

Research Article

MECHANICAL VENTILATION IN CRITICALLY ILL PEDIATRIC PATIENTS

¹Vivian Rosario Mena Miranda and ^{2,*} Vicente Manuel Martínez Cárdenas¹Hospital Universitario Pediátrico Centro Habana, Havana, Cuba²Children's Medical Center, Lake City and Live Oak, Florida, United StatesReceived 12th July 2026; Accepted 16th August 2026; Published online 11th September 2026

Abstract

Pediatric mechanical ventilation has evolved from the isolated normalization of blood gases toward individualized support that limits lung and diaphragm injury, avoids hyperoxia, preserves cardiopulmonary interactions, and facilitates timely liberation. This narrative review summarizes contemporary recommendations for noninvasive and invasive respiratory support, initial ventilator selection and settings, lung-protective ventilation in pediatric acute respiratory distress syndrome (PARDS), monitoring of respiratory mechanics and patient-ventilator interaction, and the rational use of prone positioning, high-frequency oscillatory ventilation, pulmonary vasodilators, and extracorporeal support. Current oxygenation targets, including evidence from the Oxy-PICU trial, and international guidance on ventilator liberation, spontaneous breathing trials, and extubation are reviewed. Disease-specific strategies for obstructive, restrictive, neurologic, and cardiac conditions are discussed together with emerging tools such as lung and diaphragm ultrasound, electrical impedance tomography, and automated decision-support systems. A brief special consideration addresses intubation during hemorrhagic shock, when positive-pressure ventilation may precipitate cardiovascular collapse and hemorrhage control must be integrated with airway management. Because pediatric evidence remains limited in many areas, ventilator parameters should be regarded as starting points and individualized according to pathophysiology, trends in respiratory mechanics, gas exchange, hemodynamics, and clinical response.

Keywords: Mechanical ventilation, Pediatric critical care, PARDS, Intubation, Trauma, Hemorrhagic shock, XABCDE, CAB, Ventilator-induced lung injury.

INTRODUCTION

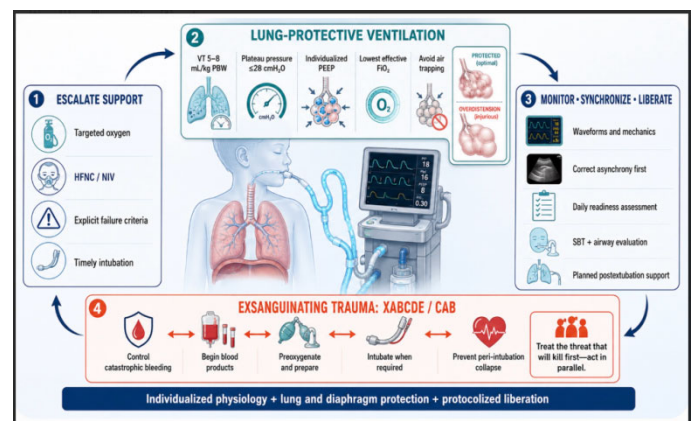
Mechanical ventilation is a supportive intervention rather than an etiologic treatment. Its benefit depends on maintaining gas exchange while limiting injury caused by overdistension, repetitive alveolar opening and closure, oxygen toxicity, patient-ventilator asynchrony, and hemodynamic disturbances. In pediatrics, the heterogeneity of ages, body sizes, pulmonary diseases, and congenital heart conditions precludes a single universal prescription. PALICC-2 and contemporary pediatric consensus statements favor individualized targets, tidal-volume measurement based on predicted or ideal body weight, pressure limitation, sufficient positive end-expiratory pressure (PEEP) to prevent derecruitment, and serial assessment of the response [1-4]. The most important update is not a new ventilatory mode but a change in purpose: ventilating the available functional lung without imposing a disproportionate load and recognizing that every increase in intrathoracic pressure affects venous return, right ventricular function, and systemic perfusion. This interaction becomes particularly important during intubation of a patient with hemorrhagic shock.

METHODS

A clinically oriented narrative review was conducted. Priority was given to international guidelines, consensus statements, clinical trials, systematic reviews, and pediatric studies published in PubMed/MEDLINE between January 2017 and August 2026. The search combined terms related to *pediatric mechanical ventilation, pediatric acute respiratory distress syndrome, PALICC-2, noninvasive respiratory support, high-flow nasal cannula, oxygen targets, patient-ventilator asynchrony, diaphragm-protective ventilation, prone positioning, high-frequency oscillatory ventilation,*

ventilator liberation, spontaneous breathing trial, extubation, and hemorrhagic shock.

Reference lists of the principal publications were also reviewed, and landmark older studies were included when they supported physiologic concepts that remain relevant.



Source: Authors' own elaboration based on the evidence synthesized in the narrative review.

Figure legend: The figure summarizes escalation of respiratory support, lung-protective ventilation, advanced monitoring, ventilator liberation, and the parallel XABCDE/CAB approach for children and adolescents with exsanguinating trauma.

Figure 1. Integrated approach to mechanical ventilation in critically ill pediatric patients.

Study selection was deliberate and was not intended to meet the requirements of a systematic review or meta-analysis. Greater weight was assigned to pediatric-specific evidence. When extrapolation from adult studies was necessary, this was explicitly acknowledged. Recommendations were organized according to clinical applicability, consistency of findings, physiologic plausibility, and the certainty recognized in the original guidelines.

*Corresponding Author: Vicente Manuel Martínez Cárdenas
Children's Medical Center, Lake City and Live Oak, Florida, United States

ESCALATION OF RESPIRATORY SUPPORT

Oxygen Therapy and Noninvasive Support

Oxygen should be prescribed to a defined target rather than regarded as a harmless intervention. In mechanically ventilated critically ill children, the Oxy-PICU trial found that a conservative peripheral oxygen saturation (SpO_2) target of 88-92% produced a modest increase in days alive and free of organ support compared with a target above 94%. However, the trial excluded patients with acute brain injury, uncorrected congenital heart disease, and pulmonary hypertension [5]. Extrapolation therefore requires caution: sustained hyperoxia should be avoided, but dangerous hypoxemia should not be accepted in patients with limited oxygen reserve or specific neurologic and cardiovascular requirements. High-flow nasal cannula and noninvasive ventilation can reduce respiratory effort and prevent intubation in selected patients. Their use requires close monitoring, an appropriate interface, and explicit failure criteria. Worsening mental status, apnea, uncorrected shock, inability to protect the airway, or a sustained increase in respiratory effort should prompt rapid reassessment. Noninvasive support must not delay an inevitable intubation [1,3].

Indications for Invasive Ventilation

The decision to intubate integrates oxygenation, ventilation, respiratory mechanics, fatigue, neurologic status, airway protection, the trajectory of illness, and the therapeutic plan. No single value can replace this integrated assessment. Firm indications include actual or impending airway obstruction, apnea or severe hypoventilation, refractory hypoxemia, exhaustion, loss of protective reflexes, the need to control ventilation because of intracranial hypertension, and urgent procedures requiring general anesthesia [6].

LUNG-PROTECTIVE INVASIVE VENTILATION

Initial Ventilator Settings

Volume-controlled or pressure-controlled ventilation is used in most children; no mode has shown universal superiority. The ventilation actually delivered matters more than the label assigned to the mode. Expiratory tidal volume, plateau pressure, peak pressure, total PEEP, driving pressure, compliance, resistance, flow-time and pressure-time waveforms, and the presence of intrinsic PEEP should be documented.

Permissive Physiologic and Mechanical Targets

Permissive hypercapnia may be accepted when it prevents injurious pressures and the pH remains tolerable, but it is not universally appropriate. Caution is required in intracranial hypertension, severe pulmonary hypertension, hemodynamic instability, selected congenital heart diseases, and metabolic disorders. In obstructive disease, limiting dynamic hyperinflation is the primary objective; an elevated arterial carbon dioxide tension (PaCO_2) may be an acceptable cost of allowing complete exhalation. In restrictive disease, reducing tidal volume and using adequate PEEP limit pulmonary stress [1-4]. Driving pressure (plateau pressure minus PEEP) and mechanical power are physiologically attractive concepts, but validated pediatric thresholds do not yet exist and these variables should not be treated as rigid targets. They are useful for identifying trends: if higher volume, pressure, respiratory rate, or flow is required to maintain the same gas exchange, the mechanical load is probably increasing.

Synchrony, Sedation, and Neuromuscular Blockade

Patient-ventilator asynchrony may increase respiratory effort, generate excessive tidal volumes, and prolong mechanical ventilation. Automatic escalation of sedation is not the appropriate first response. Pain, secretions, position, trigger sensitivity, inspiratory flow, inspiratory time, and ventilatory mode should first be corrected or optimized. Analgesia and sedation should be titrated to a validated target and reassessed daily. Neuromuscular blockade should be reserved for situations in which optimized sedation does not permit lung-protective ventilation, using the lowest effective dose for the shortest possible duration [1,7].

PARDS AND RESCUE THERAPIES

PALICC-2 broadened recognition of PARDS and classifies severity using the oxygenation index or, when an arterial blood gas is unavailable, the oxygen saturation index. During conventional ventilation, priorities include a low tidal volume, limited plateau pressure, individualized PEEP, the minimum necessary FiO_2 , and treatment of the underlying cause. After perfusion has been restored, fluid management should avoid overload; a restrictive strategy never justifies hypoperfusion [1,2]. Prone positioning may improve ventilation homogeneity and oxygenation, although pediatric evidence regarding patient-centered outcomes remains less robust than adult evidence.

Table 1. Initial guidance for invasive ventilation in pediatric patients. Values are starting points and must be individualized. Source: developed by the authors from PALICC-2 and PEMVECC [1-4]

Variable	Practical guidance	Precaution
Tidal volume	Usually 5-8 mL/kg of predicted body weight; consider 3-6 mL/kg when compliance is severely reduced.	Adjust according to pressure, gas exchange, and underlying disease; avoid actual body weight in obesity.
Plateau pressure	Keep as low as possible; PALICC-2 generally favors ≤ 28 cmH ₂ O and permits slightly higher values when chest-wall compliance is low.	Interpret together with PEEP, driving pressure, and hemodynamics.
PEEP	Titrate to prevent collapse and reduce FiO_2 while observing oxygenation, compliance, and perfusion.	Excessive PEEP may cause overdistension, increase right ventricular afterload, and reduce venous return.
Respiratory rate/inspiratory time	Provide adequate minute ventilation and allow complete exhalation.	In obstructive disease, reduce the rate and prolong expiration to prevent air trapping.
Oxygen	Use the lowest FiO_2 compatible with the individualized SpO_2 target.	Do not automatically apply an 88-92% target in brain injury, pulmonary hypertension, or cyanotic heart disease.

High-frequency oscillatory ventilation should not be used routinely; it may be considered as rescue therapy in experienced centers when lung-protective conventional ventilation fails. Inhaled nitric oxide may be used selectively for right ventricular dysfunction, pulmonary hypertension, or as a bridge. Extracorporeal membrane oxygenation (ECMO) should be considered when hypoxemia or cardiorespiratory failure persists despite optimal treatment and the underlying cause is potentially reversible [1,8].

LIBERATION FROM MECHANICAL VENTILATION

Delayed extubation increases exposure to pneumonia, weakness, delirium, and airway injury; premature extubation increases reintubation and associated morbidity and mortality. International guidelines recommend a protocolized process that includes daily assessment of readiness, a spontaneous breathing trial (SBT), separate evaluation of airway-protection capacity, and a postextubation support plan [9,10]. In standard-risk patients, a 30-60-minute SBT is usually appropriate. In high-risk patients, a 60-120-minute trial and lower assistance may be used to prevent pressure support from excessively compensating for endotracheal-tube resistance. Passing an SBT does not by itself guarantee successful extubation. Level of consciousness, cough, secretion burden, laryngeal patency, neuromuscular strength, cardiac function, and availability of rescue support should also be assessed. A cuff-leak test is not routinely recommended for every child, but it may provide useful information in patients at risk of upper-airway edema. In high-risk patients, planned continuous positive airway pressure, noninvasive ventilation, or high-flow nasal cannula immediately after extubation may reduce failure when appropriately indicated [9,10].

PATHOPHYSIOLOGY-SPECIFIC STRATEGIES

Obstructive Disease

In severe asthma, bronchiolitis with marked obstruction, and other high-resistance disorders, the objective is to prevent dynamic hyperinflation. Moderate tidal volumes, a reduced respiratory rate, adequate inspiratory flow, and a prolonged expiratory time are used. The expiratory flow-time curve should return to zero before the next breath. Peak pressure may be elevated because of resistance without indicating overdistension; plateau pressure helps distinguish these components. Permissive hypercapnia is usually preferable to a dangerous increase in minute ventilation. If sudden hypotension occurs, the patient should be briefly disconnected from the ventilator to permit exhalation, while intrinsic PEEP and pneumothorax are assessed.

Restrictive Disease and PARDS

In extensive pneumonia, pulmonary edema, and PARDS, the functional lung available for ventilation is reduced. Tidal volume should be calculated using predicted rather than actual body weight and reduced when plateau or driving pressure increases. PEEP is titrated to maintain recruitment with a reasonable FiO_2 while monitoring for overdistension and circulatory impairment. Routine or aggressive recruitment maneuvers have no established pediatric benefit and may cause hypotension or barotrauma. If considered, they should be individualized and evaluated by their mechanical and hemodynamic response [1,2].

Neurologic Disease

In acute brain injury, preventing hypoxemia and hypotension is paramount. Normocapnia is generally targeted, and abrupt changes in PaCO_2 should be avoided. Prophylactic hyperventilation may reduce cerebral blood flow and is not recommended. PEEP is not prohibited, but it should be titrated while monitoring arterial pressure, venous return, intracranial pressure, and oxygenation. Sedation should allow physiologic control without unnecessarily preventing neurologic assessment [19].

Congenital Heart Disease and Ventricular Dysfunction

Mechanical ventilation alters ventricular preload and afterload. Positive pressure may benefit a failing left ventricle by reducing its afterload, but it may impair a preload-dependent right ventricle or one exposed to pulmonary hypertension. In single-ventricle physiology, shunt-dependent circulation, or pulmonary blood-flow dependence, conventional SpO_2 , PaCO_2 , and PEEP targets may be inappropriate. Ventilator settings should be coordinated with the cardiac critical care team and directed toward an appropriate balance between pulmonary and systemic blood flow.

ADVANCED MONITORING AND PATIENT-VENTILATOR INTERACTION

Contemporary monitoring combines blood-gas analysis and pulse oximetry with waveforms, loops, and serial measurements of respiratory mechanics. An increase in peak pressure with stable plateau pressure suggests increased resistance; simultaneous elevation of both suggests reduced compliance. Flow-volume loops may reveal obstruction, leakage, and air trapping, whereas pressure-volume loops may demonstrate overdistension. Capnography confirms airway placement, detects disconnection, and permits monitoring of ventilatory trends, although the PaCO_2 -end-tidal CO_2 gradient changes with dead space and perfusion. Asynchrony should be classified before it is treated: ineffective triggering, autotriggering, double triggering, insufficient flow, premature cycling, or delayed cycling. Correcting the cause requires assessment of patient effort, secretions, leakage, trigger sensitivity, inspiratory flow, inspiratory time, and support level. Neurally adjusted ventilatory assist and proportional pressure-support ventilation may improve synchrony in selected patients, but availability remains limited and evidence regarding major outcomes is insufficient. Lung ultrasound can monitor aeration, consolidation, pleural effusion, and the response to recruitment without ionizing radiation. Diaphragm ultrasound estimates excursion and thickening fraction and may complement the assessment of readiness for liberation, although pediatric cutoff values are not uniform. Electrical impedance tomography provides continuous regional information on ventilation distribution and may facilitate PEEP titration; it is not yet a universal standard of care.

PREVENTION OF VENTILATION-ASSOCIATED COMPLICATIONS

Prevention of ventilation-associated injury includes head-of-bed elevation when not contraindicated, oral care, safe suctioning, adequate humidification, early mobilization, enteral nutrition, judicious sedation, daily assessment of devices, and

ventilator-liberation protocols. Circuits should not be changed routinely unless visibly soiled or malfunctioning. Endotracheal-tube cuff pressure should be monitored to reduce microaspiration and tracheal injury. Ventilator-induced diaphragm dysfunction may result from both excessive unloading and excessive effort. The objective is safe spontaneous breathing whenever possible: sufficient support to prevent fatigue, but not so much that respiratory effort is persistently suppressed. This approach integrates lung and muscle protection and favors light sedation, mobilization, and early weaning.

INNOVATIONS AND FUTURE DIRECTIONS

Modern ventilators increasingly incorporate leak compensation, neural synchronization, automated estimation of respiratory mechanics, and closed-loop control. Decision-support and machine-learning systems may identify patterns and predict extubation failure, but they require external validation, transparency, and clinical oversight. Poor-quality data should never be converted into therapeutic automation.

The future direction is precision ventilation that integrates pulmonary phenotype, respiratory mechanics, inspiratory effort, perfusion, functional imaging, and temporal evolution. Nevertheless, the most effective innovation remains consistent application of established principles: measuring what is actually delivered, avoiding extremes of oxygen and pressure, identifying asynchrony, reassessing the need for support every day, and protocolizing liberation.

SPECIAL CONSIDERATION: INTUBATION IN EXSANGUINATING TRAUMA

Clinical Scenario

An adolescent with multiple gunshot wounds to the abdomen and pelvis arrives pale and diaphoretic, with a blood pressure of 70/40 mmHg and a Glasgow Coma Scale (GCS) score of 8. An automatic interpretation of “GCS $\leq$ 8: intubate” may place laryngoscopy ahead of hemorrhage control, blood-product administration, and transfer to the operating room. The problem is not that the airway is unimportant; rather, neurologic deterioration may reflect cerebral hypoperfusion, and intubating a nearly exsanguinated patient is a high-risk hemodynamic intervention.

Meaning of XABCDE and CAB

XABCDE places “X,” catastrophic external hemorrhage, first: tourniquet application, wound packing, direct pressure, or a pelvic binder, depending on the bleeding site, before proceeding with the remainder of the assessment. CAB proposes that in a subgroup of exsanguinating patients whose airway can be temporarily maintained, hemorrhage control and restoration of circulation should precede intubation. This is not a slow, linear sequence. In a trained team, hemorrhage control, vascular or intraosseous access, transfusion, preoxygenation, thoracic assessment, and airway preparation occur in parallel [11-14].

Pathophysiology of Peri-intubation Collapse

A patient in hemorrhagic shock temporarily maintains perfusion through sympathetic activation, tachycardia, vasoconstriction, and negative intrathoracic pressure during

inspiration. Induction may reduce sympathetic tone and myocardial contractility. Apnea eliminates the respiratory pump, and positive-pressure ventilation increases intrathoracic pressure, reduces the gradient for venous return, and may increase right ventricular afterload. If preload is already critically low, any additional reduction may precipitate severe hypotension or pulseless electrical activity [15-17]. The claim that neuromuscular blocking agents eliminate a “muscular tamponade” around abdominal, cervical, or junctional hemorrhage has appeared in recent publications as a plausible explanation, but clinical evidence is insufficient. It should not be presented as an established mechanism. The best-supported mechanisms are loss of sympathetic tone, vasodilation, and the transition to positive-pressure ventilation.

What the Evidence Actually Shows

A recent review and prospective cohort comparison of CAB and ABC strategies in exsanguinating trauma reported a strong association between intubation before circulatory resuscitation and mortality [11,12]. This evidence is observational, however, and susceptible to confounding by indication: patients intubated first may be more severely injured, aspirating, affected by traumatic brain injury, or in need of immediate surgery. The estimate of an almost tenfold increase in mortality should not be generalized as a causal effect, particularly in pediatrics, where comparative trials are lacking. Trauma guidelines continue to recommend urgent intubation for obstruction, hypoventilation, severe hypoxemia, profound neurologic impairment, and severe hemorrhagic shock, particularly when thoracotomy or laparotomy is required [6]. The reasonable synthesis is not “never intubate first,” but rather “avoid reflexive, unprepared intubation when a few minutes of hemorrhage control and transfusion can improve physiologic reserve without jeopardizing the airway.”

Low GCS in Shock

The GCS score describes neurologic function; it does not identify the cause of impairment or independently establish airway protection. Hypoxemia, hypercapnia, hypoglycemia, medications, and shock can all lower the score. In penetrating torso trauma without signs of cranial injury, a low score should prompt immediate restoration of perfusion and reassessment, but it cannot be assumed that the airway is safe. Vomiting, blood in the oropharynx, inadequate ventilation, loss of protective reflexes, facial injury, or progressive deterioration may make intubation essential despite shock.

Practical Strategy for an Exsanguinating Child or Adolescent

Pediatric hemostatic resuscitation should follow local weight-based protocols, with active warming, prevention and correction of hypocalcemia, coagulation monitoring, and early anatomic hemorrhage control. In trauma without severe brain injury, permissive hypotension is supported by limited pediatric evidence and should not be interpreted as acceptance of clinical hypoperfusion. The goal is adequate perfusion without disrupting clot formation through excessive crystalloid administration [13,14,18]. If intubation cannot be deferred, it should be approached as a physiologically difficult airway: two teams act simultaneously; hemorrhage control is uninterrupted; transfusion has begun; the induction agent and dose are selected according to circulatory status; apnea is minimized;

Table 2. Practical integration of XABCDE/CAB with airway management. Source: developed by the authors [6,11-18]

Priority	Simultaneous actions	Error to avoid
X: hemorrhage	Apply pressure, a tourniquet, wound packing, or a pelvic binder as appropriate; activate surgery/interventional radiology.	Waiting for intubation before beginning hemorrhage control.
C: perfusion	Activate the pediatric massive-transfusion protocol; administer low-titer whole blood or balanced components according to availability; manage calcium, temperature, and coagulation.	Giving large volumes of crystalloids or delaying blood while completing a nonessential airway procedure.
A/B: oxygenation	Position and suction the airway; use an airway adjunct and oxygen; provide gentle bag-mask ventilation if necessary; prepare an expert operator and rescue plan.	Hyperventilating, applying high pressures, or allowing an isolated GCS score to dictate timing.
Intubation indicated	Preoxygenate; ensure blood products are infusing; individualize the induction dose; have a vasopressor ready as a bridge; optimize the first attempt; use capnography.	Giving a “standard dose” during profound shock or making repeated attempts without correcting physiology.
After tube placement	Confirm end-tidal CO ₂ ; use a low tidal volume, initially cautious PEEP, and a rate that avoids excessive mean airway pressure; reassess pulse, blood pressure, chest, and bleeding.	Applying settings used for a stable pulmonary patient or attributing all hypotension to the sedative.

bag-mask pressure is limited; and hypotension is anticipated and treated with blood products and, when needed, a vasopressor as a bridge, never as a substitute for hemorrhage control.

POST-INTUBATION VENTILATION IN TRAUMA

After tube placement is confirmed with capnography, the priority is to prevent the ventilator from worsening shock. Initial settings should include a lung-protective tidal volume, a high FiO₂ during initial stabilization followed by prompt titration, sufficient but not excessive PEEP, and a rate that avoids hyperinflation and high mean airway pressure. A sudden decrease in blood pressure should prompt evaluation for ongoing hemorrhage, tension pneumothorax, intrinsic PEEP, excessive ventilatory pressure, tube displacement, cardiac tamponade, and drug effects. Point-of-care ultrasound may accelerate differentiation without delaying definitive hemorrhage control. When traumatic brain injury is also present, hypoxemia and hypotension must be actively prevented. Normocapnia is the usual target, and hyperventilation should be reserved as a temporary measure for signs of impending herniation. In pulmonary contusion or secondary PARDS, the protective principles described above apply, while PEEP is balanced against right ventricular function and systemic perfusion [1,19].

CONTROVERSIES AND RESEARCH PRIORITIES

Important gaps remain, including pediatric thresholds for driving pressure and mechanical power; optimal selection between high-flow nasal cannula and noninvasive ventilation; the clinical value of biomarkers and electrical impedance monitoring; prone-positioning and oscillatory-ventilation strategies in PARDS; and phenotypes most likely to benefit from conservative oxygen targets. In trauma, multicenter pediatric studies are needed to distinguish the indication for intubation, time to transfusion and hemorrhage control, induction drugs, ventilatory pressures, and associated brain injury. Until such evidence is available, rhetorical certainty should remain lower than clinical urgency.

CONCLUSION

Contemporary pediatric ventilation is grounded in physiology, lung protection, individualized oxygen targets, monitoring of mechanical load, and daily protocolized liberation. In exsanguinating trauma, the same physiology requires recognition that intubation may precipitate cardiovascular

collapse when performed before hemorrhage control and partial restoration of circulation. XABCDE/CAB does not represent the end of airway management or a prohibition against intubating a hypotensive patient. It represents the end of applying a rigid sequence without context. The correct question is not “ABC or CAB?” but “Which threat will kill this patient first, and which actions can the team perform in parallel?” Catastrophic bleeding requires immediate control and blood products. An unmaintainable airway requires immediate intubation, but the procedure must be resuscitated, prepared, and hemodynamically informed. That integration, rather than any acronym, defines safe practice.

Key Messages

- Pediatric lung-protective ventilation is guided by the tidal volume actually delivered, plateau pressure, PEEP, respiratory mechanics, hemodynamics, and clinical trends.
- Avoiding hyperoxia does not mean accepting hypoxemia; targets must be individualized.
- Preparation for extubation begins on the first day of mechanical ventilation.
- During exsanguination, XABCDE prioritizes immediate hemorrhage control and hemostatic resuscitation.
- A low GCS score may reflect hypoperfusion, but it does not independently demonstrate that the airway is protected.
- CAB is a selective, simultaneous strategy, not a rule for delaying every intubation.
- Peri-intubation risk arises mainly from loss of sympathetic tone, decreased venous return, and positive-pressure ventilation; the “muscular tamponade” theory remains insufficiently demonstrated.
- The association with an almost tenfold increase in mortality comes from observational adult evidence and should not be presented as causal or pediatric evidence.

Editorial Declarations

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