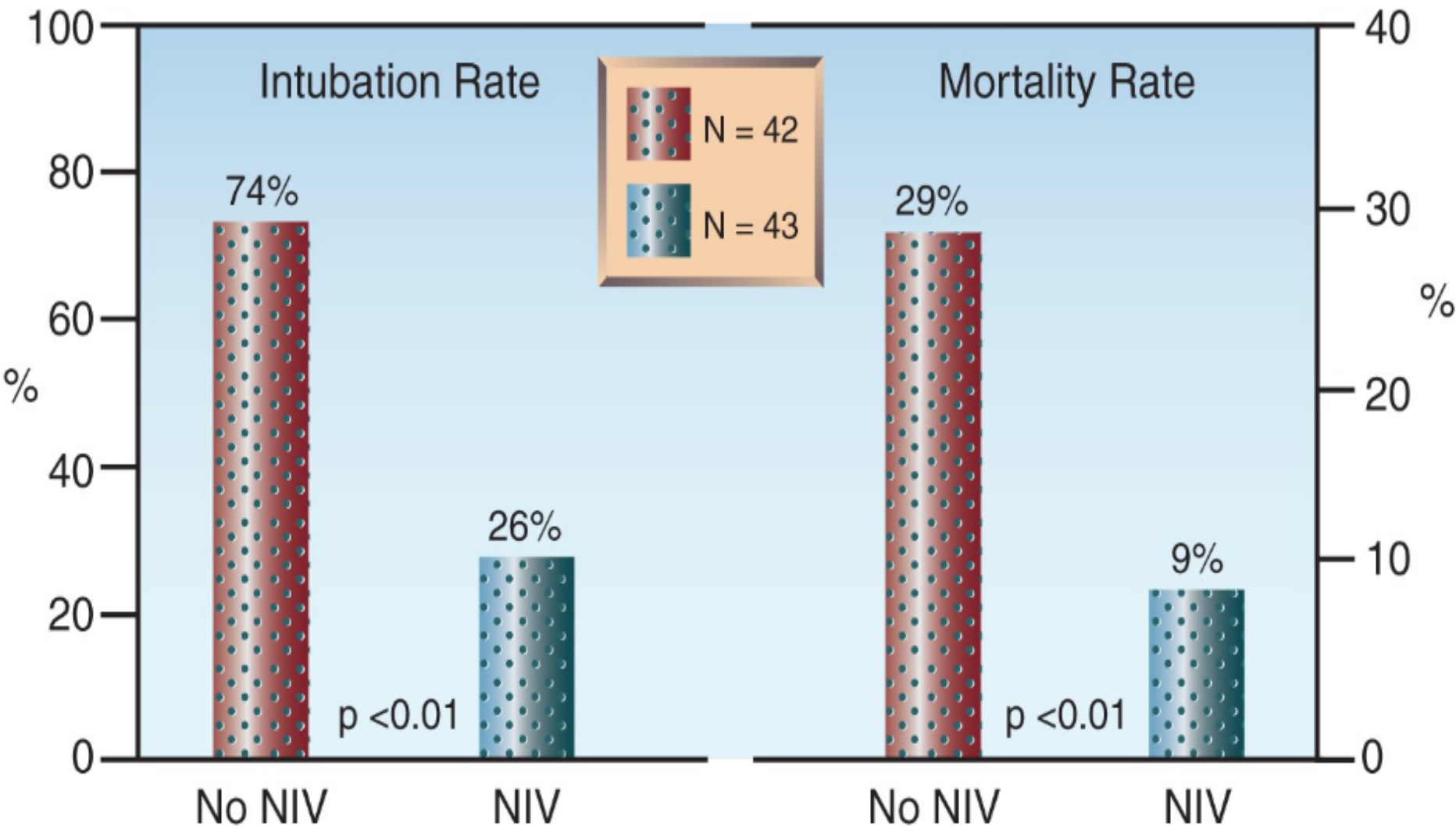


# NIV IN CORSO DI BPCO RIACUTIZZATA

Dr Livio Colombo

Pronto Soccorso Ospedale San Paolo,  
Milano





Lung Failure

Pump Failure



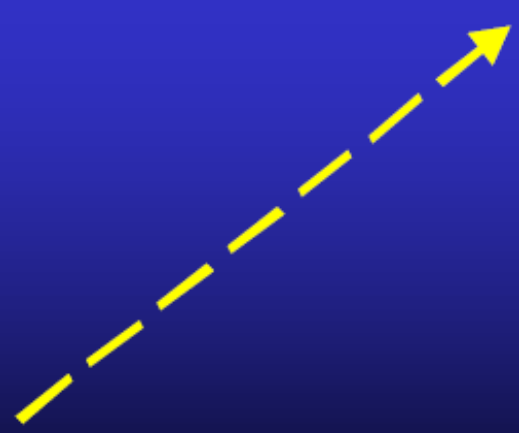
$\downarrow PaO_2$



$\uparrow PaCO_2$



$\downarrow PaCO_2$



$\downarrow PaO_2$

# Respiratory Failure

**Lung Failure**

**Gas Exchange Failure**  
↓ O<sub>2</sub>

**Pump Failure**

**Ventilatory Failure**  
↑ CO<sub>2</sub>

**Central Defect**

**Opioid Overdose**

**Mechanical Defect**

**Neuromuscular disease**

**Fatigue**

**COPD  
Asthma**

Ventilazione polmonare totale = volume corrente X frequenza respiratoria

$$\dot{V}_E = V_T \times FR$$

$$5-10 \text{ l/min} = 500 \text{ cc} \times 8-12 \text{ atti/min}$$

Volume corrente = volume alveolare + volume spazio morto

$$V_T = V_A + V_D$$

$$500 \text{ cc} = 350 \text{ cc} + 150 \text{ cc}$$

Ventilazione Alveolare = ventilaz polmonare totale - ventilaz spazio morto

$$\dot{V}_A = \dot{V}_E - \dot{V}_D = (V_T - V_D) \times FR$$

$$3.5 \text{ l/min} = 350 \text{ cc} \times 8-12 \text{ atti/min}$$

## Aumento CO<sub>2</sub> -Ipoventilazione alveolare

- Il Volume di gas fresco che arriva agli alveoli nell'unità di tempo (Ventilazione alveolare – Va) è diminuito
- Provoca sempre un aumento della pCO<sub>2</sub>

$$pCO_2 = (VCO_2/Va) \times k \quad \left\{ \begin{array}{l} VCO_2 = \text{quantità di CO}_2 \text{ eliminata nell'unità di tempo} \\ Va = \text{Ventilazione alveolare} = (Vt - Vd) \times FR \end{array} \right.$$



**Va e pCO<sub>2</sub> sono inversamente proporzionali**

# Riduzione O<sub>2</sub> -Alterazioni rapporto ventilazione/perfusione



$\dot{V}/\dot{Q}$  normale



EPA  
Polmonite

Asma  
BPCO

$\dot{V}/\dot{Q}$  ridotto



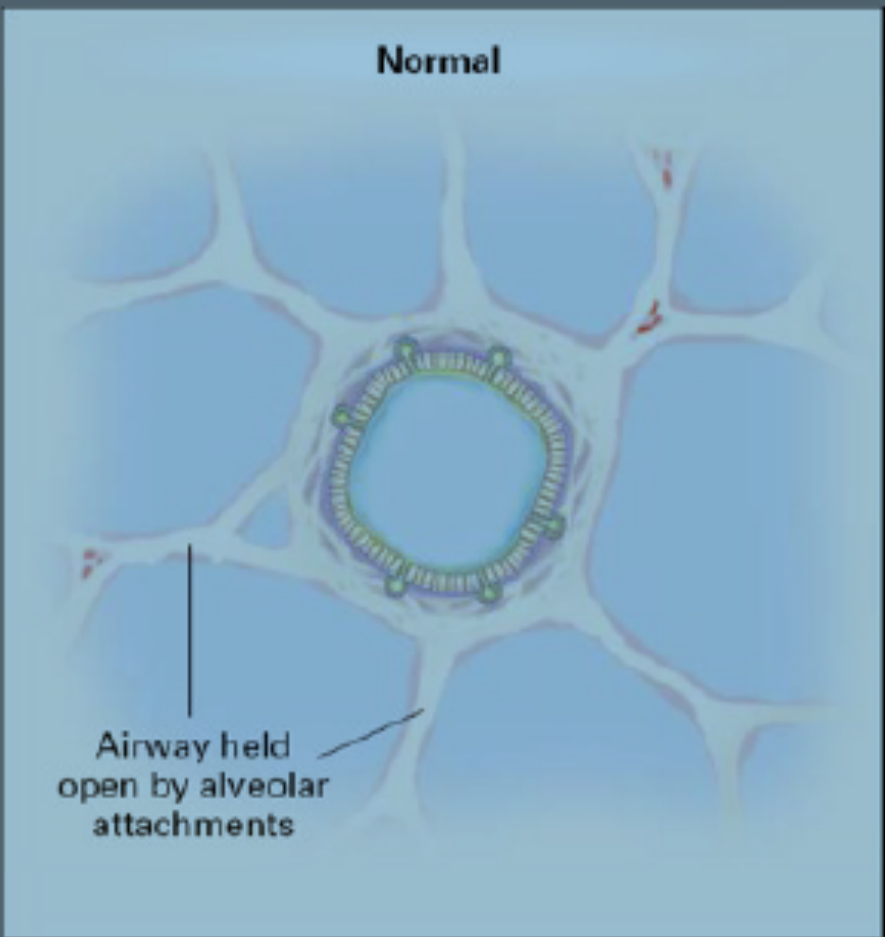
Enfisema

TEP

$\dot{V}/\dot{Q}$  aumentato

# Alterazioni morfologiche delle vie aeree in corso di BPCO

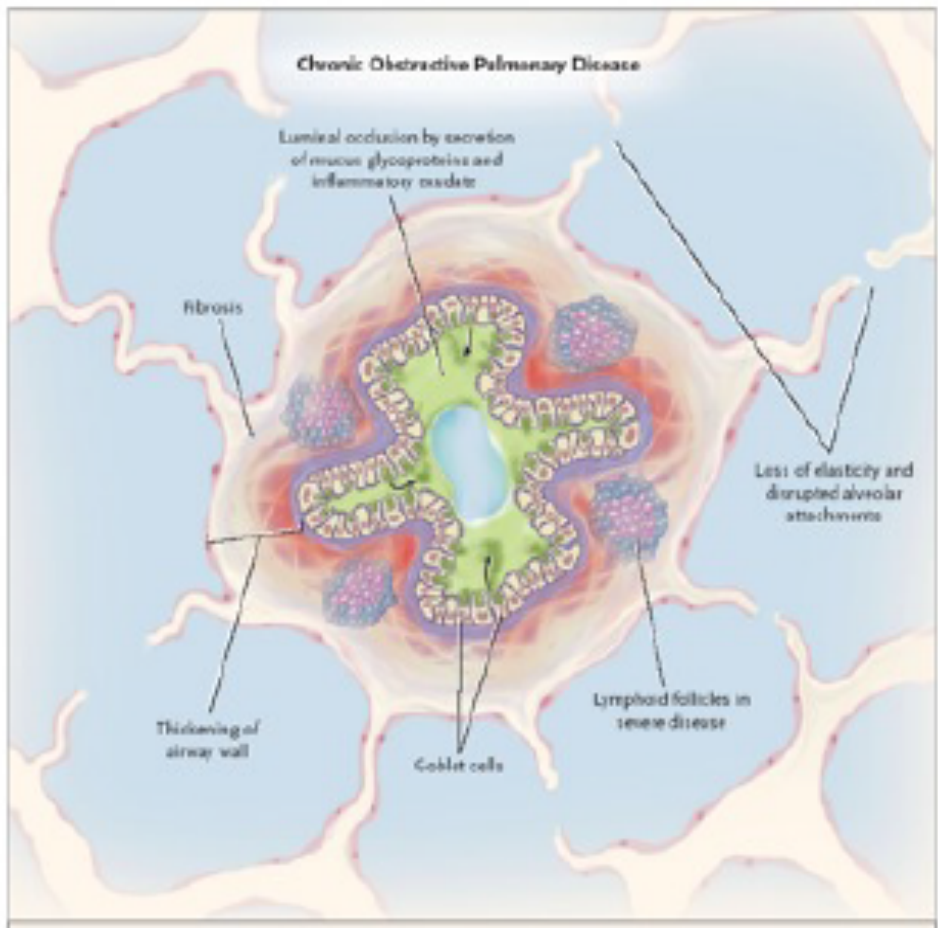
Normal



Airway held open by alveolar attachments

This diagram shows a normal airway cross-section. The airway is circular and surrounded by a thin wall. It is held open by numerous alveolar attachments, which are represented by small green dots along the inner surface of the airway wall. The surrounding lung tissue is light blue and has a porous, alveolar appearance.

Chronic Obstructive Pulmonary Disease



Luminal occlusion by secretion of mucus, glycoproteins and inflammatory exudate

Fibrosis

Less of elasticity and disrupted alveolar attachments

Thickening of airway wall

Goblet cells

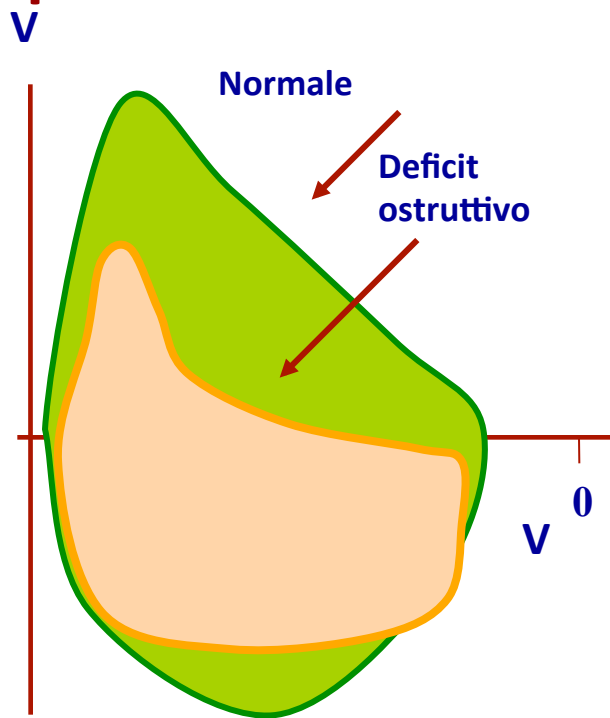
Lymphoid follicles in severe disease

This diagram illustrates the morphological changes in an airway affected by COPD. The airway is significantly narrowed and distorted. The lumen is partially blocked by a thick, irregular mass of mucus, glycoproteins, and inflammatory exudate. The airway wall is markedly thickened and contains numerous goblet cells, which are shown as purple clusters. There is also evidence of fibrosis, indicated by the dense, pinkish-red staining of the surrounding tissue. The alveolar attachments are disrupted, and the overall lung tissue appears less elastic and more scarred. Lymphoid follicles are visible in the severe disease stage.



# Curva flusso-volume

## • Curva flusso-volume espiratoria normale e in un paziente ostruito



Nel paziente con ostruzione bronchiale si rileva una riduzione dei flussi a tutti i volumi polmonari con riduzione del PEF e concavità verso l'alto della curva espiratoria (freccia).

# CHIUSURA DINAMICA VIE AEREE

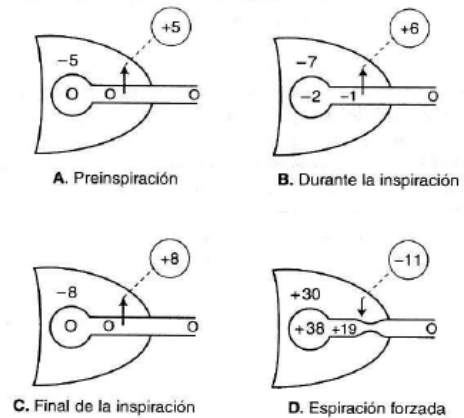
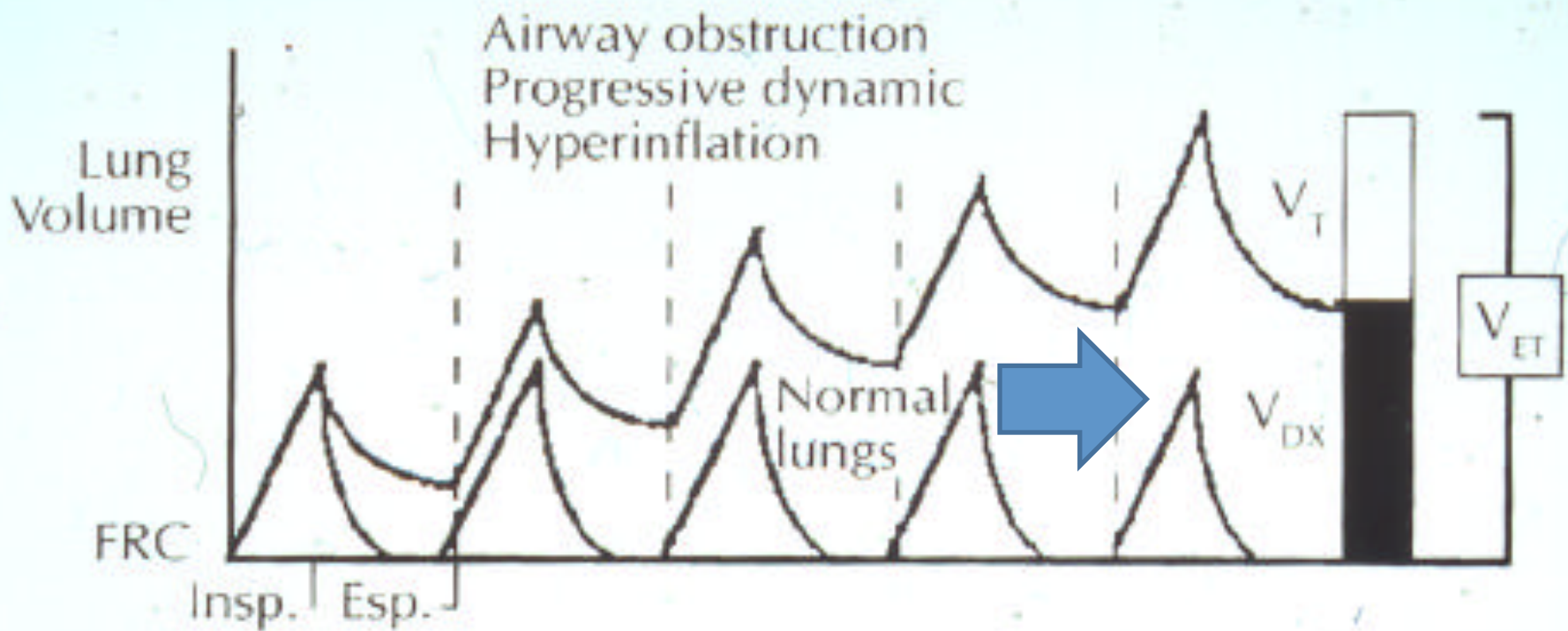


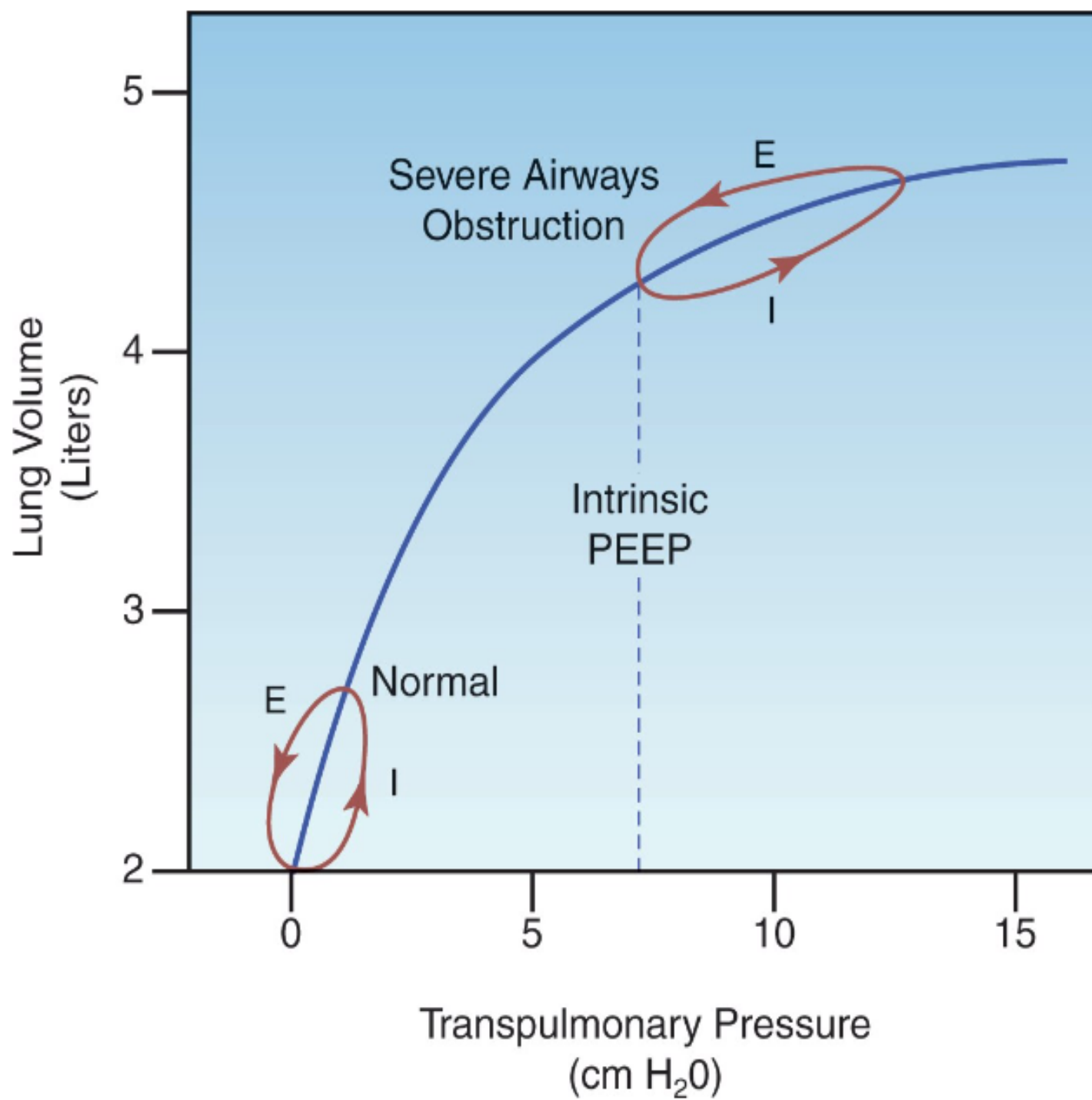
Fig. 7-18. Esquema que demuestra por qué las vías aéreas se comprimen durante la espiración forzada. La presión a través de la vía aérea mantiene abierta a ésta, salvo durante la espiración forzada. Véanse los detalles en el texto.

## Compresión dinámica de las vías aéreas

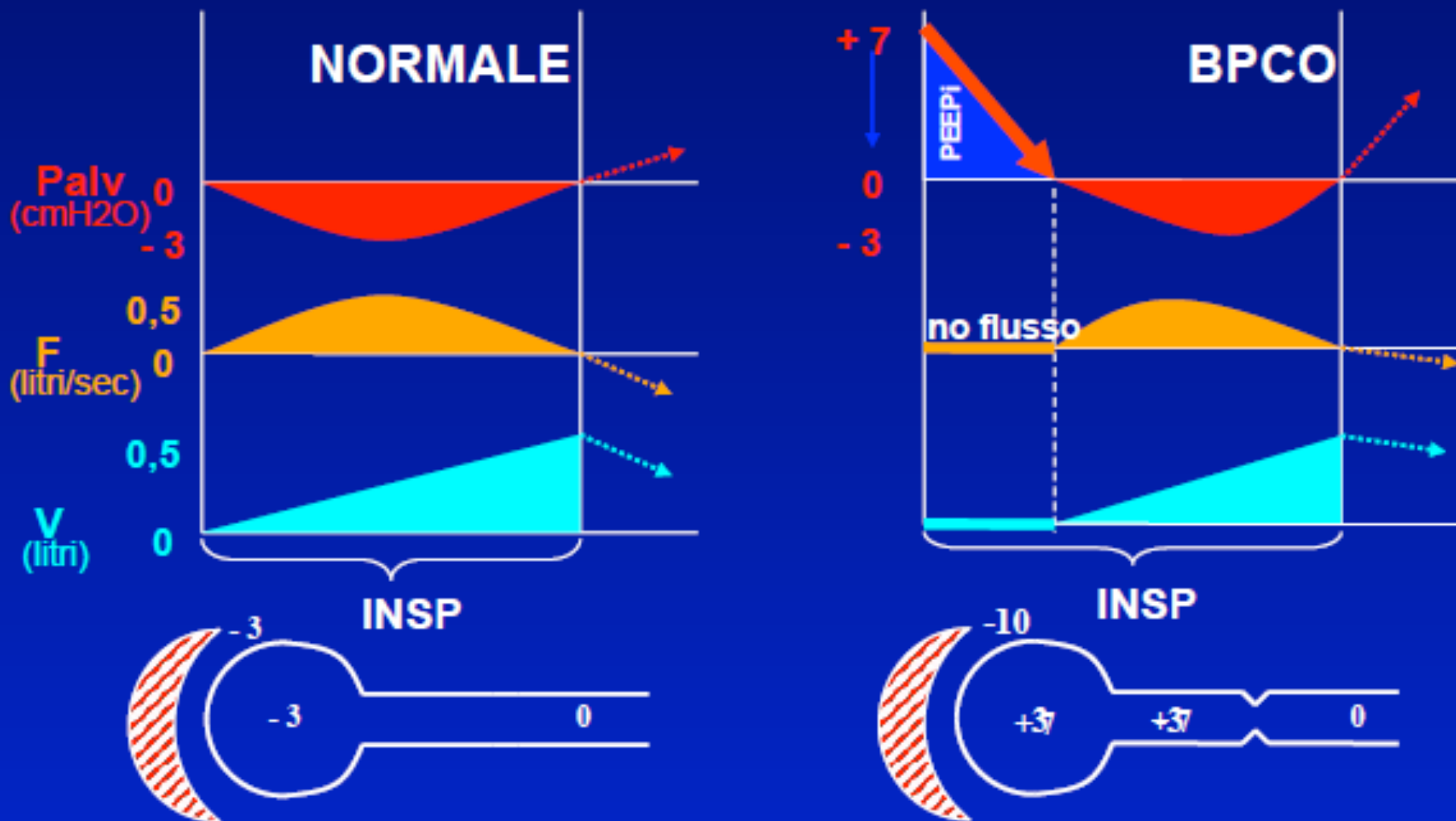
- Limita el flujo aéreo en personas normales durante la espiración forzada
- Puede aparecer en las neumopatías con flujos espiratorios relativamente bajos, reduciendo la capacidad de realizar ejercicio
- Durante la compresión dinámica el flujo se halla determinado por la presión alveolar menos la presión pleural (no la presión en la boca)
- Está exagerada en algunas enfermedades pulmonares por la disminución del retroceso elástico pulmonar y la pérdida de la tracción radial en las vías aéreas



**PEEP INTRINSECA (occulta o autoPEEP):**  
 per iperinflazione dinamica la fase esp  
 non si completa prima della successiva  
 insp >  $\uparrow$  volume intrappolato >  $\uparrow$  CFR >  
 modifica della curva volume/pressione

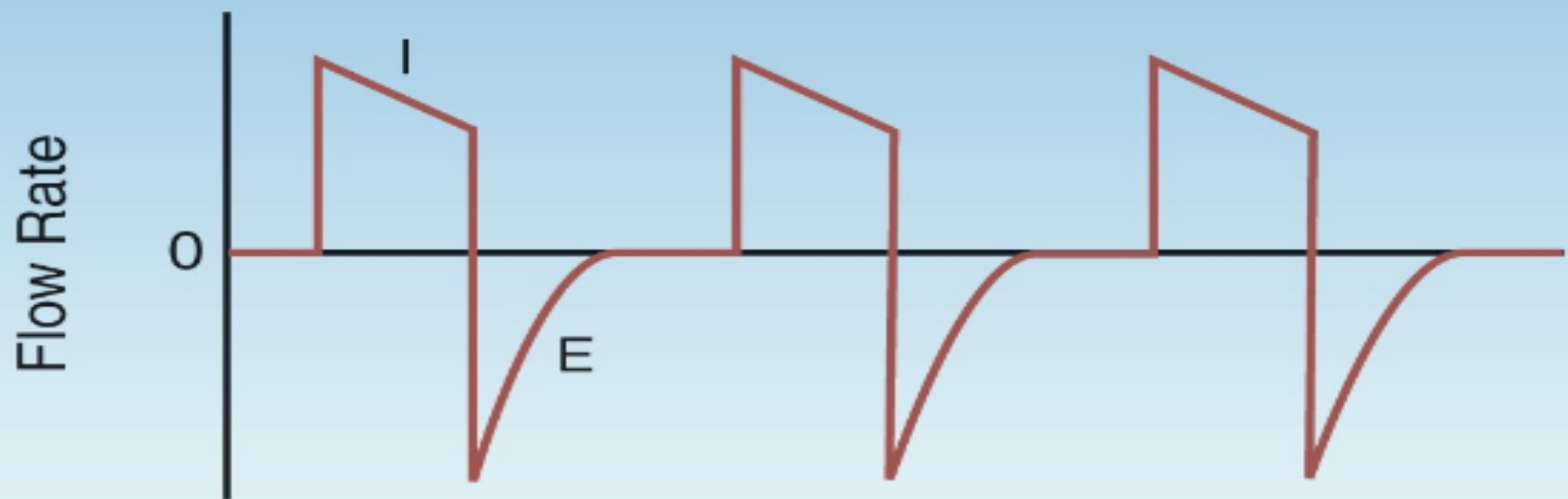


# Inspirazione e PEEPi

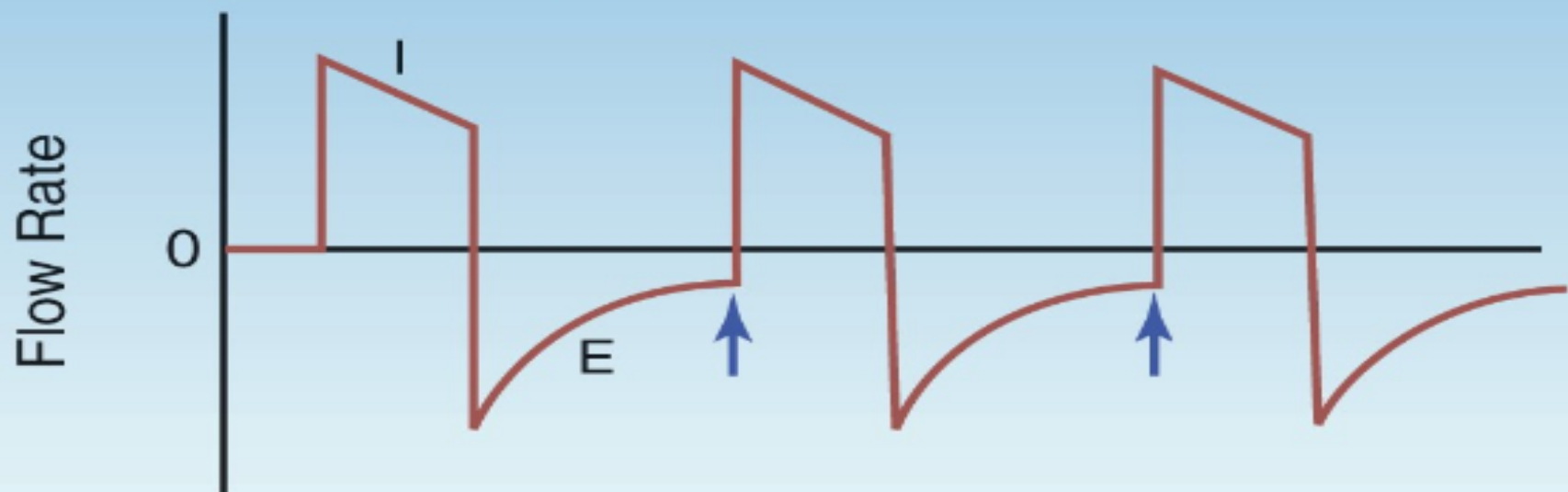


La PEEP intrinseca agisce come un carico soglia meccanico che i muscoli inspiratori devono superare prima di poter generare flusso inspiratorio, ad ogni ciclo respiratorio.

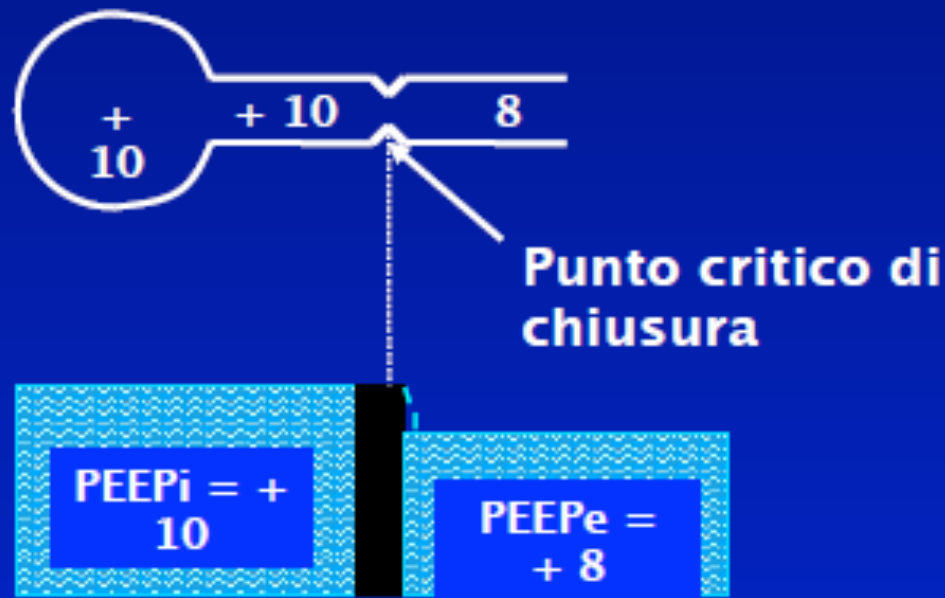
### Normal



### Dynamic Hyperinflation



# PEEP intrinseca + PEEP estrinseca



## Riduzione del WOB inspiratorio

Lavoro elastico (isometrico): CPAP (PEEPe) → controbilanciare la PEEPi

# LAVORO RESPIRATORIO

INSPIRATORIO

ESPIRATORIO

**DINAMICO**, resistivo ed isotonico

RESISTENZE AL FLUSSO DI ARIA NELLE  
VIE AEREE

**ELASTICO**, statico ed isometrico

RESISTENZE ELASTICHE DEL SISTEMA  
TORACO-POLMONARE



# L'aumento del Lavoro Respiratorio nella BPCO

Lavoro Inspiratorio Dinamico  
(resistivo ed isotonic)

Lavoro Inspiratorio Elastico  
(statico ed isometrico)

Lavoro Espiratorio Dinamico  
(resistivo ed isotonic)

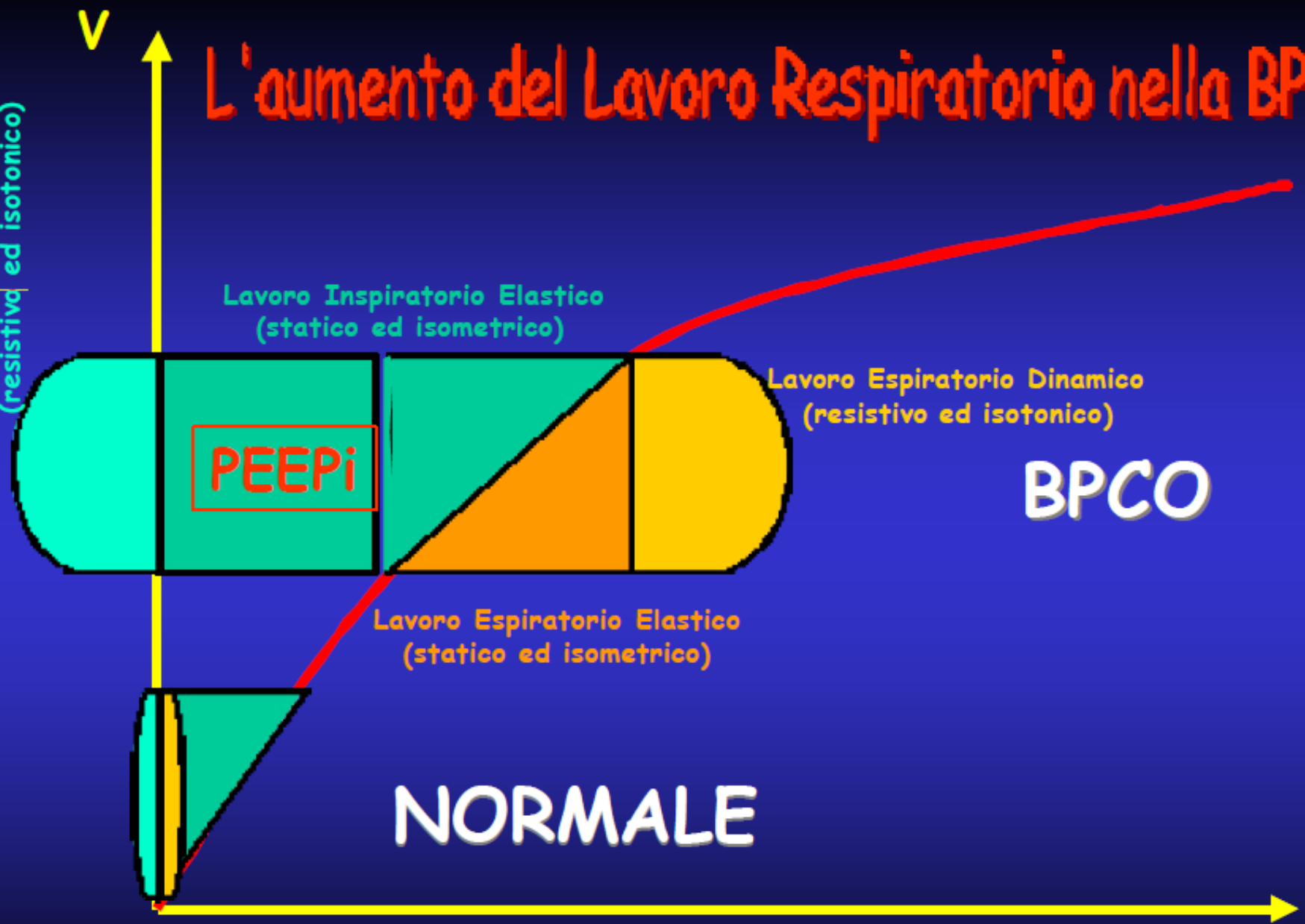
PEEPi

BPCO

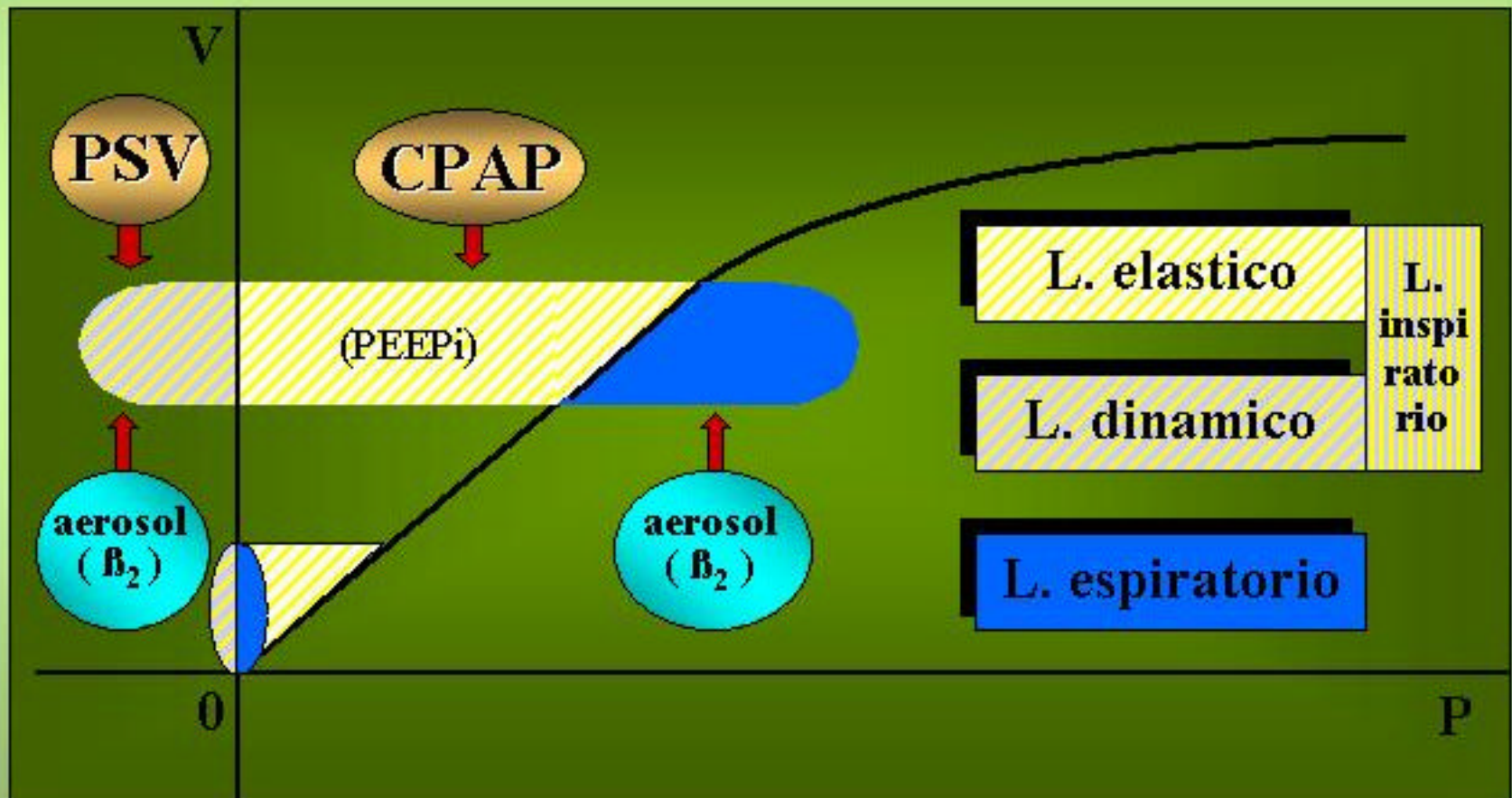
Lavoro Espiratorio Elastico  
(statico ed isometrico)

NORMALE

P



# Lavoro respiratorio nel pz. BPCO e nel soggetto sano: effetti della NIMV



# Criteria di selezione per l'impiego della NIV

## Indications

### Bedside observations

- Increased dyspnoea—moderate to severe
- Tachypnoea (>24 breaths per min in obstructive, >30 per min in restrictive)
- Signs of increased work of breathing, accessory muscle use, and abdominal paradox

### Gas exchange

- Acute or acute on chronic ventilatory failure (best indication),  $\text{PaCO}_2 > 45$  mm Hg,  $\text{pH} < 7.35$
- Hypoxaemia (use with caution),  $\text{PaO}_2/\text{F}_i\text{O}_2$  ratio  $< 200$

# Il pH

## Emogasanalisi

**pH < 7,20**

**NIV molto fortemente indicata**

- Anche con NIV 50% → intubazione.
- Se NIV applicata con successo migliora mortalità intraospedaliera e ad 1 anno



**UTI, semi intensiva**

**pH < 7,30**

**NIV fortemente indicata**

Senza NIV 50 %  
peggiora sino ad  
intubazione o morte

**pH < 7,35**

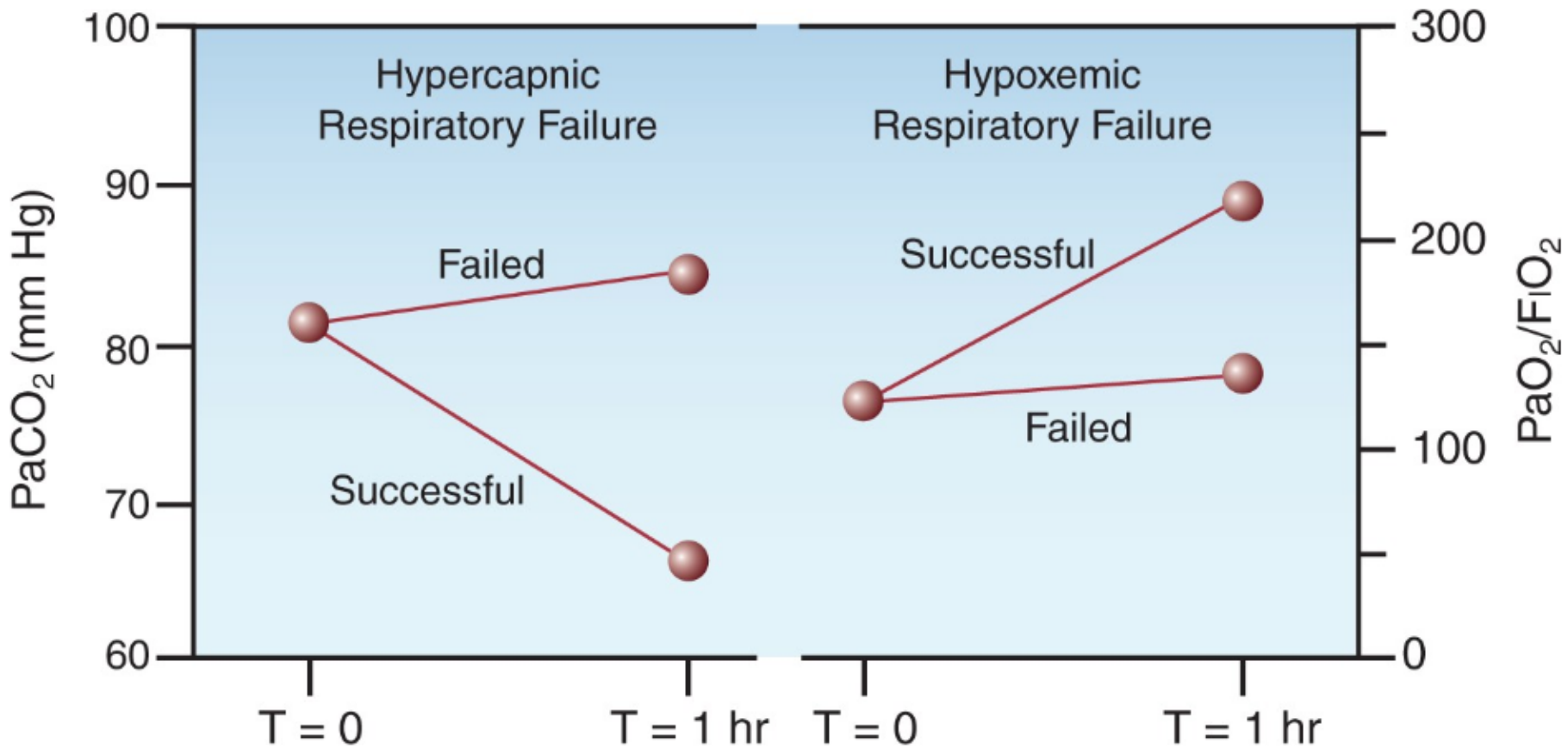
**NIV indicata**

- 80% migliora con terapia medica senza NIV
- Per 10 pz trattati evitata 1 intubazione
- Più rapida risoluzione dispnea



**Reparto pneumologia, medicina generale**

**- A condizione che: intubazione urgente disponibile;  
capacità personale gestione ventilatori**

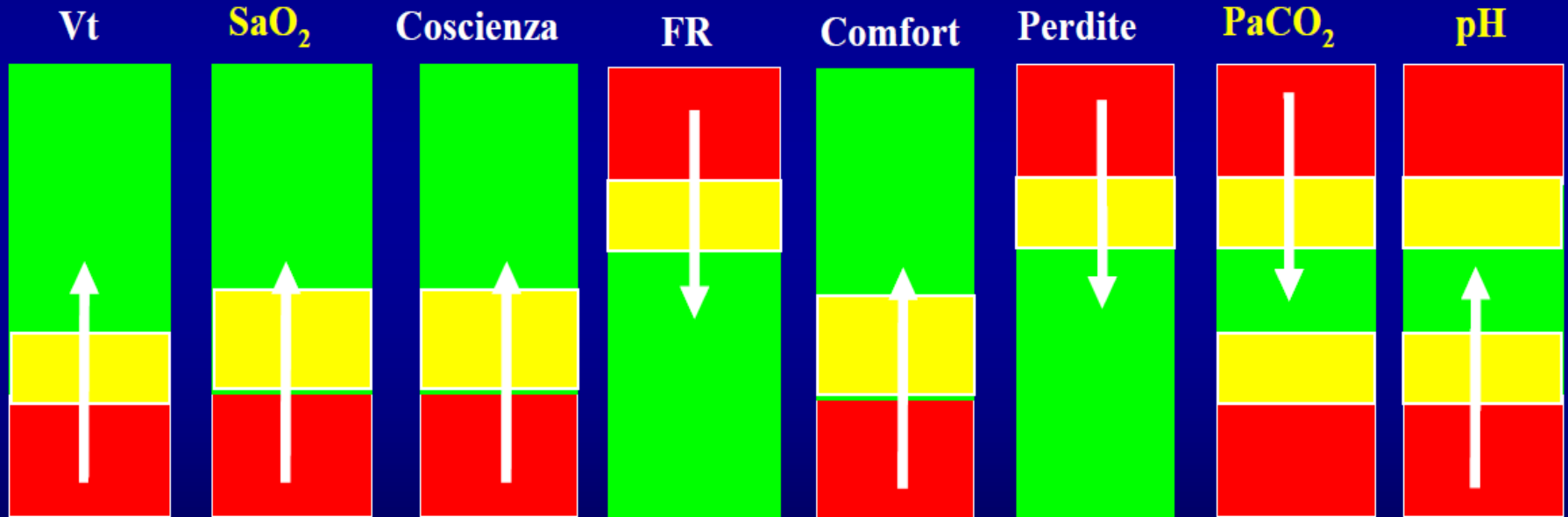


Antonelli et al. Int Care  
Med 2001; 27:1718-1728

# GLI OBIETTIVI

- Riduzione della dispnea
- Incremento del *comfort* del respiro
- Miglioramento della vigilanza
- Riduzione FR (20%)
  
- Incremento  $V_T$  (5-7 mL/kg)
  
- SpO<sub>2</sub> 90-92%
- Incremento pH (< 7,35)
- Riduzione pCO<sub>2</sub> (15-20%)

Individua degli indicatori le cui variazioni permettono di valutare il raggiungimento o meno degli obiettivi prefissati



Movimento paradossico dell'addome, utilizzo muscolatura accessoria, P/F, PA, FC..

## Where to perform noninvasive ventilation?

M.W. Elliott\*, M. Confalonieri<sup>#</sup>, S. Nava<sup>¶</sup>

The ideal location for noninvasive positive-pressure ventilation will vary from country to country and indeed from hospital to hospital, depending upon local factors. However, **the most important factor is that staff be adequately trained** in the technique and be available throughout the 24-h period.

*Eur Respir J* 2002; 19: 1159–1166.



# Complicanze

## Iperdistensione alveolare

- $\uparrow V_D$  alveolare
- $\downarrow$  compliance polmonare
- $\uparrow$  lavoro respiratorio

## Ipotensione

- $\uparrow$  Pressione intratoracica
- $\downarrow$  precarico
- Effetti emodinamici negativi sulla gittata cardiaca

## Barotrauma

- Pneumotorace, pneumomediastino  
con pressioni  $> 25$  cm  $H_2O$

# Fallimento terapeutico

## FATTORI DI RISCHIO

Risk stratification of NPPV failure was assessed in 1,033 consecutive patients admitted to experienced hospital units, including two intensive care units, six respiratory intermediate care units, and five general wards. NPPV was successful in 797 patients.

		pH admission <7.25		pH admission 7.25–7.29		pH admission >7.30	
RR		APACHE ≥29	APACHE <29	APACHE ≥29	APACHE <29	APACHE ≥29	APACHE <29
GCS 15	<30	29	11	18	6	17	6
	30–34	42	18	29	11	27	10
	≥35	52	24	37	15	35	14
GCS 12–14	<30	48	22	33	13	32	12
	30–34	63	34	48	22	46	21
	≥35	71	42	57	29	55	27
GCS ≤11	<30	64	35	49	23	47	21
	30–34	76	49	64	35	62	33
	≥35	82	59	72	44	70	42

■: 0–24%; ■: 25–49%; ■: 50–74%; ■: 75–100%.

Eur Respir J 2005; 25: 348–355

A chart of failure risk for noninvasive ventilation in patients with COPD exacerbation

M. Confalonieri<sup>1</sup>, G. Garuti<sup>2</sup>, M.S. Cattaruzza<sup>3</sup>, J.F. Osborn<sup>4</sup>, M. Antonelli<sup>5</sup>, G. Conti<sup>6</sup>, M. Kodric<sup>7</sup>, O. Resta<sup>8</sup>, S. Marchese<sup>9</sup>, C. Gregoretti<sup>10</sup> and A. Rossi, on behalf of the Italian noninvasive positive pressure ventilation (NPPV) study group<sup>11</sup>

# After two hours

		pH after 2 h <7.25		pH after 2 h 7.25–7.29		pH after 2 h ≥7.30	
RR		APACHE ≥29	APACHE <29	APACHE ≥29	APACHE <29	APACHE ≥29	APACHE <29
GCS 15	<30	72	35	27	7	11	3
	30–34	88	59	49	17	25	7
	≥35	93	73	64	27	38	11
GCS 12–14	<30	84	51	41	13	19	5
	30–34	93	74	65	28	39	12
	≥35	96	84	78	42	54	20
GCS ≤11	<30	93	74	65	28	39	12
	30–34	97	88	83	51	63	26
	≥35	99	93	90	66	76	40

■: 0–24%; ■: 25–49%; ■: 50–74%; ■: 75–100%.

A. Does the patient have:	YES	NO
1. Signs of respiratory distress?	<input checked="" type="checkbox"/>	<input type="checkbox"/>
2. $\text{PaO}_2/\text{FiO}_2 < 200$ and/or $\text{PaCO}_2 > 45$ mm Hg?	<input checked="" type="checkbox"/>	<input type="checkbox"/>
B. If the answer is YES to both, answer the following questions.		
C. Does the patient have:	YES	NO
1. Respiratory failure that is an immediate threat to life?	<input type="checkbox"/>	<input checked="" type="checkbox"/>
2. A life-threatening circulatory disorder (e.g., shock)?	<input type="checkbox"/>	<input checked="" type="checkbox"/>
3. Coma, severe agitation, or uncontrolled seizures?	<input type="checkbox"/>	<input checked="" type="checkbox"/>
4. Inability to protect the airways?	<input type="checkbox"/>	<input checked="" type="checkbox"/>
5. Hematemesis or recurrent vomiting?	<input type="checkbox"/>	<input checked="" type="checkbox"/>
6. Laryngeal edema, facial trauma, or recent head and neck surgery?	<input type="checkbox"/>	<input checked="" type="checkbox"/>
D. If the answer is NO to all of the above questions, the patient is a candidate for noninvasive ventilation.		

## A FINAL WORD

### Don't Forget to Intubate

The rising popularity of noninvasive ventilation tends to overshadow the value of endotracheal intubation. The following simple rules about endotracheal intubation deserve mention.

**Rule 1: Hesitation invites trouble.** There is a tendency to rely on noninvasive ventilation and delay intubation as long as possible in the hopes that it will be unnecessary. However, delays in intubation create unnecessary dangers for the patient because emergency intubations in patients who are *in extremis* can be troublesome as well as dangerous. As soon as intubation becomes a serious consideration, you should intubate the patient and get control of the airway without delay.

**Rule 2: Endotracheal intubation is not the 'kiss of death'.** The perception that “once on a ventilator, always on a ventilator” is a fallacy that should never influence the decision to intubate a patient for full ventilatory support. Being on a ventilator does not create ventilator dependence, having a severe cardiopulmonary or neuromuscular disease does.

Marino's, The ICU Book 4 ed.

