

Chronic Respiratory Failure

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Chronic Respiratory Failure

Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions:

oxygenation

carbon dioxide elimination

Chronic Respiratory Failure

Hypoxemic respiratory failure (type I) is characterized by an arterial oxygen tension (PaO_2) lower than 60 mm Hg with a normal or low arterial carbon dioxide tension (PaCO_2).

Chronic Respiratory Failure

Hypercapnic respiratory failure (type II) is characterized by a PaCO_2 higher than 50 mm Hg. Hypoxemia is common in patients with hypercapnic respiratory failure who are breathing room air.

The respiratory System

Lungs

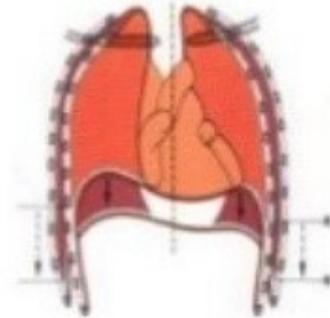


Pulmonary Failure

- PaO₂ ↓↓
- PaCO₂ N/ ↓

Hypoxic
Respiratory
Failure

Respiratory pump



Ventilatory Failure

- PaO₂ ↓
- PaCO₂ ↑↑

Hypercapnic
Respiratory
Failure

Acute vs Chronic Respiratory Failure

Chronic Respiratory Failure

While **acute respiratory failure** is characterized by **life-threatening** derangements in arterial blood gases and acid–base status, the manifestations of **chronic respiratory failure** are more **indolent** and may be clinically **inapparent**.

Chronic Respiratory Failure

Chronic hypercapnic respiratory failure develops over several days or longer, allowing time for renal compensation and an increase in bicarbonate concentration. Therefore, the pH usually is only slightly decreased.

Chronic Respiratory Failure

Acute hypercapnic respiratory failure develops over minutes to hours; therefore, pH is less than 7.3.

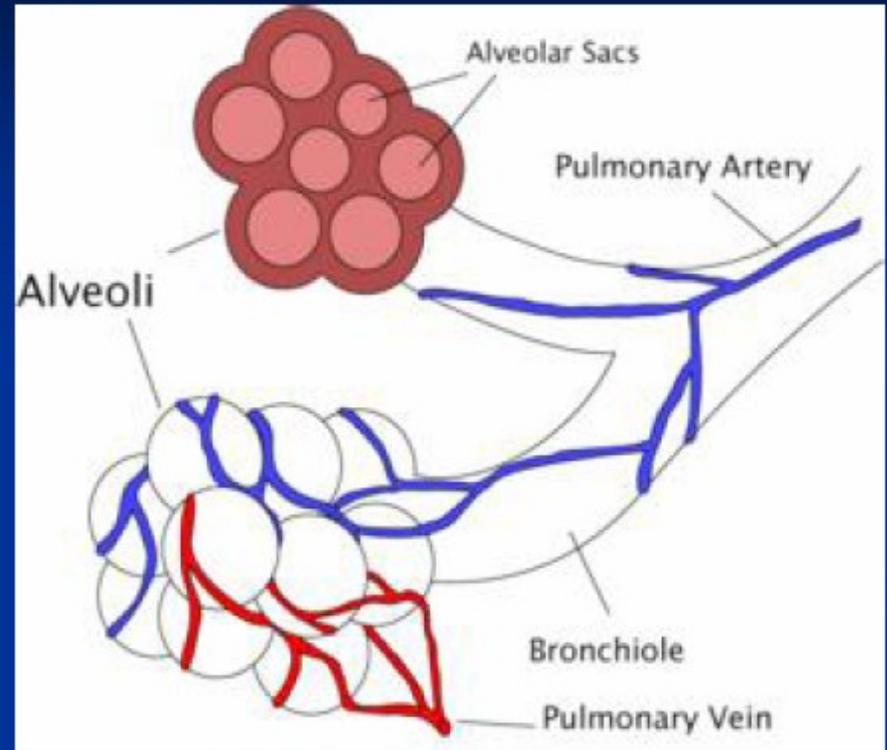
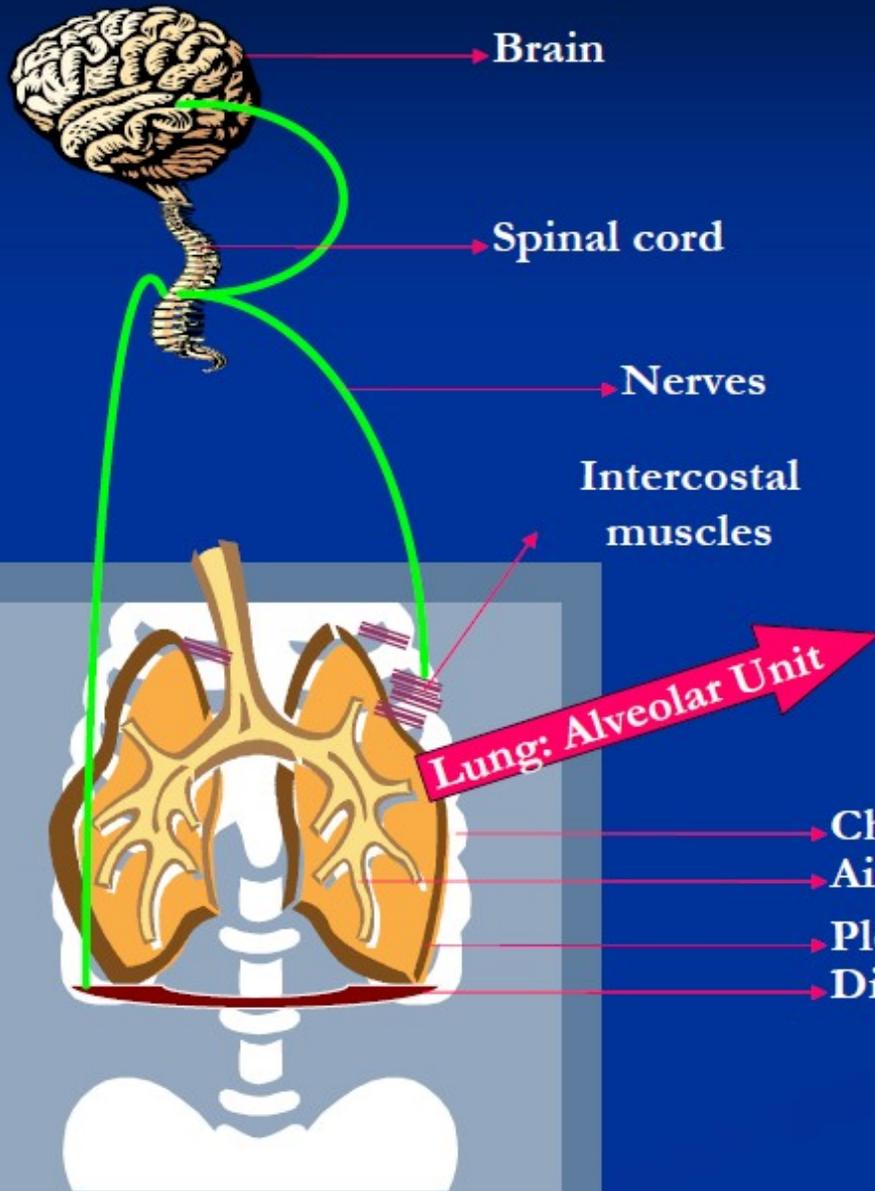
Chronic Respiratory Failure

The clinical markers of chronic hypoxemia, such as **polycythemia** or **cor pulmonale**, suggest a long-standing disorder.

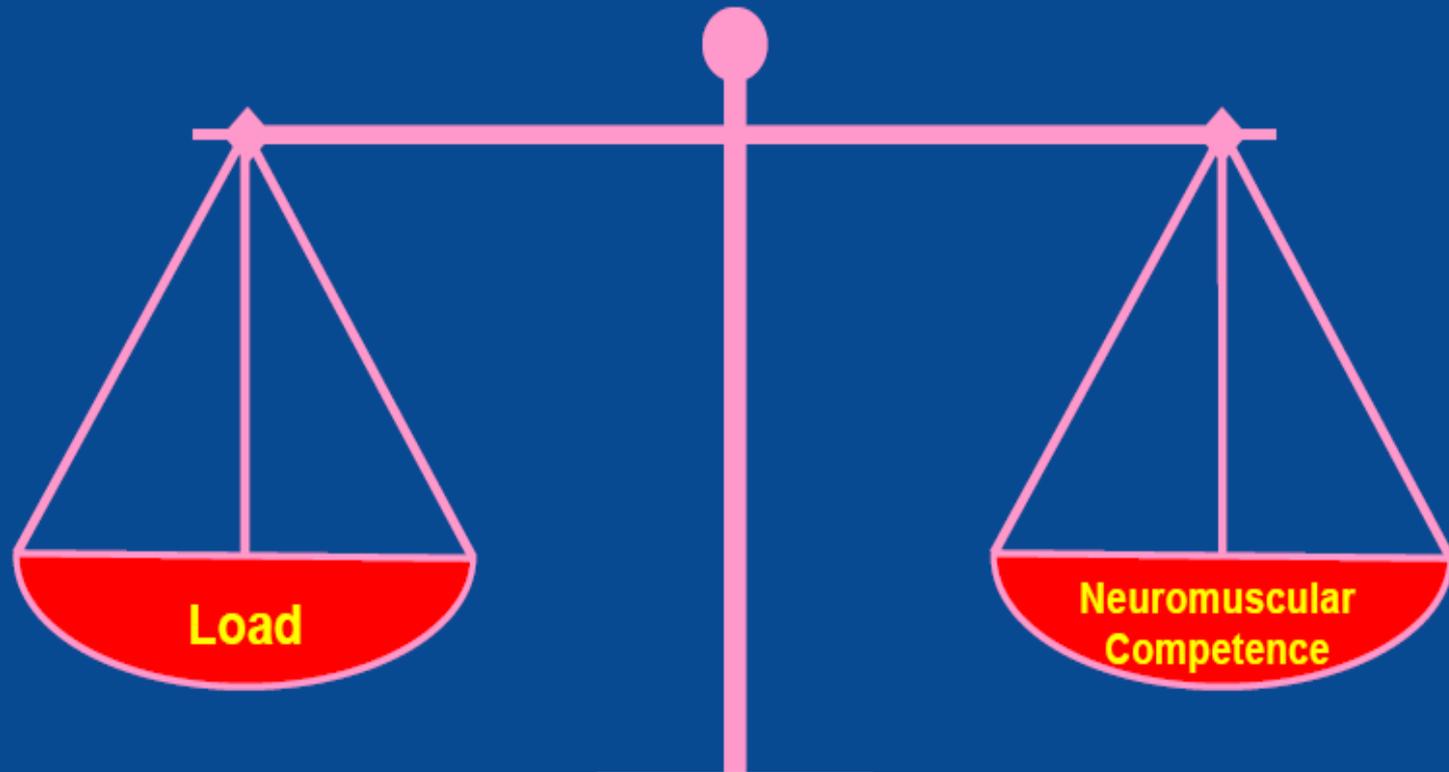
Chronic Respiratory Failure

No specific symptoms are related to chronic hypercapnia, and these patients can tolerate marked elevations in PaCO₂ without experiencing adverse consequences.

Respiratory System



Sustaining Oxygenation and Alveolar Ventilation



The balance between load (resistive, elastic, and minute ventilation) and neuromuscular competence (drive, transmission, and muscle strength)

Chest wall elastic loads

- Abdominal distention
- Ascites
- Obesity
- Pleural effusion
- Pneumothorax
- Rib fracture
- Tumor

Lung elastic loads

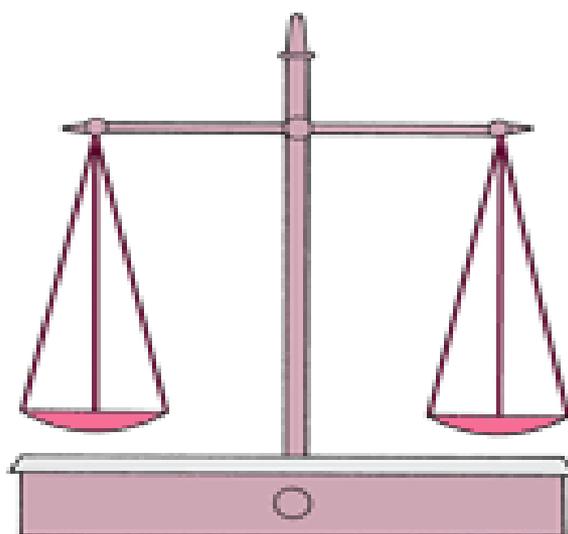
- Alveolar edema
- Atelectasis
- Infection
- Fibrosis

Minute ventilation loads

- Excess calories
- Hypovolemia
- Pulmonary embolus
- Sepsis

Resistive loads

- Bronchospasm (eg, asthma, bronchiolitis, COPD)
- Edema, secretions, or scarring of airway
- Obstructive sleep apnea
- Upper airway obstruction (eg, croup, epiglottitis)



Load

Neuromuscular competence

Impaired respiratory drive

- Brain stem lesion
- Drug overdose
- Hypothyroidism
- Sleep-disordered breathing

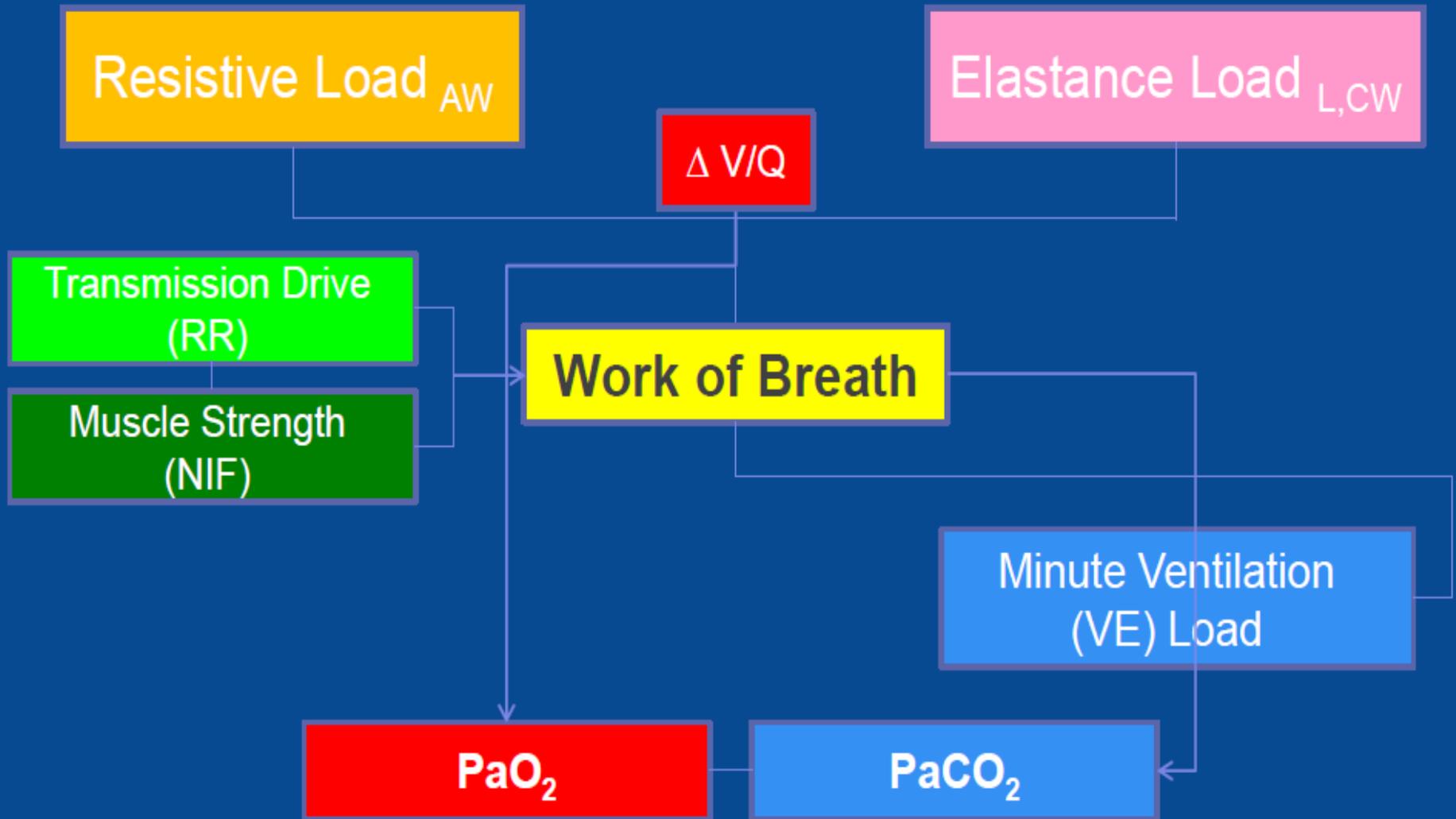
Impaired neurotransmission

- Aminoglycosides
- Amyotrophic lateral sclerosis
- Botulism
- Spinal cord lesion
- Guillain-Barré syndrome
- Myasthenia gravis
- Neuromuscular blockers
- Phrenic nerve injury

Muscle weakness

- Electrolyte abnormalities
- Fatigue
- Hypoperfusion states
- Hypoxemia
- Myopathy
- Undernutrition

Sustaining Oxygenation and Alveolar Ventilation



Respiratory Failure due to ↑ Resistance Load

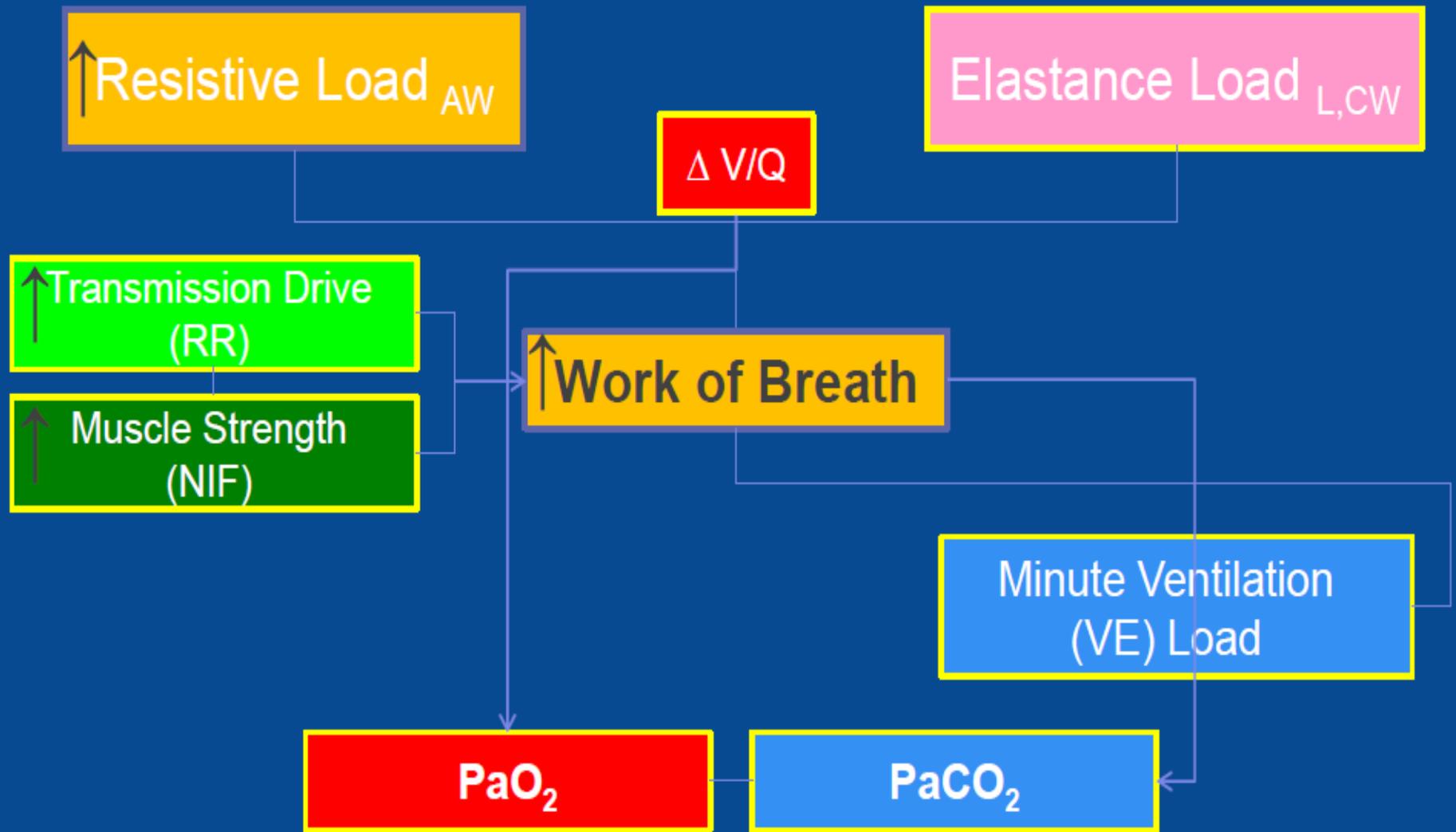
Respiratory Failure due to ↑ Resistance Load

COPD and Sever Asthma

Respiratory Failure due to ↑ Resistance Load

Airway narrowing results in a greater transthoracic pressure gradient requirement for inspiratory airflow. The resistive component of the work of breathing is increased.

Respiratory Failure due to ↑ Resistance Load



Balanced Load and Competence

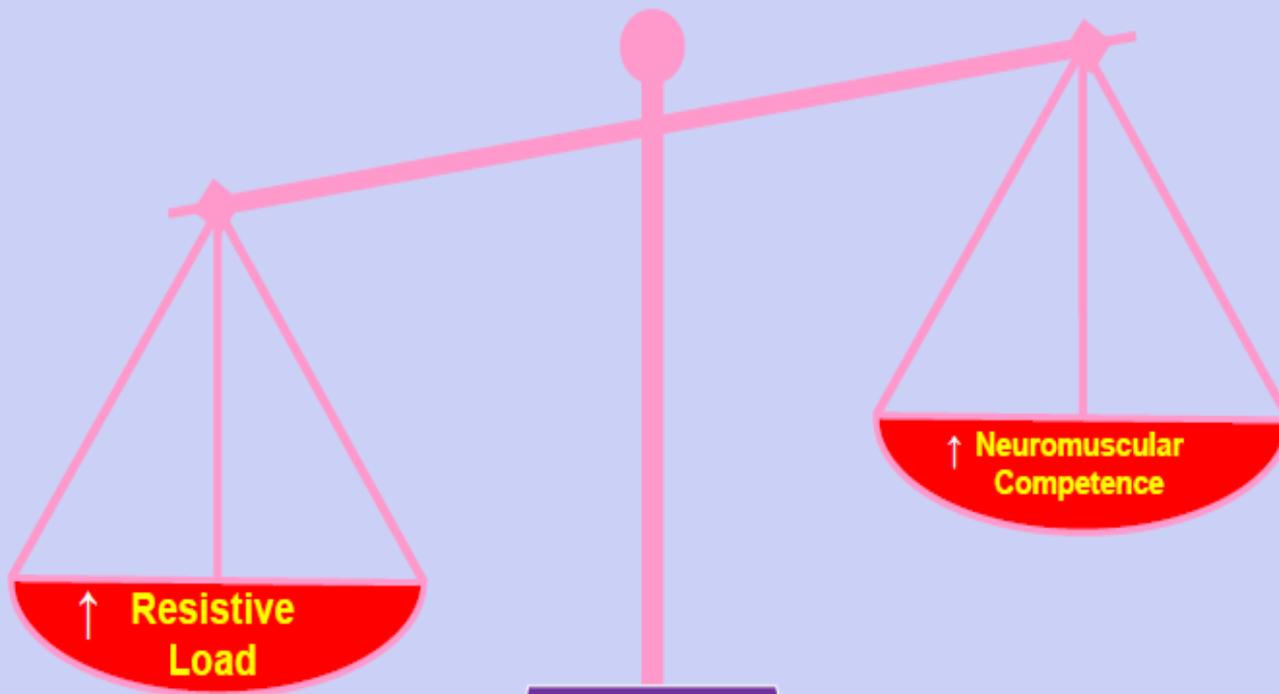


ABGs: PH: 7.42, PCO₂: 33, PO₂: 88, HCO₃: 20, SAT: 95%

Respiratory Failure due to ↑ Resistance Load

With Increased expiratory airway **resistance**,
Hypoxemia from **ventilation/perfusion
mismatch** will occur.

Imbalanced Load and Competence



ABGs: PH: 7.39, PCO₂: 44, PO₂: 58, HCO₃: 26, SAT: 88%

Respiratory Failure due to ↑ Resistance Load

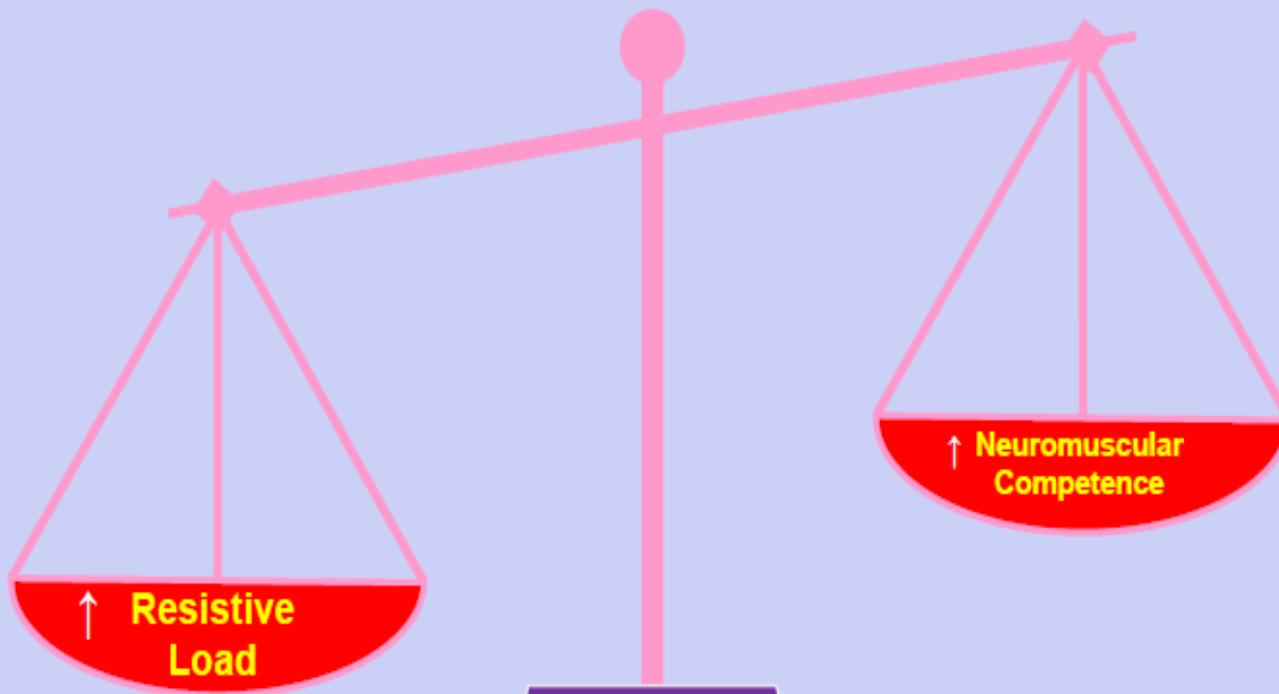
Hypercapnea occurs due to

Increased inspiratory airway resistance causes increased dead space (V_d/V_t), tidal volume falls

Dynamic hyperinflation

Abnormal respiratory muscle length-tension relationship

Imbalanced Load and Competence



ABGs: PH: 7.39, PCO₂: 50, PO₂: 57, HCO₃: 29, SAT: 85%

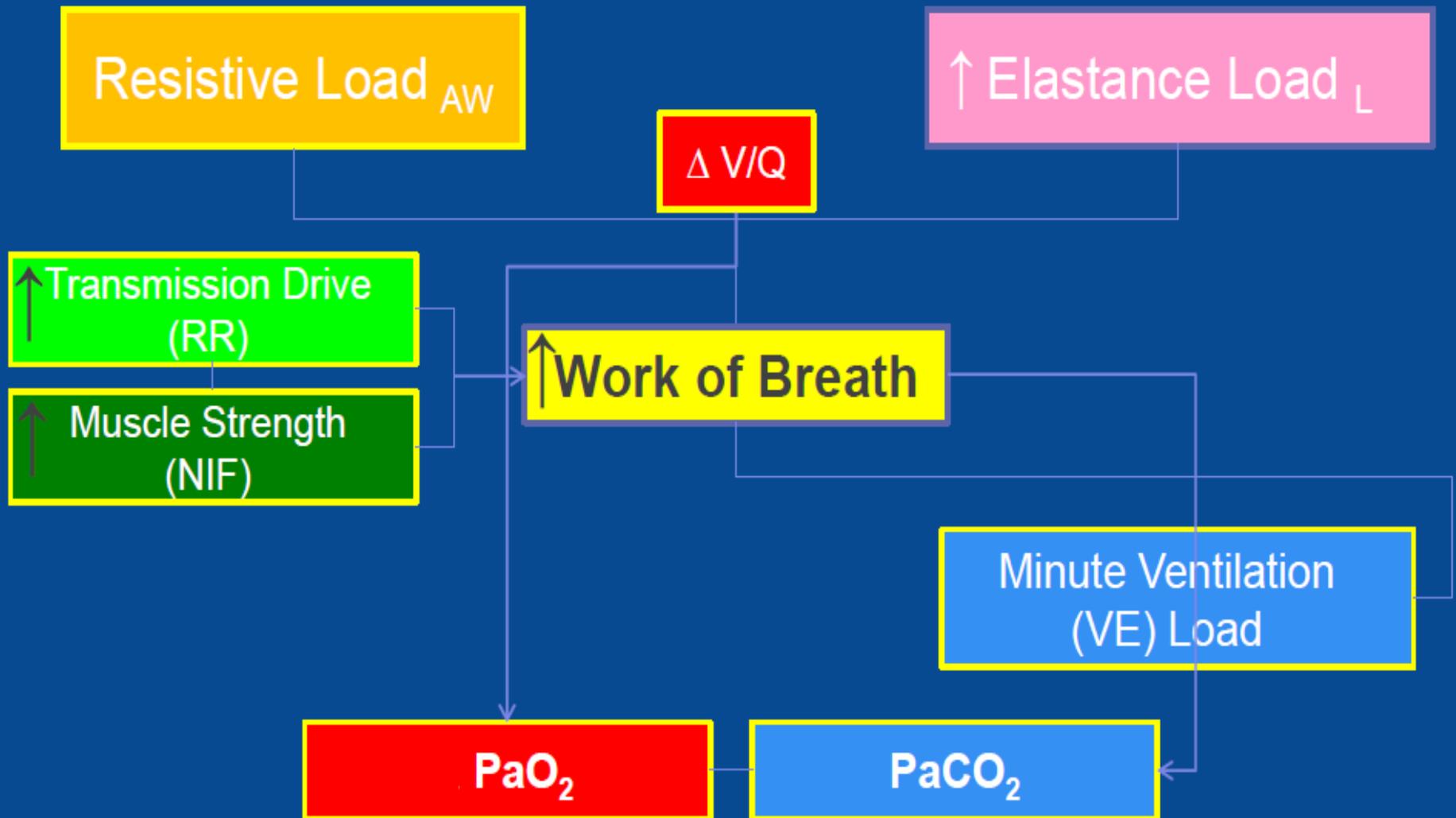
Respiratory Failure due to Compliance Decrease

Pulmonary Fibrosis

Respiratory Failure due to Compliance Decrease

Interstitial Fibrosis may impair diffusion across the alveolar-capillary membrane, The Elastance component of the work of breathing is increased.

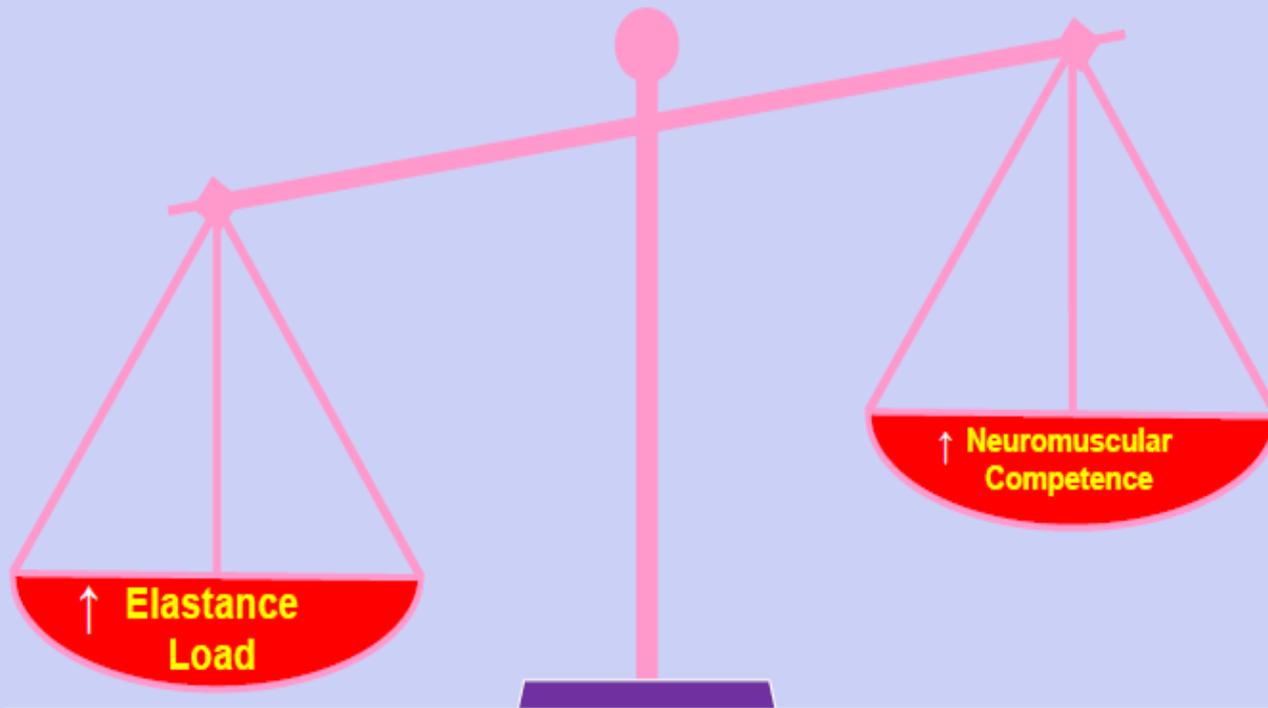
Respiratory Failure due to Compliance Decrease



Respiratory Failure due to Compliance Decrease

Increased Interstitial Fibrosis further impairs **diffusion** across the alveolar-capillary membrane, and further impairing oxygenation of venous blood.

Imbalanced Load and Competence



ABGs: PH: 7.41, PCO₂: 33, PO₂: 58, HCO₃: 20, SAT: 85%

Respiratory Failure due to Compliance Decrease

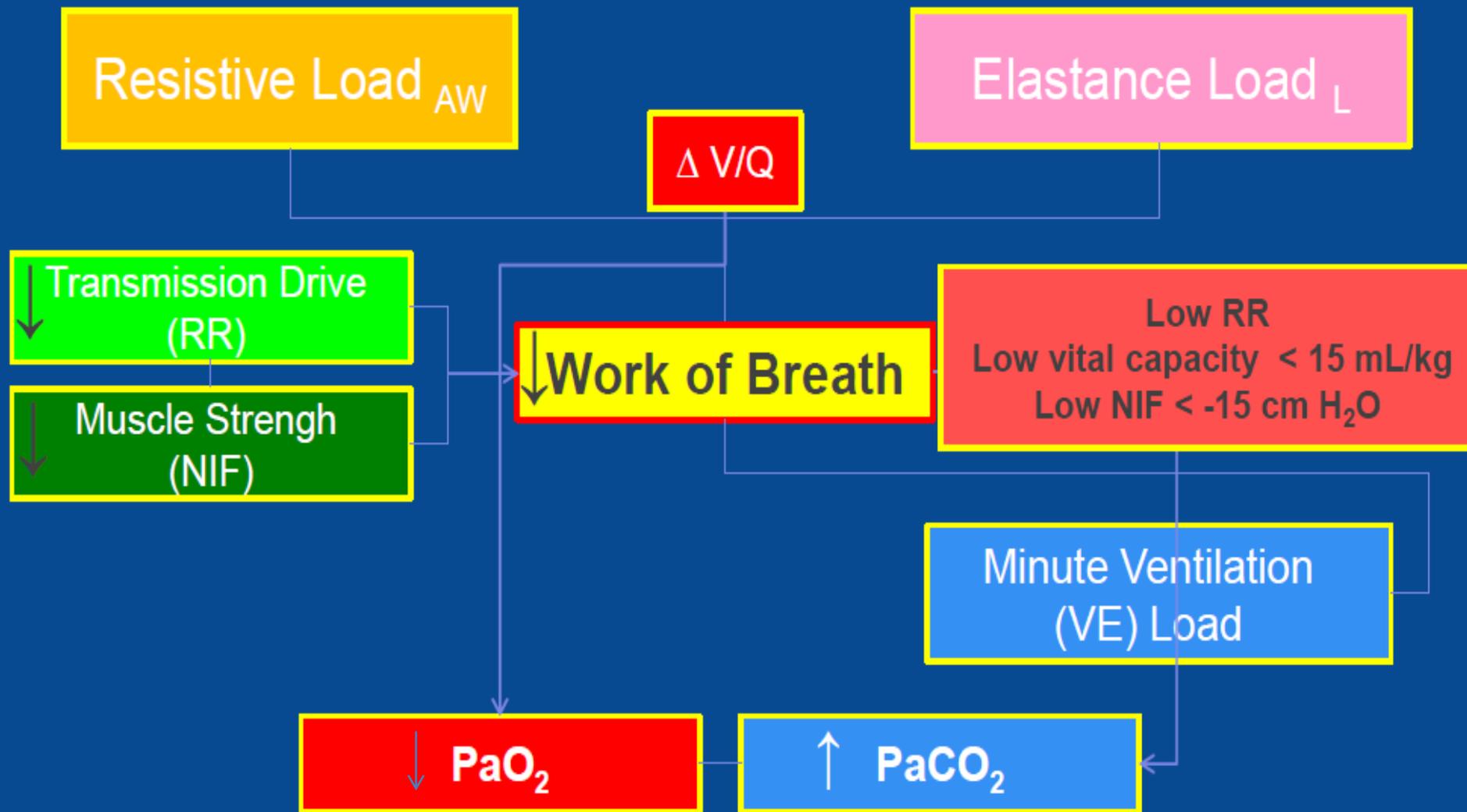
If control of ventilation is intact, **ventilation increases** in response to a raised P_{aco2} , so that the *excess CO₂ is excreted* by the *normal* areas of the lungs.

Respiratory Failure due to Compliance Decrease

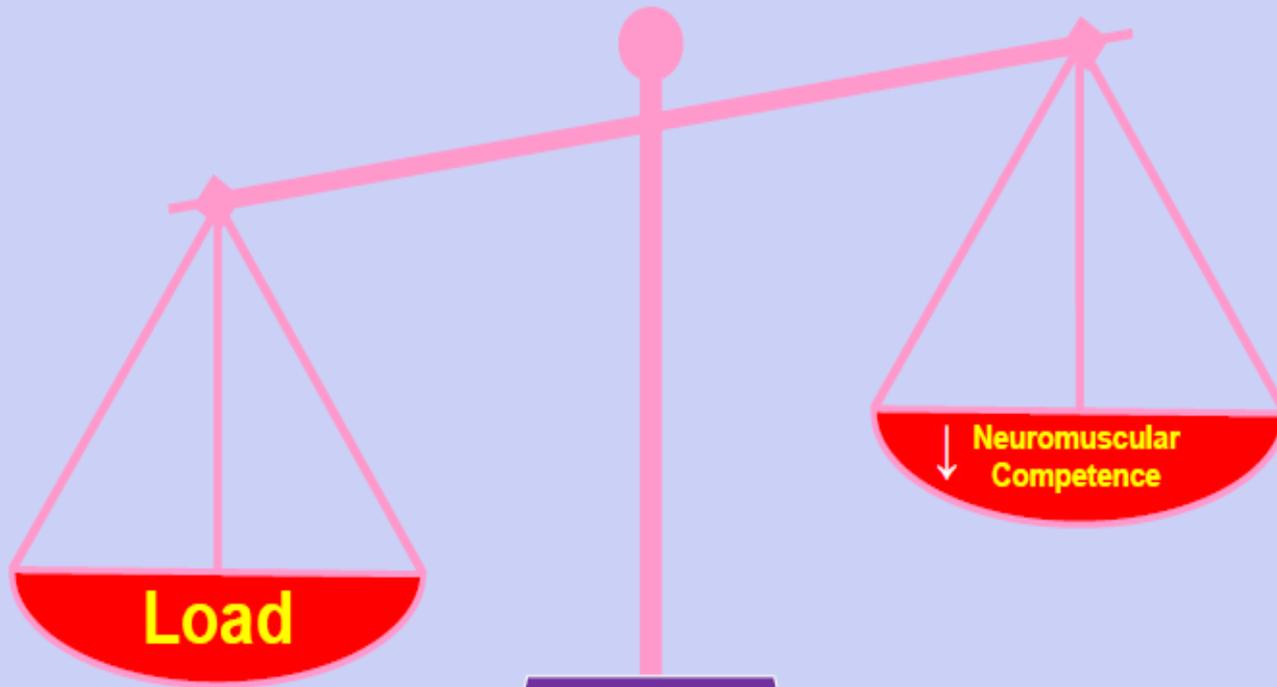
Hyperventilation **cannot** result in much more oxygen being taken up in the normal areas of the lungs, since the blood there is already **fully oxygenated**.

Respiratory Failure due to ↓ Neuromuscular Competence

Respiratory Failure due to ↓ Neuromuscular Competence



Imbalanced Load and Competence



ABGs: PH: 7.39, PCO₂: 50, PO₂: 57, HCO₃: 29, SAT: 85%

Management of Chronic Respiratory Failure

Management

Management of **underlying cause** is essential in
type I and II respiratory failure

Management

Oxygen is the critical therapy in the management of **type I** respiratory failure and can be given with impunity in this condition since there is **no hypercapnia**.

Management

The goal is to assure **adequate oxygen delivery** to tissues, generally achieved with an arterial oxygen tension (PaO_2) of 60 mm Hg or an arterial oxygen saturation (SaO_2) greater than 90%.

Management

Supplemental O₂ is usually indicated in patients with Type II chronic hypercapnic respiratory failure.

Management

Supplemental O₂ may produce exaggerated increases in PaCO₂ in patients with disorders of ventilatory control in whom the ventilatory response to CO₂ is blunted but the O₂ response is preserved.

Management

Accordingly, **blood-gas** tensions should be **monitored** closely when **O₂ is applied** initially.

Management

In particular, selected patients with **type II**

- **Obesity hypoventilation syndrome**
- **kyphoscoliosis**
- **neuromuscular disease**

have been successfully maintained on **NIV** for prolonged periods.

Management

NIV decreases the need for airway intubation, provides considerable patient comfort, and is easy to use.

Management

In patients with **sever chronic hypercapnia failure in COPD** and a history of **hospitalization** for acute respiratory failure, **long term NIV** may be considered (**Evidence B**).

Complications of Chronic Respiratory Failure

Pulmonary Hypertension

Pulmonary Hypertension

Group 3 PH — WHO Classification

Pulmonary hypertension owing to lung diseases or hypoxemia.

Pulmonary Hypertension

This group includes PH due to

Chronic obstructive pulmonary disease

Interstitial lung disease

Sleep-disordered breathing

Alveolar hypoventilation disorders

Pulmonary Hypertension

The initial symptoms of PH are the result of an inability to adequately increase cardiac output during exercise. These include exertional dyspnea, lethargy, and fatigue.

Pulmonary Hypertension

Primary therapy for group 3 PH consists of

Treatment of the underlying cause of hypoxemia

Correction of the hypoxemia with supplemental oxygen

Pulmonary Hypertension

Oxygen is the only modality with proven mortality benefit in some patients with group 3 PH.

Cor Pulmonale

Cor Pulmonale

Cor pulmonale refers to the **altered structure** (eg, hypertrophy or dilatation) and/or **impaired function** of the **right ventricle** that results from **pulmonary hypertension** that is associated with diseases of the lung

Cor Pulmonale

Symptoms attributable to cor pulmonale include dyspnea on exertion, fatigue, lethargy, exertional syncope, and exertional angina.

Cor Pulmonale

TREATMENT — The treatment of cor pulmonale can be conceptualized as having three major physiological goals:

Reduction of **right ventricular afterload**

Decrease of **right ventricular pressure**

Improvement of **right ventricular contractility**.

Cor Pulmonale

Right ventricular afterload can be reduced by

- Administering supplemental oxygen to patients with hypoxemia, since the oxygen will mitigate any hypoxic vasoconstriction.
- Treatment of the underlying cause of the pulmonary hypertension may also reduce right ventricular afterload.

Cor Pulmonale

Right ventricular Pressure

Diuretic therapy appears to improve the hemodynamic performance of patients with such markedly elevated right ventricular pressure.

Cor Pulmonale

Right ventricular contractility

There are **no oral inotropic agents** that are appropriate for routine use in patients with cor pulmonale.

Cor Pulmonale

Digoxin is an oral cardiac glycoside with **inotropic effects** that provides symptomatic benefit to patients with **left ventricular systolic dysfunction**.

Cor Pulmonale

Digoxin is **not indicated** for the treatment of cor pulmonale in **the absence of coexisting** atrial fibrillation or left ventricular dysfunction because there is **no evidence** that it improves clinical outcomes and it may have **detrimental effects**.

Thank You