ARTICLE IN PRESS

Journal of Cardiothoracic and Vascular Anesthesia 000 (2026) 1−20

Contents lists available at ScienceDirect

Journal of Cardiothoracic and Vascular Anesthesia

journal homepage: www.jcvaonline.com

Review Article

Weaning From Mechanical Ventilation in Cardiac Surgery Patients: Current Strategies, Monitoring Innovations, and Future Perspectives

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Weaning from mechanical ventilation after cardiac surgery is a dynamic, multifaceted process that extends beyond respiratory function alone. While most patients experience timely extubation, others face delayed or failed weaning due to the complex interplay of perioperative factors. This review provides an integrative overview of the weaning process, beginning with key definitions and classifications specific to the cardiothoracic population. It then explores the underlying pathophysiological determinants, including cardiopulmonary interactions, impaired lung mechanics, right heart failure, and altered intrathoracic pressure dynamics. Surgical strategies and anesthesia management also critically influence weaning trajectories, with evidence suggesting a variable impact based on surgical approach, cardiopulmonary bypass use, and intraoperative ventilation settings. The role of spontaneous breathing trials and protocolized weaning is reviewed, highlighting their applicability and limitations in this unique setting. An extensive analysis of monitoring tools—such as esophageal manometry, diaphragm ultrasound, and electrical impedance tomography—follows, emphasizing their role in phenotyping and individualizing care. Sedation, analgesia, and delirium management are discussed as key drivers of liberation success, along with the impact of preexisting and acquired neuromuscular dysfunction. Nutritional status, muscle wasting, and sarcopenia are also addressed as systemic barriers to ventilator liberation. Importantly, this review considers the long-term consequences of weaning failure, including functional disability and persistent neurocognitive impairment. We propose an integrated, multimodal framework for assessing readiness to wean and underscore the importance of postextubation trajectories. Future research should focus on the development of digital dashboards, predictive indices, and personalized protocols to optimize ventilator liberation and improve long-term outcomes in cardiac surgery patients.

Keywords: Weaning; mechanical ventilation; cardiac surgery; spontaneous breathing trial; future perspectives; heart−lung interaction; risk stratification; monitoring; extubation failure; evidence-based medicine

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https://doi.org/10.1053/j.jvca.2026.02.009

Introduction

Weaning from mechanical ventilation (MV) is a complex, multifactorial process that accounts for a significant portion of the time spent on ventilatory support in intensive care units (ICUs). In cardiac surgical patients, the process of liberation from MV presents unique challenges due to preexisting comorbidities, the physiological impact of cardiopulmonary bypass (CPB), postoperative pulmonary complications, and the frequent use of sedatives and opioids during and after surgery.[1]

While most patients undergoing cardiac surgery are extubated within the first 6 to 12 hours postoperatively, a subset experiences prolonged weaning, defined variably in the literature but commonly as the failure to achieve successful extubation within 7 days after the first spontaneous breathing trial (SBT).[2] Prolonged weaning is associated with increased ICU and hospital length of stay, higher health care costs, and worse outcomes, including increased mortality.[3] Despite its clinical relevance, weaning failure remains poorly predicted and inconsistently managed, especially in the context of cardiac surgery.

Traditional weaning classifications (simple, difficult, and prolonged) do not adequately capture the complexity of the cardiac surgical population, which often includes patients with diastolic dysfunction, residual myocardial ischemia, high pulmonary artery pressures, or postoperative low cardiac output syndrome.[4] Moreover, extracardiac factors such as diaphragmatic dysfunction, fluid overload, or impaired chest wall mechanics due to sternotomy or thoracotomy contribute to weaning difficulties.[5]

Recent years have seen growing interest in personalizing the weaning process through physiological monitoring, early rehabilitation protocols, and integration of advanced ventilatory strategies. However, no specific guidelines currently exist for ventilator weaning in adult cardiac surgical patients, and most protocols are extrapolated from general ICU populations.[6] This lack of standardization leads to significant heterogeneity in clinical practice and outcomes. It should be acknowledged that part of the literature on ventilator weaning after cardiac surgery includes patients with congenital heart disease. However, these populations differ substantially from adult cardiac surgery patients in terms of baseline physiology, surgical complexity, and postoperative trajectories. The present review primarily focuses on adult cardiac surgery patients, in whom weaning failure is more commonly driven by acquired cardiopulmonary dysfunction, comorbidities, and perioperative factors rather than congenital physiology. Therefore, findings derived from congenital heart disease populations should be interpreted with caution when extrapolated to adult cardiac surgical patients.

The present review aims to provide a comprehensive, multidisciplinary synthesis of the available evidence on weaning from mechanical ventilation in cardiac surgery patients. We address the epidemiology, pathophysiology, predictors of weaning failure, monitoring strategies, and therapeutic interventions, including the role of tracheostomy. Particular attention is given to post−cardiac surgical patients at risk of prolonged weaning and the development of a tailored, physiology-based approach to liberation from mechanical ventilation.

Epidemiology and Definitions of Prolonged Weaning in Cardiac Surgery Patients

The process of weaning from mechanical ventilation after cardiac surgery is typically rapid in most patients, with extubation occurring within 6 to 12 hours in 85% to 90% of cases.[7] This is especially true for patients undergoing elective procedures with preserved ventricular function and no major intraoperative complications. However, a significant minority—estimated between 5% and 15%—develop postoperative respiratory failure requiring prolonged ventilatory support, which may result in difficult or prolonged weaning.[8] However, the definition of prolonged weaning and related outcomes remains highly heterogeneous across studies, particularly in cardiac surgical populations. Table 1 summarizes the most commonly used definitions, incidence ranges, and classification frameworks currently applied in this setting.

Table 1
Epidemiology and Definitions of Weaning Outcomes After Cardiac Surgery

Item	Definition/Data	Source
Successful weaning	Extubation without reintubation within 48-72 h	Boles et al.[9]
Difficult weaning	Failure of first SBT, success within ≤3 attempts	Boles et al.[9]
Prolonged weaning	>7 days after first SBT	Boles et al.[9]
WIND classification	Short (<24 hours), difficult (1-7 days), prolonged (≥7 days) weaning after the first separation attempt, independent of extubation status	Béduneau et al.[10]
Prolonged MV (cardiac surgery)	MV ≥24 h after surgery	STS/Sankar et al.[11]
Incidence of prolonged MV	10%-20% (≈15% in TRICS III)	Sankar et al.[11]
Tracheostomy rate	2%-11%	Observational studies

This table summarizes the most commonly used definitions of weaning success, difficult and prolonged weaning, as well as prolonged mechanical ventilation specifically applied to cardiac surgery patients. Major classification systems (International Consensus and WIND) and reported incidence ranges from large multicenter studies are presented to highlight heterogeneity in definitions and clinical implications.
Abbreviations: MV, mechanical ventilation; SBT, spontaneous breathing trial; STS, Society of Thoracic Surgeons; WIND, Weaning according to a New Definition.

The International Consensus Conference organized by Boles et al.[9] established the first standardized classification of weaning from mechanical ventilation, distinguishing 3 categories based on the number of SBTs and the total duration of the weaning process: simple, difficult, and prolonged weaning. Subsequent validation studies confirmed that prolonged weaning identifies a distinct high-risk clinical phenotype associated with increased morbidity and mortality, even outside the cardiac surgery population.[12]

The introduction of the WIND classification by Béduneau et al.[10] represented a major step forward in standardizing the definition of weaning outcomes. In a multicenter study including more than 2,700 mechanically ventilated patients, the authors proposed a pragmatic categorization based on the duration of the weaning process—short (<1 day), difficult (1-7 days), and prolonged (>7 days)—irrespective of extubation status. While this classification remains broadly accepted in general ICU populations, it is less frequently applied in cardiac surgery cohorts, in whom the etiology and timing of respiratory failure are distinct.

Prolonged weaning in cardiac surgery patients is often multifactorial and may be driven by cardiac complications (eg, low output syndrome, myocardial stunning), pulmonary dysfunction (eg, atelectasis, pulmonary edema, acute respiratory distress syndrome [ARDS]), or systemic issues such as sepsis or acute kidney injury.[7,13] Additionally, the CPB circuit itself contributes to an inflammatory cascade, capillary leak, and potential diaphragmatic dysfunction, all of which may delay successful extubation.[14,15]

Several observational studies have reported that approximately 3% to 10% of cardiac surgery patients fall into the prolonged weaning category, although the exact incidence varies depending on institutional practices, definitions used, and patient characteristics.[16] A large multicenter cohort demonstrated that prolonged weaning in this population is independently associated with increased mortality, particularly when associated with failed SBTs or reintubation. Importantly, each additional day on the ventilator after cardiac surgery correlates with higher risks of nosocomial pneumonia, ICU delirium, muscle weakness, and long-term functional decline.[17]

Contemporary evidence from large, multicenter cardiac surgery cohorts confirms that prolonged mechanical ventilation remains a frequent postoperative complication. In a secondary analysis of the TRICS III trial, including more than 4,800 patients across 71 centers worldwide, Sankar et al.[11] reported an incidence of prolonged mechanical ventilation of approximately 15%, defined as ventilation lasting ≥24 hours. Prolonged ventilation was independently associated with surgical complexity (eg, cardiopulmonary bypass duration, prior cardiac surgery) and patient-related factors, such as left ventricular dysfunction, renal failure, and pulmonary hypertension, and was strongly associated with perioperative complications and mortality.

The heterogeneity in definitions across studies poses a barrier to synthesizing evidence and implementing standardized weaning pathways. Some centers define "prolonged ventilation" as >24 or >48 hours of MV, while others consider a duration of >96 hours as a threshold for concern.[18,19] These inconsistencies underscore the need for tailored definitions and risk stratification tools specific to cardiac surgical patients.

Observational studies in specialized weaning units confirm that cardiac surgery patients requiring prolonged ventilation represent a high-risk subgroup with poor short-and long-term outcomes.[11,15,18,19]

Moreover, the need for reintubation or unplanned noninvasive ventilation after apparent successful extubation is frequent in this population, often revealing a latent failure in respiratory or cardiovascular reserve. These events should not be regarded simply as extubation failures but rather as indicators of a mismatch between weaning readiness and physiologic capacity.[20-22]

Understanding the epidemiology and refining definitions are essential steps toward designing interventional studies and implementing structured weaning protocols. Importantly, it also helps in identifying patients who may benefit from preemptive measures such as prophylactic noninvasive ventilation, individualized sedation protocols, or delayed extubation in high-risk subsets.[23,24]

Predictors of Weaning Failure and Patient Risk Stratification

In this review, the term *weaning failure* is used to describe unsuccessful liberation attempts or intolerance to spontaneous breathing trials, rather than a permanent inability to discontinue mechanical ventilation.

Predicting which cardiac surgery patients are at risk of weaning failure remains a critical challenge in perioperative care. Early identification of these patients allows for timely interventions and optimization of supportive strategies.[25] However, the complexity of perioperative physiology in this setting requires a multifactorial approach that considers cardiac, respiratory, neurologic, and systemic parameters.[26,27] Table 2 provides an overview of the main predictors of weaning failure and their clinical relevance, highlighting the multidimensional nature of postoperative risk assessment.

Clinical and Demographic Risk Factors

Several preoperative variables have been associated with prolonged mechanical ventilation and weaning failure.[25] These include advanced age, high body mass index, poor baseline functional status, chronic obstructive pulmonary disease, obstructive sleep apnea, chronic kidney disease, and left ventricular systolic dysfunction.[28,29] Patients with a high EuroSCORE II or Society of Thoracic Surgeons score are more likely to require prolonged ventilatory support after surgery.[30] Intraoperative factors such as prolonged CPB time, high intraoperative transfusion requirements, hypothermia, and low cardiac output at weaning from CPB have also been implicated in weaning failure.[31] Intraoperative ventilation strategies may also influence the trajectory of postoperative respiratory mechanics and, consequently, the ease of ventilator weaning after cardiac surgery. Lung-protective approaches, including the use of low tidal volumes, avoidance of excessive driving pressures, and judicious application of positive end-expiratory

Table 2
Predictive Indices and Scores for Weaning Success

Index/Score	Formula/Components	Physiological Domain	Typical Cutoff for Success	Notes/Limitations
RSBI (Rapid Shallow Breathing Index)	RR/VT (breaths/min/L)	Respiratory pattern	$<$105 breaths/min/L	Poor specificity in sedated/poststernotomy patients
NIF/MIP (negative inspiratory force)	Max inspiratory pressure (cmH_2O)	Respiratory muscle strength	<-20 cmH_2O	Effort-dependent; limited in weak or delirious patients
IWI (Integrative Weaning Index)	(Cstat × SaO_2)/(RR/Vt)	Global performance	$>$25	Combines compliance and oxygenation
CROP Index	Cdyn × (PaO_2/PAO_2) × (MIP/RR)	Cardiopulmonary integration	$>$13	Sensitive to both cardiac and respiratory load
CORE-SCORE	Composite (RR, HR, MIP, P0.1, PaO_2/FiO_2)	Multisystem	Variable (AUC ≈ 0.85)	Promising, but requires validation in cardiac surgery
BNP/NT-proBNP	Biomarker of LV overload	Cardiogenic component	$<$200 pg/mL	Distinguishes WIPO from noncardiogenic failure

Overview of commonly used respiratory, cardiopulmonary, and integrative indices to predict weaning readiness and extubation outcomes. The table summarizes their formula or main components, physiological domain, typical cutoffs associated with successful weaning, and principal limitations—particularly relevant in postoperative cardiac surgery patients, where sedation, reduced respiratory drive, altered chest wall mechanics, and transient myocardial dysfunction may affect test performance.

Abbreviations: BNP/NT-proBNP, B-type natriuretic peptides; Cdyn, dynamic compliance; Cstat, static compliance; HR, heart rate; LV, left ventricle; MIP, maximal inspiratory pressure; NIF/MIP, negative inspiratory force/maximal inspiratory pressure; PAO_2, alveolar oxygen pressure; PaO_2, partial pressure of arterial oxygen; PaO_2/FiO_2, Ratio of arterial partial oxygen pressure to fractional inspired oxygen; PO.1, occlusion pressure at 0.1 seconds; RR, respiratory rate; SaO_2, arterial oxygen saturation; VT, tidal volume; WIPO, weaning-induced pulmonary edema.

pressure, may reduce atelectasis, lung edema, and postoperative loss of compliance. In addition, minimizing intraoperative lung overdistension and cyclic derecruitment may attenuate diaphragmatic dysfunction and respiratory muscle unloading in the immediate postoperative period. Although direct evidence linking specific intraoperative ventilation strategies to weaning outcomes in cardiac surgery patients remains limited, these approaches may contribute to a more favorable physiological substrate for early postoperative spontaneous breathing and ventilator liberation.[32]

Likewise, postoperative complications, including bleeding, acute kidney injury, delirium, and pulmonary infections, markedly increase the risk of extubation failure or need for reintubation.[19]

Beyond isolated cardiopulmonary mechanisms, the burden and interaction of comorbidities play a pivotal role in determining weaning outcomes. Advanced age, multimorbidity, and frailty frequently coexist in cardiac surgery patients and substantially affect tolerance to spontaneous breathing. Recent data from the WEAN SAFE study demonstrated that comorbidities—including chronic kidney disease, neurologic impairment, and metabolic disorders—are independently associated with prolonged weaning and worse outcomes, underscoring the multifactorial nature of weaning failure in contemporary ICU populations. These findings reinforce the need for an integrated, patient-centered approach rather than reliance on single physiological parameters.[33]

While early studies laid the foundation for understanding weaning physiology after cardiac surgery, substantial advances in monitoring technologies and ventilatory strategies have occurred over the past 2 decades. Contemporary approaches increasingly incorporate dynamic assessment of respiratory effort, cardiac−pulmonary interactions, and bedside imaging, allowing a more individualized and physiology-driven weaning process.

Cardiovascular Predictors

In the cardiac surgical population, weaning failure is often due to cardiovascular instability rather than pure respiratory insufficiency.[34] The concept of cardiogenic weaning failure is increasingly recognized and refers to the inability of the heart to tolerate the hemodynamic changes induced by spontaneous breathing—namely, increased venous return and afterload.[35] This may manifest as hypotension, pulmonary edema, or decreased mixed venous oxygen saturation during or after the SBT.[36]

Elevated B-type natriuretic peptide (BNP) levels before or during SBT have been shown to predict cardiovascular weaning failure with reasonable accuracy.[37,38] Transthoracic echocardiography may also be informative, particularly in assessing left ventricular filling pressures, right ventricular dysfunction, and dynamic changes in ejection fraction or stroke volume during spontaneous breathing trials.[39,40] More specifically:

- Right ventricular (RV) function: The transition from mechanical ventilation to spontaneous breathing can increase venous return and RV afterload. Assessing RV function is therefore crucial.[41] While increased venous return during spontaneous breathing trials may unmask right ventricular dysfunction, it should also be acknowledged that positive pressure ventilation can increase pulmonary vascular resistance through lung overdistension and compression of the pulmonary vasculature. Consequently, in selected patients, the transition to spontaneous breathing and extubation may actually improve right ventricular performance by reducing RV afterload.[42]

- Echocardiographic measures: Parameters such as tricuspid annular plane systolic excursion and right ventricular fractional area change can provide insight into RV performance; however, tricuspid annular plane systolic excursion should be interpreted with caution in the presence of severe tricuspid regurgitation, where longitudinal annular motion may overestimate effective RV systolic function. An impaired RV may struggle to accommodate the increased preload, potentially leading to right heart failure during weaning.[43]
- Systolic pulmonary artery pressure (sPAP): Estimations of sPAP during echocardiography can help assess pulmonary vascular resistance, which, if elevated, may compromise RV function during spontaneous breathing[44,45] but can also result from elevated left-sided filling pressures in the setting of left ventricular dysfunction; therefore, sPAP should be interpreted within the broader hemodynamic and echocardiographic context.
- Hemodynamic monitoring: Continuous monitoring of heart rate, blood pressure, and central venous pressure (CVP) allows clinicians to gauge the heart's response during weaning to detect adverse shifts in preload and afterload, as well as integrates pulse contour analysis and echocardiography to evaluate the impact of weaning on cardiac function.[46] Sudden changes in these parameters can be indicative of an underlying cardiac limitation. For example, a significant rise in CVP or a drop in blood pressure during an SBT may signal that the heart is not tolerating the increased preload that accompanies spontaneous breathing.[47,48]
- Pulmonary artery catheter**:** In selected patients, pulmonary artery catheterization during spontaneous breathing trials may help distinguish predominant right, left, or biventricular dysfunction by integrating changes in filling pressures, cardiac output, and pulmonary vascular resistance during the transition to spontaneous breathing.[47]

Respiratory Predictors

Pulmonary predictors of weaning failure include a low PaO_2/FiO_2 ratio, poor cough strength, elevated $PaCO_2$, high ventilatory demand, and low respiratory system compliance.[49,50] The rapid shallow breathing index (RSBI), calculated as the ratio of respiratory rate to tidal volume, is traditionally used as a predictor of SBT success, with values <105 breaths/min/L associated with successful weaning.[51-53] Cardiac surgery patients may experience significant hemodynamic shifts upon transitioning from positive-pressure ventilation to spontaneous breathing. RSBI focuses solely on respiratory mechanics (respiratory rate and tidal volume) and does not account for cardiac factors such as changes in preload, afterload, or myocardial performance.[52] Moreover, postoperative changes—such as myocardial stunning, fluid shifts, or residual ischemia—can affect breathing patterns and respiratory muscle performance in ways that are not reflected by the RSBI. Therefore, a patient might have an acceptable RSBI value while still harboring cardiac dysfunction that could lead to extubation failure.[53]

Respiratory muscle weakness, including diaphragmatic dysfunction, is increasingly recognized as a significant contributor to weaning failure. Studies employing diaphragm ultrasound have demonstrated reduced thickening fraction or paradoxical motion in patients with weaning difficulty.[54,55] The mechanisms include CPB-induced diaphragmatic inflammation, phrenic nerve dysfunction, and critical illness myopathy.[56]

Integrated Weaning Risk Scores

To better stratify weaning risk, several integrative scoring systems have been proposed. The Integrative Weaning Index combines RSBI, static compliance, and oxygenation parameters and has shown promise in predicting successful weaning in mixed ICU populations.[57] However, its utility in cardiac surgery remains to be specifically validated.

More recently, machine learning models incorporating high-dimensional data (vital signs, ventilator waveforms, laboratory values) have been trained to predict extubation outcomes with greater accuracy than traditional scores.[58] Although these models are still in the early stages of validation, they offer a glimpse into the future of personalized weaning pathways.

Spontaneous Breathing Trials: Timing, Modalities, and Monitoring

SBTs represent the cornerstone of the ventilator weaning process, providing a controlled assessment of a patient's ability to sustain spontaneous ventilation.[59] In cardiac surgery patients, the optimal timing, duration, and mode of SBT remain debated, especially given the unique perioperative alterations in cardiorespiratory physiology and sedation depth. This section explores the current evidence on SBT practices and their role in optimizing ventilator liberation after cardiac surgery.[7,11,60] Figure 1 presents an integrated clinical framework for weaning from mechanical ventilation after cardiac surgery, designed to support individualized decision-making; Figure 2 illustrates the core cardiopulmonary physiological changes occurring during the transition from mechanical ventilation to spontaneous breathing, independently of the underlying cause of weaning failure.

Timing of SBT Initiation

The timing of the first SBT is a critical determinant of weaning success. Initiating SBT too early, particularly in the presence of hemodynamic instability, residual neuromuscular blockade, or unresolved pulmonary pathology, increases the risk of failure and reintubation.[60] Conversely, delaying the first SBT may lead to unnecessary prolongation of mechanical ventilation, sedation accumulation, and ICU-related complications such as delirium and muscle weakness.[11,61]

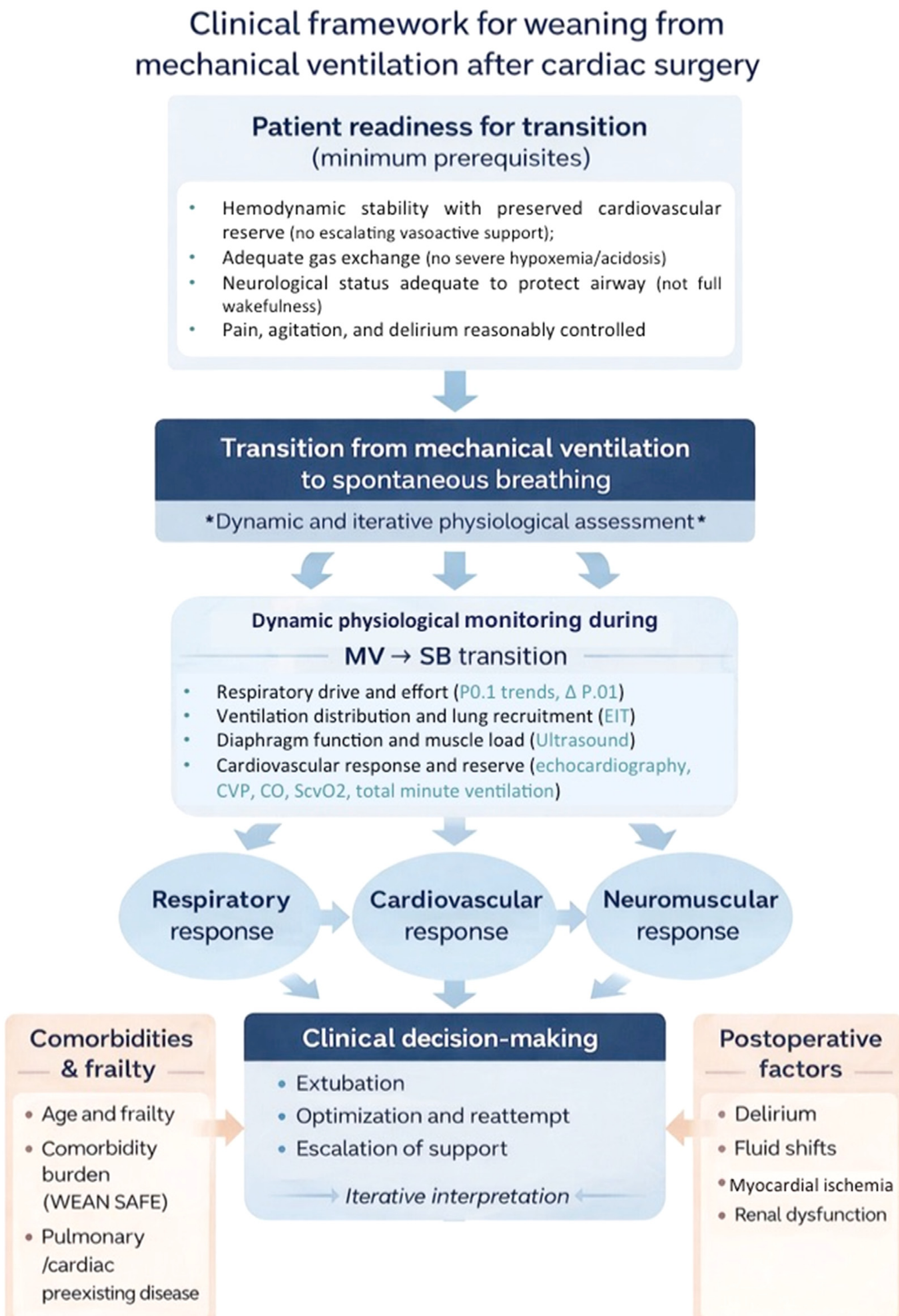

Figure 1. Clinical framework for weaning from mechanical ventilation after cardiac surgery. This figure illustrates an integrated and nonalgorithmic framework for the transition from mechanical ventilation to spontaneous breathing. It emphasizes minimum readiness prerequisites; dynamic physiological monitoring across respiratory, cardiovascular, and neuromuscular domains; and iterative clinical decision-making informed by comorbidities, frailty, and postoperative factors. The framework is intended to support individualized clinical judgment rather than prescribe a rigid weaning pathway. CO, cardiac output; CVP, central venous pressure; EIT, electrical impedance tomography; MV, mechanical ventilation; P0.1, airway occlusion pressure at 100 ms; ΔP0.1, change in P0.1; SB, spontaneous breathing; $ScvO_2$, central venous oxygen saturation.

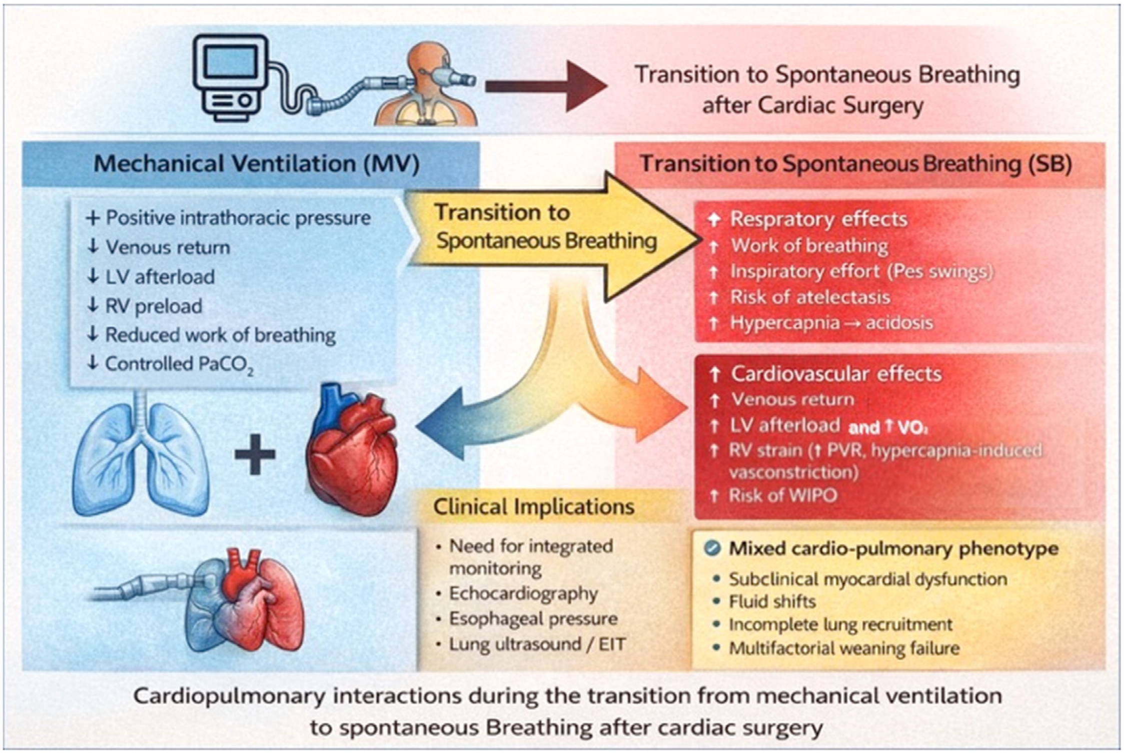

Figure 2. Cardiopulmonary interactions during the transition from mechanical ventilation to spontaneous breathing after cardiac surgery. The figure illustrates the major respiratory and cardiovascular physiological changes occurring during the shift from positive-pressure mechanical ventilation to spontaneous breathing. Removal of positive intrathoracic pressure leads to increased venous return, left ventricular afterload, and right ventricular preload, while respiratory effort and work of breathing increase. These combined effects may unmask subclinical myocardial dysfunction and promote mixed cardiopulmonary phenotypes, contributing to weaning failure. The figure highlights the need for integrated respiratory and hemodynamic monitoring during this transition. EIT, electrical impedance tomography; LV, left ventricle; MV, mechanical ventilation; $PaCO_2$, carbon dioxide partial pressure; Pes, esophageal pressure; PVR, pulmonary vascular resistance; RV, right ventricle; SB, spontaneous breathing; VO_2, oxygen consumption; WIPO, weaning-induced pulmonary edema.

Current guidelines recommend a daily screening protocol for SBT readiness, but these were primarily developed for general ICU populations.[62] Parameters often used to trigger the first SBT include stable hemodynamics (with or without minimal vasopressor support), adequate oxygenation (eg, $PaO_2/FiO_2 > 200$), controlled secretions, appropriate mental status, and normalized acid−base balance.[9,63] The assessment of residual effects of anesthesia and neuromuscular blocking agents is particularly relevant in this setting.[64]

SBT Modalities

Different SBT modalities have been proposed, including (1) T-piece trial, which requires the patient to breathe spontaneously through a simple oxygen circuit without any ventilatory assistance; (2) low-level pressure support ventilation (PSV), typically 5 to 7 cmH_2O with positive end-expiratory pressure (PEEP) 5 cmH_2O; (3) continuous positive airway pressure trials, using continuous airway pressure without inspiratory support; and (4) automatic tube compensation, which compensates for endotracheal tube resistance.[9]

The T-piece has traditionally been considered the most demanding form of SBT and has been widely used in cardiac surgical ICUs due to its ability to unmask cardiopulmonary insufficiency.[11,65] However, it may not adequately replicate the conditions postextubation, where upper airway resistance and intrinsic PEEP may play a larger role.

PSV trials, on the other hand, may underestimate the work of breathing required postextubation, potentially resulting in overoptimistic assessments.[66] Nonetheless, in selected patients, low-level PSV has been associated with a reduced duration of weaning without increasing the risk of reintubation.[67] It should be acknowledged that not all SBT modalities generate comparable intrathoracic pressure changes. SBTs performed with a T-piece or zero-pressure support are more likely to reproduce fully spontaneous breathing and negative intrathoracic pressure swings, whereas SBTs conducted with pressure support and PEEP may partially attenuate these effects. Accordingly, the hemodynamic and respiratory stress imposed by an SBT may vary depending on the selected modality.[65-67]

Several studies have compared these modalities in heterogeneous ICU populations, but very few have specifically addressed post−cardiac surgery patients. The most recent evidence on the optimal approach to spontaneous breathing trials comes from the multicenter TIP-EX trial by Thille et al.,[68,69] which randomized 969 ICU patients at high risk of extubation failure to undergo SBTs with either pressure support ventilation (8 cmH_2O, zero PEEP) or a T-piece. The study found no significant difference between groups in the number of ventilator-free days at day 28 (median 27 *v* 27 days, p = 0.31), rates of successful extubation, or reintubation within 7 days (14.9% *v* 13.8%). These results suggest that both methods may be effective for assessing readiness for extubation in selected high-risk ICU populations. Importantly, most patients received postextubation high-flow oxygen or noninvasive ventilation, which likely mitigated differences in postextubation outcomes. Therefore, current evidence supports the use of either PSV or T-piece for SBTs in general ICU populations, while their applicability in post−cardiac surgery patients should be interpreted with caution and guided by cardiovascular tolerance.

Duration and Repetition of SBTs

The duration of SBTs typically ranges from 30 to 120 minutes. Trials shorter than 30 minutes may not sufficiently stress the cardiorespiratory system, while prolonged trials may expose patients to undue fatigue. A 30- to 60-minute trial is generally considered adequate in cardiac surgery patients, although some protocols extend to 2 hours in high-risk settings.[9,59,70]

In cases of SBT failure, repeated daily SBTs remain the standard approach, ideally accompanied by optimization strategies (eg, diuretic therapy, bronchodilation, sedation weaning) between attempts. A failed SBT should prompt a careful assessment of reversible causes and the implementation of targeted interventions prior to rechallenge.[9,62,71]

Monitoring During SBTs

Careful physiological monitoring during SBT is essential to identify early signs of failure and avoid unsafe extubation. Key monitoring variables include parameters such as respiratory rate >35/min or <8/min; SpO_2 (Periferal Oxygen Saturation) <90% or PaO_2 drop with FiO_2 ≥0.4; tachycardia, arrhythmias, or hypotension; signs of respiratory distress, paradoxical breathing, or diaphoresis; and $PaCO_2$ rising >10 mm Hg from baseline or pH <7.32.

Post-SBT Evaluation

Even when an SBT is tolerated without clear signs of distress, it does not guarantee successful extubation. Posttrial evaluation should include assessment of cough strength, using objective (peak cough flow) or subjective scales; secretion load and suctioning frequency; airway patency, including risk of upper airway obstruction and neurologic function; and ability to protect the airway.[72,73] In cardiac surgery patients, the risk of negative-pressure pulmonary edema due to dynamic upper airway collapse is not negligible and must be anticipated, particularly in patients with obstructive sleep apnea or high body mass index.[74-76] In selected high-risk patients, extubation failure predictors such as failed cuff-leak tests, low cough peak flow (<60 L/min), or excessive secretion burden may prompt consideration of prophylactic noninvasive ventilation or high-flow nasal oxygen immediately after extubation.[77,78]

Role of Respiratory Mechanics and Esophageal Pressure Monitoring

The analysis of respiratory mechanics plays a pivotal role in the weaning process. In cardiac surgery patients, this assessment becomes even more relevant due to transient alterations in thoracic compliance, diaphragm dysfunction, and postoperative fluid shifts.[79-84] The integration of esophageal pressure (Pes) monitoring offers a unique opportunity to separate the contributions of the lung and chest wall to overall respiratory effort, thus refining clinical decision-making during weaning.[85] In this context, advanced monitoring should not be viewed as a means to identify a single “cause” of weaning failure but rather as a tool to characterize dominant contributors within a multifactorial process. In most postoperative cardiac surgery patients, routine SBTs remain appropriate and sufficient, with advanced monitoring reserved for those with recurrent SBT failure, borderline physiological reserve, or discordant clinical signals.

Table 3 summarizes the key mechanical parameters used during weaning, including their physiological significance and recommended targets in postoperative cardiac surgery patients.

Basic Principles of Respiratory Mechanics in Weaning

Mechanical ventilation imposes external support on a system composed of the lung parenchyma, chest wall, and respiratory muscles. The ability to wean depends not only on gas exchange capacity but also on the balance between respiratory load and muscle capacity.[86] Key parameters derived from ventilator waveforms or advanced measurements include driving pressure (ΔP = Pplat − PEEP): associated with lung stress and strain; respiratory system compliance (Crs): reflects the ease of lung and thoracic expansion; work of breathing: inferred or

Table 3
Key Mechanical Parameters During Weaning

Parameter	Physiological Significance	Optimal Target During Weaning
ΔPes	Inspiratory muscle effort	<10-15 cmH_2O
ΔP	Global lung stress	<15 cmH_2O
PL,insp	End-inspiratory transpulmonary pressure	<25 cmH_2O
MP	Energy load	<12-17 J/min
Strain	Volume change/FRC	<2

Summary of the main physiological variables used to assess respiratory workload, lung stress, and transpulmonary pressures during the weaning phase. For each parameter, the table reports its physiological significance and the optimal target range associated with a safer transition from supported to spontaneous breathing in postoperative cardiac surgery patients.
Abbreviations: ΔPes, esophageal pressure swing; ΔP, driving pressure; PL, insp, end-inspiratory transpulmonary pressure; MP, mechanical power; Strain, ratio between tidal volume and functional residual capacity (VT/FRC).

directly measured as an index of patient effort; pressure−time product (PTP) and esophageal pressure swing (ΔPes): directly measure muscular effort. In post−cardiac surgery patients, decreased compliance may result from atelectasis, pleural effusions, or thoracic stiffness after sternotomy.[87,88]

Chest Wall and Lung Mechanics: The Importance of Partitioned Assessment

Traditional measures of Crs combine the mechanical properties of the lung and chest wall, potentially masking pathological conditions. For example, a low Crs may reflect either lung overdistension (low compliance due to high alveolar pressures) or chest wall stiffness (eg, obesity, surgical dressings, pain-induced splinting) and abdominal pressure effects (eg, ileus, obesity, elevated intra-abdominal pressure).[89]

By using an esophageal balloon catheter, clinicians can separate the pressure generated by the chest wall (Pcw) from that of the lung (PL = Pao − Pes), allowing for the calculation of transpulmonary pressure (PL), which represents the true distending force across the lung.[90]

This distinction is critical in cardiac surgery patients, where postoperative changes in chest wall mechanics (eg, from median sternotomy or thoracotomy) may not reflect true lung compliance. Adjusting ventilatory settings based on global Crs alone may therefore be misleading.

Esophageal Pressure Monitoring: Technique and Interpretation

Esophageal pressure monitoring involves placement of a balloon-tipped catheter in the lower third of the esophagus, serving as a surrogate for pleural pressure. Proper placement is confirmed by occlusion testing and waveform validation.[91]

Key variables derived from Pes include ΔPes: respiratory muscle pressure swing during inspiration; PTP: area under the pressure−time curve, a measure of cumulative effort; PL-insp: end-inspiratory transpulmonary pressure, indicating risk of overdistension; PL-exp: end-expiratory transpulmonary pressure, used for PEEP titration.[92]

During spontaneous breathing trials, excessive ΔPes or PTP suggests high effort and predicts failure.[93] Conversely, minimal effort may reflect overassistance or respiratory muscle weakness.

Importantly, high PL-insp (>25 cmH_2O) may signal overdistension, while negative PL-exp may indicate an alveolar collapse risk. Thus, Pes monitoring may be instrumental in setting optimal PEEP and avoiding both atelectrauma and volutrauma.[94]

Monitoring and Predicting Weaning Outcome

Several studies have demonstrated that Pes-derived variables outperform traditional indices (eg, RSBI) in predicting SBT outcome.[95-97] For example, patients with SBT failure often show ΔPes >10 to 15 cmH_2O, indicating increased respiratory drive or effort; high PTP per minute correlates with early fatigue and SBT failure; lack of reduction in PL during assisted ventilation may indicate ineffective support or respiratory muscle fatigue.

Recent data suggest that esophageal pressure monitoring can also predict the need for postextubation support, such as noninvasive ventilation or HFNO (High Flow Nasal Oxygen), particularly when ΔPes remains elevated despite acceptable oxygenation and hemodynamics.[98]

Mechanical Power, Stress, and Strain in the Context of Weaning

Building on the previously discussed concepts of respiratory load, effort, and patient−ventilator interaction, mechanical power, stress, and strain provide an integrative physiological framework rather than additional independent weaning criteria.

Early optimization of ventilatory support after cardiac surgery has been associated with physiologic correlates of improved outcomes and reduced incidence of ventilator-induced complications. Simeone et al.[99] conducted a randomized clinical study in 49 elective cardiac surgery patients comparing a fast-track weaning protocol with conventional management. The protocol group showed a significantly shorter duration of mechanical ventilation, reduced ICU stay, and improved oxygenation (PaO_2/FiO_2 ratio), with no adverse hemodynamic effects. The authors highlighted that prolonged mechanical ventilation may trigger systemic inflammatory responses through cytokine release (IL-6, IL-8, and TNF), thereby promoting ventilator-induced lung injury and postoperative organ dysfunction. These findings reinforce the rationale for early individualized ventilatory adjustment to minimize lung stress and enhance postoperative recovery.

Beyond static indices such as compliance or driving pressure, the concept of mechanical power (MP) has recently emerged as an integrative parameter quantifying the total energy delivered to the respiratory system per minute.[100] Mechanical power encompasses the combined effects of tidal volume, respiratory rate, flow, airway pressure, and PEEP.[101] During assisted or spontaneous ventilation, excessive mechanical power may lead to patient self-inflicted lung injury even in patients without ARDS, particularly when inspiratory effort is high.[102]

MP can be estimated as:

$MP = 0.098 \times RR \times VT \times (Ppeak - \frac{1}{2} \times \Delta P)$ where MP is expressed in J/min, RR is respiratory rate (breaths/min), VT is tidal volume (L), Ppeak is peak inspiratory pressure (cmH_2O), and ΔP is driving pressure. For controlled ventilation, simplified forms also exist, incorporating flow-dependent terms.[103] During weaning, patient effort contributes additional power, which can be indirectly estimated from esophageal pressure swings (ΔPes) and work of breathing.[104]

In cardiac surgery patients, the transient reduction in compliance (due to atelectasis, edema, or chest wall stiffness) can increase MP even under protective settings.[84,88] Importantly, higher MP during SBT or pressure-support ventilation may not always be deleterious if accompanied by lower driving pressure and better recruitment, suggesting a potential physiological redistribution of energy, the clinical implications of which remain uncertain, rather than excessive cyclic stress.[105] However, sustained MP above 12 to 17 J/min has been associated with weaning failure and postoperative pulmonary complications in observational studies.[106]

Despite their strong physiological rationale, the clinical role of mechanical power, stress, and strain during weaning—particularly in post−cardiac surgery patients—remains incompletely defined. Current evidence is largely derived from small single-center studies or extrapolated from ARDS and controlled ventilation settings, limiting its generalizability to spontaneous or assisted breathing during weaning. Importantly, no large prospective studies have validated MP-, stress-, or strain-based thresholds as decision-making targets during spontaneous breathing trials or extubation readiness assessment. The randomized study by Simeone et al.,[99] while hypothesis-generating, was limited by sample size and was not designed to establish causal relationships between these variables and weaning outcomes. Accordingly, in the context of weaning after cardiac surgery, mechanical power, stress, and strain should be regarded as integrative descriptors of respiratory load and patient−ventilator interaction rather than as established clinical targets. Their primary value currently lies in supporting physiological interpretation, risk stratification, and future research, rather than in guiding routine bedside decisions.

Lung Stress and Strain: Translating Mechanics Into Tissue Load

While mechanical power quantifies energy delivery, stress and strain describe the mechanical consequences on the lung parenchyma.[107]

Stress corresponds to the transpulmonary pressure (PL)—that is, the distending pressure across the lung—calculated as PL = Paw − Pes; strain represents the relative change in lung volume compared with the functional residual capacity (FRC):

$$Strain = \left(VT + \Delta EELV\right)/FRC$$

Experimental and clinical evidence suggests that excessive stress (>25 cmH_2O) or strain (>2) induces microstructural damage and alveolar rupture.[108] During the weaning phase, particularly in patients with strong inspiratory effort or derecruited lung regions, local stress and strain may far exceed global values derived from airway pressure alone.[109]

Role of Diaphragmatic Ultrasound and Electrical Impedance Tomography in Monitoring

In recent years, the use of noninvasive bedside monitoring tools such as diaphragmatic ultrasound and electrical impedance tomography (EIT) has gained increasing attention for its ability to provide real-time physiological insights during the weaning process.[110-114] These tools allow clinicians to assess respiratory muscle function and regional lung aeration, both of which are critical determinants of weaning success, particularly in high-risk populations such as post−cardiac surgery patients.[115]

Diaphragmatic Ultrasound: A Window Into Muscle Function

The diaphragm is the principal muscle of respiration, and its dysfunction is strongly associated with weaning failure.[116] After cardiac surgery, diaphragm function may be compromised due to phrenic nerve injury (eg, during internal mammary artery harvesting), postoperative inflammation or edema, sedation, opioids, neuromuscular blocking agents, and disuse atrophy from prolonged mechanical ventilation.[117]

Ultrasound of the diaphragm offers a rapid, reproducible, and noninvasive technique to assess diaphragm activity at the bedside.[118-120] Two main parameters are typically measured:

- Diaphragm excursion (DE): the cranio-caudal movement during inspiration, usually measured in M-mode at the zone of apposition
- Diaphragm thickening fraction (TF): calculated as

$$TF = (Thicknessatend - inspiration - Thicknessatend \\ -expiration)/Thicknessatend - expiration \times 100\%.$$

Values of DE <1 cm or TF <20% to 30% have been associated with diaphragm dysfunction and predict weaning failure in several populations.[121]

In cardiac surgical patients, studies have shown that diaphragm dysfunction may persist for several days after surgery, even in the absence of overt phrenic nerve injury. This may manifest as reduced DE and TF, with incomplete recruitment during SBTs.[122] Ultrasound-guided assessment thus serves as an early warning tool for clinicians.

Moreover, diaphragmatic ultrasound may help differentiate central (neurologic or sedative-induced) from peripheral (muscle fatigue or phrenic nerve injury) causes of respiratory insufficiency, guiding personalized strategies such as physiotherapy or targeted weaning modalities.[121,123]

Electrical Impedance Tomography: Visualizing Lung Aeration and Ventilation

EIT is a noninvasive imaging modality that generates real-time cross-sectional images of regional lung ventilation.[113,114] A belt of electrodes is placed around the thorax, and changes in electrical impedance during the respiratory cycle are used to infer ventilation distribution and aeration patterns.

In the context of weaning, EIT provides critical information on ventilation heterogeneity: differences between dependent and nondependent lung regions, regional overdistension or collapse, effects of PEEP adjustment or position change, dynamic lung compliance and regional tidal volume estimation, and the pendelluft phenomenon (intrapulmonary air shifts during spontaneous efforts).[124]

After cardiac surgery, EIT has been employed to evaluate the impact of PEEP, optimize ventilatory settings, and monitor recruitment strategies. More recently, its use has been extended to assess weaning readiness, particularly in obese patients or those with pleural effusions, in whom regional collapse may hinder extubation success.[125,126]

EIT enables visualization of dorsal recruitment during spontaneous breathing trials or changes in ventilatory pattern post-extubation. Patients who maintain homogeneous ventilation during weaning are less likely to experience respiratory failure.[127,128]

Furthermore, EIT can detect asynchronies or paradoxical breathing patterns (eg, inverse tidal distribution) that might not be apparent during conventional monitoring.[129] This is especially relevant in post−cardiac surgery patients with altered chest wall mechanics or diaphragmatic dysfunction.

Combined Use and Predictive Role

The combined use of diaphragm ultrasound and EIT offers a comprehensive assessment of the "pump" and the "bellows" components of respiration. When used together, diaphragm ultrasound quantifies the contractile function of the primary respiratory muscle, and EIT evaluates the effectiveness of muscle effort in generating regional ventilation and alveolar inflation.

This integrated approach allows clinicians to discriminate between central fatigue, neuromechanical uncoupling, and lung derecruitment as causes of weaning failure. For instance, a patient with preserved TF but asymmetric or dorsal ventilation loss on EIT may benefit from postural changes or PEEP titration, while a patient with reduced TF and homogeneous ventilation may require neuromuscular recovery or inspiratory muscle training.

Limited evidence suggests EIT-derived indices such as the global inhomogeneity index, center of ventilation, and end-expiratory lung impedance change as surrogate markers of weaning readiness, although threshold values remain to be standardized.[130]

Advantages and Limitations

Both techniques are noninvasive and repeatable at the bedside, making them ideal for serial assessments during the weaning process. However, limitations exist: operator dependency in ultrasound imaging; artifacts in EIT due to chest tubes, wires, or electrode displacement; limited penetration depth of EIT in obese patients or those with distorted thoracic anatomy; and lack of standardized cutoff values and algorithms integrating these tools into weaning protocols.

Despite these limitations, their growing adoption in clinical research and practice suggests that these modalities may soon become cornerstones of personalized weaning strategies, particularly in complex populations such as cardiac surgery patients. Although most postoperative cardiac surgery patients will undergo spontaneous breathing trials, advanced physiological monitoring may be particularly useful in identifying patients at higher risk of trial failure or extubation-related complications, allowing for tailored timing, modality selection, and preventive strategies rather than indiscriminate delays or repeated failed attempts. Despite their physiological appeal, advanced monitoring tools such as esophageal pressure, diaphragm ultrasound, or electrical impedance tomography are not routinely employed in clinical practice. This likely reflects the absence of large randomized trials demonstrating improved weaning outcomes, the multifactorial nature of weaning failure, and the technical expertise required for correct interpretation.

Criteria for Extubation and Postextubation Strategies

The decision to extubate a mechanically ventilated patient represents a critical turning point in the management of patients recovering from cardiac surgery. Premature extubation may lead to reintubation, increased morbidity, and mortality, while unnecessary prolongation of ventilation increases the risk of ventilator-associated complications.[11,12,73] Therefore, defining robust and reliable criteria for extubation readiness is essential. The transition from positive to spontaneous pressure represents a major hemodynamic challenge, particularly in patients with limited cardiac reserve. In a prospective hemodynamic study, De Backer et al.[131] examined 80 postoperative patients undergoing successful weaning after different types of surgery, including cardiac surgery, heart transplantation, and abdominal aortic repair. The authors observed that oxygen consumption (VO_2) and cardiac index increased significantly during the weaning phase, reflecting the augmented metabolic and circulatory load associated with spontaneous breathing. However, the magnitude of the cardiac index rise was markedly lower after cardiac surgery and transplantation than after abdominal aortic surgery, indicating a limited ability to augment cardiac output in these populations. This blunted response underscores that even successful weaning can expose postoperative cardiac patients to hemodynamic stress, predisposing them to weaning-induced pulmonary edema when left ventricular compliance is impaired. Figure 3 depicts a conceptual physiological framework illustrating the predominant mechanisms contributing to weaning failure. The relative contribution of respiratory versus hemodynamic factors reflects the dynamic balance of cardiopulmonary interactions during the transition from positive-pressure ventilation to spontaneous breathing. From a clinical perspective, this distinction has pragmatic implications, as respiratory-dominant patterns may primarily benefit from ventilatory optimization (eg, load reduction, support adjustment, PEEP titration), whereas hemodynamic-dominant patterns warrant targeted cardiovascular assessment and management, including evaluation of preload, afterload, and ventricular function.[76,132] In addition to left ventricular loading conditions, right ventricular function plays a pivotal role during weaning. Hypercapnia-induced acidosis may increase pulmonary vascular resistance, while changes in venous return during mode transitions can further challenge RV performance. These phenotypes are not mutually exclusive and may evolve dynamically during the weaning process, further supporting the need for repeated reassessment rather than static classification.

Weaning-Induced Pulmonary Edema

A distinctive cause of weaning failure in cardiac surgery patients is weaning-induced pulmonary edema (WIPO), a

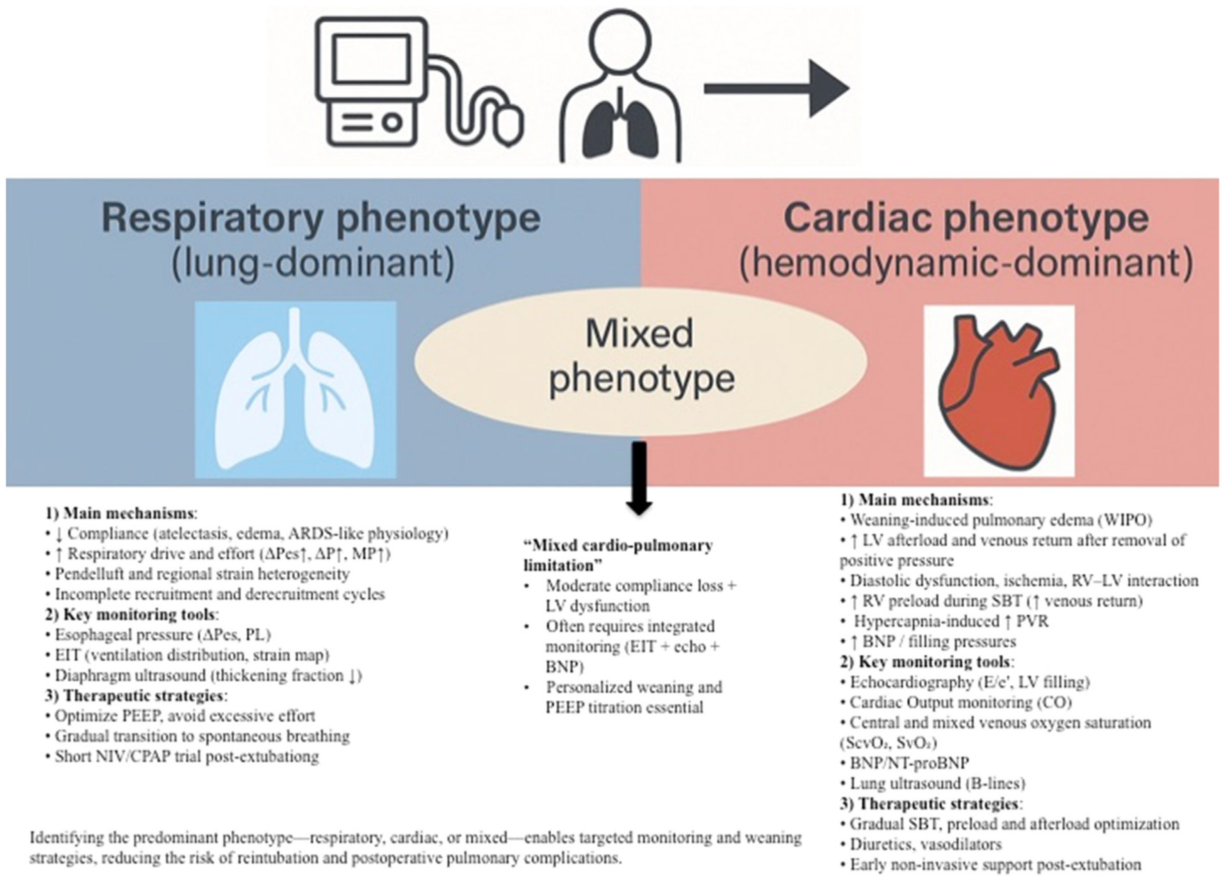

Figure 3. Respiratory, cardiac, and mixed phenotypes of weaning failure after cardiac surgery. The figure illustrates 3 major physiological phenotypes that may underlie weaning intolerance following cardiac surgery. (1) Respiratory phenotype (lung-dominant): It is characterized by decreased lung compliance, atelectasis, increased respiratory drive and effort, and heterogeneous regional ventilation. Key monitoring tools include esophageal manometry (ΔPes, PL), electrical impedance tomography (EIT), and diaphragm ultrasound. Management prioritizes optimization of PEEP, reduction of excessive inspiratory effort, gradual transition to spontaneous breathing, and selective use of noninvasive ventilation/CPAP after extubation. (2) Cardiac phenotype (hemodynamic-dominant): The cardiac-dominant phenotype includes both left and right ventricular mechanisms, with right ventricular dysfunction influenced by changes in venous return, pulmonary vascular resistance, and hypercapnia-related acidosis. Driven by weaning-induced pulmonary edema (WIPO), diastolic dysfunction, LV afterload increase, or RV/LV interaction during the shift from positive- to negative-pressure breathing. Echocardiography (E/E′, LV filling pressure), BNP/NT-proBNP, and lung ultrasound (B-lines) are central monitoring tools. Management focuses on preload/afterload optimization, controlled SBTs, diuretics/vasodilators, and early noninvasive support when needed. (3) Mixed phenotype: This represents overlapping respiratory and hemodynamic limitations, with moderate compliance loss and combined ventilatory−cardiac stress. These patients often require integrated monitoring and individualized strategies, including personalized PEEP titration and careful hemodynamic management. This phenotype-based framework supports individualized weaning strategies and may reduce the risk of extubation failure and postoperative pulmonary or cardiovascular complications. ABGs, arterial blood gases; BNP, B-type natriuretic peptide; CPAP, continuous positive airway pressure; E/E′, ratio of early transmitral flow to early diastolic mitral annular velocity; EIT, electrical impedance tomography; HFNC, high-flow nasal cannula; LV, left ventricle; MP, mechanical power; NT-proBNP, N-terminal pro-BNP; PEEP, positive end-expiratory pressure; PL, transpulmonary pressure; PVR, pulmonary vascular resistence; RV, right ventricle; SBT, spontaneous breathing trial; US, ultrasound; WIPO, weaning-induced pulmonary edema; ΔPes, esophageal pressure swing.

cardiogenic event triggered by abrupt hemodynamic changes during the transition from positive- to spontaneous-pressure breathing.[133-135] Positive pressure ventilation reduces venous return and left ventricular afterload; once discontinued, the increase in venous return and transmural pressure may precipitate acute pulmonary congestion in patients with limited diastolic reserve or left ventricular dysfunction.[136,137] The underlying mechanisms include increased venous return leading to elevated pulmonary capillary hydrostatic pressure, increased left ventricular (LV) afterload due to the loss of intrathoracic positive pressure, and decreased lung compliance caused by interstitial edema and neurohumoral activation (catecholamine surge and vasoconstriction).[75,138-140] Clinically, WIPO typically manifests during or shortly after SBT with sudden tachypnea, hypoxemia, and hypertension. Additional signs include new B-lines or pleural effusions on lung ultrasound, elevated LV filling pressures on echocardiography, and a rise in BNP or N-terminal pro-BNP within 1 to 2 hours after failed SBT. An echocardiographic (E/e′ >14) and BNP > 200 gt; 200 pg/mL are considered strong predictors of WIPO.[141-143] Preventive measures include gradual reduction of ventilatory support, preload optimization with diuretics or nitrates, and, in selected cases, noninvasive positive pressure immediately after extubation.[25]

Cardiovascular dysfunction may play a pivotal role in weaning failure, particularly in cardiac surgery patients with limited ventricular reserve. The transition from positive to negative intrathoracic pressure during spontaneous breathing can unmask latent left or right ventricular dysfunction, leading to pulmonary congestion and hemodynamic intolerance. Recognizing this "weaning-induced cardiovascular failure" phenotype supports the integration of echocardiographic, hemodynamic, and biomarker monitoring into the weaning assessment of high-risk cardiac surgical patients.[144] In line with the pathophysiological mechanisms described by Routsi et al.,[25] recent advances have refined the concept of liberation from mechanical ventilation in cardiac patients, shifting from a purely respiratory assessment toward an integrated cardiopulmonary approach.[145]

Recent evidence has emphasized that liberation from mechanical ventilation in cardiac patients requires an integrated cardiopulmonary approach. Shahu et al.[18] proposed a comprehensive framework for ventilator liberation in the cardiac intensive care unit, highlighting the unique hemodynamic challenges during the transition from positive to spontaneous breathing. The authors underscored the role of combined cardiopulmonary assessment—including echocardiography and lung and diaphragmatic ultrasound—and individualized strategies for postextubation support, such as noninvasive ventilation or high-flow nasal oxygen in high-risk cardiac patients. This state-of-the-art review provides a modern, cardiac ICU-specific reference that extends classical weaning paradigms to the postoperative cardiac population.

Comparison of Postextubation Strategies

Recent studies have explored the use of high-flow nasal cannula (HFNC) therapy in patients following cardiac surgery, focusing on its efficacy in improving postoperative respiratory outcomes. Moreover, Chaudhuri et al.[146] showed that prophylactic use of HFNC in the immediate postoperative period after cardiothoracic surgery reduced reintubation and the need for escalation of respiratory support compared to conventional oxygen therapy (COT). This effect was particularly notable in high-risk and obese patients. Patil[147] indicated that HFNC therapy reduced atelectasis, improved oxygenation, and prevented respiratory failure, thereby decreasing the need for prolonged intubation and reducing the risk of ventilator-associated pneumonia in high-risk patients. Liu et al.[148] compared HFNC therapy to COT in post−cardiac surgery patients. The findings suggested that HFNC therapy presents a promising approach to oxygen therapy, offering benefits over COT. Corley et al.[149] conducted a randomized controlled trial to investigate the effects of extubating obese patients (body mass index $\geq$30 kg/m^2) directly onto HFNC versus standard oxygen therapy. While no significant difference in atelectasis scores was observed between the groups, the HFNC group reported reduced subjective dyspnea levels, indicating potential benefits in respiratory comfort for obese patients. Zhu et al.[150] in a meta-analysis demonstrated that, compared with COT, HFNC significantly reduced postextubation respiratory failure and respiratory rates and increased PaO_2, and it was safely administered in patients after planned extubation. Furthermore, the NOTACS (Nasal High-Flow Oxygen Therapy After Cardiac Surgery) trial aims to assess the efficacy, safety, and cost-effectiveness of HFNC therapy in patients after cardiac surgery. Results from this trial are pending.[151]

Weaning Failure and Tracheostomy in Cardiac Surgery Patients

Weaning failure in the cardiac surgical population is a clinically significant event associated with increased morbidity, prolonged ICU stay, higher risk of ventilator-associated complications, and mortality. The clinical relevance of detailed physiological assessment during weaning lies not in deciding whether to attempt liberation from mechanical ventilation but in modifying how weaning is conducted and how failure is managed—including optimization of preload and afterload, adjustment of ventilatory support during SBTs, early use of noninvasive ventilation, or timely consideration of tracheostomy in selected patients.

Tracheostomy becomes a relevant consideration when extubation is deemed unsafe or when prolonged ventilatory support is anticipated. In general ICU practice, early tracheostomy (defined as within the first 7-10 days of mechanical ventilation) has been proposed to reduce sedation requirements, improve patient comfort, facilitate airway hygiene, and potentially shorten ICU stay.[152-155] However, the role of early versus late tracheostomy remains controversial in the cardiac surgery population, as many patients who initially have unsuccessful weaning may recover rapidly after correction of transient postoperative issues. Earlier concerns regarding an increased risk of mediastinitis following tracheostomy in cardiac surgery patients have not been confirmed by contemporary studies. With modern surgical techniques and percutaneous approaches, tracheostomy is no longer considered an independent risk factor for mediastinitis and may facilitate safer prolonged weaning in selected patients.[155] In contemporary cardiac surgical ICUs, percutaneous dilatational tracheostomy (PDT) has increasingly replaced surgical tracheostomy as the preferred technique in patients requiring prolonged ventilatory support. Recent observational data suggest that PDT can be safely performed after cardiac surgery, including in poststernotomy patients, with low rates of major complications, bleeding, or mediastinitis. In a national cohort analysis, early tracheostomy after cardiac surgery was associated with reduced ICU length of stay and lower in-hospital mortality without an increase in sternal wound infections, supporting its integration within structured weaning pathways rather than as a last-resort intervention.[155] More recent single-center data specifically evaluating PDT in a cardiac surgical ICU confirmed its feasibility and safety, even in patients receiving antiplatelet or anticoagulant therapy, with acceptable complication rates and relatively short tracheostomy duration.[156]

These findings support the inclusion of PDT as a technically viable and clinically relevant component of individualized weaning strategies in selected cardiac surgery patients. The reported incidence of tracheostomy after cardiac surgery ranges from 2% to 11%, depending on patient characteristics, institutional practices, and case mix (eg, redo surgeries, emergency interventions, or patients with preexisting chronic lung disease).[157,158] Factors independently associated with the need for tracheostomy in this population include advanced age, reduced left ventricular ejection fraction, prolonged cardiopulmonary bypass time, high EuroSCORE II, postoperative delirium, pneumonia, and renal dysfunction.[159]

While tracheostomy can facilitate weaning in selected patients, it also carries risks such as bleeding, infection, tracheal stenosis, and the psychological burden of prolonged mechanical ventilation. Therefore, identifying predictors of weaning failure early—such as a persistently high RSBI, poor cough peak flow, or elevated Integrative Weaning Index—may help guide decisions and avoid delayed tracheostomy, which has been associated with worse outcomes.[19,28,73]

Several studies have proposed weaning protocols or decision algorithms that integrate clinical judgment with objective measurements to guide the timing of tracheostomy. The use of daily screening and structured SBTs, combined with physiologic monitoring (eg, respiratory drive, P0.1, and esophageal pressure swings), may identify patients who would benefit from early tracheostomy or allow successful extubation in borderline cases.[160-162] Parameters such as P0.1 should not be interpreted as absolute criteria for weaning readiness but rather as indicators of excessive respiratory drive that may signal an imbalance between load and capacity. Similarly, adequate alertness for weaning does not require full neurologic recovery but sufficient consciousness to maintain airway patency, protect against aspiration, and cooperate with spontaneous breathing trials.[163-164]

In recent years, the implementation of multidisciplinary weaning teams and "ventilator liberation" bundles has been associated with a reduction in time to extubation and tracheostomy rates, suggesting that organizational factors may also influence outcomes.[165] In cardiac ICUs, collaboration between anesthesiologists, intensivists, surgeons, and physiotherapists is essential to optimize the weaning trajectory. Large contemporary datasets suggest that prolonged mechanical ventilation after cardiac surgery reflects the combined burden of patient vulnerability and surgical complexity, rather than isolated respiratory failure, reinforcing the need for individualized weaning and airway management strategies.[11]

In conclusion, tracheostomy in cardiac surgery patients should not be viewed merely as a rescue intervention for weaning failure but rather as a strategic option within a personalized weaning pathway. The decision should be based on a combination of clinical stability, objective indices, and patient-centered factors, ideally integrated into a structured weaning protocol to improve outcomes and resource utilization.[165,166]

Systemic Effects and Long-Term Outcomes

Beyond cardiopulmonary limitations, prolonged weaning in cardiac surgery patients often reflects the cumulative impact of nonrespiratory organ dysfunctions and systemic stressors, including delirium, acute kidney injury, and ICU-acquired weakness. These factors rarely represent isolated barriers to liberation but rather modulate tolerance to repeated spontaneous breathing trials and recovery trajectories after initial weaning failure.

Neurocognitive Outcomes and Delirium

The relationship between weaning failure and neurocognitive dysfunction is bidirectional. Prolonged mechanical ventilation and sedation increase the risk of delirium, which in turn impairs weaning ability by reducing cooperation and impairing drive.[167] In cardiac surgery patients, the incidence of postoperative delirium ranges from 20% to 50%, particularly in elderly or frail patients.[168]

Limited evidence suggests that delirium is independently associated with weaning failure, reintubation, longer ICU stay, and higher mortality.[167-169] Delirium prevention strategies—early mobilization, light sedation, circadian rhythm preservation, multimodal analgesia, and pharmacologic protocols—must be integrated into the weaning strategy. Use of CAM-ICU (Confusion Assessment Method for the Intensive Care Unit) and ICDSC (Intensive Care Delirium Screening Checklist) tools is crucial for daily assessment.[170] Long-term consequences include postintensive care syndrome with persistent cognitive decline, which correlates with cumulative sedative exposure and ventilatory days.[171]

ICU-Acquired Weakness and Muscle Dysfunction

ICU-acquired weakness is a major determinant of weaning difficulty and is often underdiagnosed in postoperative cardiac surgery. It results from critical illness myopathy, neuropathy, or disuse atrophy, and it manifests as diaphragm dysfunction and global muscle weakness.[172] Sarcopenia, present preoperatively in many cardiac patients, worsens during an ICU stay due to immobilization, inflammation, and catabolism. Studies have shown that diaphragm dysfunction assessed via ultrasound correlates with weaning failure, especially when combined with peripheral weakness.[173] Early mobilization protocols and electrical muscle stimulation may preserve function and improve outcomes. Sedation minimization, glycemic control, and nutritional adequacy are cornerstones of muscle preservation.[174] Muscle ultrasound and nerve conduction studies are gaining traction as diagnostic tools, although clinical assessment (eg, Medical Research Council score) remains key in the ICU setting.[172-174]

Nutrition and Weaning Readiness

Nutritional status is both a predictor and a consequence of weaning success. Malnutrition impairs respiratory muscle strength, immune function, and healing capacity, all of which are vital in the postoperative period.[175] In cardiac surgery patients, preoperative nutritional risk (eg, using NRS-2002 Nutrition Risk Screening) or MUST (Malnutrition Universal Screening Tool)) correlates with ICU length of stay and delayed extubation.[176] During weaning, underfeeding worsens muscle wasting, while overfeeding may increase CO_2 production and prolong ventilator dependency.[177] Indirect calorimetry should be used to guide energy targets where available. Protein intake >1.3-1.5 g/kg/d is recommended to support muscle anabolism during weaning.[175] In high-risk patients, early enteral nutrition should be initiated within 24 to 48 hours, avoiding parenteral nutrition unless enteral is contraindicated.[178] Micronutrient deficits, especially vitamin D, thiamine, and selenium, have been linked to poor weaning outcomes, although more evidence is needed.[175,178]

Long-Term Outcomes and Emerging Frontiers

Weaning failure is associated with poor long-term outcomes, including increased readmission, mortality, and reduced quality of life. Patients who require tracheostomy or prolonged weaning often experience post-ICU functional decline, with persistent fatigue, dyspnea, and psychological distress.[179] Cardiac surgery patients, in particular, are susceptible to physical deconditioning, sleep disorders, and neurocognitive impairment even months after ICU discharge.[180,181]

Emerging strategies include weaning bundles integrating sedation, mobilization, delirium prevention, and weaning protocols; use of machine learning algorithms to predict weaning success based on multimodal input data; development of "weaning centers" or specialized post-ICU rehabilitation pathways to improve functional recovery; telemonitoring; and structured follow-up after ICU discharge, especially in patients with tracheostomy or frailty.

A comprehensive weaning strategy in cardiac surgery patients should not end at extubation but extend into postdischarge rehabilitation, with multidisciplinary support and attention to quality of life.

Monitoring and Future Perspectives in Weaning From Mechanical Ventilation in Cardiac Surgery

Effective weaning from mechanical ventilation in cardiac surgery patients requires not only structured clinical protocols but also advanced and dynamic monitoring systems capable of capturing the complexity of cardiorespiratory interactions. As postoperative respiratory failure may result from both cardiac and pulmonary dysfunction, multimodal monitoring plays a central role in personalizing weaning strategies and improving outcomes.

Artificial Intelligence and Decision Support Systems

The integration of artificial intelligence (AI) and machine learning into ICU practice offers new frontiers for weaning management. Predictive algorithms trained on large datasets can identify patterns associated with weaning success or failure, enabling early interventions.[182] Examples include neural networks that continuously analyze ventilator waveforms to detect ineffective efforts, double triggering, or excessive workload.[183]

Prototype "weaning dashboards" are being developed to synthesize physiologic variables, ventilator settings, and trends over time into actionable insights. These tools aim to reduce variability in clinical decision-making and promote timely extubation or tracheostomy when appropriate.[184]

AI-powered risk calculators may also support clinicians in stratifying patients based on the probability of weaning success, guiding the use of stepwise pressure support reduction, high-flow oxygen postextubation, or prophylactic noninvasive ventilation in high-risk individuals.[182]

Future Research Directions

Despite technological advances, robust randomized trials specifically addressing monitoring-guided weaning in cardiac surgery patients are still lacking. Most existing studies are extrapolated from general ICU populations or small, single-center cohorts.

Future research should aim to validate composite indices that integrate respiratory mechanics, neurologic readiness, and cardiac function in a single predictive model, being able to compare protocolized versus individualized weaning strategies based on real-time monitoring tools (eg, EIT, esophageal pressure, ultrasound); to investigate the impact of early diaphragmatic rehabilitation, inspiratory muscle training, or neuromuscular stimulation on weaning outcomes in high-risk cardiac surgery patients; and, finally, to explore whether dynamic monitoring (including AI-based prediction) can reduce reintubation rates and tracheostomy needs, ultimately improving long-term functional recovery and reducing ICU costs.[185,186]

Concluding Remarks

The future of weaning in cardiac surgery will likely be driven by the fusion of physiology, technology, and personalization. A monitoring-based approach—integrating ventilator mechanics, diaphragm function, lung imaging, and AI—may help overcome the limitations of one-size-fits-all protocols and support safer, more efficient liberation from mechanical ventilation. The development and validation of such strategies should become a priority in the next generation of perioperative critical care research.

Funding

None.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Michela Rauseo: Writing − original draft, Methodology, Investigation, Data curation, Conceptualization. **Antonella Cotoia:** Supervision. **Francesco Paolo Padovano:** Investigation. **Enrico Squiccimarro:** Investigation. **Domenico Paparella:** Supervision. **Paolo Vetuschi:** Investigation. **Stefania Da Lima:** Investigation. **Giuseppina Mollica:** Investigation.

Lucia Mirabella: Supervision. **Gianluca Paternoster:** Writing − review & editing, Validation. **Gilda Cinnella:** Writing − review & editing, Validation.

Acknowledgments

We acknowledge the contributions of our colleagues.

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