Mechanical Ventilation in Severe Asthma



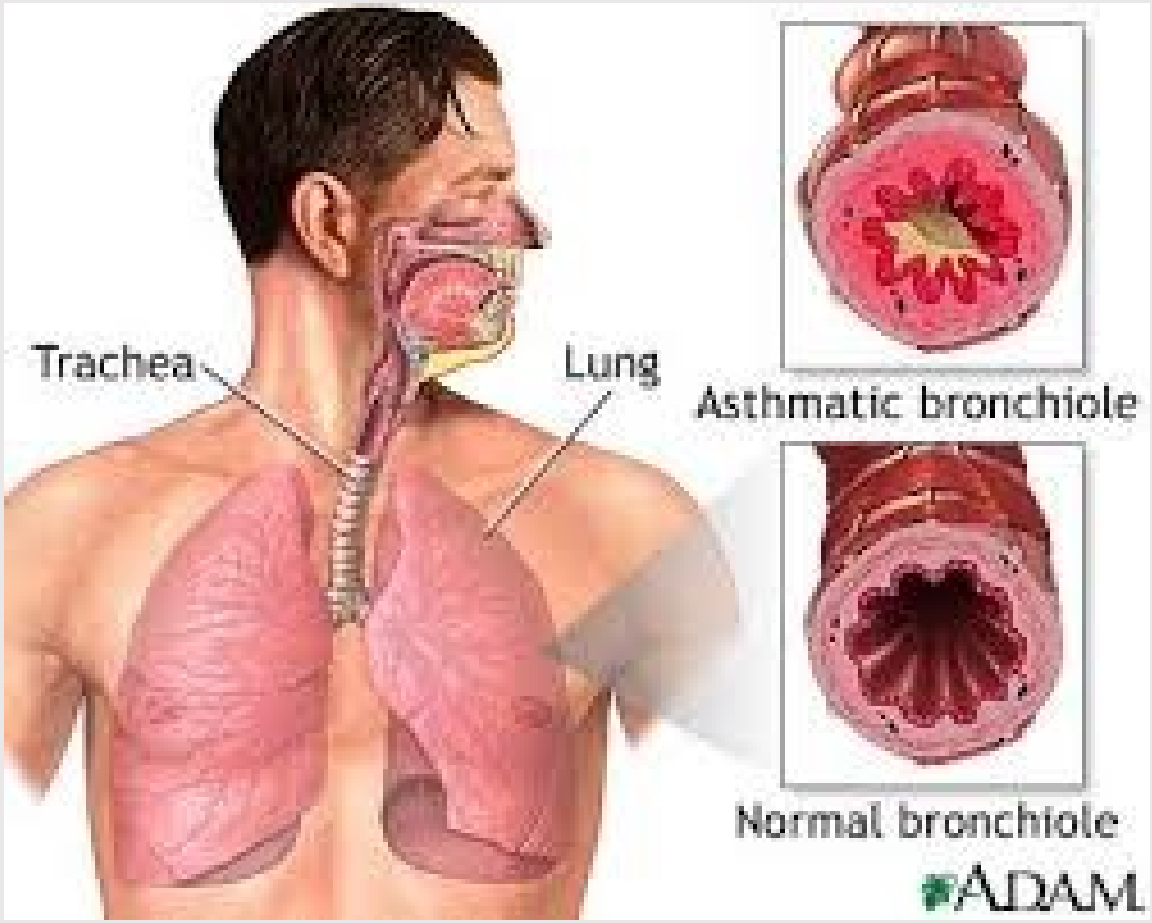
Dr. M. Hadi Alakkad

it is estimated that about **10%** of individuals admitted to hospital for asthma go to the **intensive care unit**, with **2%** of all admitted patients being **intubated**.

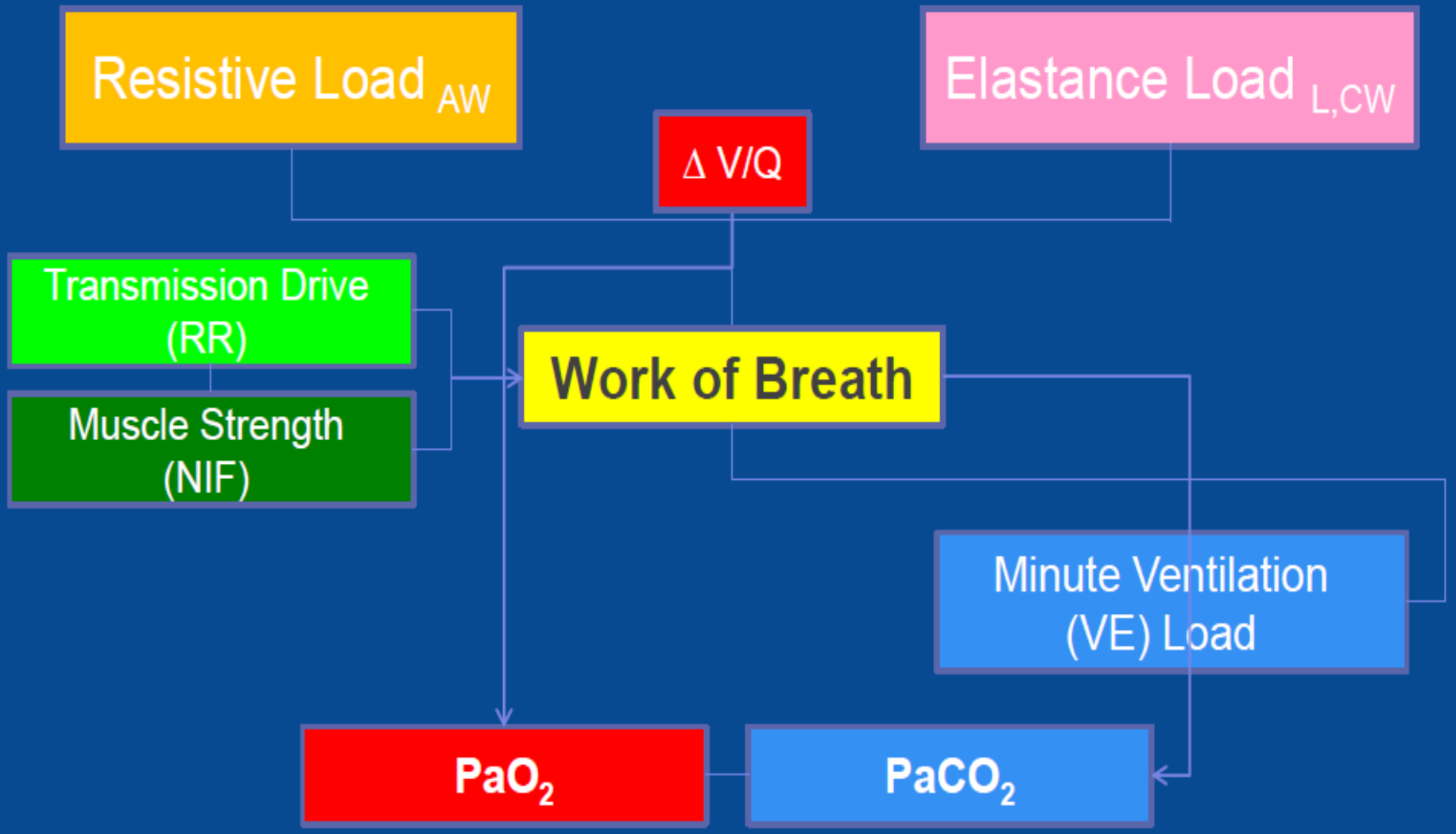
Camargo CA Jr., Smithline HA, Malice MP, et al. A randomized controlled trial of intravenous montelukast in acute asthma. *Am J Respir Crit Care Med. 2003;167:528–533.*

Why Asthmatic Patients Get Worse and Admitted to The ICU

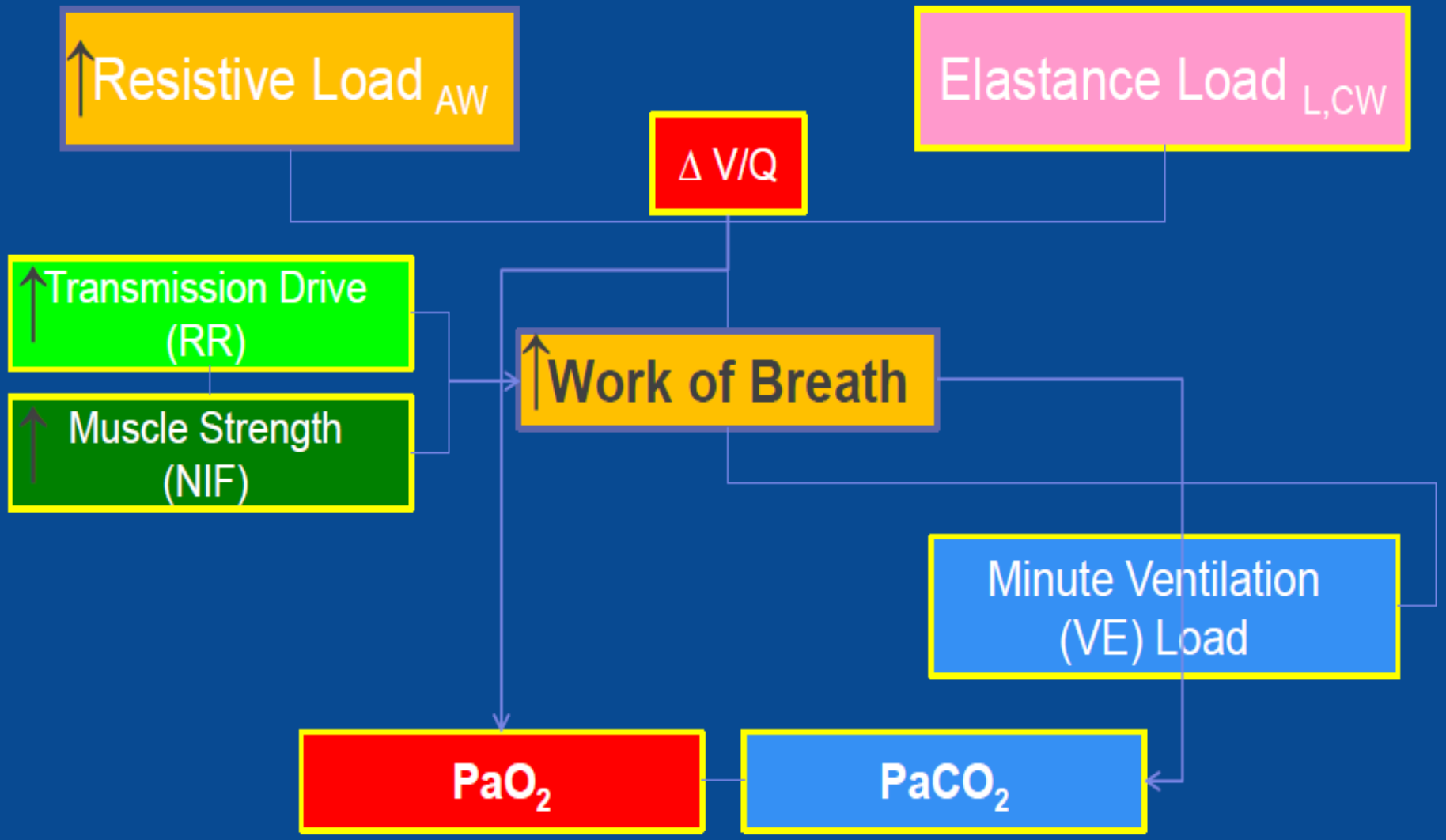
Increased Resistance



Sustaining Oxygenation and Alveolar Ventilation



Respiratory Failure due to ↑ Resistance Load



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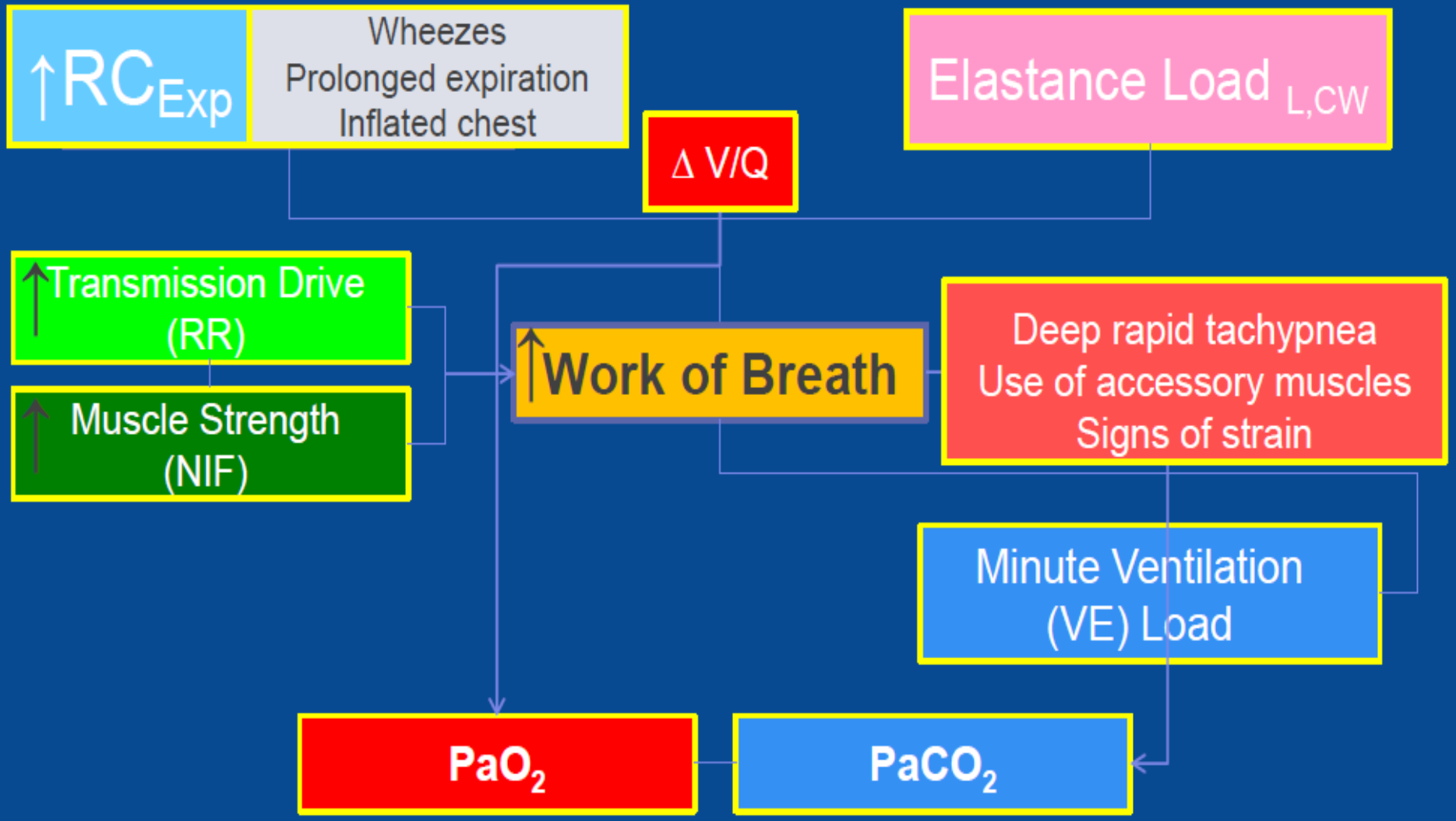


Obstructive Airway Disease

- **Beware of auto-PEEP!**



PATHOPHYSIOLOGY

Severe Airway Obstruction & Gas Trapping

Severe Bronchospasm

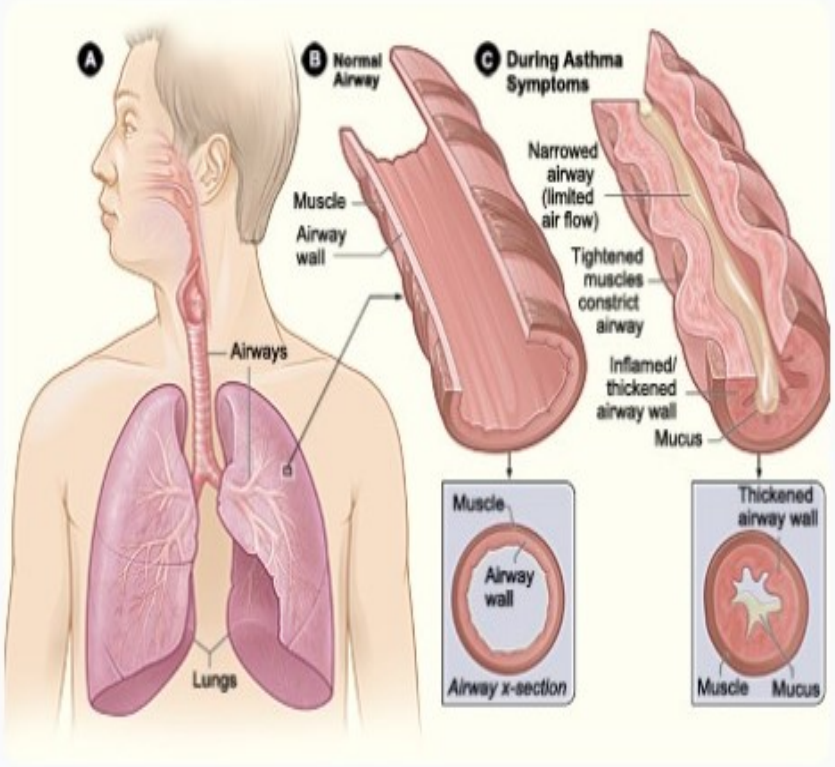
Smooth muscle constriction with mucosal edema and dense intraluminal mucus plugging.

Expiratory Flow Limitation

Profound increase in airway resistance prolongs required exhalation time significantly.

Dynamic Hyperinflation

Incomplete exhalation creates intrinsic PEEP (Auto-PEEP) and dynamic lung overdistension.



Auto PEEP

- Increased Resistence

- Increased RR

When **death** does occur it is most commonly a result of one of the complications of **severe gas-trapping**. These complications include:

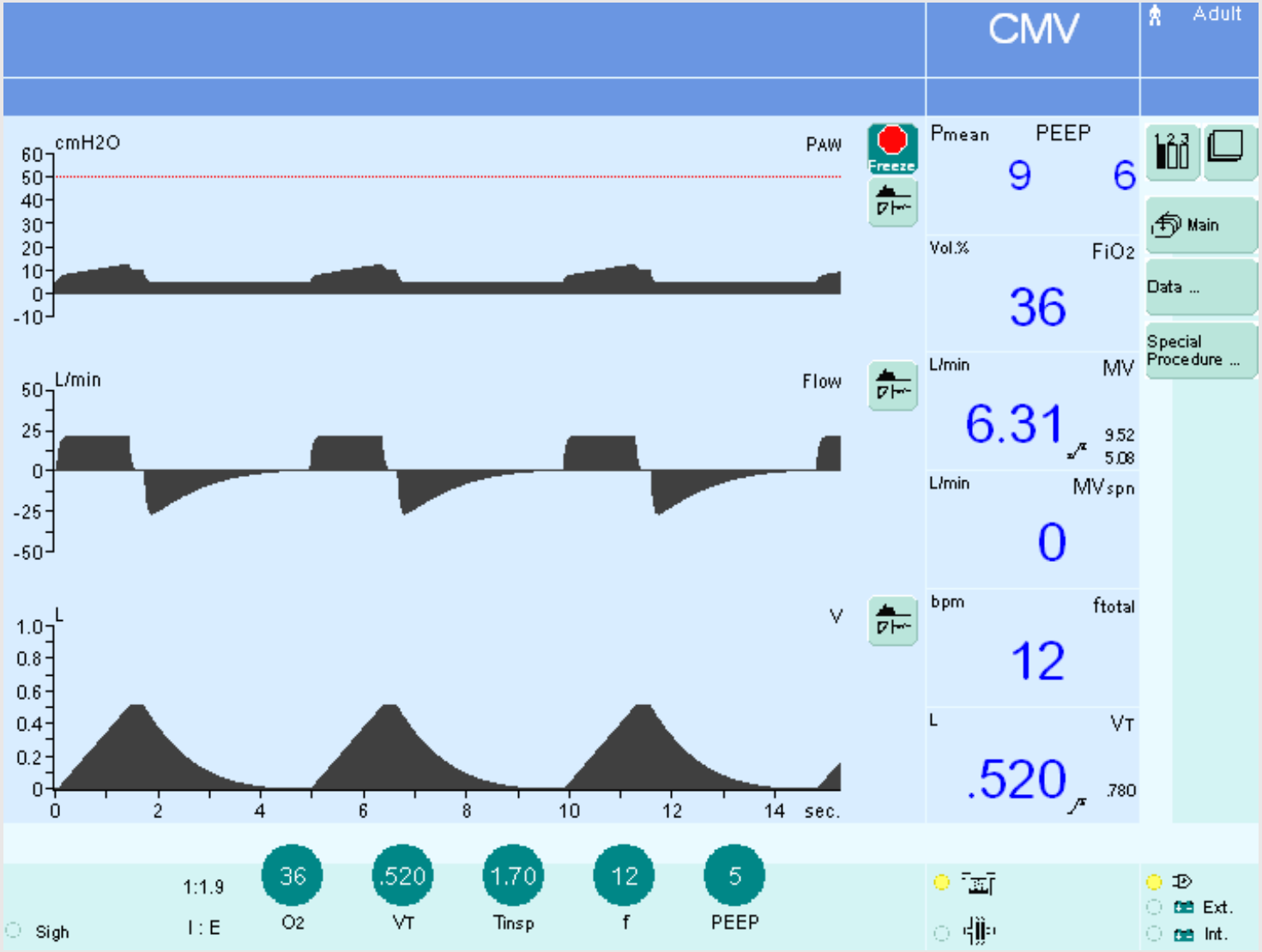
Barotrauma

Hypotension

Refractory respiratory acidosis.

Richards GN, Kolbe J, Fenwick J, Rea HH. Demographic characteristics of patients with severe life threatening asthma: comparison with asthma deaths. Thorax. 1993;48:1105–1109.

NO AUTOPEEP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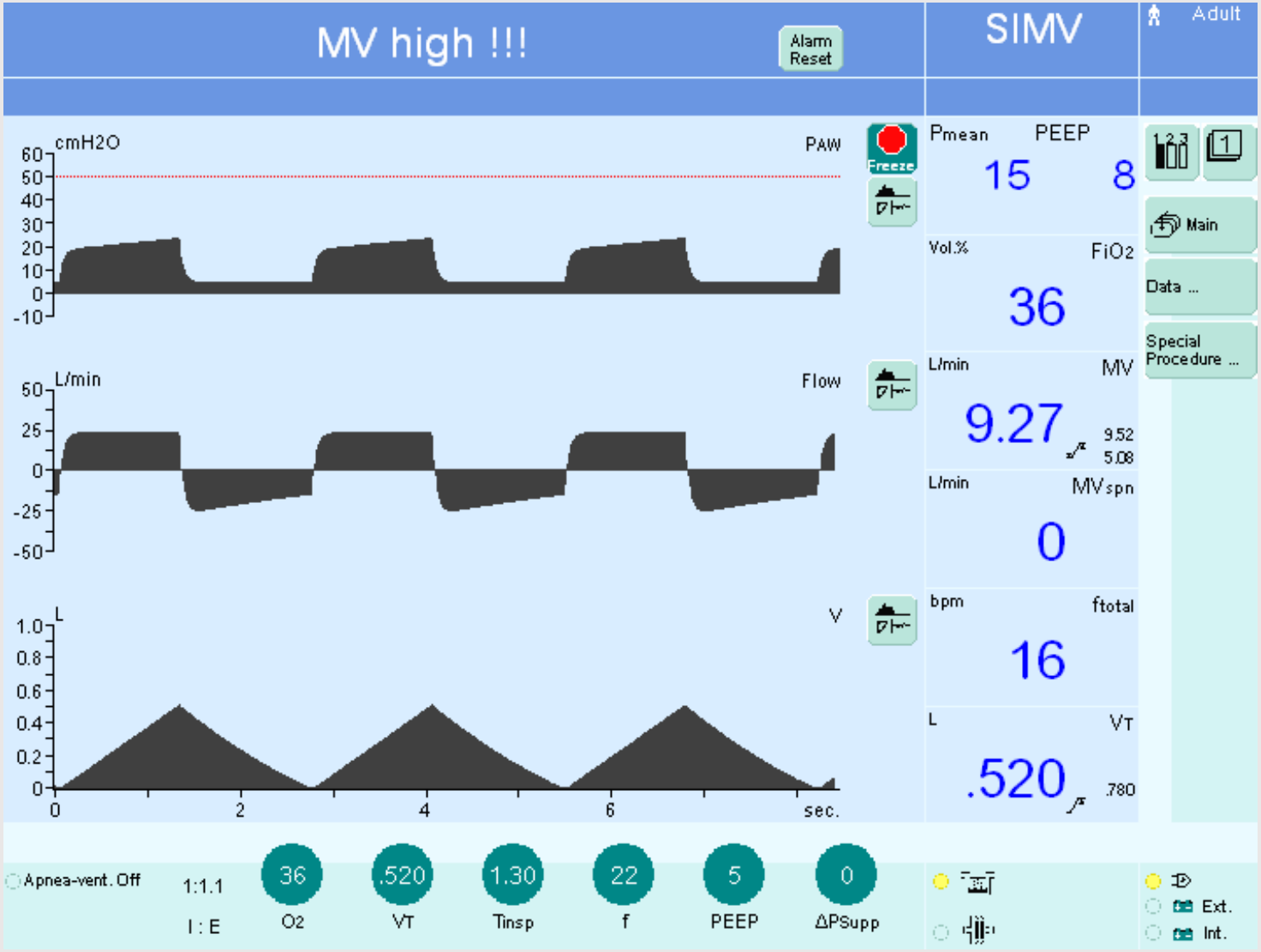
AUTO-PEEP


MV high !!!
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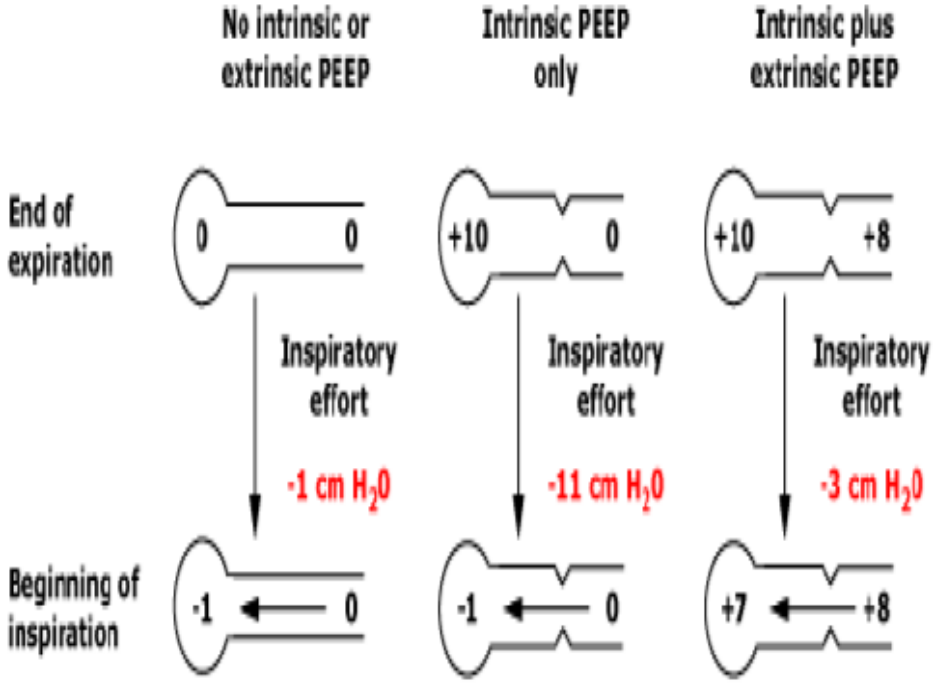
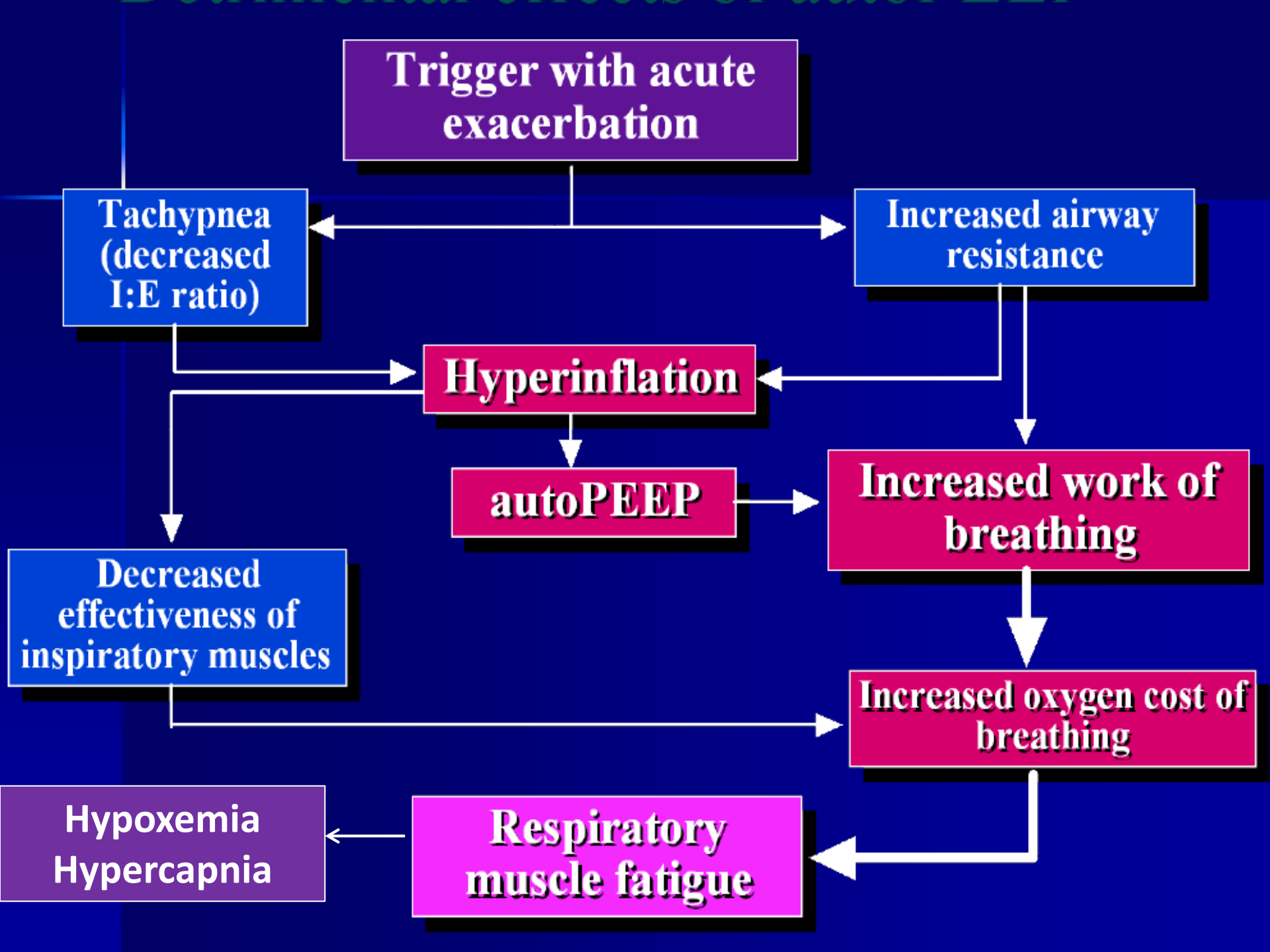
↑ Work of Breath



Intrinsic PEEP (cm H_2O)	0	10	10
Extrinsic PEEP (cm H_2O)	0	0	8
Gradient (intrinsic-extrinsic PEEP, cm H_2O)	0	10	2

Why Asthmatic Patients Get Worse and Admitted to The ICU


Trigger with acute exacerbation
Tachypnea (decreased I:E ratio)
Increased airway resistance
Hyperinflation
autoPEEP
Increased work of breathing
Decreased effectiveness of inspiratory muscles
Increased oxygen cost of breathing
Hypoxemia Hypercapnia
Respiratory muscle fatigue

Noninvasive Ventilation in Asthma patients

Noninvasive Ventilation in Asthma patients

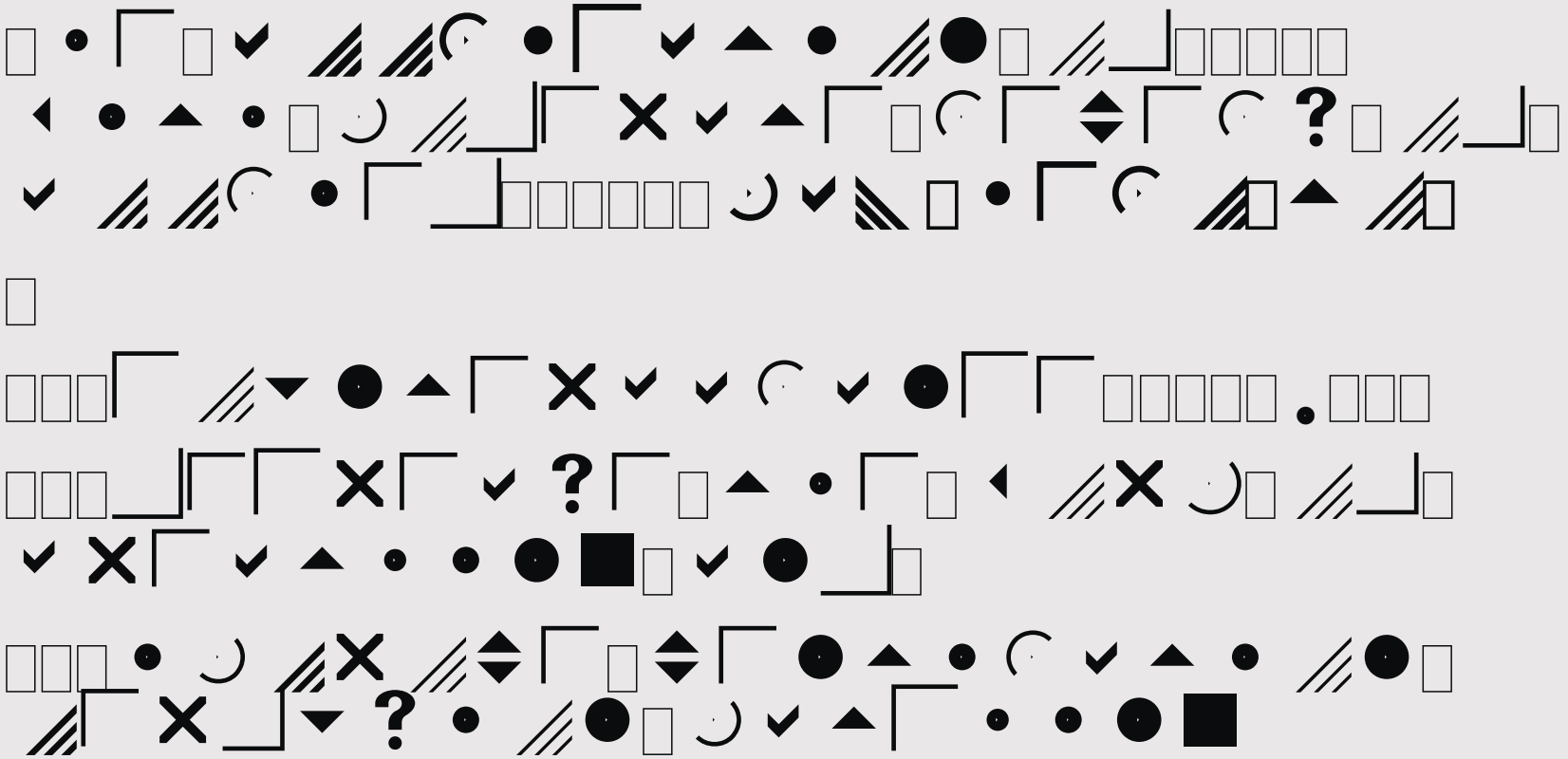
The use of NIV in SAE remains **controversial**. However, its application might be **considered** in order to **decrease the need for invasive MV and its deleterious consequences**

Noninvasive Ventilation in Asthma patients

It is most likely to be successful when instituted **early** before progression to life-threatening **co2 retention** and **impending respiratory arrest.**

Scala R. noninvasive ventilation in sever acute asthma ? Still far from the truth. Respir Care 2010; 55:630-637.

Noninvasive Ventilation in Asthma patients



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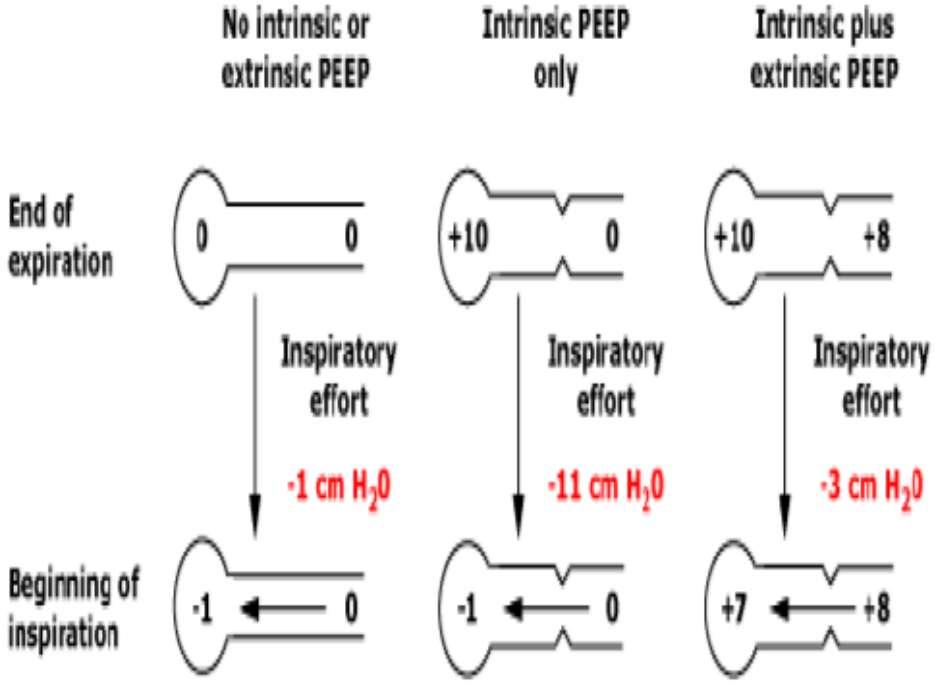

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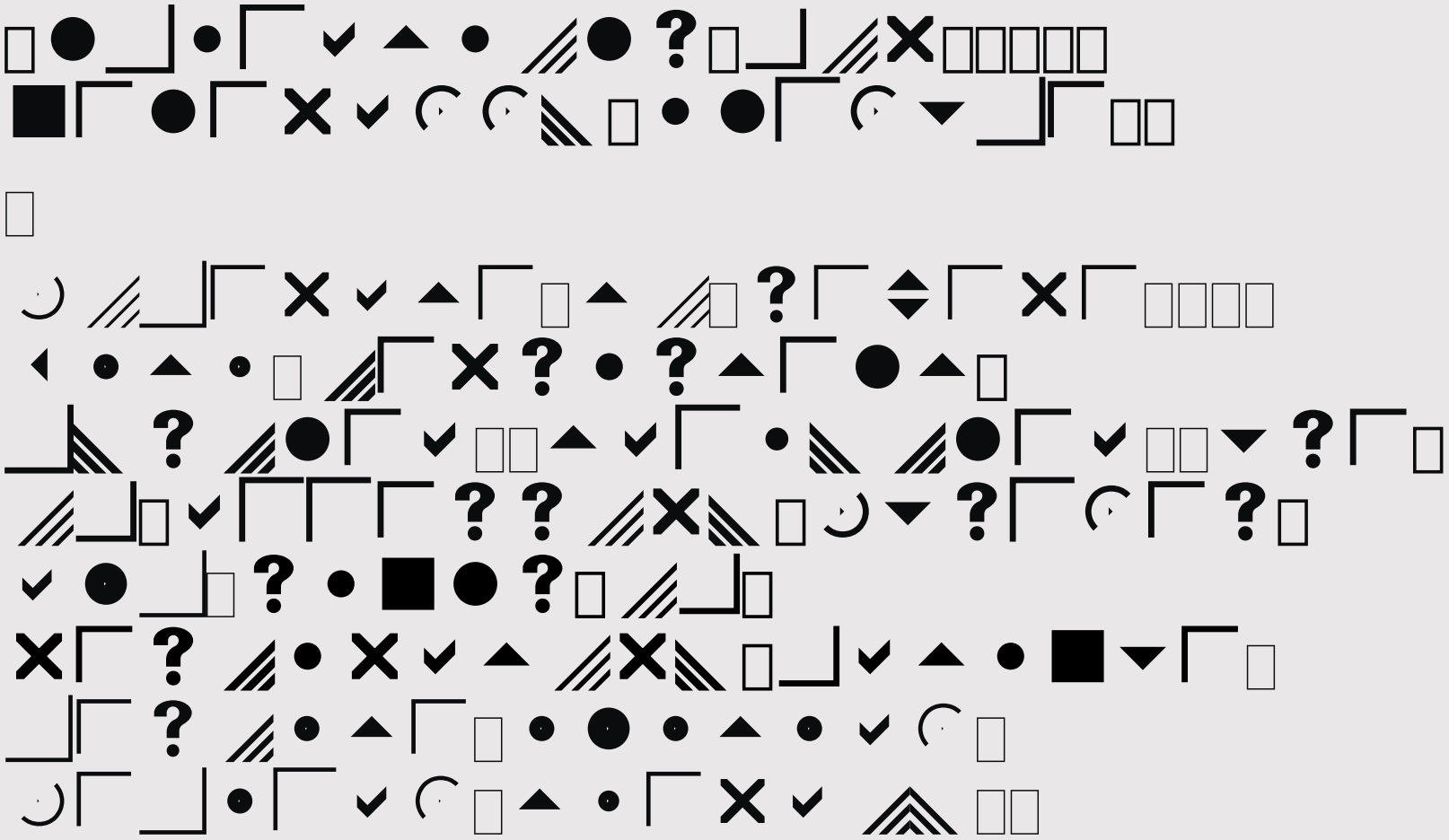
Noninvasive Ventilation in Asthma patients



Noninvasive Ventilation in Asthma patients

PRE-INTUBATION SUPPORT

Non-Invasive Ventilation (NIV) in Acute Asthma

Clinical Role & Goals

Unloads respiratory muscles, reduces breathing work, and bridges to medical therapy response.

Auto-PEEP Offsetting

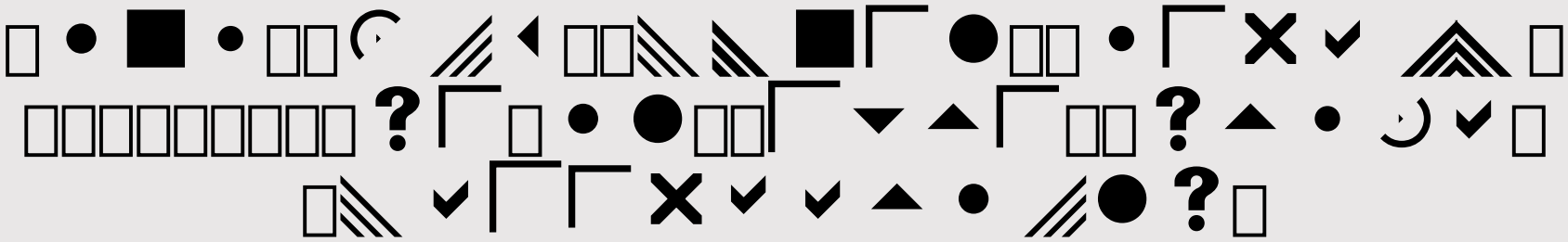
Low EPAP (3–5 cmH2O) counteracts intrinsic PEEP threshold without worsening gas trapping.

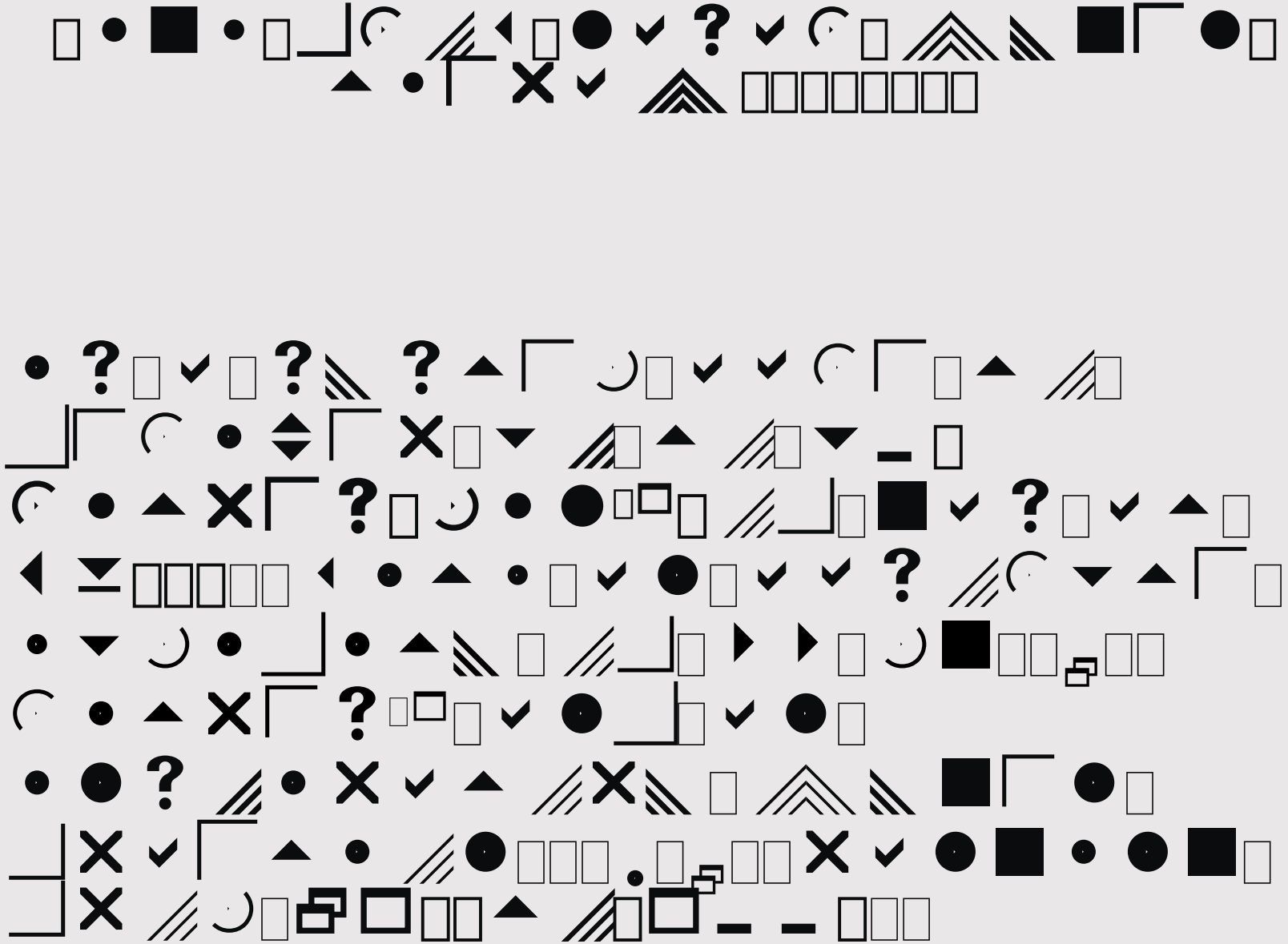
Initial Settings

IPAP 8–10 cmH2O, EPAP 4–5 cmH2O; titrate for low tidal volume (6–8 mL/kg) and comfort.

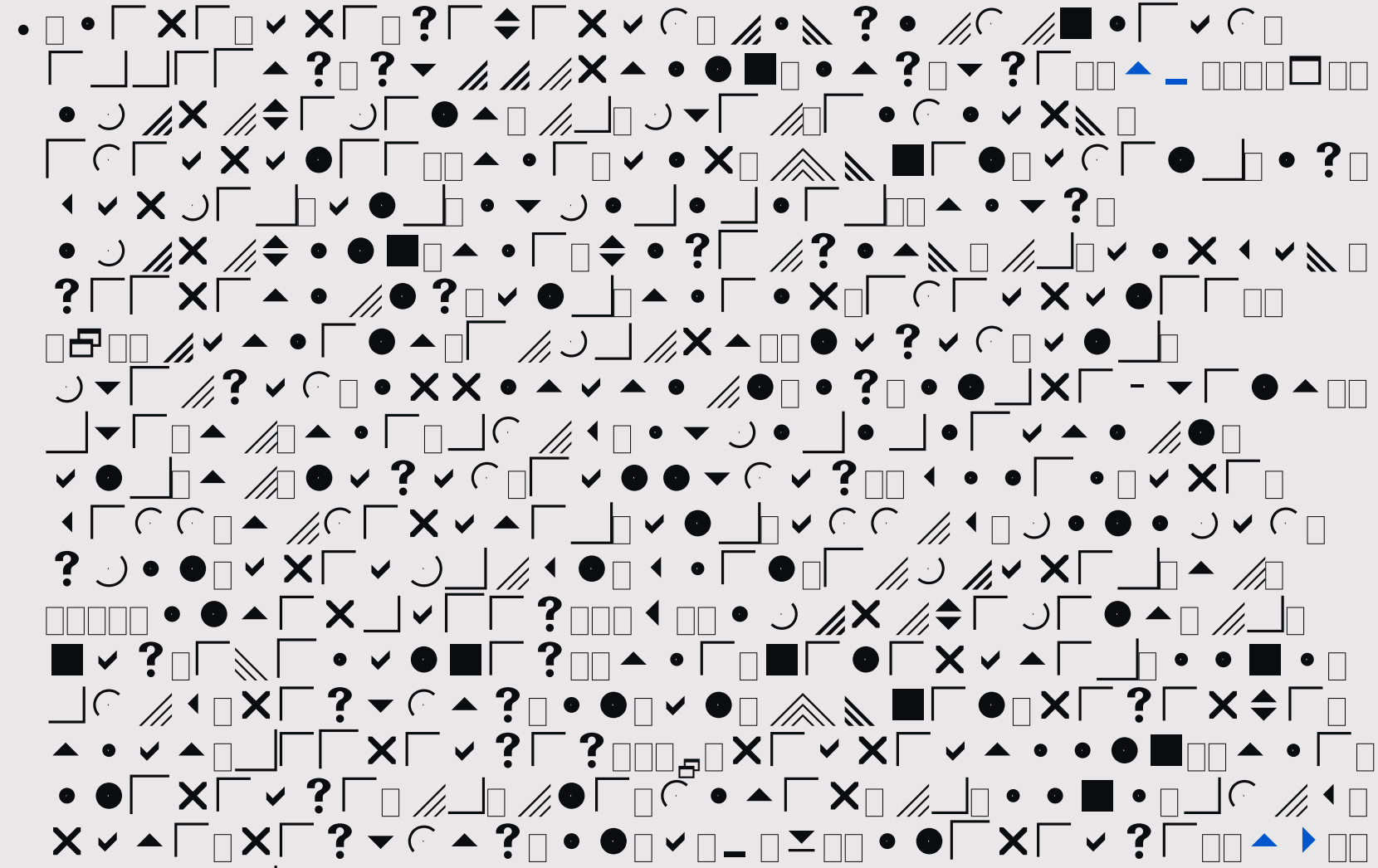
Strict Time Limit

Re-evaluate within 1–2 hours; intubate immediately if dyspnea, acidosis, or exhaustion progresses.





(3) improvement of gas exchanges
(4) work of breathing reduction
(5) PEEP effect



Mechanical Ventilation

Mechanical Ventilation

The decision to **intubate** should be based mainly on **clinical judgment**.

CLINICAL ASSESSMENT

Indications for Endotracheal Intubation

Clinical Arrest

Respiratory arrest or coma.

Profound muscle exhaustion.

Mental Status Change

Agitation or lethargy.

Inability to protect airway.

Refractory Failure

Worsening severe acidosis.

Hypoxemia despite oxygen.

Mechanical Ventilation

The **goals** of **mechanical ventilation** during an acute asthma exacerbation are to

- maintain adequate **oxygenation**,
- reduce the **work of breathing**, and
- prevent **barotrauma due to airtrapping**,

while waiting for bronchodilator and glucocorticoid medications to reverse the airway edema, inflammation, and bronchoconstriction.

Mechanical Ventilation

Be patient; the “attack” will “brake.”

Mechanical Ventilation

Invasive mechanical ventilation in acute severe asthma may **be life saving**, but can be associated with **significant morbidity and mortality**.

Williams T, Tuxen D, Scheinkestel C, et al. Risk factors for morbidity in mechanically ventilated patients with acute severe asthma. Am Rev Respir Dis 1992;146(3):607-

AIRWAY MANAGEMENT

High-Risk Intubation Protocol

STEP 1

Fluid Loading

Rapid IV crystalloid bolus to prevent post-intubation collapse.

STEP 2

Induction Agent

Ketamine 1.5–2.0 mg/kg IV for bronchodilating effect.

STEP 3

Paralytic Choice

Rocuronium 1.2 mg/kg IV to achieve rapid relaxation.

STEP 4

Large ETT Sizing

Size 8.0 mm or larger to minimize airway resistance.

PRESCRIPTION

Recommended Initial Ventilator Settings

Ventilator Parameter	Initial Setting	Physiological Rationale
Ventilation Mode	Volume Control (VC)	Guarantees consistent tidal volume delivery despite changing resistance.
Tidal Volume (Vt)	5–7 mL/kg IBW	Minimizes end-inspiratory alveolar overdistension and hyperinflation.
Respiratory Rate (RR)	10–12 breaths/min	Provides sufficient expiratory time window to minimize gas trapping.
Inspiratory Peak Flow	80–100 L/min	Shortens inspiratory time (Ti) to maximize exhalation duration.
Extrinsic PEEP	0–5 cmH2O	Avoids adding baseline pressure to severe pre-existing Auto-PEEP.

VENTILATOR TARGETS

Core Mechanical Ventilation Goals

< 30	> 7.20	1:4
Plateau Pressure (cmH2O)	Arterial pH Limit	Minimum I:E Ratio

⚠ **Clinical Priority:** Avoid lung overdistension and barotrauma over maintaining normal arterial carbon dioxide levels.

Mechanical Ventilation

Assessment of AutoPEEP

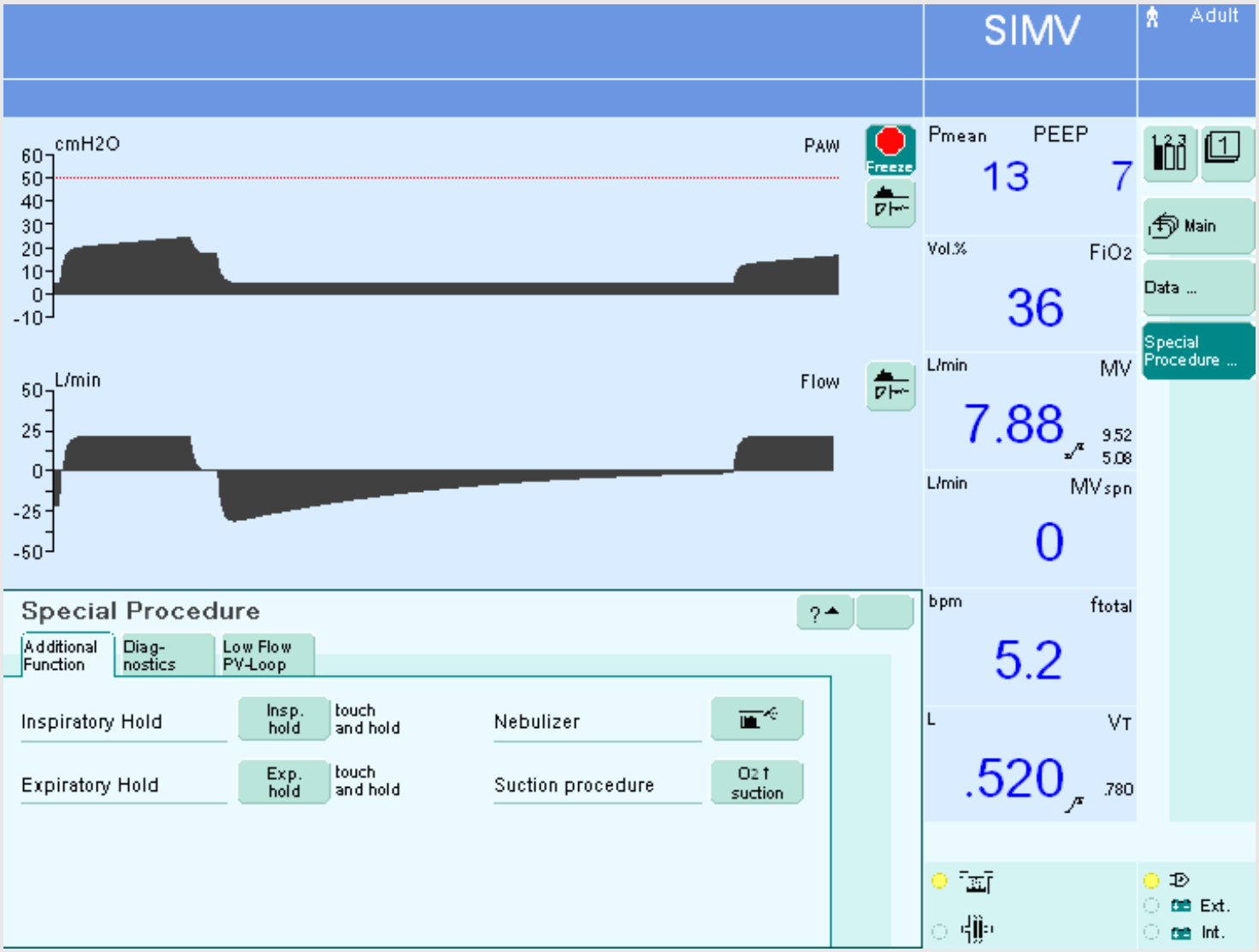
Mechanical Ventilation

Assessment of AutoPEEP

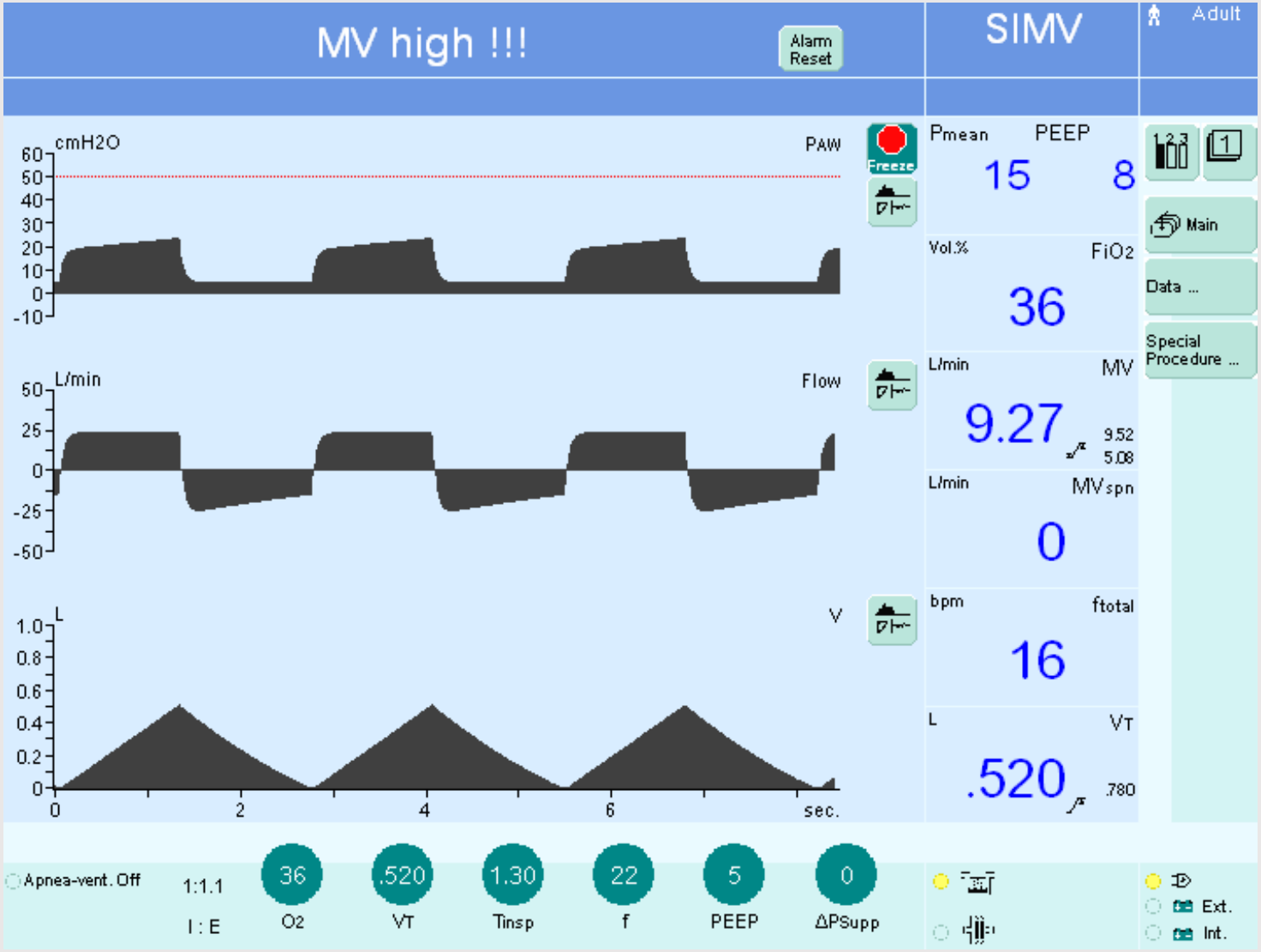
The presence of auto-PEEP can be detected by

- An end expiratory hold valve, enabling the auto-PEEP to be measured directly.
- Flow Time Curve

Expiratory Hold



AUTO-PEEP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Dynamic Hyperin

AUTO-PEEP

Auto-PEEP is treated by

- Changing the **I:E ratio**, allowing more time for exhalation by:
  - Decreasing rate.
  - Increasing inspiratory flow.
  - Decreasing tidal volume.

$$PaCO_2 = \frac{VCO2 \times 0.863}{VA}$$

$$VA = VE - VD$$

VCO2 = CO2 production

VE = minute (total) ventilation = resp. rate x tidal volume

VD = dead space ventilation = resp. rate x dead space volume

0.863 converts VCO2 and VA units to mm Hg

$$\uparrow PaCO_2 = \frac{\uparrow VCO2 \times 0.863}{VA = VE - VD}$$

Increased CO2 production but not able to hyperventilate:

Fever
Sepsis
Hyperthyroidism
Overfeeding with carbohydrates
Agitation

$$\uparrow PaCO_2 = \frac{\leftrightarrow VCO2 \times 0.863}{\downarrow VA = \downarrow VE - VD}$$

Decreased Alveolar Ventilation due to Decreased Minute Ventilation (VE= ↓VT X ↓RR)

Sedative drug overdose

Respiratory muscle paralysis or fatigue

Central hypoventilation

$$\uparrow PaCO_2 = \frac{\leftrightarrow VCO2 \times 0.863}{\downarrow VA = VE - \uparrow VD}$$

Decreased Alveolar Ventilation due to Increased Dead Space Ventilation (VD= Dead Space Volume X RR)

High PEEP or Autopeep
Pulmonary hypertension
Pulmonary embolism

VENTILATOR STRATEGY

Permissive Hypercapnia Protocol

Core Rationale

Accept high arterial $PaCO_2$ to avoid dynamic hyperinflation and barotrauma.

Safe Limits

Target pH ≥ 7.20 with slow buffering via sodium bicarbonate if required.

Contraindications

Avoid in acute intracranial hypertension or severe pulmonary hypertension.

HEMODYNAMIC & MECHANICS MONITORING

Peak vs. Plateau Airway Pressures

Peak Pressure (Ppeak)

Flow Dependent

Reflects airway resistance plus alveolar elasticity.

Frequently elevated (> 45 cmH2O) due to high bronchospasm flow resistance.

High Ppeak alone does not indicate alveolar barotrauma.

Plateau Pressure (Pplat)

Alveolar Target

Measured during end-inspiratory pause maneuver.

Reflects true end-inspiratory alveolar distension pressure.

Must maintain strictly below 30 cmH2O.

SEDATION STRATEGY

Sedation & Analgosedation in MV Asthma

Ketamine Infusion

First-line sedative providing direct smooth muscle bronchodilation and analgesia.

Propofol Titration

Rapid drive suppression and bronchodilation; monitor for hypotensive response.

Fentanyl Analgosedation

Preferred opioid without histamine release; strictly avoid morphine.

Neuromuscular Blockade

Cisatracurium for refractory dyssynchrony; limit to < 48h to prevent acute myopathy.

Complications of Mechanical Ventilation in Asthma

Complications of Mechanical Ventilation in Asthma

Hypotension

Sedation

AutoPEEP

Pneumothorax

Arrhythmias

Tuxen D , Lane S. The effect of ventilatory pattern on hyperinflation, airway pressures, and circulation in mechanical ventilation of patients with sever airflow obstruction. Am Rev Respir Dis1987;126:166-9.

ICU EMERGENCY PROTOCOL

Management of the Crashing Ventilated Asthmatic

ACTION 1

Disconnect Vent

Immediately remove circuit to allow prolonged passive exhalation.

ACTION 2

Manual Compression

Perform manual chest compression ("Status Asthmaticus Hug").

ACTION 3

IV Fluid Bolus

Administer rapid IV fluid volume to restore right atrial preload.

ACTION 4

Rule Out Pneumothorax

Perform urgent needle decompression for suspected tension pneumothorax.

ICU COMPLICATIONS

Acute Steroid-Paralytic Myopathy (AQMS)

Synergistic Toxicity

High-dose IV steroids combined with continuous NMBAs cause thick-filament (myosin) loss.

Clinical Features

Diffuse flaccid weakness, decreased deep tendon reflexes, and failure to wean from ventilator.

NMBA Duration Limit

Strictly limit neuromuscular blockade to < 24–48 hours; discontinue as dyssynchrony resolves.

Monitoring Protocol

Utilize Train-of-Four (TOF) peripheral nerve stimulation to minimize NMBA exposure.

Complications of Mechanical Ventilation in Asthma

Recovery can be slow (takes weeks to months).

The role of muscle relaxants and corticosteroid. Chest 1999;115(6):1627-31

Complications of Mechanical Ventilation in Asthma

Acute Steroid Paralytic Myopathy management include

- Minimise the dose of parenteral corticosteroids.
- Use nebulised agents.
- Minimise deep sedation.
- Avoid NMBA.
- Physical Therapy

The role of muscle relaxants and corticosteroid. Chest 1999;115(6):1627-31


THANK YOU