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Review

Ventilating Pediatric Critical Asthma: Why Validated, Disease-Specific Targets Are Still Missing—A Narrative Review and Proposed Research Framework

Serafeia Kalamitsou [1], Vasiliki Avramidou [2], Elpis Chochliourou [3] and Asterios Kampouras [1,*]

[1] 4th Pediatric Department, Papageorgiou Hospital, Aristotle University of Thessaloniki, 56403 Thessaloniki, Greece; serfkal@yahoo.gr
[2] School of Medicine, Aristotle University of Thessaloniki, 54124 Thessaloniki, Greece; vavramid@yahoo.gr
[3] 3rd Pediatric Department, Hippokration Hospital, Aristotle University of Thessaloniki, 54642 Thessaloniki, Greece; elpismd@hotmail.com
* Correspondence: akampoug@auth.gr

Highlights

What are the main findings?

- No asthma-specific guideline covers invasive ventilation of pediatric critical asthma; validated, outcome-linked ventilator targets and a severity metric for the intubated child are missing.
- Five explanations are weighed, including heterogeneity of obstruction and a haemodynamic hazard; adult critical care shares the same gap.

What are the implications of the main findings?

- A research framework is proposed in which physiological validation and consensus development proceed in parallel.
- A severity metric that applies to the intubated child is the most tractable first step towards disease-specific guidance.

Abstract

Pediatric acute respiratory distress syndrome (PARDS) has an international, evidence-graded consensus guideline (PALICC-2). PALICC-2 excludes respiratory failure caused solely by airway obstruction. The Paediatric Mechanical Ventilation Consensus Conference (PEMVECC) provides general recommendations for obstructive airway disease, and the 2025 AARC/PALISI guideline on paediatric critical asthma does not address invasive ventilation. Validated, outcome-linked ventilator targets for critical asthma and a severity metric for the intubated child are still missing. This narrative review examines five possible explanations: heterogeneity of obstruction and overlap with parenchymal disease; loss of severity assessment after intubation; the small number of ventilated children; a haemodynamic hazard that develops over minutes to hours; and the brevity and instability of the illness. Adult critical care has the same gap. We pay most attention to the missing severity metric, a daily obstacle for clinicians, although solving it would not alone produce a guideline. We propose a research framework in which physiological validation and consensus development proceed in parallel. PARDS is used only as a comparator; the two conditions are distinct diseases.

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1. Introduction

PALICC-2 states plainly that possible PARDS and at-risk-for-PARDS diagnoses should not be applied to children whose respiratory failure arises solely from airway obstruction, naming critical asthma as an explicit example [1]. This exclusion is physiologically defensible, and it is not the central gap this review addresses: the Paediatric Mechanical Ventilation Consensus Conference (PEMVECC) already provides recommendations specifically for paediatric obstructive airway disease, including plateau pressure, tidal volume, expiratory timing, PEEP, and monitoring [2]. We do not claim that no guidance exists. The actual knowledge gap is narrower, and we argue, more consequential: the lack of validated, disease-specific ventilator targets supported by paediatric clinical outcomes and of a severity metric that applies once the child is intubated.

Acute hypoxaemic respiratory failure from parenchymal lung injury has a detailed guideline covering tidal volume, PEEP, plateau pressure, and escalation to extracorporeal support [1]. Status asthmaticus requiring invasive ventilation, in which almost all asthma-attributable PICU mortality is concentrated, has no equivalent graded guideline: the 2025 AARC/PALISI paediatric critical asthma guideline makes 11 recommendations on drugs, noninvasive support, and care pathways, but none on invasive ventilation [3]. Clinicians rely on retrospective case series, several dating from the period of 1984–1992, expert statements, and physiological reasoning [4–7].

This review has three aims: (1) to document the asymmetry precisely, including its presence in adult practice; (2) to set out and weigh the candidate explanations for it; and (3) to propose—explicitly as a starting point for discussion and future study, not as a validated protocol—what would have to be established before a consensus process could succeed. PARDS is used throughout as a comparator that shows where disease-specific guidance is missing, not as a template for obstructive disease. We focus on critical asthma; other paediatric conditions with airflow obstruction, such as bronchiolitis, bronchopulmonary dysplasia, and upper airway obstruction, differ in mechanism, age group, and evidence base and are outside the scope of this review.

2. Background

2.1. Epidemiology of Paediatric Status Asthmaticus Requiring Mechanical Ventilation

Analysis of the Virtual Pediatric Systems database from 2009 to 2019 found that approximately 5% of children admitted to the PICU with status asthmaticus required intubation and invasive mechanical ventilation, a proportion that decreased over the study period, coinciding with wider adoption of noninvasive respiratory support [4,8]. Roughly 98% of asthma-attributable PICU deaths occur in mechanically ventilated patients [4]. Noninvasive support may now avert intubation in some children, but invasive ventilation remains necessary for those who do not improve, and outcomes are the worst in that group. The population in which a guideline would matter most is therefore small, reportedly shrinking, and the least available for study.

2.2. What PARDS Has

Published in 2023, PALICC-2 updated the paediatric ARDS definition and centred its management recommendations on a lung-protective bundle adapted from adult ARDS Network principles: tidal volumes of 6–8 mL/kg predicted body weight (reduced to 4–6

mL/kg if pressures are high), plateau pressure limits of 28–32 cmH2O, driving pressure limited to 15 cmH2O, and permissive hypercapnia to a pH floor of approximately 7.20 [1]. These recommendations rest on a severity metric. In PALICC-2, PaO2/FiO2 and SpO2/FiO2 are used for noninvasive assessment, whereas the oxygenation index (OI) and oxygen saturation index (OSI) are the preferred severity indices in invasively ventilated children; they allow PARDS to be graded, stratified, and enrolled. Without such a variable, the numeric targets above could not have been derived, tested, or graded.

2.3. What Obstructive Failure Has

By contrast, asthma-specific guidance for ventilated status asthmaticus is largely qualitative: reduce the rate, prolong exhalation, and tolerate hypercapnia [5,6,9]. Where a numeric figure does appear—a suggested PEEP of 5–8 cmH2O, for example—it comes without any grading of obstruction severity to attach it to. Recent narrative reviews describe example starting settings, but these draw largely on textbooks and observational reports and are not validated against outcomes [10]. There is no severity index applicable to the ventilated child, no numeric target validated against outcome, and no asthma-specific consensus document. Table 1 sets the two conditions side by side along the dimensions that matter for guideline development rather than for pathophysiology.

Table 1. The evidential asymmetry between PARDS and paediatric obstructive respiratory failure.

Dimension	PARDS (Diffuse Parenchymal Injury)	Paediatric Obstructive Respiratory Failure (Critical Asthma)
International consensus guideline	Yes—PALICC (2015), updated PALICC-2 (2023), with graded recommendations	No guideline on invasive ventilation in critical asthma. PEMVECC provides general recommendations for obstructive airway disease, PALICC-2 excludes isolated airway obstruction, and the 2025 AARC/PALISI critical asthma guideline does not address invasive ventilation.
Accepted severity metric	PaO2/FiO2 for noninvasive assessment; oxygenation index (OI) and oxygen saturation index (OSI) are the preferred graded metrics once a child is invasively ventilated—used for classification, enrolment, and stratification	Validated clinical scores (PRAM, PASS, PIS) and peak flow exist for the non-intubated child, but none survives intubation, sedation, and paralysis. In the invasively ventilated child, there is no accepted metric at all.
Numeric ventilation targets	Tidal volume of 4–8 mL/kg PBW; plateau pressure of 28–32 cmH2O; driving pressure of ≤ 15 cmH2O; pH floor ≈ 7.20	Mostly qualitative ("low rate, prolonged expiration, tolerate hypercapnia"). No numeric target for tidal volume, rate, PEEP, plateau or peak pressure, peak flow, or inspiratory/expiratory time has been validated against outcomes.
Randomised trial base	Limited in children but substantial by extrapolation from adult ARDS Network trials	Effectively absent in children and in adults. Practice rests on case series, several from the period of 1984–1992, and expert opinion.
Adult counterpart guideline	Mature and widely implemented (ARDSNet lineage; Berlin definition)	Also absent. Adult reviews state directly that guidance for ventilating acute severe asthma is not supported by strong clinical evidence.
Dominant hazard	Ventilator-induced lung injury from alveolar overdistension; oxygenation failure	Dynamic hyperinflation causing hypotension, air leak syndromes and cardiac arrest; ventilation failure with oxygenation often preserved until late—until exhaustion, severe ventilation–perfusion mismatch or critical dynamic hyperinflation develops.
Time course of the disease	Underlying cause (sepsis, pneumonia, burns) resolves over days to weeks; ventilation is prolonged	Bronchospasm is often reversible within hours of effective bronchodilation; mechanical ventilation may be brief, and respiratory mechanics change continuously throughout.

Trial denominator	PARDS occurs in a substantial proportion of PICU admissions with respiratory failure	Approximately 5% of PICU status asthmaticus admissions are intubated, a proportion reported to be falling.

2.4. Review Approach

This is a narrative review. We searched PubMed/MEDLINE for English-language publications up to September 2025 and made a further web-based check for newer guidelines and reviews in October 2026. The search used combinations of the terms status asthmaticus, critical asthma, mechanical ventilation, paediatric, PEMVECC, PALICC, dynamic hyperinflation, auto-PEEP, and the candidate metrics in Section 4.2 (VEI, expiratory time constant, VTi − VTe). We specifically looked for existing consensus or guideline documents on paediatric obstructive ventilation, including society statements and national guidance; PEMVECC, PALICC-2, and the AARC/PALISI critical asthma guideline were identified this way. Reference lists of relevant reviews and primary studies were handsearched. Adult, bench, and animal studies are used only where paediatric data are lacking or a mechanism requires them, and the type of evidence is stated with each finding. We did not restrict the search by study design.

3. Documenting the Asymmetry

The asymmetry is not specific to paediatrics. Adult critical care, with a larger denominator and a longer research history, has the same gap: reviews of ventilation in acute severe asthma state that guidance in this area is not supported by strong clinical evidence [9], and a recent adult cohort of life-threatening asthma called for multicentre trials and intensive-care guidelines that do not yet exist [11]. If under-study of children were the whole explanation [12], the adult literature would have filled the gap. It has not; so, the cause more probably lies in the disease than in the age group.

There is reasonable evidence for the decision to intubate a child with severe asthma, and the complications of invasive ventilation are well described. What is missing lies after that decision: validated ventilator parameters and a validated way to choose the mode and settings.

4. Why Are Validated, Disease-Specific Targets Missing? Five Candidate Explanations

4.1. Heterogeneity of Obstruction and Overlap with Parenchymal Disease

"Status asthmaticus" names a clinical presentation, not a mechanically uniform state. The degree of obstruction, the relative contributions of bronchospasm, mucosal oedema and mucus plugging, and the presence or absence of intercurrent infection all vary widely between patients and within the same patient over hours.

A related hypothesis, which requires verification, is that asthma alone is unlikely to produce PARDS, so that a second process should be sought when a ventilated asthmatic child meets PARDS criteria. We found no literature addressing this question.

4.2. Intubation Removes the Instruments That Measure Severity

This is, we suggest, the central obstacle. Severity assessment in children who are breathing spontaneously is well served by validated clinical scores. The Preschool/Pediatric Respiratory Assessment Measure (PRAM), based on retractions, wheezing, air entry, and oxygen saturation, stratifies severity from preschool children to teenagers [13,14]; the Pediatric Asthma Severity Score (PASS), based on wheezing, prolonged expiration, and work of breathing, has good to excellent interobserver reliability [15]; and peak expiratory flow adds an objective measure in children old enough to perform it [15,16].

Every one of these instruments requires a breathing, cooperating, unsupported patient. Wheeze, retractions, and work of breathing are observations of spontaneous respiratory effort; peak flow requires volitional effort and is in any case unavailable in the preschool children who make up much of the acute asthma population. No validated severity score can be used in the intubated child.

The endotracheal tube also adds resistance in series with the obstructed airway; so, pressure- and time-based indices of obstruction in children need normalisation for tube size; this is discussed in Section 4.2.1.

4.2.1. How Much of the Signal Is the Tube?

Bench work in a paediatric model spanning tube sizes from 3.0 to 8.0 mm found that the difference between peak and plateau pressure during pressure-controlled ventilation was independently associated with respiratory system compliance and resistance, with the set pressure above PEEP, and—most strongly—with inspiratory time, and that peak inspiratory pressure overestimated plateau pressure across all tube sizes [17]. Expiratory resistance imposed by small tubes is a recognised problem in neonatal and infant ventilation, where it impairs gas exchange and generates intrinsic PEEP independently of disease [18].

Clinical physiological studies concern overall respiratory effort, not these pressure measurements. In 409 children, respiratory effort during CPAP without pressure support was lower than after extubation, including after excluding children with post-extubation upper airway obstruction, which argues against a large tube-attributable effort penalty in that setting [19]. Adult studies are less consistent [20,21]. An observational study in adults reported an association between smaller endotracheal tube size and mortality in ventilated status asthmaticus, but confounding by indication (sicker or smaller patients may both require, and tolerate poorly, smaller tubes) limits causal interpretation [22].

For the purposes of this review, the practical implication is narrow: dynamic, flow-dependent measurements (peak pressure, the peak-to-plateau gradient, expiratory time constants) need age-, body-size-, and tube-size-specific normalisation before they can be compared between children or used as a severity index, while adequately equilibrated static measurements (plateau pressure, VEI) are far less affected by tube size. Validation studies of any candidate severity metric should therefore report tube size explicitly and, where the metric is dynamic, correct for it.

4.2.2. Candidate Replacements for a Severity Metric

In a series of studies, Tuxen and colleagues measured end-inspiratory lung volume above functional residual capacity (VEI) from the total gas volume exhaled during 40–60 s of apnoea in paralysed adults with acute severe asthma, and used it to titrate minute ventilation to a safe level irrespective of PaCO2—reducing the rate when VEI exceeded 20 mL/kg and increasing it when VEI fell below [23]. VEI proved the most reliable predictor of ventilator-related complications, with VEI > 20 mL/kg predicting hypotension and air leak [24], and it correlated closely with directly measured functional residual capacity ($r = 0.85$), which fell in parallel with VEI as obstruction resolved [23]. In these adult studies, VEI predicted harm better than the pressure measurements available. It is not in routine use, probably because it requires apnoea, paralysis, and dedicated spirometry, but the reasons for its limited adoption have not been studied. Its thresholds have not been validated in children, and we therefore regard VEI as a candidate reference comparator against which bedside measures can be tested, not as a validated target.

Table 2 lists the candidates, what each measures, its evidence status, and its feasibility in children. Driving pressure is not included: it describes alveolar distension, not airway obstruction, and in severe obstruction, it may read falsely high because of overinflation

rather than injury. POCUS and capnography detect complications of hyperinflation and do not grade obstruction severity.

Table 2. Candidate metrics of obstruction severity and hyperinflation in the ventilated child.

Candidate Metric	What It Measures	Evidence Status	Feasibility in Children
Expiratory flow–time waveform	Whether expiratory flow returns to zero before the next breath	Universally taught but never formalised as a graded variable. Shows ongoing trapping; may look normal once a new steady state is reached.	The highest of any candidate—displayed by default on every ventilator
Ppeak—Pplateau	Pressure consumed by airway resistance; a gradient of roughly 5–8 cmH2O indicates a resistive load, but with no validated grading scale of obstruction severity	Not a candidate outcome variable, since resistive pressure is dissipated in the airways and does not reach the alveolus. Its value is indirect: as a proxy for the magnitude of obstruction	High, but the gradient varies with tube size, inspiratory time, and set pressure; so, it needs weighting for these.
VTi − VTe	Positive value: gas that entered has not left (leak-free circuit)	Shows ongoing retention only. May return to zero once a new steady state is reached; so, it cannot identify established hyperinflation. Not evaluated as a severity or safety index.	High if leak-free; limited with uncuffed tubes or a chest drain
Auto-PEEP	End-expiratory alveolar pressure above set PEEP	Widely measured; the measured value is an unreliable index of trapping for the reasons set out in §5.2, though it remains essential for setting external PEEP in the spontaneously breathing patient	High—expiratory hold, but requires a passive patient (adequately sedated and, where indicated, neuromuscular-blocked)
VEI (end-inspiratory volume above FRC)	Trapped gas volume, from total exhaled volume during 40–60 s of apnoea	Best-validated predictor of air leak and hypotension in adult asthma (small historical cohorts, 1987–1992). A reference comparator for children, not yet validated there.	Low as originally described—apnoea plus dedicated spirometry; simplified bedside surrogates are under study
Expiratory time constant (RCEXP)	Speed of lung emptying; by definition the product of resistance and compliance	Measurable by several concordant methods in adults, where a threshold near 0.8 s marks prolonged emptying. No paediatric data and no outcome linkage; the adult threshold should not be assumed to transfer	High—computed as R × C, displayed directly by many modern ventilators, or derived from the expiratory flow–volume relationship, with the available methods showing good agreement
Regional τ by EIT	Pixel-level heterogeneity of emptying	Feasible and valid against flow-derived τ in adults with severe respiratory failure; untested in asthma	Research-grade only; EIT is present in very few PICUs.
POCUS (cardiac and lung)	Right ventricular dilatation, septal D-shaping, non-compressible IVC, low stroke volume; lung ultrasound identifies pneumothorax	Established in obstructive shock; not studied as monitoring in ventilated asthma. Detects a complication, not severity.	High where operator skill exists
End-tidal CO2 / capnography	Shark-fin waveform suggests bronchospasm; a falling end-tidal CO2 may reflect low cardiac output, not improvement	Waveform sign well described. Lower end-tidal CO2 predicted admission in non-intubated acute asthma, with weak correlation to other markers [25]; not studied as monitoring in ventilated asthma. A monitoring tool, not a severity index.	The highest—continuous, noninvasive, standard on every ventilated patient

4.3. The Small Ventilated Denominator

With roughly 5% of PICU status asthmaticus admissions intubated and that fraction reported to have fallen over the past decade (a trend that coincides with wider noninvasive support but is not shown to be caused by it, since case mix, intubation thresholds, and coding may also have changed) [4,8], the population available for a randomised trial of ventilator strategy is small. This is a genuine constraint and partly explains the reliance on case series. It is not sufficient on its own: extracorporeal support—comparably rare in children, and in refractory asthma delivered specifically as extracorporeal CO2 removal (Section 5.8)—has nonetheless acquired structured registry infrastructure and organisational guidance through the Extracorporeal Life Support Organization, which shows that rarity raises the cost of evidence without by itself preventing organised practice [26].

4.4. The Dominant Hazard Is Haemodynamic, and It Accumulates over Minutes to Hours

Ventilator-induced lung injury accumulates over days, whereas the hazard in ventilated obstruction accumulates over minutes to hours. Each breath delivered before the previous one has been fully exhaled adds to the volume of trapped gas. Hyperinflation does not necessarily increase without limit: it may reach a new steady state in which the volume exhaled per breath again equals the volume inhaled, but at a higher end-expiratory lung volume and with persisting haemodynamic effects. The transition itself is dangerous: moving a child from the negative intrathoracic pressures of spontaneous breathing to the high positive pressures of the ventilator at intubation raises intrathoracic pressure abruptly, and in the severely obstructed asthmatic, this alone can precipitate haemodynamic collapse. Rising intrathoracic pressure reduces venous return and cardiac preload and increases pulmonary vascular resistance with consequent right ventricular impairment; depending on the severity of obstruction and the appropriateness of the ventilator settings, the interval from the first stacked breath to circulatory collapse may be minutes or hours rather than days [27]. The event is mechanical and reversible by disconnecting the circuit and allowing full exhalation—a manoeuvre with no analogue in parenchymal disease.

Avoiding dynamic hyperinflation and the resulting haemodynamic compromise is a key objective of ventilation in severe asthma, and these complications are plausible safety endpoints for future trials. Methods built to detect slow parenchymal injury are poorly suited to a hazard measured in minutes, which may partly explain the lack of a framework.

Trapping has two further consequences. The first is air leak (pneumothorax, pneumomediastinum, and subcutaneous emphysema), the classical complication of ventilated asthma and the endpoint against which VEI was validated [24]. Air leak can occur before positive-pressure ventilation; ventilation may increase the risk when it worsens dynamic hyperinflation, and the risk depends on ventilator settings and the patient's own effort. Strong spontaneous inspiratory effort can itself generate excessive lung stress in experimental lung injury [28], but its effect on air-leak incidence in paediatric asthma is uncertain. The second consequence appears once the patient breathes spontaneously. Intrinsic PEEP acts as an inspiratory threshold load: the child must generate enough negative pressure to overcome it before the ventilator senses any effort, which causes ineffective triggering, dyspnoea and respiratory muscle fatigue [29]. Ineffective efforts, seen on the waveform as inspiratory attempts that produce no breath, are therefore a direct clinical sign of hyperinflation.

4.5. The Condition Is Brief, Unstable, and Rapidly Reversible

A final explanation concerns the shape of the illness. ARDS has causes (sepsis, pneumonia, burns, trauma) that take days to weeks to resolve, which provides a stable enough

substrate for protocolised ventilation and for trials with meaningful ventilator-day endpoints. Status asthmaticus differs: bronchodilation can act within hours; so, ventilation may be needed for a very short period, and a paediatric cohort examining duration of ventilation in life-threatening asthma identified an acute asphyxial subgroup with a still shorter course [30].

Two consequences follow. For research, a therapy applied for hours in a condition that resolves in hours is hard to study with the usual outcome measures, which may contribute to the empty trial literature independently of rarity. For practice, respiratory mechanics shift rapidly, sometimes within minutes to hours, as bronchospasm waxes and resolves; so, settings cannot be fixed at intubation. Ventilation in status asthmaticus is better managed as an alarm-driven process, with alarms set to alert the clinician to volume retention, to expiratory flow reaching zero sooner as obstruction resolves, or to resolving bronchospasm that makes previously appropriate pressures excessive. A future framework should specify monitoring and alarm strategy as explicitly as initial settings.

5. What Is Nonetheless Known: Physiologic Constraints on Any Future Framework

Considerable physiology is established, and any future framework is constrained by it. Table 2 lists candidate measurements; Table 3 states what is already known about the mechanics that any such measurement, and any framework, must respect. The subsections that follow expand the points of greatest uncertainty.

Table 3. Physiologic constraints on a future obstructive ventilation framework.

Variable	Behaviour in Obstructive Failure	Consequence for Any future Framework
Airway resistance	Markedly elevated; the primary lesion is the airway, not the alveolus. This is established physiology, not a research question	Resistance must be quantified before it can serve as a severity metric. Peak pressure alone cannot do this, since it depends on flow, waveform, inspiratory time, and tube size.
Compliance	Often near-normal or increased distal to the obstruction	Driving-pressure and tidal-volume-per-kg logic, built to protect a low-compliance lung, addresses a problem that is not the limiting one here
Expiratory time constant (τ)	The product of resistance and compliance; prolonged and regionally heterogeneous, with complete emptying requiring roughly 3τ	Respiratory rate, I:E ratio, and inspiratory flow should follow measured expiratory mechanics. In children, τ needs normalisation for age, body size, and tube size (Section 4.2.1).
Endotracheal tube size	A fixed resistor in series with the airway; tube size, inspiratory time, and set pressure all shape the measured resistive pressure	Indices of obstruction need normalisation for tube size (Section 4.2.1)
Dynamic hyperinflation	The principal hazard, and it is cumulative: trapped gas accumulates breath by breath, over minutes to hours rather than days	The safety ceiling must track trapped volume, not inspiratory pressure
Auto-PEEP	Frequently 10–15 cmH2O in severe obstruction and measurable at the bedside. The phenomenon is unquestionably harmful; what has not been demonstrated is that the measured airway-opening value tracks that harm, because expiratory muscle activity inflates it and severe airway closure can make it falsely low	A necessary monitor but an unreliable safety target. Its limitation is one of measurement, not of physiology
End-inspiratory lung volume (VEI)	The only measure shown to predict hyperinflation-related complications in adults (Table 2)	A candidate reference comparator; requires apnoea and specific equipment and is unvalidated in children

Expiratory flow reaching zero	Gas is being trapped if inspiratory flow begins before expiratory flow returns to baseline; may look normal at a new steady state (Table 2)	The simplest available bedside signal, and the first thing a clinician looks at. Any framework should name it explicitly rather than assume it
VTi − VTe difference	A positive value means gas is being retained; it cannot detect established hyperinflation at a new steady state (Table 2)	A candidate continuous, noninvasive signal of ongoing retention; not formally evaluated and merits study
Asynchrony and ineffective triggering	Auto-PEEP acts as an inspiratory threshold load that the patient must overcome before a breath can be triggered, producing ineffective efforts, dyspnoea, and muscle fatigue; asynchrony in turn worsens trapping	Trapping persists in spontaneously breathing and weaning patients; so, the framework must address them as well as paralysed patients
Applied PEEP	Lower external PEEP is generally favoured in passive, paralysed patients; added PEEP in severe obstruction has increased hyperinflation and caused hypotension	The logic differs from recruitment strategies and may change once the patient breathes spontaneously; PEEP needs individual titration (Section 5.4)
Haemodynamics	Rising intrathoracic pressure reduces venous return and raises pulmonary vascular resistance, impairing right ventricular function	An important safety endpoint for future studies

5.1. Expiratory Time Constants and the Setting of Respiratory Rate

The instruction to "use a low rate and allow time to exhale" is universal and unquantified. The expiratory time constant (τ) is the product of resistance and compliance and describes the speed of lung emptying; about three time constants are needed to exhale 95% of the tidal volume, which converts into a maximum respiratory rate for a given inspiratory time [31,32]. τ can be computed from resistance and compliance, read from many ventilators, or derived from the expiratory flow–volume relationship, and a prospective comparison of six methods found concordant values in both pressure- and volume-controlled ventilation [32].

Clinicians do not set rate well unaided. In a multicentre study of ventilated adults, τ was prolonged in about half of the cohort, those patients had markedly higher intrinsic PEEP, and the clinician-set rate exceeded the rate predicted from the measured time constant in most patients [33].

Two points apply before this is carried into paediatrics. First, the adult threshold of about 0.8 s for prolonged emptying should not be assumed to transfer to children. In a ventilated child, τ also reflects the tube (Section 4.2.1); so, paediatric reference values need to be established by age and body size and normalised for tube size before any τ-based rate rule is applied [17,18]. Second, a single global τ averages lung units that empty at very different speeds. Electrical impedance tomography can derive expiratory time constants pixel by pixel, which has been validated against flow-derived values in adults with severe respiratory failure [34], and could in principle map regions that are not emptying. Because EIT is present in very few paediatric intensive care units, we regard it as a mechanistic research tool and not as a component of a framework.

5.2. Peak Pressure, Plateau Pressure, and What a Pressure Limit Means in Obstruction

Peak inspiratory pressure comprises a resistive component (the product of resistance and flow) and an elastic component (the product of elastance and tidal volume, clinically the driving pressure) [35]. In obstruction the resistive term dominates and is dissipated across the airways rather than transmitted to the alveolus. A peak pressure of 60 cmH2O with a plateau of 20 cmH2O describes an airway problem; the same peak with a plateau of 35 cmH2O describes a lung at risk. The number alone is uninterpretable.

A very high peak pressure still matters. It signals the size of the resistive load, and the need to raise the ventilator's pressure limit above its default is itself a marker of severity that is worth documenting. A sustained extreme peak pressure, or an abrupt rise,

should prompt a search for mechanical obstruction (mucus plug, kinked or malpositioned tube, aspirated foreign body) rather than being attributed to asthma alone.

Early descriptions of controlled hypoventilation targeted a peak pressure below 50 cmH2O [7], and paediatric series used comparable ceilings while tolerating hypercapnia [6]. However, peak pressure depends heavily on inspiratory flow and waveform: patients ventilated with high flows routinely exceed 50 cmH2O without increased air-leak risk, and changes in peak pressure do not reliably track the reduction in hyperinflation achieved by prolonging expiratory time [36,37]. A peak-pressure ceiling can therefore be met by lowering flow, which shortens expiratory time and worsens the actual hazard. The question is which variable should anchor the safety limit; peak pressure carries information about the airway, not the alveolus.

Auto-PEEP is the natural candidate for a safety ceiling and can be measured with an expiratory hold, and the phenomenon it represents is dangerous. The difficulty is the measurement. Expiratory muscle activity raises measured intrinsic PEEP without dynamic hyperinflation [38], and in severe asthma, the measured value can be dangerously low, because small airways close early in expiration and trap gas behind them at a pressure that the expiratory hold does not capture (hidden auto-PEEP) [39]. A value that can be raised without trapping and lowered by severe trapping is unsuitable as a safety ceiling, although it remains necessary for setting PEEP (Section 5.4). VEI, not auto-PEEP, has a demonstrated relationship to harm in adults [23,24].

5.3. Mode Selection: An Open Question and a Moving One

Neither PALICC-2 nor PEMVECC endorses a mode; both leave the choice to clinician experience and patient characteristics [1,2]. No consensus on the superiority of either mode in severe paediatric status asthmaticus has been established, and in practice, the choice rests on institutional preference and clinical rationale, not on comparative outcome evidence. An adult meta-analysis in acute respiratory failure found no difference in air leak between pressure- and volume-controlled ventilation, but in populations drawn from ARDS and postoperative surgery, not asthma [40]. The evidence is outlined below by mode, with bench findings kept separate from clinical evidence.

Pressure-controlled ventilation. Clinical evidence: a retrospective single-centre paediatric series found it an effective and reasonable option [41], but the accompanying editorial treated this as a description of feasible practice, not evidence of superiority [42]. Physiological rationale: delivered tidal volume varies with mechanics and effort; so, it may fall when obstruction is severe and rise as mechanics improve at the same pressure setting, and a pressure ceiling does not prevent excessive volume. Bench evidence: simulations under high-resistance conditions found that pressure-targeted modes delivered less than the programmed tidal volume, which raises a concern about hypoventilation [43].

Volume-controlled ventilation. Delivered volume is fixed, but airway pressures may rise as mechanics worsen, and actual delivery still depends on pressure limits, leaks, and ventilator performance. Some clinicians start with volume control because of the bench concern above [43]. Comparative clinical data in paediatric status asthmaticus are lacking.

Pressure-regulated volume control. The algorithm adjusts inspiratory pressure breath by breath according to the volume achieved on the preceding breath. In a lung whose resistance changes continuously, the applied pressure may lag behind the patient; so, the stable minute ventilation for which the mode is chosen may not be achieved. It has also been suggested, without independent confirmation in a primary source, that dynamic hyperinflation could cause the algorithm to adjust pressure inappropriately; we regard this as a hypothesis.

Whichever mode is chosen, status asthmaticus is a changing picture: resistance falls, sometimes abruptly, as bronchodilators act, and may rise again as their effect wanes. A mode suited to intubation, when resistance is at its peak, may be unsuitable a few hours later. Mechanics should therefore be reassessed at intervals in a structured way, with readiness to change mode or alarm settings as the patient's needs change.

5.4. Applied PEEP in Obstructive Disease: Individualised Titration

PEEP is used in both conditions, for different reasons. In parenchymal injury, PEEP is added to recruit collapsed units and reduce shunt, and contemporary practice increasingly titrates it against plateau and driving pressure rather than by any fixed schedule [1]. In obstruction, the question is entirely different, and—importantly—the appropriate level depends on mechanics and on whether the patient is making respiratory effort.

During controlled ventilation of a passive, paralysed patient, the conventional position is to minimise external PEEP, and this is one of the few areas where direct experimental evidence in obstruction exists. In a controlled physiologic study of paralysed patients with severe airflow obstruction, PEEP was applied at 5, 10, and 15 cmH2O with hyperinflation quantified by VEI; increasing PEEP progressively increased functional residual capacity, and two of the six patients could not be studied at the highest PEEP level because of hypotension [44]. The accompanying editorial framed the question that has still not been settled—whether PEEP has any role at all in airflow obstruction [45]. Paediatric practice reflects this, commonly using zero or minimal PEEP during the acute paralysed phase.

Once the child breathes spontaneously, the calculation changes. Intrinsic PEEP acts as an inspiratory threshold load, and applied external PEEP counteracts it in two ways: it splints the collapsible intrathoracic airways against dynamic compression during expiration, and it narrows the pressure difference the patient must generate to trigger the next breath, which reduces ineffective efforts and the work of breathing [29,46]. In patients with obstructive airway disease weaning on pressure support, intrinsic PEEP averaged about 9.5 cmH2O, measured from the pressure deflection needed to start inspiratory flow rather than by an end-expiratory hold (which requires a passive patient); progressive circuit PEEP progressively reduced the effort needed to overcome it, without altering the delivered tidal volume or frequency [29]. A commonly cited target is external PEEP at about 75–80% of the measured auto-PEEP. A review of auto-PEEP detection and prevention provides this figure with the qualifier that the benefit applies to spontaneously breathing patients, and that there is no evidence that such PEEP helps during controlled ventilation when the patient makes no inspiratory effort [47]. Where expiratory flow limitation is present, applied PEEP below auto-PEEP reduces the intrinsic component while leaving total PEEP unchanged; without flow limitation it simply adds, which is why exceeding the measured auto-PEEP is advised against [48,49].

Two caveats keep this from being a rule. The measured auto-PEEP on which the 75–80% target depends is itself unreliable in exactly the circumstances where it matters most, for the reasons set out in Section 5.2—it can be falsely low where airway closure is severe, and falsely high where expiratory muscles are active [38,39]. And evidence from chronic obstructive pulmonary disease, where modest PEEP produced a more uniform pattern of lung emptying than zero PEEP [50], may or may not apply to paediatric asthma. We therefore favour individualised PEEP titration, re-assessed as mechanics and respiratory effort change, over fixed recommendations for each ventilatory phase. Lower external PEEP is supported in the passive, paralysed patient, where it avoids adding to trapped volume, whereas in the spontaneously breathing child a modest level of applied PEEP, set below and guided by the measured intrinsic value, is physiologically justified. These are tendencies, not thresholds: the appropriate level depends on the presence of expiratory flow

limitation, the reliability of the auto-PEEP measurement, respiratory effort, and haemodynamic tolerance, and none of these approaches has been tested against outcomes in children.

5.5. Permissive Hypercapnia and pH Targets

Both fields tolerate hypercapnia without evidence for any particular threshold. PALICC-2 and PEMVECC both recommend a pH floor near 7.20, and PEMVECC notes the absence of outcome data on an optimal CO2 target or the lowest tolerable pH [1,2]. Asthma-specific sources vary between floors of 7.15 and 7.20 [51]. Adult asthma reviews conclude that permissive hypercapnia may reduce morbidity and mortality compared with conventional normocapnic ventilation, without establishing a threshold [7,9]. The similar floors in different frameworks do not in themselves provide outcome evidence for any threshold.

5.6. Noninvasive Respiratory Support: An Asymmetric Comparison

Noninvasive support occupies different positions in the two conditions. In established severe PARDS, it is not expected to avert intubation, and noninvasive modalities are relevant chiefly in milder or at-risk disease [1]. In status asthmaticus, noninvasive positive pressure and high-flow nasal cannula are used with the explicit aim of avoiding intubation in children who are already severe, and their expanding use may contribute to the reported fall in intubation rate, although changes in case mix and intubation thresholds cannot be excluded [4,8]. The evidence base remains thin: no standardised threshold defines which patients warrant a noninvasive trial, and a 2025 narrative review concluded that robust evidence in paediatric critical asthma remains limited and heavily observational [4,52]. The 2025 AARC/PALISI guideline suggests NIV over conventional oxygen for persistent hypoxaemia or respiratory distress, with very low certainty of evidence [3].

5.7. Bedside Signals That Are Already Available

Several informative signals need no equipment beyond what is already at the bedside, and they have not been formalised.

The flow–time waveform is the first display a clinician looks at, and it answers the central question directly: if inspiratory flow begins before expiratory flow has returned to zero, gas is being trapped. It is continuous and available on every ventilator, and it should be named explicitly in any framework. Its limitation is that, once a new steady state has been reached, the waveform may look normal despite persistent hyperinflation. The difference between inspired and expired tidal volume, VTi − VTe (a positive value signals retention), offers a quantitative counterpart under leak-free conditions, that is, with no cuff leak, chest drain, tracheo-oesophageal fistula, or other escape route. The leak-free requirement is a real constraint in paediatrics, where uncuffed tubes remain in use. VTi − Vte has a more fundamental limit: it detects ongoing accumulation, not the volume already trapped. Once hyperinflation has reached a new steady state, Vti − Vte returns to about zero although end-expiratory lung volume remains high; so, it cannot reliably identify established hyperinflation and a value of zero is not reassuring. A positive value suggests worsening, and a negative value suggests that trapped gas is being released as obstruction resolves. We are not aware of any formal evaluation of VTi − VTe as a severity or safety index and propose it as a candidate for study, to be interpreted together with other signals. Of the measures in Table 2, only VEI quantifies trapped volume directly; because it derives from adult studies in paralysed patients and is impractical at the bedside, we propose it as a reference comparator against which bedside signals can be tested in children.

When circulatory compromise develops, point-of-care ultrasound can show that positive pressure is impairing the right heart: right ventricular dilatation, septal flattening with D-shaping of the left ventricle, a non-compressible inferior vena cava, and reduced stroke volume, corresponding to the sonographic profile of obstructive shock [53]. Capnography is covered in Table 2. A shark-fin waveform is the classic capnographic sign of bronchospasm, and as cardiac output falls, alveolar dead space rises and end-tidal CO2 may drop sharply [54]; a falling end-tidal value in a deteriorating asthmatic child should therefore not be read as improving ventilation. Lung ultrasound in the same examination identifies pneumothorax, the other complication that presents as sudden decompensation. None of this has been studied as a monitoring strategy in ventilated paediatric asthma.

5.8. Extracorporeal Carbon Dioxide Removal

Extracorporeal support deserves more targeted treatment than a general reference to ECMO, because the physiology of obstruction points to a specific modality. The failure in status asthmaticus is one of ventilation rather than oxygenation: the problem is carbon dioxide clearance through obstructed airways, and oxygenation is frequently preserved until late. Extracorporeal carbon dioxide removal (ECCO2R) addresses exactly this and has been proposed for refractory status asthmaticus where severe dynamic hyperinflation or life-threatening respiratory acidosis persists despite optimal medical and ventilator management—the situation in which the ventilator itself has become the limiting factor [55]. ECCO2R as an adjunct to invasive ventilation in refractory asthma was first reported in 1981, and several case series have followed [26].

Veno-venous ECMO has a reasonably established role in refractory status asthmaticus, whereas evidence for genuinely low-flow, exclusively CO2-removing circuits is sparse: the largest reported series used a two-cannula veno-venous configuration at blood flows closer to VV-ECMO than to low-flow ECCO2R [56]. No paediatric indication criteria exist. Because the mechanistic rationale is arguably cleaner in obstruction than in hypoxaemic failure, and registry infrastructure for paediatric extracorporeal support exists, defining when a ventilated child with asthma should be considered for CO2 removal is a tractable question for the consensus process.

6. Toward a Framework: What Would Have to Come First

The following are offered explicitly as a starting point for discussion and future study, not as a validated protocol. They are framed as prerequisites rather than recommendations, because the main gaps are in validated measurement and outcome evidence.

Table 4 summarises the five candidate explanations examined in Section 4 and the corresponding elements of the proposed framework, so that each evidence gap can be read against the study or consensus task intended to address it.

Table 4. The five candidate explanations for the lack of validated, disease-specific ventilation targets in paediatric critical asthma, and the proposed framework.

Candidate Explanation (Section)	What Is Known	Why It Impedes Consensus	Proposed Response (Sections 6 and 7)
1. Heterogeneity of obstruction and overlap with parenchymal disease (Section 4.1)	"Status asthmaticus" is a clinical label; bronchospasm, oedema, plugging, and intercurrent infection vary between and within patients. Mixed physiology occurs.	No single mechanical phenotype to write targets for; mixed cases fit neither PARDS nor obstruction frameworks.	Define the target population and prespecify a mixed-physiology subgroup in trials and registries.
2. No severity metric usable after intubation (Sections 4.2 and 4.2.1)	Validated scores (PRAM, PASS, peak flow) need spontaneous effort. VEI predicted	Targets cannot be graded, stratified or tested against	Validate bedside candidates (flow–time waveform, Ppeak − Pplateau, VTi − VTe) against VEI (an adult-derived

	complications in adults. Candidate bedside surrogates are unvalidated; tube effects need normalisation.	outcome in the intubated child.	reference comparator), then outcomes—as a parallel priority, not a precondition.
3. Small ventilated denominator (Section 4.3)	About 5% of PICU status asthmaticus admissions are intubated, and the fraction has reportedly fallen.	Randomised trials are difficult; practice rests on case series.	Multicentre cohorts, using registry data only if suitable, with stated limits: confounding by indication, limited ventilator detail.
4. Dominant hazard is haemodynamic and develops over minutes to hours (Section 4.4)	Dynamic hyperinflation reduces venous return and raises pulmonary vascular resistance; air leak risk depends on settings and effort.	Methods built for slow parenchymal injury poorly; endpoints are unstandardised.	Prespecify haemodynamic, air-leak fitand ineffective-triggering safety endun-points; specify monitoring and alarm strategy.
5. Brief, unstable and rapidly reversible condition (Section 4.5)	Mechanics change within minutes to hours as bronchospasm resolves; ventilation may last hours.	Static settings and ventilator-free-days endpoints behave differently from ARDS.	Trial designs suited to short courses; be-scheduled reassessment of mode, settings, and alarms.
Proposed framework (Sections 6 and 7)	Existing physiological principles and PEMVECC recommendations are available now.	A validated metric is not strictly required to begin.	Run structured consensus (Delphi, with adult intensivists) alongside validation, registry, and physiological studies.

- Pursue a severity metric for the intubated child as a parallel priority, not a precondition: existing physiological principles and the PEMVECC recommendations can support a structured consensus process while validation proceeds. The most plausible near-term path pairs a feasible bedside variable from Table 2 (the flow–time waveform, the peak-to-plateau gradient normalised for tube size and inspiratory time, or VTi − VTe, which signals ongoing retention but cannot identify established hyperinflation) with VEI as a reference comparator in a physiological study, and then with outcomes (Steps 1–3, Section 7).
- Anchor the safety ceiling to trapped volume, not to inspiratory pressure—while acknowledging that the only variable with demonstrated predictive value in adults (VEI) is currently impractical and unvalidated in children, and that the convenient surrogate (auto-PEEP) is unreliable as a measurement in precisely the patients of greatest concern [23,24,38,39] (Steps 1 and 3).
- Derive respiratory rate from measured expiratory mechanics rather than convention, and establish paediatric reference values for the expiratory time constant normalised for age, body size, and endotracheal tube size. The adult 0.8 s threshold should not be assumed to transfer [17,31–33] (Steps 1 and 2).
- Treat mode selection as an open question and as a recurring one. A coherent physiologic rationale and paediatric practice favour pressure targeting; bench data raise a concern about volume delivery at high resistance; no study has compared clinical outcomes between modes in children, and the choice currently rests on local preference. Beyond resolving this empirically, a framework should require scheduled reassessment of mechanics and permit mode change—or, at minimum, reconfiguration of alarms—as bronchospasm resolves, rather than treating mode as fixed at intubation [41,43] (Steps 3 and 4).
- Individualise PEEP titration instead of prescribing fixed levels for each ventilatory phase. Lower external PEEP is generally supported in the passive, paralysed patient, and low applied PEEP set below the measured intrinsic value in the spontaneously breathing child, but these are tendencies to be re-titrated against mechanics, effort,

and haemodynamics in each child, and neither rests on paediatric outcome data [29,44,47] (Step 4).

- Adopt haemodynamic endpoints alongside respiratory ones and include air leak and ineffective triggering as prespecified outcomes. Circulatory collapse in these patients is not confined to the peri-intubation period; it can develop later, from the progressive accumulation of trapped gas under inappropriate settings [24,27,53] (Step 3).
- Specify a monitoring and alarm strategy, not only initial settings. In a condition with highly dynamic airway mechanics that can shift rapidly and potentially resolve within minutes or hours, the alarm thresholds that alert the clinician to volume retention, to resolving bronchospasm, or to a shortening expiratory flow pattern are as consequential as the settings chosen at intubation (Step 4).
- Define indications for extracorporeal CO2 removal in children, given that the failure in obstruction is hypercapnic rather than hypoxaemic and that no paediatric criteria currently exist [55,56] (Step 4).
- Retain a pH floor as a pragmatic guide while stating plainly that no outcome data justify any specific threshold over another [2,9] (Step 4).
- Treat noninvasive escalation criteria as a distinct, prospectively studiable decision point rather than an extrapolation from either PARDS or adult asthma practice [4,52] (Step 4).

7. Research Agenda

The research agenda has four steps, numbered as in Figure 1 and linked to the Section 6 bullets in which each is cited. They are not strictly sequential: consensus development (step 4) can begin now and be revised as results from steps 1–3 accrue, whereas outcome validation (step 3) depends on measures first tested in steps 1 and 2.

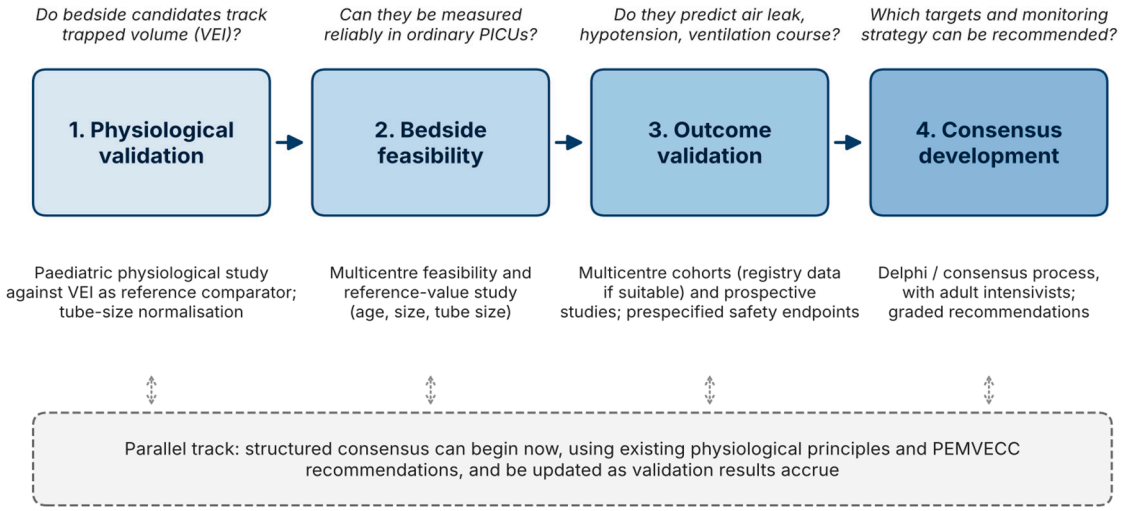


Figure 1. Proposed evidence-development pathway for ventilator targets in paediatric critical asthma. VEI, end-inspiratory lung volume above functional residual capacity; PEMVECC, Paediatric Mechanical Ventilation Consensus Conference.

Figure 1 shows the pathway, with consensus development running in parallel rather than waiting for the final stage.

Step 1, physiological validation. A physiological study in ventilated children with obstruction should test feasible bedside candidates from Table 2 (flow–time waveform, Ppeak − Pplateau, VTi − VTe, expiratory time constant) against VEI as a reference comparator, reporting tube size explicitly [23].

Step 2, bedside feasibility and reference values. A multicentre study should show that these measures can be obtained reliably in ordinary PICUs, and should establish reference values for the expiratory time constant (and, if useful, the peak-to-plateau gradient) by age, body size, and endotracheal tube size, not as a single figure [17,18]. Regional distribution could be explored with electrical impedance tomography where available, as a mechanistic sub-study [34,57].

Step 3, outcome validation. Candidate measures, and the settings guided by them, should be linked to air leak, hypotension and the course of ventilation in a multicentre cohort. Existing registries might supply some of these data, but their suitability has not been verified. Virtual Pediatric Systems (VPS) is a subscription-based clinical registry with standardised, manually validated data on diagnoses, severity of illness, interventions, and outcomes; the Pediatric Health Information System (PHIS) is an administrative database of children's hospitals with diagnoses, procedures, and resource use but no physiological detail. Neither was designed to record breath-level ventilator variables (mode-specific settings, resistance, compliance, auto-PEEP), and we have not confirmed that either records ventilation mode reliably enough to compare pressure- with volume-controlled ventilation. This should be checked against the registries' data dictionaries before a protocol is written; if mode cannot be ascertained, data would have to be collected prospectively or from electronic records. Mode selection is also not random; so, confounding by indication would remain after adjustment, and the lack of an obstruction-specific severity measure limits severity adjustment. Any retrospective comparison is therefore best regarded as hypothesis generating [4,41].

Step 4, consensus development. A formal Delphi or consensus process on paediatric obstructive respiratory failure, structured like PALICC and run jointly with adult intensivists because the gap is shared [1,11], can start now from PEMVECC and existing physiological principles, and be revised as results from steps 1–3 become available.

Two further points sit alongside the agenda. Future paediatric ventilation trials should consider including status asthmaticus subgroups instead of excluding them [58,59], and trial design should allow for the short, variable duration of ventilation in this condition, which makes ventilator-free-days endpoints behave differently than in ARDS [30]. The unexplained racial and ethnic disparities in PICU asthma outcomes also warrant dedicated investigation [8].

8. Limitations

This is a narrative, not a systematic, review, consistent with its aim of conceptual synthesis and argument rather than pooled effect estimation. Much of the underlying primary literature on ventilated paediatric status asthmaticus is decades old, retrospective, and drawn from small single-centre series.

A limitation specific to paediatrics conditions every measurement proposal in this paper. "Children" spans infants to adolescents, across which lung volumes, airway calibre, compliance, resistance, and normal respiratory rate vary by an order of magnitude, and endotracheal tube size varies as well (Section 4.2.1). Any candidate metric must be indexed to something, and age, weight, height, predicted body weight, and tube size each capture only part of the variation. Establishing the normalisation is part of steps 1 and 2.

Several of the physiologic findings we rely on most heavily—VEI, expiratory time constants, the mode-selection bench data, the PEEP studies—are adult or bench-derived, and their extrapolation to children is uncertain; we present them as hypotheses requiring paediatric validation. Finally, we searched explicitly for existing guidance, but absence is harder to establish than presence, and national or institutional guidance not indexed in the sources searched may have been missed.

9. Conclusions

Paediatric critical asthma requiring invasive ventilation has no validated, outcome-linked set of ventilator targets, although PEMVECC provides general guidance for paediatric obstructive ventilation, PALICC-2 for lung-protective ventilation, and the 2025 AARC/PALISI critical asthma guideline does not address invasive ventilation. A comparable gap exists in adult critical care, which suggests that it reflects the disease more than paediatric neglect. Of the five explanations we examined, the lack of a severity metric usable after intubation is in our view the most tractable, although resolving it would not by itself produce a guideline.

The strongest existing candidate is end-inspiratory lung volume above functional residual capacity (VEI), which predicted hyperinflation-related complications in adults with acute severe asthma but requires apnoea, paralysis, and dedicated spirometry. It has not been validated in children. The main unanswered questions are whether feasible bedside signals (the flow–time waveform, the peak-to-plateau gradient normalised for tube size, VTi − VTe) track trapped volume in children; which reference values apply by age, body size, and tube size; whether these measures predict air leak, hypotension, and the course of ventilation; and how PEEP, mode, and rate should be individualised as mechanics change.

We propose that physiological validation, bedside feasibility and reference-value studies, and outcome validation proceed together with a structured consensus process, as shown in Figure 1, so that recommendations can be revised as evidence accrues.

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