Key Terms

Alveolar deadspace	alveolar that are ventilated but not perfused, and where, as a result, no gas exchange occur.
Deadspace ventilation	ventilation in excess of perfusion; High V/Q
Shunt perfusion	perfusion in excess of ventilation; Low V/Q
Pathology	it is the cause and effect of diseases; the typical behavior of a disease

Example

PH	7.52	VT	400	BP	140/70
PCO2	27 mmHg	RR	30 min	Pulse	110 min
PO2	60 mmHg	MV	12 L	Temp	37.8 C

A 60-year-old, 48-kg woman with a 2-day history of thrombophlebitis of the right calf suddenly complains of chest pain and SOB. While breathing a fraction of inspired oxygen (FI02) of 0.21, the following clinical data are available above.

Result: Acute alveolar hyperventilation (respiratory alkalosis) with mild hypoxemia.

Example (cont)

PH	7.48	VT	400 mL	BP	120/80 mmHg
PCO2	33 mmHg	RR	20 min	Pulse	90 min
PO2	90 mmHg	MV	8 L	Temp	37.8 C

After 20 minutes of oxygen therapy (FIO2 0.50):

Result: Acute alveolar hyperventilation (respiratory alkalosis) secondary to hypoxemia.

Example (contt)

FIO2: 0.21						
	PH	7.48	VT	600 mL	BP	140/70
	PCO2	33 mmHg	RR	25 min	Pulse	110 min
	PO2	60 mmHg	MV	15 L	Temp	37.8 C

FIO2: 0.50						
	PH	7.45	VT	600 mL	BP	120/70
	PCO2	35 mmHg	RR	25 min	Pulse	100 min
	PO2	110 mmHg	MV	15 L	Temp	37.8 C

On further evaluation, this patient was found to have a left lower lobe pneumonia.

If the diagnosis were pulmonary embolus, the following clinical data would have been:

Example (conttt)

FIO2: 0.21						
	PH	7.48	VT	600 mL	BP	140/70
	PCO2	33 mmHg	RR	25 min	Pulse	110 min
	PO2	60 mmHg	MV	15 L	Temp	37.8 C

FIO2: 0.50						
	PH	7.45	VT	600 mL	BP	120/70
	PCO2	35 mmHg	RR	25 min	Pulse	100 min
	PO2	110 mmHg	MV	15 L	Temp	37.8 C

The minute ventilation is about three times normal, and yet the PACO2 is only slightly less than normal, suggesting an increased deadspace ventilation.

At 50% inspired O2, there is a significant increase in the PAO2 without significant changes in the ventilatory status – a circumstance that suggests deadspace-producing pathology.

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Increase dead space occurs in pulmonary embolism

Lung areas that are ventilated but not perfused form part of the dead space. Alveolar dead space is potentially large in pulmonary embolism.

Acute PE impairs the efficient transfer of oxygen and carbon dioxide across the lung (Tables 2).

Decreased arterial Po2 (hypoxemia) and increase in the alveolar-arterial oxygen tension gradient are the most common gas exchange abnormalities. Total dead space increases.

Ventilation and perfusion become mismatched, with blood flow from obstructed pulmonary arteries redirected to other gas exchange units. Shunting of venous blood into the systemic circulation may occur.