Approach to a case of Respiratory failure - Acute and Chronic

Pulmonary medicine

A 55 years old, smoker male having exertional breathlessness, cough with scanty expectoration since 4-5 years. Now presented with worsening dyspnea since 4 days following upper respiratory tract infection.

History

HOPI

- Cough
- Expectoration
- Breathlessness
- Systemic symptoms

History

- Past History
- Allergic History
- Medication History
- Family History
- Personal History

Examination

- General Physical Examination

Drowsy, oriented to person but not to time and place

Vitals:

PR – 100, BP – 90/60 mmHg, RR- 30/min

Spo2 – 89 % RA

Systemic Examination:

Inspection :

Barrel shaped chest,
tracheal descent present,
widened intercostal space,
intercostal retraction present,
apex impulse shifted downward

Palpation:

Widened intercostal space,
apex beat shifted downward

- **Percussion:**

B/L hyperresonant note,

Liver shifted downward

- **Auscultation:**

B/L wheezing present

Investigation

- ABG
- Chest X-ray PA View
- Routine investigations

ABG

Test	Results	Units	Test Ranges Low	High
BLOOD GAS				
pH	7.208		6.500	8.000
pCO2	67.3	mmHg	3.0	200.0
pO2	47.5	mmHg	0.0	800.0
SO2%	72.1		30.0	100.0
Hct	46	%	12	70
Hb	14.9	g/dL	4.0	24.0
CALCULATED				
A	56.9	mmHg		
a/A	0.8			
AaDO2	9.5	mmHg		
HCO3-	27.1	mmol/L		
BEecf	-1.1	mmol/L		
BEb	-2.1	mmol/L		
O2Ct	15.1	mL/dL		
O2Cap	20.8	mL/dL		
P50	26.8	mmHg		
RI	0.2			
SBC	22.1	mmol/L		
pO2/FIO2	227.0	mmHg		
CHEMISTRY				
Na+	133.0	mmol/L	80.0	200.0
K+	4.10	mmol/L	1.00	20.00
Ca++	0.74	mmol/L	0.10	2.70
Lac	0.9	mmol/L	0.3	20.0
BUN	25	mg/dL	3	100
CALCULATED CHEMISTRY				
nCa	0.67	mmol/L		
TCO2	29.1	mmol/L		

Reported by _____ Time: _____

Notes

pH – 7.208

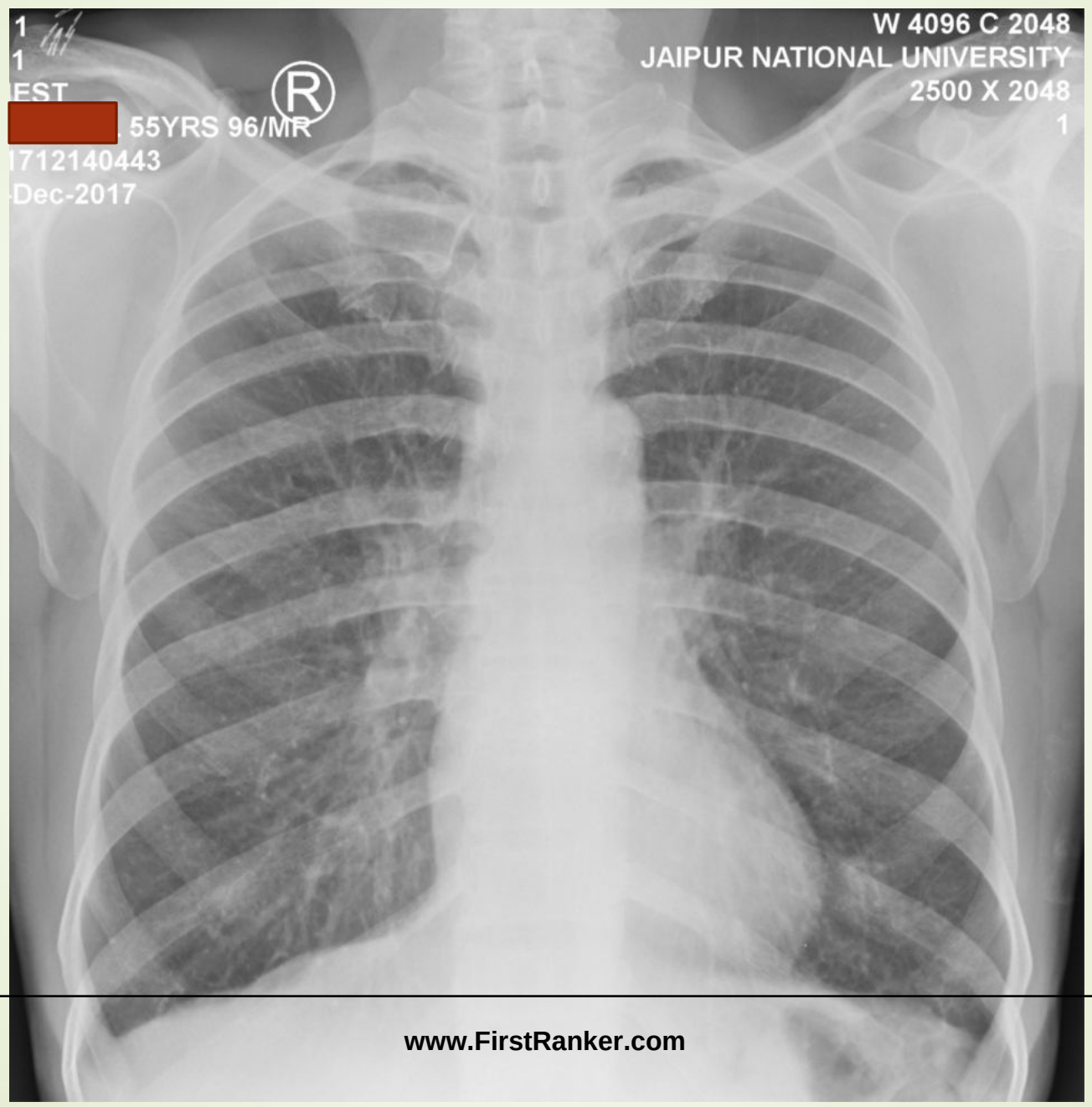
pCO2 – 67.3

pO2 – 47.5

SO2 – 72.1

HCO3 – 27.1

Chest Xray



Complete Hemogram

HAEMATOLOGY ANALYSIS REPORT

EDTA BLOOD

Investigations	Status	Result	Unit	Biological Reference Interval
Haemoglobin		13.5	Gm%	13-17
TLC.		4.6	Thousand cu/mm	4-11
Neutrophils		72	%	40-80
Lymphocytes		22	%	20-40
Monocytes		02	%	2-10
Eosinophils		04	%	1-6
Basophils		00	%	0-2
TRBC		4.80	millions/cumm	4.5-5.5
Platelet Count		287	Thousand /cumm	150-500
MCV		83.3	FL	83-101
MCH		28.1	pg	27-32
MCHC		33.8	g/dl	30--35
PCV		40.0	%	38-45

*** End of Report ***

Management

Respiratory failure

- Respiratory failure is a condition in which the respiratory system fails in one or both of its gas-exchanging functions; that is,
  - Oxygenation, and
  - Carbon dioxide elimination

Patho-physiology

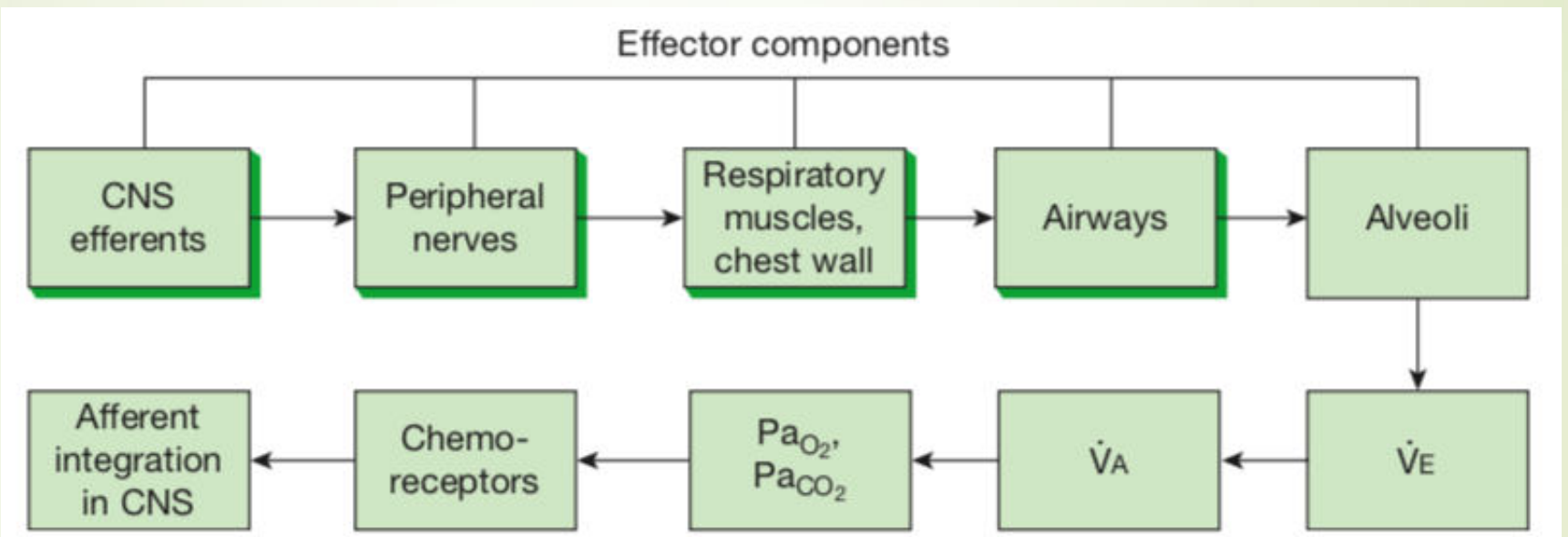
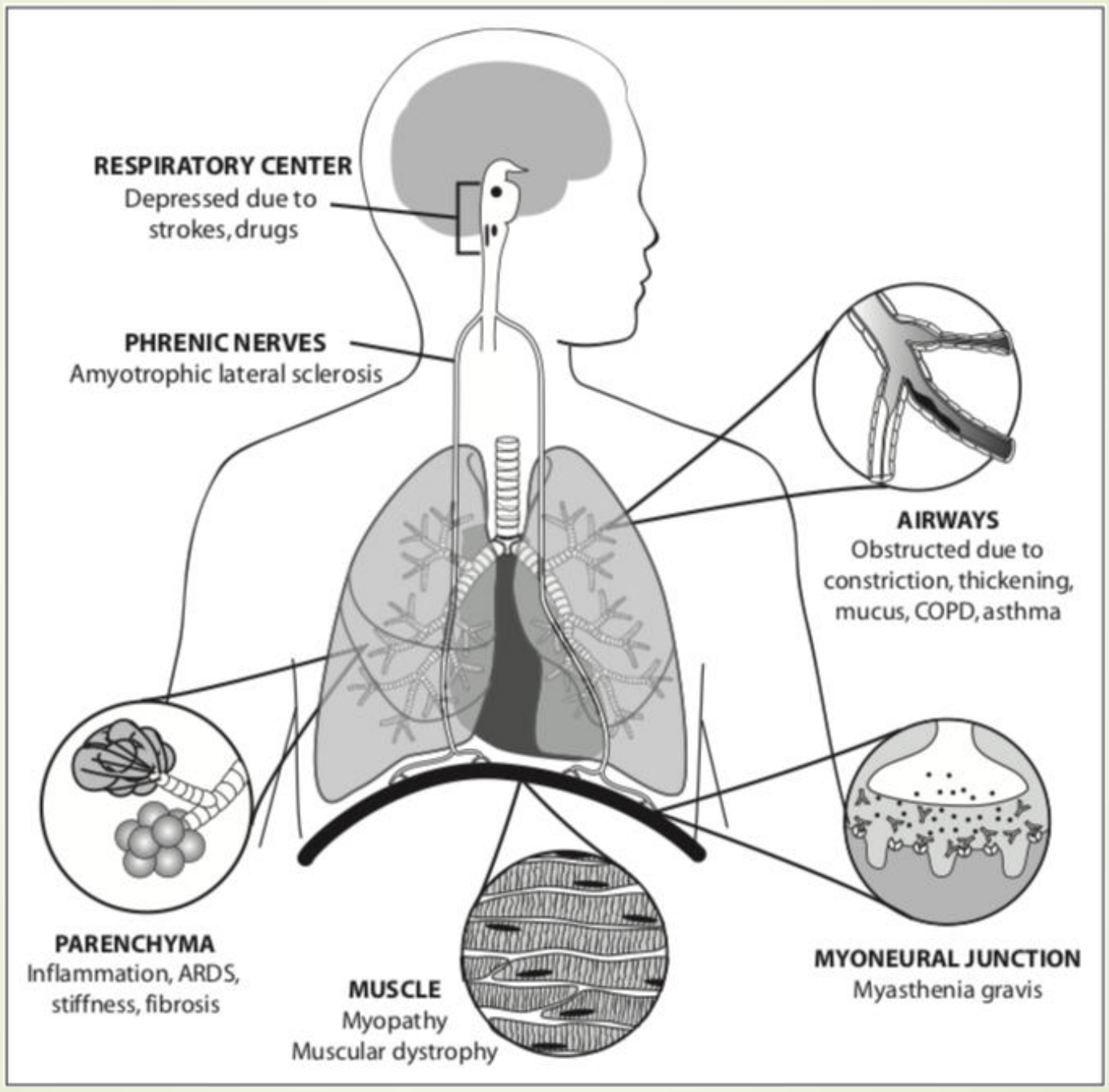
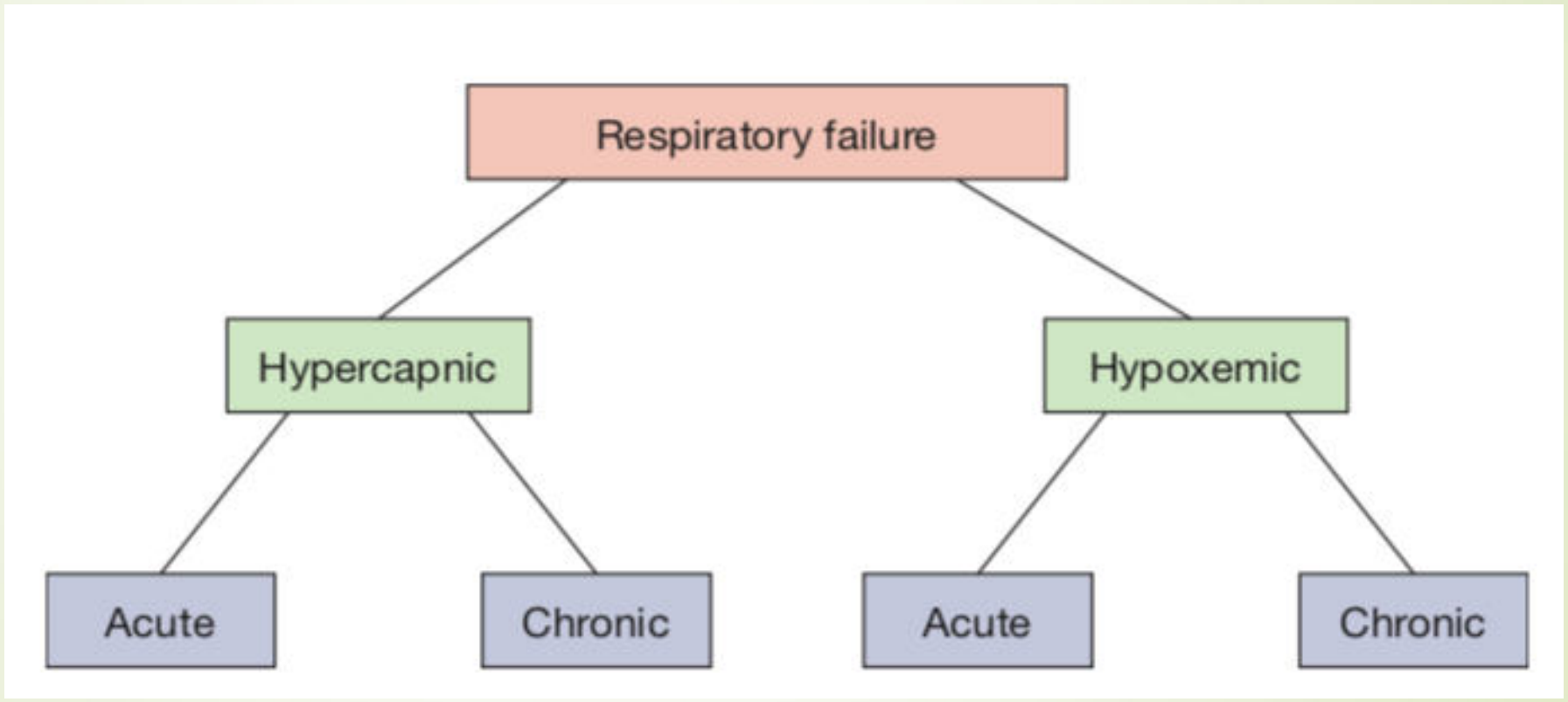


Figure 139-2 Functional components of the respiratory system and its controller. Abnormalities in any of the effector components can result in respiratory failure. The central and peripheral nervous systems, respiratory muscles and chest wall, and airways constitute the "respiratory pump" (*shaded boxes*). Hypercapnia is the hallmark of respiratory pump failure, while hypoxemia constitutes the primary disturbance in alveolar disorders producing respiratory failure. *(Reproduced with permission from Lanken PN. Pathophysiology of respiratory failure. In: Grippi MA, ed. Pulmonary Pathophysiology. Philadelphia. PA: JB Lippincott; 1995.)*



Classification of Respiratory failure



Category	Characteristic
Hypercapnic respiratory failure	Pa_{CO_2} >45 mm Hg
Acute	Develops in minutes to hours
Chronic	Develops over several days or longer
Hypoxemic respiratory failure	Pa_{O_2} <55 mm Hg
Acute	Develops in minutes to hours
Chronic	Develops over several days or longer

Type I (hypoxemic) respiratory failure:

- COPD
- Pneumonia
- Pulmonary edema
- Pulmonary fibrosis
- Asthma
- Pneumothorax
- Pulmonary embolism
- Pulmonary arterial hypertension
- Pneumoconiosis
- Granulomatous lung diseases
- Cyanotic congenital heart disease
- Bronchiectasis
- Acute respiratory distress syndrome (ARDS)
- Fat embolism syndrome
- Kyphoscoliosis
- Obesity

Type II (hypercapnic) respiratory failure:

- COPD
- Severe asthma
- Drug overdose
- Poisonings
- Myasthenia gravis
- Polyneuropathy
- Poliomyelitis
- Primary muscle disorders
- Porphyria
- Cervical cordotomy
- Head and cervical cord injury
- Primary alveolar hypoventilation
- Obesity-hypoventilation syndrome
- Pulmonary edema
- ARDS
- Myxedema
- Tetanus

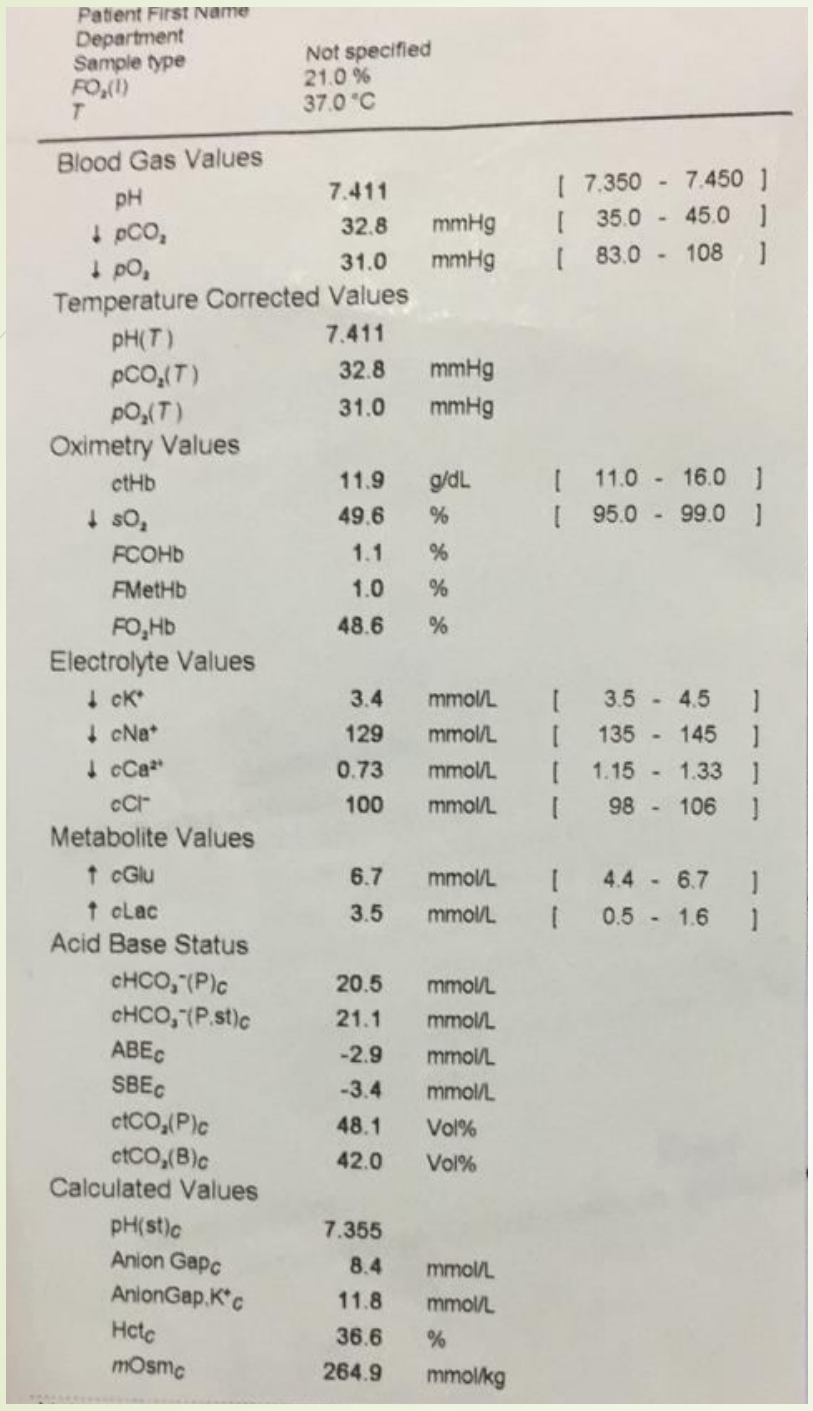
pH – 7.411

pCO2 – 32.8

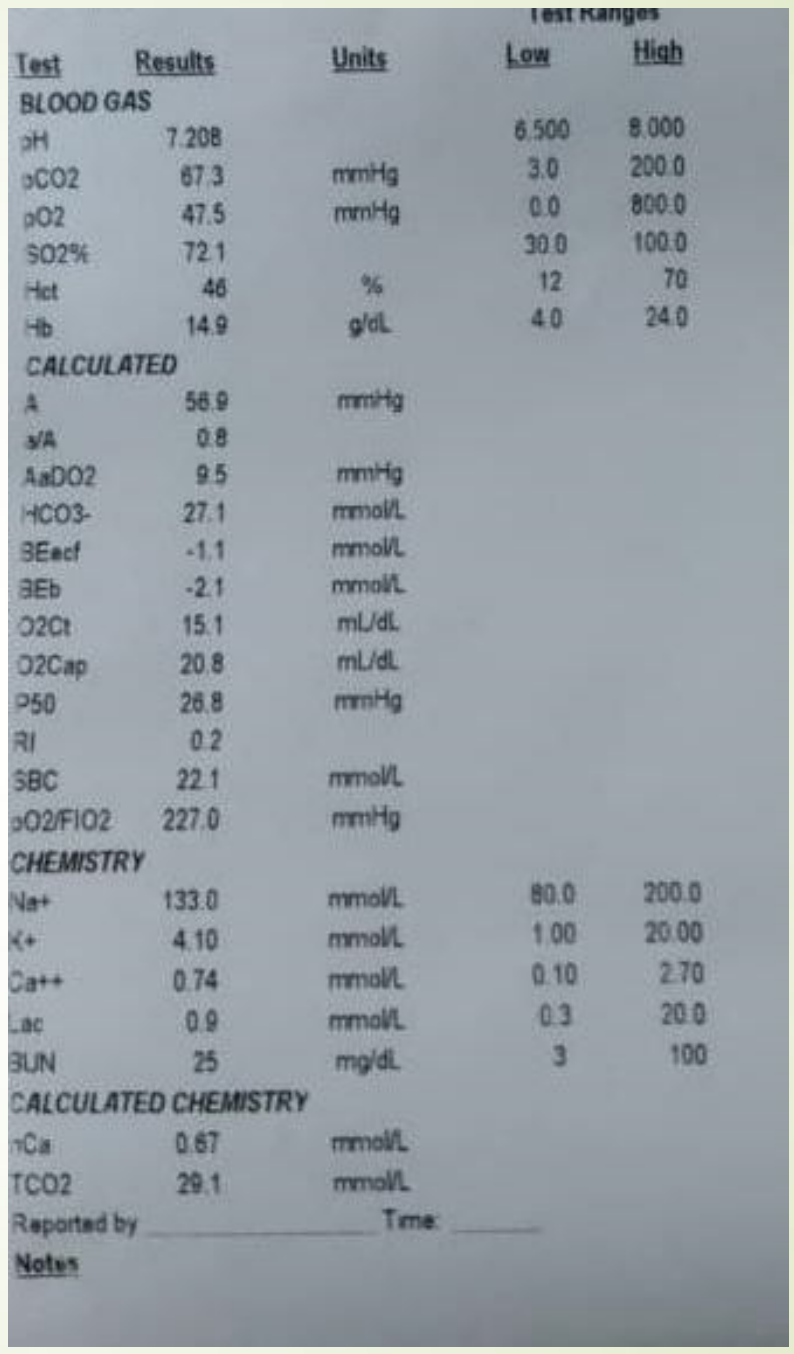
pO2 – 31.0

SO2 – 49.6

HCO3 - 20.5



Patient First Name			
Department			
Sample type	Not specified		
$FO_2(I)$	21.0 %		
T	37.0 °C		
Blood Gas Values			
pH	7.411		[7.350 - 7.450]
↓ pCO_2	32.8	mmHg	[35.0 - 45.0]
↓ pO_2	31.0	mmHg	[83.0 - 108]
Temperature Corrected Values			
pH(T)	7.411		
$pCO_2(T)$	32.8	mmHg	
$pO_2(T)$	31.0	mmHg	
Oximetry Values			
ctHb	11.9	g/dL	[11.0 - 16.0]
↓ sO_2	49.6	%	[95.0 - 99.0]
FCOHb	1.1	%	
FMetHb	1.0	%	
FO_2Hb	48.6	%	
Electrolyte Values			
↓ cK^+	3.4	mmol/L	[3.5 - 4.5]
↓ cNa^+	129	mmol/L	[135 - 145]
↓ cCa^{2+}	0.73	mmol/L	[1.15 - 1.33]
cCl^-	100	mmol/L	[98 - 106]
Metabolite Values			
↑ cGlu	6.7	mmol/L	[4.4 - 6.7]
↑ cLac	3.5	mmol/L	[0.5 - 1.6]
Acid Base Status			
$cHCO_3^-(P)_c$	20.5	mmol/L	
$cHCO_3^-(P,st)_c$	21.1	mmol/L	
ABE_c	-2.9	mmol/L	
SBE_c	-3.4	mmol/L	
$ctCO_2(P)_c$	48.1	Vol%	
$ctCO_2(B)_c$	42.0	Vol%	
Calculated Values			
$pH(st)_c$	7.355		
Anion Gap_c	8.4	mmol/L	
$AnionGap.K^+_c$	11.8	mmol/L	
Hct_c	36.6	%	
$mOsm_c$	264.9	mmol/kg	



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- The presence of markers of chronic hypoxemia (e.g., polycythemia or cor pulmonale) provides clues to a long -standing disorder, whereas abrupt changes in mental status suggest an acute event.

- Marker of chronic hypercapnia – Bicarbonate levels

Hypoxemic Respiratory failure

- Alveolar hypoventilation

  Normal alveolar–arterial oxygen gradient $P_AO_2 = F_iO_2(P_{atm} - P_{H_2O}) - \frac{P_aCO_2}{0.8}$

- Ventilation–perfusion mismatch,
- Shunt, and
- Diffusion limitation

Hypercapnic Respiratory failure

$$\dot{V}_A = K \cdot \dot{V}_{CO_2}/Pa_{CO_2}$$

where

$\dot{V}_A$ = minute alveolar ventilation

K = a constant

$\dot{V}_{CO_2}$ = rate of CO_2 production.

Ventilatory supply Versus demand

- Ventilatory supply : maximal spontaneous ventilation that can be maintained without development of respiratory muscle fatigue. AKA *maximal sustainable ventilation* (MSV)
- Ventilatory demand : spontaneous minute ventilation, which, when maintained constant, results in a stable PaCO2
- A 70-kg adult has an MVV of about 160 L/ min, an MSV of 80. L/min, and, under basal conditions, a Ve of approximately 6 to 7 liters per minute (90 mL/kg/ min).
- Normally, therefore, there is a 10- to 15-fold difference between resting V.e and MSV.
- In disease states, the V. e requirement may approach a markedly reduced MSV.
- Further reductions in MSV result in ventilatory demand exceeding supply, and hypercapnia occurs.

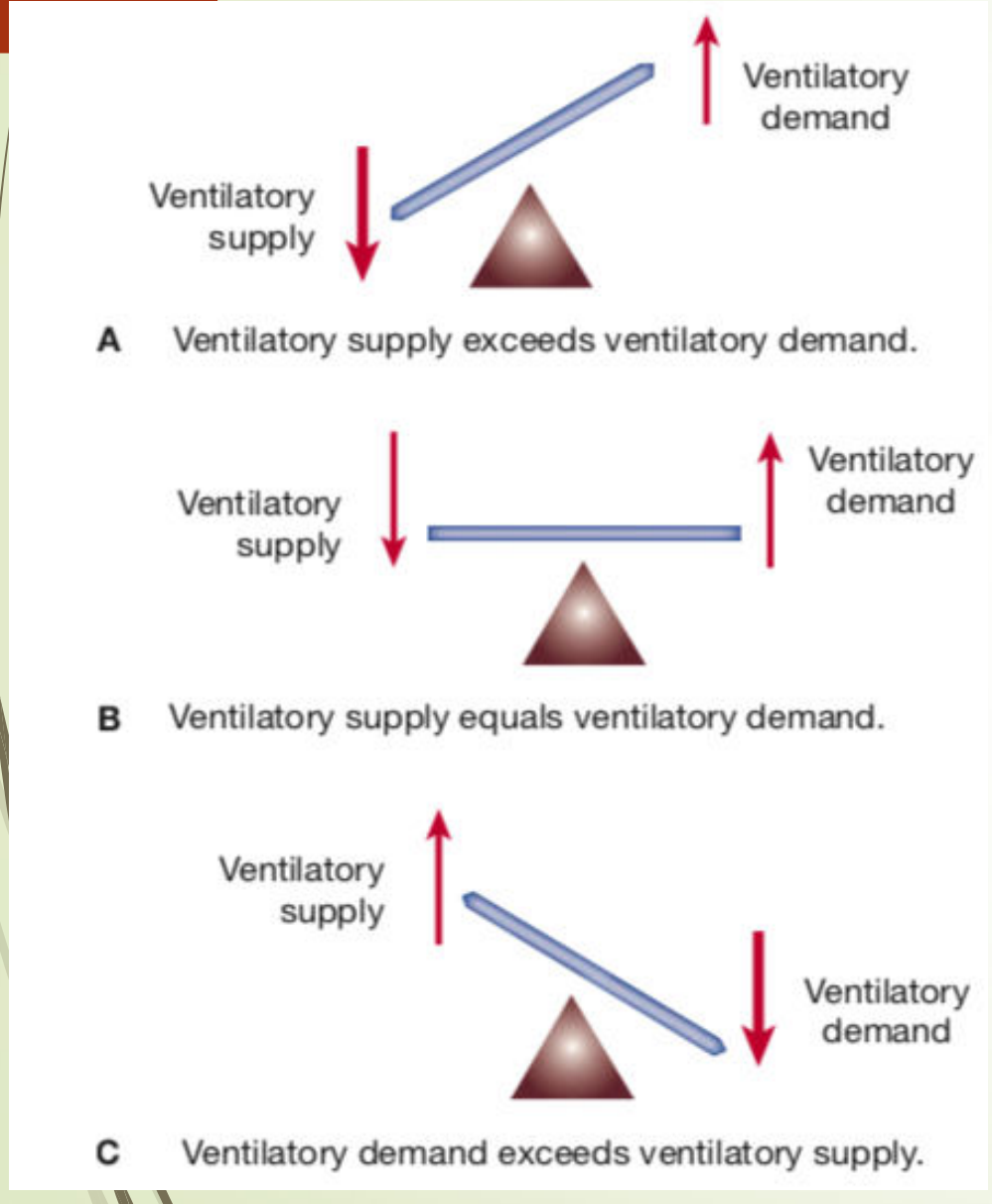

Figure 139-3 Relationship between ventilatory supply (maximal sustainable ventilation) and ventilatory demand (overall level of ventilation specified by the CNS controller). Relative size of the arrows indicates levels of supply and demand in each of the three circumstances illustrated. **A.** Normal. Ventilatory supply greatly exceeds ventilatory demand. Physiological "reserve" is maintained. **B.** Ventilatory supply is decreased and ventilatory demand increased (e.g., acute asthma attack). "Borderline" respiratory failure exists. **C.** Ventilatory demand exceeds ventilatory supply (e.g., sepsis in a patient with COPD). Respiratory muscle fatigue develops, and hypercapnic respiratory failure ensues. See text for details. *(Reproduced with permission from Lanken PN. Pathophysiology of respiratory failure. In: Grippi MA, ed. Pulmonary Pathophysiology. Philadelphia, PA: JB Lippincott; 1995.)*

TABLE 139-2 Factors That Diminish Ventilatory Supply

Factor	Clinical Examples
Decreased respiratory muscle strength	
Muscle fatigue	Recovery from acute respiratory failure, high respiratory rates, increased inspiratory time
Disuse atrophy	Prolonged mechanical ventilation, following phrenic nerve injury
Malnutrition	Protein–calorie starvation
Electrolyte abnormalities	Low serum phosphate or potassium concentrations
Arterial blood gas abnormalities	Low pH, low Pa_{O_2}, high Pa_{CO_2}
Fatty infiltration of diaphragm	Obesity
Unfavorable alteration in diaphragm length–tension relationship	Flattened domes of diaphragm caused by hyperinflation
Increased muscle energy requirement or decreased substrate supply	
High elastic work of breathing	Low lung or chest wall compliance, high respiratory rate
High resistive work of breathing	Airway obstruction
Reduced diaphragm perfusion	Shock, anemia
Decreased motor neuron function	
Decreased phrenic nerve output	Polyneuropathy, Guillain–Barré syndrome, phrenic nerve transection or injury, poliomyelitis
Decreased neuromuscular transmission	Myasthenia gravis, use of paralyzing agents
Abnormal respiratory mechanics	
Airflow limitation	Bronchospasm, upper airway obstruction, excessive airway secretions
Loss of lung volume	After lung resection, large pleural effusion
Other restrictive defects	Pain-limited inspiration; tense abdominal distention due to ileus, peritoneal dialysis fluid, or ascites

TABLE 139-3 Factors That Increase Ventilatory Demand

Factor	Clinical Examples
Increased V_D/V_T	Acute asthma, emphysema, late phase of acute respiratory distress syndrome, pulmonary emboli
Increased $\dot{V}_{O_2}$	Fever, sepsis, trauma, shivering, increased work of breathing, massive obesity
Increased RQ	Excessive carbohydrate feeding
Decreased Pa_{CO_2}	Hypoxemia, metabolic acidosis, anxiety, sepsis, renal failure, hepatic failure

Source: Data from Lanken PN. Pathophysiology of respiratory failure. In: Grippi MA, ed. Pulmonary Pathophysiology. Philadelphia. PA: JB Lippincott; 1995.

Approach to the patient

- Clinical suspicion:

Signs of

- underlying disease process – pneumonia, pulmonary edema, asthma, COPD, cor pulmonale etc
- Hypoxemia- restlessness, anxiety, tachycardia, dyspnea, cyanosis, use of accessory muscles, arrhythmias, seizures etc
- Hypercapnia – Confusion, drowsiness, somnolence, asterixis, tachycardia etc

ABG :

- PaO_2
- $PaCO_2$
- AaDO2
- pH
- HCO3
- O2 saturation

Difference between Acute & Chronic

Principles of Management

1. Triage decision

- OPD
- Ward
- HDU
- ICU

- ✓ At one end of the spectrum is the patient with fulminant hypoxemic respiratory failure, metabolic acidosis, and imminent cardiovascular collapse, who needs emergent intubation, mechanical ventilation, and admission to a critical care unit.
- ✓ At the other end of the spectrum is the patient with COPD and chronic, compensated hypercapnic respiratory failure, who requires observation in an intermediate care unit.

2. Airway Management:

Intubation and mechanical ventilation

Non invasive ventilation (Pros & Cons)

3. Correction of Hypoxemia and Hypercapnia

Hypoxemia : most life-threatening aspect of acute respiratory insufficiency

Goal is – assure adequate oxygen delivery to tissues (Generally PaO_2 > 60 mmHg),

Coronary or cerebrovascular disease (a slightly higher level)

Hypercapnia:

NIV/Invasive

4. Search of an underlying cause

Monitoring patients with acute respiratory failure

- Simple observation of respiratory rate, tidal volume.
- Use of accessory muscles, and presence of paradoxical breathing movements provides evidence of worsening respiratory failure and the need for intubation and mechanical ventilation
- On mechanical ventilation, the patient must be care-fully monitored for ventilator-associated complications. In addition, placement of indwelling arterial and venous catheters, patient immobilization, and use of a broad range of pharmacologic agents present additional potential threats to the acutely ill patient

Thank you