guideline suggested propofol over benzodiazepines after cardiac surgery and either propofol or dexmedetomidine over benzodiazepines in general mechanically ventilated adults. The 2025 focused update conditionally favors dexmedetomidine over propofol when light sedation or delirium reduction is a high priority. This recommendation addresses treatment goals in the ICU; it is not proof of hemodynamic superiority in every cardiac surgical subgroup [30,31].

Physiological considerations during postoperative reassessment

Physiology-guided selection is already described in cardiovascular anesthesia and ICU reviews [9,10]. Here, circulatory pattern refers to the dominant findings at postoperative assessment rather than a fixed phenotype. Heart rate and conduction, ventricular performance, vascular tone, and changes in vasoactive support help interpret observed tolerance. Table *2* and Figure *1* provide an educational framework for reassessment; this framework has not been validated for assigning sedatives or improving outcomes [1-6,17,25].

Circulatory pattern	Dexmedetomidine consideration	Propofol consideration	Reassessment focus
Slow heart rate or conduction vulnerability	Further rate reduction may limit output.	Bradycardia and hypotension remain possible.	Review rhythm, rate-limiting drugs, and pacing status.
Low vascular tone or increasing vasopressors	Do not assume protection against hypotension.	Vasodilation and reduced venous return may limit tolerance.	Assess perfusion and the cause of increasing support.
Tachycardia or sympathetic activation	Sympatholysis may be useful if compensatory support is not essential.	Sedation may reduce agitation; pressure response still matters.	Identify pain, agitation, hypovolemia, or low output.
Ventricular dysfunction or low output	Consider dependence on heart rate and adrenergic drive.	Consider preload, vascular tone, and required depth.	Interpret imaging or output measurements in clinical context.
Mixed or changing post-CPB circulation	Reassess response rather than retain a fixed category.	Reassess response rather than retain a fixed category.	Compare achieved sedation with rhythm, perfusion, and support trends.

TABLE 2: Physiological considerations during early postoperative sedation.

These considerations are physiological inferences and have not been validated as treatment assignments.

CPB: cardiopulmonary bypass

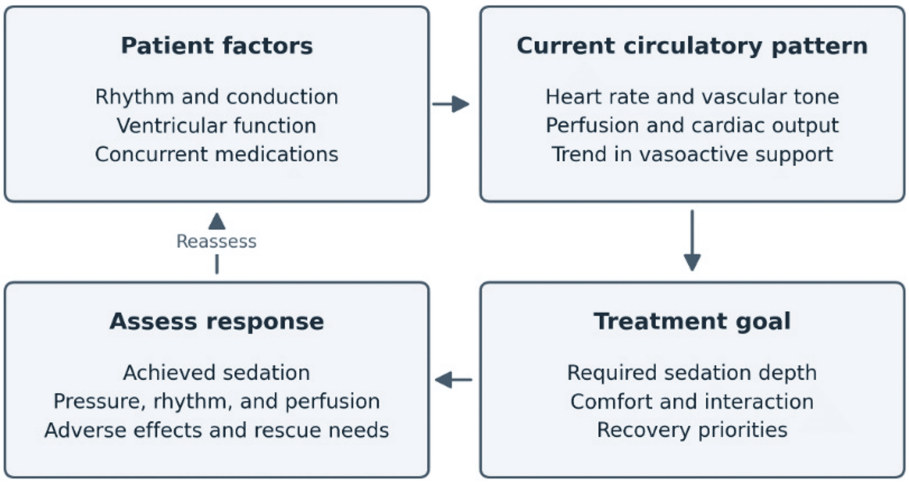


FIGURE 1: Conceptual process for physiological assessment during early postoperative sedation.

Patient factors inform interpretation of the current postoperative circulation and sedation goal. Observed responses prompt reassessment as physiology changes. This educational model specifies no preferred drug, dose, or numerical threshold and has not been prospectively validated.

Application begins with the indication and desired sedation depth, followed by the circulatory feature most likely to limit tolerability. Analgesics and other cardiovascular drugs should be considered alongside the primary sedative.

For a patient with slow heart rate and limited stroke-volume reserve, further sympathetic suppression may be poorly tolerated. For a patient with low vascular tone and increasing support, propofol-related vasodilation warrants attention, while dexmedetomidine remains capable of causing hypotension. In a tachycardic patient, the cause of sympathetic activation matters as follows: analgesia, agitation, hypovolemia, and low output imply different management priorities. These considerations support reassessment of the cause of circulatory instability and the observed response to sedation [1,3,5,25].

After initiation or adjustment, compare achieved sedation with trends in pressure, rhythm, perfusion, and vasoactive agents. Record whether deterioration required dose reduction, rescue treatment, crossover, or discontinuation. This distinguishes a transient monitored event from treatment-limiting intolerance and helps interpret the response to the whole regimen. Alternative causes of deterioration must be assessed concurrently.

Limitations and research priorities

This targeted narrative synthesis is vulnerable to selection and interpretation bias and cannot establish exhaustive coverage. Overlapping reviews, differing sedation targets, and inconsistent adverse-event definitions limit synthesis. Observational recovery findings may reflect secular changes and residual confounding. Patients with marked instability may be underrepresented in direct trials, so extrapolation to severe vasoplegia or ventricular failure requires particular restraint. Adjunct and general ICU studies remain indirect evidence.

Two untested hypotheses warrant prospective evaluation - baseline rhythm and conduction status may modify the comparative risk of treatment-limiting bradycardia with dexmedetomidine versus propofol, and ventricular dysfunction may modify the comparative risk of hypotension requiring treatment escalation. These are candidate treatment-effect modifiers, not established criteria for assigning a sedative.

Future postoperative trials should compare intravenous dexmedetomidine and propofol with comparable sedation targets, standardized analgesia, and explicit rescue and extubation criteria. Randomization could be stratified by rhythm or conduction status and ventricular function, with prespecified, adequately powered treatment-by-subgroup interactions. Reporting should distinguish transient threshold events from sustained compromise and include vasoactive dose and duration, treatment-limiting bradycardia, rescue interventions, discontinuation, achieved sedation, and recovery. A prospective registry using these definitions could assess feasibility and event frequency before a stratified trial; exploratory associations would not establish treatment-effect modification.

Conclusions

For early postoperative sedation after adult cardiac surgery, neither dexmedetomidine nor propofol has demonstrated universal superiority across hemodynamic safety and recovery outcomes. Direct trials suggest selected benefits with dexmedetomidine, but bradycardia, hypotension, and conflicting recovery findings require consideration of the actual regimen and clinical setting. Perioperative adjunct and prolonged general ICU studies inform context rather than directly resolve this postoperative comparison.

The practical contribution is to interpret adverse-event definitions, sedation targets, co-interventions, and rescue treatment alongside recovery outcomes. Physiological reassessment helps place these findings in the patient's current clinical context. Standardized reporting and prospective testing are needed to determine whether patient characteristics modify comparative treatment effects.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ahmed Salim

Acquisition, analysis, or interpretation of data: Ahmed Salim

Drafting of the manuscript: Ahmed Salim

Critical review of the manuscript for important intellectual content: Ahmed Salim

Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Lomivorotov VV, Efremov SM, Kirov MY, Fominskiy EV, Karaskov AM: Low-cardiac-output syndrome after cardiac surgery. J Cardiothorac Vasc Anesth. 2017, 31:291-308. 10.1053/j.jvca.2016.05.029
2. Omar S, Zedan A, Nugent K: Cardiac vasoplegia syndrome: pathophysiology, risk factors and treatment . Am J Med Sci. 2015, 349:80-8. 10.1097/MAJ.0000000000000341
3. Sahinovic MM, Struys MM, Absalom AR: Clinical pharmacokinetics and pharmacodynamics of propofol. Clin Pharmacokinet. 2018, 57:1539-58. 10.1007/s40262-018-0672-3
4. de Wit F, van Vliet AL, de Wilde RB, et al.: The effect of propofol on haemodynamics: cardiac output, venous return, mean systemic filling pressure, and vascular resistances. Br J Anaesth. 2016, 116:784-9. 10.1093/bja/aew126
5. Boncyk C, Rolfsen ML, Richards D, Stollings JL, Mart MF, Hughes CG, Ely EW: Management of pain and sedation in the intensive care unit. BMJ. 2024, 387:10.1136/bmj-2024-079789
6. Montaseri M, Saberian M, Jackson AC, Bahramnejad F, Bahramnezhad F: Hemodynamic effects of dexmedetomidine versus propofol in cardiac surgery: a systematic review and meta-analysis. BMC Anesthesiol. 2026, 26:10.1186/s12871-026-04027-z
7. Sattar L, Reyaz I, Rawat A, et al.: Comparison between dexmedetomidine and propofol for sedation on outcomes after cardiac surgery in patients requiring mechanical ventilation: a meta-analysis of randomized-control trials. Cureus. 2023, 15:10.7759/cureus.42212
8. Abowali HA, Paganini M, Enten G, Elbadawi A, Camporesi EM: Critical review and meta-analysis of postoperative sedation after adult cardiac surgery: dexmedetomidine versus propofol. J Cardiothorac Vasc Anesth. 2021, 35:1134-42. 10.1053/j.jvca.2020.10.022
9. Safiullah ZN, Applefeld WN, Li W, et al.: Hemodynamic effects of anesthetics, sedatives, and analgesics in the CICU: clinical decision guidance. JACC Adv. 2026, 5:10.1016/j.jacadv.2026.103017
10. Martin NJ, Brinkman HM, Ellis S, et al.: Suitability of sedative usage in patients undergoing cardiovascular surgery. Expert Rev Cardiovasc Ther. 2026, 24:715-27. 10.1080/14779072.2026.2703069
11. Djaiani G, Silverton N, Fedorko L, Carroll J, Styra R, Rao V, Katznelson R: Dexmedetomidine versus propofol sedation reduces delirium after cardiac surgery: a randomized controlled trial. Anesthesiology. 2016, 124:362-8. 10.1097/ALN.0000000000000951
12. Liu X, Zhang K, Wang W, Xie G, Fang X: Dexmedetomidine sedation reduces atrial fibrillation after cardiac surgery compared to propofol: a randomized controlled trial. Crit Care. 2016, 20:10.1186/s13054-016-1480-5
13. Subramaniam B, Shankar P, Shaefi S, et al.: Effect of intravenous acetaminophen vs placebo combined with propofol or dexmedetomidine on postoperative delirium among older patients following cardiac surgery: the DEXACET randomized clinical trial. JAMA. 2019, 321:686-96. 10.1001/jama.2019.0234
14. Turan A, Duncan A, Leung S, et al.: Dexmedetomidine for reduction of atrial fibrillation and delirium after

cardiac surgery (DECADE): a randomised placebo-controlled trial. Lancet. 2020, 396:177-85. 10.1016/s0140-6736(20)30631-0

15. Lei C, Zheng Z, Han J, et al.: Effects of dexmedetomidine on outcomes after cardiac surgery (DOCS): a randomised double-blind, placebo-controlled trial. Br J Anaesth. 2026, 136:55-64. 10.1016/j.bja.2025.09.026
16. Heybati K, Zhou F, Ali S, Deng J, Mohananey D, Villablanca P, Ramakrishna H: Outcomes of dexmedetomidine versus propofol sedation in critically ill adults requiring mechanical ventilation: a systematic review and meta-analysis of randomised controlled trials. Br J Anaesth. 2022, 129:515-26. 10.1016/j.bja.2022.06.020
17. Peng J, Wu Y, Li L, Xia P, Yu P, Zhang J, Liu X: Dexmedetomidine vs. propofol on arrhythmia in cardiac surgery: a meta-analysis of randomized controlled trials. Front Cardiovasc Med. 2024, 11:10.3389/fcvm.2024.1433841
18. Herr DL, Sum-Ping ST, England M: ICU sedation after coronary artery bypass graft surgery: dexmedetomidine-based versus propofol-based sedation regimens. J Cardiothorac Vasc Anesth. 2003, 17:576-84. 10.1016/S1053-0770(03)00200-3
19. Preveden M, Zdravković R, Vicković S, et al.: Dexmedetomidine vs. propofol sedation reduces the duration of mechanical ventilation after cardiac surgery - a randomized controlled trial. Eur Rev Med Pharmacol Sci. 2023, 27:7644-52. 10.26355/eurrev_202308_33418
20. Liu X, Zhang K, Wang W, et al.: Dexmedetomidine versus propofol sedation improves sublingual microcirculation after cardiac surgery: a randomized controlled trial. J Cardiothorac Vasc Anesth. 2016, 30:1509-15. 10.1053/j.jvca.2016.05.038
21. Euteneuer AA, Radosevich MA, Weingarten TN, Seelhammer TG, Schroeder D, Wittwer ED: Dexmedetomidine versus propofol for postoperative recovery after cardiac surgery: a historical cohort study. Can J Anaesth. 2025, 72:409-16. 10.1007/s12630-024-02877-0
22. Li A, Wu H, Zhang K, Ying J: Efficacy and hemodynamic safety of perioperative dexmedetomidine for the prevention of postoperative delirium in elderly patients undergoing cardiac surgery: an updated systematic review and meta-analysis of randomized controlled trials. Front Neurol. 2026, 17:10.3389/fneur.2026.1895312
23. Hunt T, Payne T, Brophy JM, et al.: Perioperative dexmedetomidine for the prevention of postoperative delirium after cardiac surgery: a systematic review, Bayesian meta-analysis, and Bayesian re-analysis of the DECADE trial. Br J Anaesth. 2025, 134:1671-82. 10.1016/j.bja.2025.02.031
24. Chen YJ, Xu SS, Shen QH, Yu DH: Dexmedetomidine for the prevention of postoperative atrial fibrillation: a systematic review and meta-analysis. J Int Med Res. 2026, 54:10.1177/03000605261465578
25. Vincent JL, De Backer D: Circulatory shock. N Engl J Med. 2013, 369:1726-34. 10.1056/NEJMra1208943
26. Jakob SM, Ruokonen E, Grounds RM, et al.: Dexmedetomidine vs midazolam or propofol for sedation during prolonged mechanical ventilation: two randomized controlled trials. JAMA. 2012, 307:1151-60. 10.1001/jama.2012.304
27. Shehabi Y, Howe BD, Bellomo R, et al.: Early sedation with dexmedetomidine in critically ill patients . N Engl J Med. 2019, 380:2506-17. 10.1056/NEJMoa1904710
28. Hughes CG, Mailloux PT, Devlin JW, et al.: Dexmedetomidine or propofol for sedation in mechanically ventilated adults with sepsis. N Engl J Med. 2021, 384:1424-36. 10.1056/NEJMoa2024922
29. Walsh TS, Parker RA, Aitken LM, et al.: Dexmedetomidine- or clonidine-based sedation compared with propofol in critically ill patients: the A2B randomized clinical trial. JAMA. 2025, 334:32-45. 10.1001/jama.2025.7200
30. Devlin JW, Skrobik Y, Gélinas C, et al.: Clinical practice guidelines for the prevention and management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. Crit Care Med. 2018, 46:825-73. 10.1097/CCM.0000000000003299
31. Lewis K, Balas MC, Stollings JL, et al.: A focused update to the clinical practice guidelines for the prevention and management of pain, anxiety, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. Crit Care Med. 2025, 53:711-27. 10.1097/CCM.0000000000006574