

VOLUME 2 • CHAPTER 32 • PART 1

Pulmonology

Foundations, Pathophysiology, and Assessment

Three things can fail — ventilation, diffusion, perfusion. Every disease in this chapter is one of them.
Aligned to Region SOPs

145 minutes, three parts — and where today sits

1 Foundations — today

Anatomy and physiology review, the pathophysiology framework, assessment, and management principles. 55 minutes.

2 Obstruction and obstructive disease

Upper airway obstruction, ARDS, emphysema, chronic bronchitis, and asthma — the diseases that narrow or destroy the airway.

3 Infection, embolism, and the rest

URI, pneumonia, coronaviruses, lung cancer, inhalation injury, PE, pneumothorax, hyperventilation, and neurologic causes.

One framework carries all three sessions

Three processes have to work: VENTILATION (moving air), DIFFUSION (gas crossing the membrane), and PERFUSION (blood arriving to carry it).

Every disease in this chapter breaks one or more of them, and the treatment follows from which one is broken. Asthma breaks ventilation. Pneumonia and ARDS break diffusion. Pulmonary embolism breaks perfusion. Hold that, and the eighteen diseases in Parts 2 and 3 stop being a list to memorise.

Today — minute by minute

5 Case Study

Read now, solved in Part 3.

5 Introduction

Why this chapter matters, and who is at risk.

10 Anatomy review

Upper airway to alveolus, and the three processes.

15 Patho

The framework. The largest block today.

10 Assessment

Scene, primary, secondary, and the three diagnostics.

10 Management

Priorities, and oxygen treated as a drug.

CASE STUDY

Read now. We solve it at the end of Part 3.

Dispatch

Difficulty breathing, 1420 hours, private residence.

On arrival

A 54-year-old woman, sitting upright, visibly anxious and breathing hard. She tells you the shortness of breath came on suddenly about an hour ago while she was watching television, along with a sharp pain in her right chest that is worse when she breathes in. She has no history of lung disease and takes no inhalers. She had a knee replacement eleven days ago and has been mostly in a recliner since.

RR 30 and shallow · HR 124 · BP 108/70 · SpO₂ 89% on room air · lungs CLEAR and equal bilaterally · ETCO₂ 26 mmHg with a normal waveform · her right calf is warm and slightly swollen.

Hold these questions:

- 1 Her lungs are CLEAR and she is hypoxic. What does that combination rule out?
- 2 Which of the three processes — ventilation, diffusion, perfusion — is failing here?
- 3 Why is her ETCO₂ low when her respiratory rate is high?
- 4 What would a bronchodilator accomplish in a patient whose bronchi are already open?

Why this chapter, and who gets these diseases

1 These are among the most common calls you will run

Not a specialty chapter. The respiratory system provides oxygen to the tissues and removes metabolic waste, and a significant fraction of every shift you work will be a failure of that.

2 INTRINSIC risk — from within the patient

The most important intrinsic risk factor is GENETIC PREDISPOSITION. Asthma is the clearest example: inherited susceptibility plus individual trigger sensitivity.

3 EXTRINSIC risk — external to the patient

The most important extrinsic risk factor is CIGARETTE SMOKING. Emphysema, chronic bronchitis, and the vast majority of lung cancers are extrinsic diseases.

4 And the division changes your history

Intrinsic-dominant → ask about family history, prior episodes, known triggers.
Extrinsic-dominant → ask about pack-years, occupation, home environment, exposures. Most people ask neither well.

Three processes have to work. Everything in this chapter breaks one of them.

VENTILATION — moving air. DIFFUSION — gas crossing the membrane. PERFUSION — blood arriving to carry it.

Write those three on the board and leave them there for all three sessions. They are the reason the eighteen diseases ahead are three categories rather than a list.

Respiratory Anatomy and Physiology

A review — but with a different lens. Chapter 22 asked how to open an airway. This asks what disease closed it.

Upper airway · Lower airway · Ventilation · Diffusion and perfusion

The upper airway — warming, humidifying, filtering

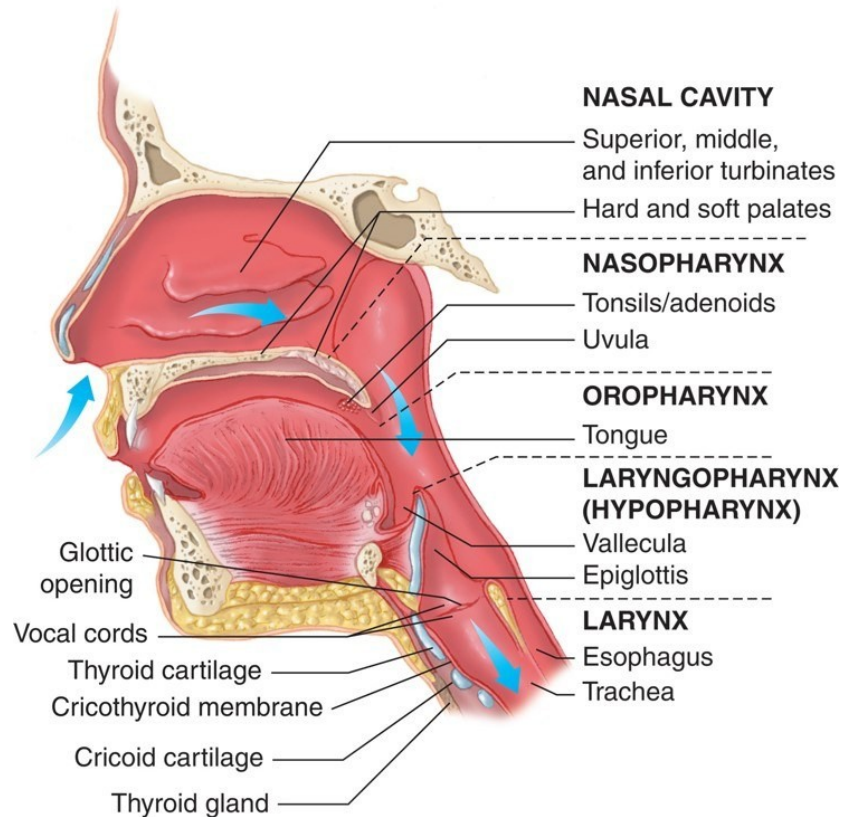


Figure 32-2 Anatomy of the upper airway.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

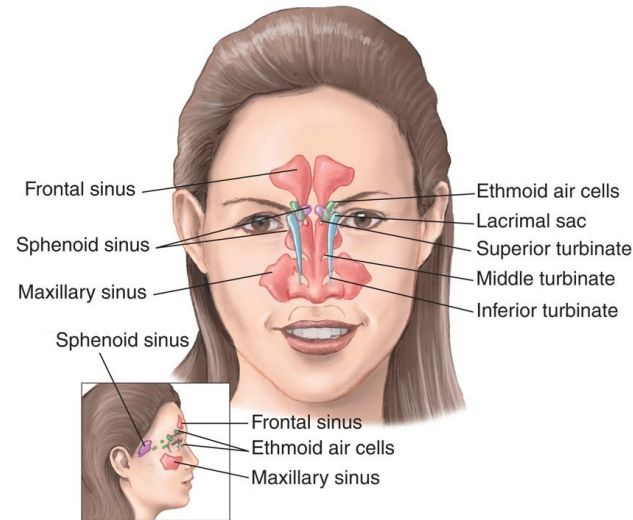


Figure 32-3 The paranasal sinuses.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

REGION SOP Your anaphylaxis protocol: “New onset hoarseness or underpowered voice indicates vocal cord edema and should prompt concern for airway compromise.” That is a **VOICE** change, not a breath sound — and it is available to you from the doorway.

The job: warm, humidify, filter

That is what the upper airway is FOR — and it is why intubated patients dry out and why cold dry air triggers asthma.

Turbinates create turbulence

Air is deliberately made to swirl so inhaled particles are thrown onto the mucosa and removed rather than carried onward.

The larynx is a filtering device

As well as a voice. The epiglottis closes during swallowing to divert food and liquid into the esophagus — the crossroads where air and food are separated.

And it is where edema kills

Laryngeal edema appears in obstruction, anaphylaxis and inhalation injury alike. It is what turns a breathing problem into an airway emergency.

The lower airway — trachea to alveolus

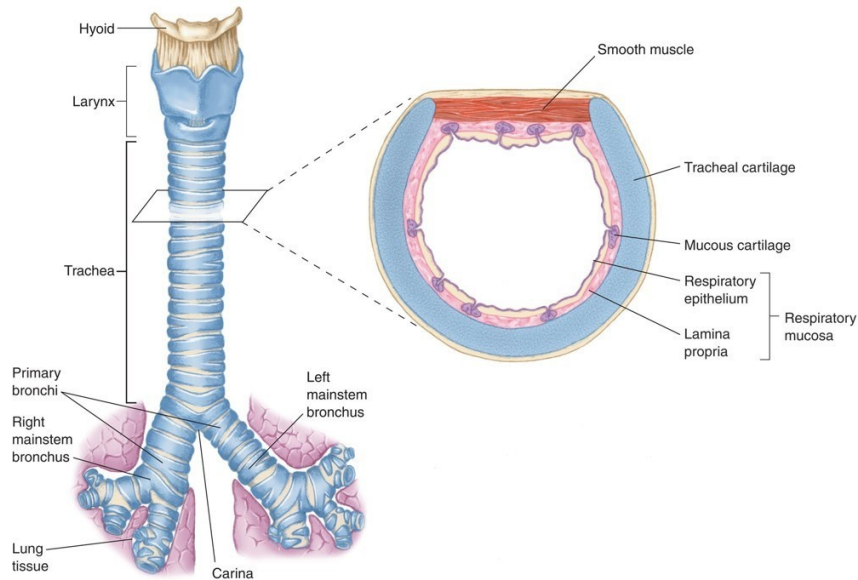


Figure 32-4 The lower airway and tracheal cross section.

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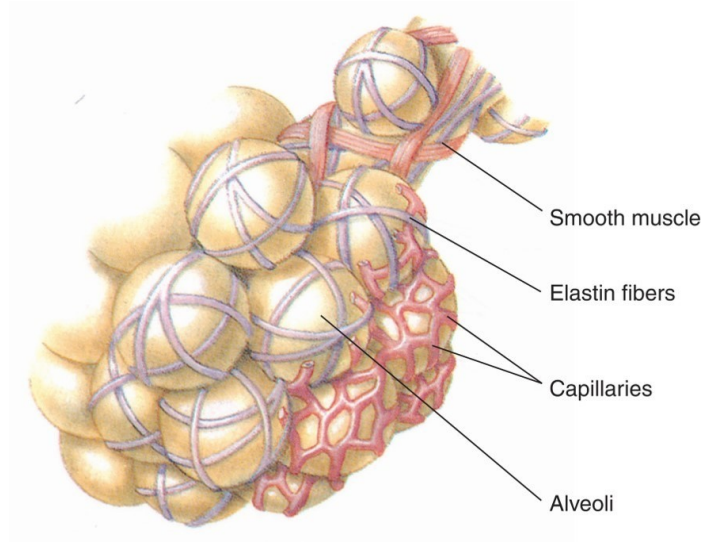


Figure 32-5 Alveoli and pulmonary capillaries.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

Name the disease at each level

Trachea and mainstem — foreign body, aspiration
 Bronchi — chronic bronchitis
 Bronchioles — asthma
 Alveoli — emphysema, pneumonia, ARDS
 Capillaries — pulmonary embolism

1 Trachea and carina

C-shaped rings, lined with the same cells as the nares. The RIGHT mainstem is almost a straight continuation — which is why aspiration favours the right lower lobe.

2 Bronchioles — 1 mm, with muscle around them

“Approximately 1 mm thick and contain SMOOTH MUSCLE THAT CAN CONTRACT, reducing the diameter of the airway.” That sentence is the entire pathophysiology of asthma — and no cartilage holds them open.

3 The respiratory membrane

Alveolar lining + supportive tissue + capillaries. Diffusion happens across it, and several diseases here work by THICKENING it: ARDS with fluid and inflammatory cells, pneumonia by filling the alveoli, edema by flooding the interstitium.

4 Surfactant

Keeps the alveoli moist and open. It is lost in ARDS — which is why alveoli collapse there.

Lungs, pleura, and two separate blood supplies

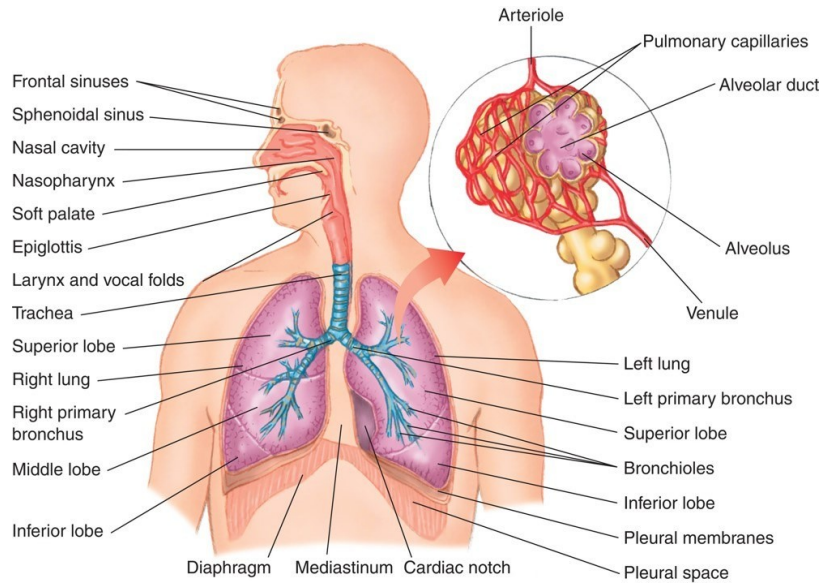


Figure 32-1 The respiratory system.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

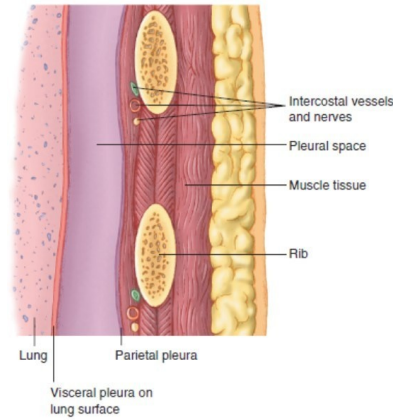


Figure 32-7 Pleura and the chest wall.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

Coming in Part 2

Emphysema: pulmonary capillaries decrease in number → pulmonary hypertension → right heart failure.

Chronic bronchitis: raised PaCO_2 → pulmonary vasoconstriction → pulmonary hypertension → cor pulmonale.

Both are stories about the pulmonary circuit — and they only make sense if this slide landed.

Lobes and pleura

Right lung three lobes, left lung two. Visceral pleura on the lung, parietal lining the cavity — and only the parietal has nerve fibres, which is why pleuritic pain exists at all.

PULMONARY circulation — gas exchange

Arteries carry DEOXYGENATED blood from the heart to the lungs; veins return oxygenated blood to the heart. The one place in the body where that is reversed.

BRONCHIAL circulation — nutrition

Branches from the AORTA to feed the lung tissue itself; bronchial veins return to the superior vena cava. A completely separate circuit with a different job.

Which explains something surprising

A pulmonary embolus stops gas exchange in that segment — but the lung tissue is still fed by the bronchial circulation, so infarction is relatively uncommon.

Ventilation — resistance and compliance

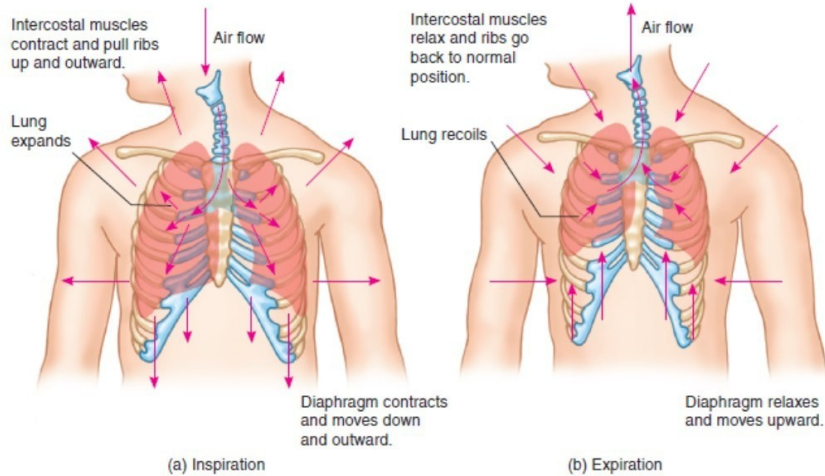


Figure 32-8 Inspiration and expiration.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

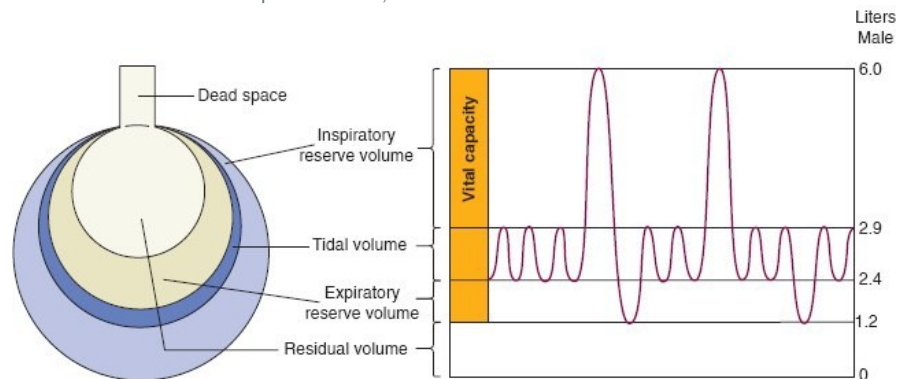


Figure 32-9 Lung volumes and capacities.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

Paramedic Education • Ch 32 Pulmonology • Part 1

1 Five things must be intact

The CHEST WALL, the NERVE PATHWAYS, the DIAPHRAGM, the PLEURAL CAVITY, and the BRAINSTEM. That is a checklist of what can break — flail chest, Guillain-Barré, high cervical injury, pneumothorax, opioid overdose.

2 AIRWAY RESISTANCE

More resistance, less airflow. Asthma causes bronchospasm, reducing airway diameter — and resistance rises steeply as diameter falls, so a little narrowing costs a lot of work.

3 LUNG COMPLIANCE

The ease with which the chest expands. More expansion, more volume. Loss of elasticity in the chest wall with age decreases compliance.

4 Control sits in the MEDULLA

The lower brainstem, adjusting volume to the metabolic needs of the patient — which is why CNS depressants and brainstem injury both appear in the pathophysiology block.

Resistance and compliance fail in different diseases — and give you different patients

A RESISTANCE problem — asthma, chronic bronchitis, emphysema — wheezes, has a prolonged expiratory phase, and struggles to get air OUT.

A COMPLIANCE problem — pulmonary edema, ARDS, pneumonia, a stiff chest wall — breathes fast and shallow, and struggles to get air IN.

That distinction is available at the bedside with no equipment at all, and it cuts the differential in half before you have touched a monitor.

Diffusion and perfusion — the other two processes

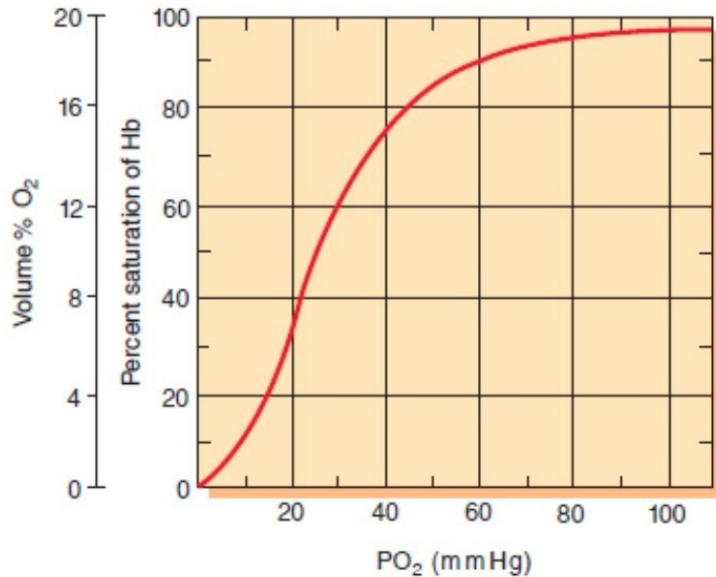


Figure 32-10 The oxyhemoglobin dissociation curve.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

The reason this slide is the hinge

It explains the patients whose lungs sound FINE and who are still hypoxic.

A clear chest reads as reassurance — and two of these three perfusion failures produce a completely clear chest.

DIFFUSION — passive, free, and breakable in exactly three ways

Gases flow from HIGH concentration to LOW, across the respiratory membrane. Oxygen alveolus → capillary; carbon dioxide capillary → alveolus. It costs no energy, so the only ways to break it are: change the GRADIENT, thicken the MEMBRANE, or take away the BLOOD on the far side.

Which is why high-concentration oxygen helps here — it steepens the gradient. That is the physiologic justification for oxygen in pneumonia and ARDS.

PERFUSION depends on three conditions — and each fails in a different disease

1 Adequate blood volume

Because of the concentration of hemoglobin in the blood. FAILS IN: hemorrhage, severe anemia — perfect lungs, no delivery.

2 Intact pulmonary capillaries

Where the gas exchange actually takes place. FAILS IN: pulmonary embolism, and the capillary destruction of emphysema.

3 Efficient pumping by the heart

To deliver oxygen and eliminate carbon dioxide. FAILS IN: cardiogenic shock, massive myocardial infarction.

Pathophysiology

The largest block today — and the one that makes the next two sessions teachable.

Ventilation · Diffusion · Perfusion

Three processes, three ways to fail

1 VENTILATION — air is not moving

Obstructed conducting pathways, impaired chest wall function, or an abnormality in nervous system control. The patient cannot move air in or out.

2 DIFFUSION — gas is not crossing

Altered alveolar structure or patency, a thickened respiratory membrane, or changed capillary permeability. Air arrives; it just cannot get across.

3 PERFUSION — blood is not arriving

Reduced circulating volume, blocked or damaged pulmonary capillaries, or a failing pump. Gas crosses fine; there is nothing there to carry it away.

If this is broken	It looks like	And treatment means
VENTILATION	Wheezing, stridor, prolonged expiration, poor air movement, abnormal rate or depth. RISING ETCO_2	MOVE AIR. Positioning, bronchodilators, positive pressure, assisted ventilation
DIFFUSION	Crackles, consolidation, fever, hypoxia with reasonable air movement	STEEPEN THE GRADIENT and address the membrane. Oxygen, CPAP, treat the cause
PERFUSION	CLEAR lungs with real hypoxia. Tachycardia. Often a LOW ETCO_2 despite a high rate	FIX THE CIRCUIT. Volume, pump support, rapid definitive care — and expect the least from oxygen alone

Many real patients break more than one — emphysema destroys alveolar walls (diffusion), reduces pulmonary capillaries (perfusion) AND weakens the small bronchioles (ventilation). That is not a flaw in the framework. It is why those patients are so sick.

Disruption of ventilation — three routes in

1 The pathways are OBSTRUCTED

Upper airway trauma, infections, foreign bodies, and other structures — including the tongue, which is the most common cause of upper airway obstruction and which opens Part 2.

2 The CHEST WALL cannot do its job

Pneumothorax, hemothorax, flail chest, diaphragmatic rupture. Ventilation needs negative pressure generated inside an INTACT container — break the container, or its rigidity, and the gradient cannot form.

3 The NERVOUS SYSTEM stops asking

Any process impairing regulation of breathing: CNS depressants such as alcohol or barbiturates — and opioids, which is the version you will actually meet, several times a month.

Three routes in — and three completely different things to do about it

For obstruction

Open it, remove it, or bypass it. Position, suction, forceps, or an advanced airway — the Chapter 22 ladder, applied to a cause.

For a chest wall problem

Decompress a pneumothorax, support the flail segment with positive pressure, and ventilate. You are substituting for a broken container.

For a control problem

Ventilate FOR them and reverse the cause if you can. The lungs are fine; nothing is asking them to work.

The mechanism tells you the intervention — which is why this framework is worth 15 minutes

You do not need a diagnosis to know that an obstructed patient needs the obstruction relieved, a flail chest needs pressure, and an overdose needs someone to breathe for them. Three different problems, three different answers, all reachable without naming the disease.

Five patterns produced by specific brain injury

Pattern	What it looks like	What it points to
Cheyne–Stokes	Crescendo–decrescendo depth with apneic pauses	Older patients with terminal illness or brain injury — and commonly severe heart failure
Kussmaul's	Deep, rapid, laboured — sustained	A CORRECTIVE measure against metabolic acidosis, classically diabetic ketoacidosis. This patient is compensating, not failing
Central neurogenic hyperventilation	Sustained, deep, rapid — no pauses	Strokes or injury to the brainstem
Ataxic (Biot's)	Irregular and unpredictable, with irregular pauses	Increased intracranial pressure. More ominous than Cheyne–Stokes
Apneustic	Prolonged inspiratory gasps with inadequate expiration	Stroke or severe central nervous system disease. Rare, and a poor sign

Four of these five are NEUROLOGIC findings that happen to be visible in the breathing

They are not respiratory diseases, and treating them as such is the error this slide exists to prevent.

A patient with Kussmaul respirations does not need their rate slowed — they need their acidosis treated. A patient with Biot's respirations does not need a bronchodilator — they need a CT scanner.

A respiratory pattern is one of very few findings available from the doorway that actually localises a lesion. That is worth the thirty seconds it takes to look.

Disruption of diffusion, and of perfusion

DISRUPTION OF DIFFUSION — four mechanisms

Oxygen concentration falls

Lower the concentration in the alveolus and the gradient flattens. Altitude, and confined spaces with displaced oxygen.

Alveolar structure or patency is altered

Emphysema destroys alveolar walls. Atelectasis collapses them. Pneumonia fills them.

The respiratory membrane thickens

The most common cause is named explicitly: accumulation of FLUID AND INFLAMMATORY CELLS in the interstitial space. That is ARDS, pulmonary edema and interstitial pneumonia in one mechanism.

Capillary permeability changes

Leaky capillaries let fluid into the interstitium, which thickens the membrane. This is the mechanism of ARDS specifically.

Ventilation without perfusion = DEAD SPACE. Perfusion without ventilation = SHUNT.

DISRUPTION OF PERFUSION — two mechanisms

Circulating volume falls

Any disease state reducing normal circulating blood volume limits perfusion in the lungs — hemorrhage, dehydration, profound anemia.

A portion of lung loses blood flow

“If a portion of lung is without blood flow, it becomes unavailable for perfusion REGARDLESS OF OXYGENATION.” That is the whole pathophysiology of pulmonary embolism, in one clause.

The prediction that framework makes

You can ventilate an embolised lung segment perfectly, fill it with 100% oxygen, and it will contribute NOTHING — because there is no blood there to load.

Which is why a PE patient's saturation often improves disappointingly little on high-flow oxygen. That is not equipment failure and it is not a patient who is less sick than you thought. It is the physiology doing exactly what this slide says it will.

Assessment of the Respiratory System

From the doorway to the monitor — and the nine findings that mean this patient is dying.

Scene size-up · Primary · Secondary · Diagnostics

Scene size-up and the general impression

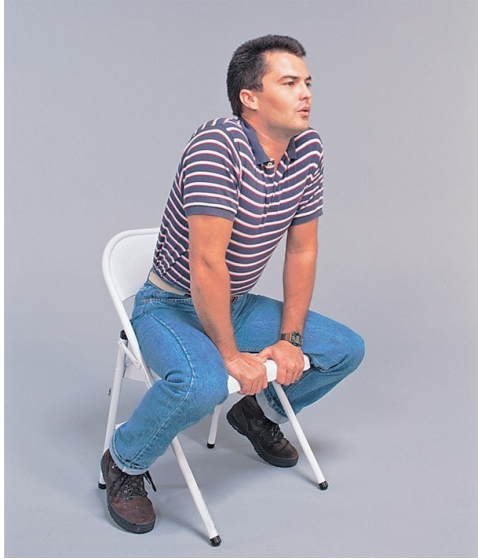


Figure 32-14 Tripod position.

Tripoding is a TREATMENT, not a symptom

The patient has already found the position that maximises their mechanics. Laying them down to “assess them properly” removes the only intervention that was working.

REGION SOP The general impression takes about eight seconds and is often more informative than the next ten minutes. Stop in the doorway and look before you kneel down.



Figure 32-15 Cyanosis.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 Two questions on approach

Is the scene safe? And are there VISUAL CLUES about the medical complaint? The second is underused — concentrators, nebulisers, inhalers, a CPAP by the bed, cigarettes, a wood stove, a running generator. All of it is diagnosis, and all of it disappears once you load.

2 Five things from the doorway

POSITION · COLOUR · MENTAL STATUS · ABILITY TO SPEAK · RESPIRATORY EFFORT. Count the words per breath — one- to two-word dyspnea is a vital sign that needs no equipment.

3 Signs of distress

Nasal flaring · intercostal retraction · cyanosis · PURSED LIPS · tracheal tugging. Pursed lips is auto-PEEP — the patient generating their own CPAP to hold small airways open, and a sign of a resistance problem specifically.

4 Airway first, and it stops you

Airway assessment is done IMMEDIATELY — open, patent, abnormal noises. If it is compromised it MUST BE CORRECTED BEFORE YOU MOVE ON. Only then does breathing get assessed.

Nine findings that mean this patient is dying

The brain is failing

Alterations in mental status — and note that a patient who becomes CALM after being agitated has often not improved. Rising carbon dioxide is sedating.

Oxygen is not reaching tissue

Severe CENTRAL cyanosis — lips, tongue, mucous membranes, meaning the arterial blood itself is desaturated. Plus pallor and diaphoresis.

Air is not moving

ABSENT breath sounds • audible stridor. Noise requires airflow — a silent chest in a patient who was wheezing has got worse, not better.

No air to spare for speech

One- to two-word dyspnea. Count the words per breath. No equipment, no delay, and it tracks severity closely.

Compensating at maximum

Tachycardia ≥ 130 bpm • intercostal and sternocleidomastoid retractions • use of accessory muscles. The body spending everything it has.

Seven of the nine require no equipment at all

Which means this list is available in the first thirty seconds, in a dark bedroom, before a bag is opened. Commit it to memory — it is the highest-yield content in the assessment section, and it is the difference between recognising a dying patient and documenting one.

This list is not for documentation. It is for a decision.

Any one of these findings changes your tempo. It means you are MANAGING rather than assessing, you are moving toward the ambulance, and you are anticipating the airway — not waiting to see what the next set of vitals shows.

Secondary assessment — history and physical examination

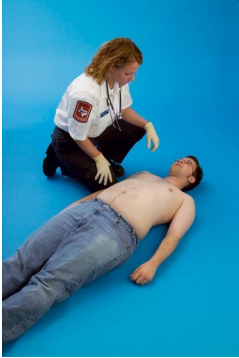


Figure 32-16 Inspection.



Figure 32-19 Auscultation.

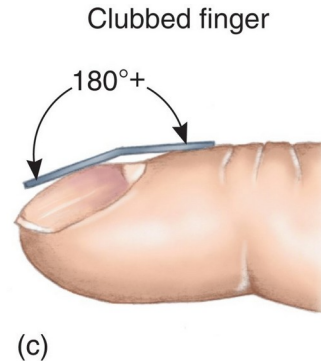


Figure 32-20 Clubbing of the fingers.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

A patient who cannot tell you their diagnosis will usually hand you a bag of bottles that does

Respiratory medications are disease-specific in a way most drug classes are not. Tiotropium suggests COPD. Anticoagulants raise both bleeding risk and the question of a prior clot. Take the bottles with you.

Clubbing tells you about the BASELINE, not the episode

It is a sign of chronic hypoxemia over years, and it is common in emphysema. Which matters when you are judging how sick this patient is compared with their own normal — not compared with yours.

1 SAMPLE and OPQRST — plus three

Has this happened BEFORE? Is there a KNOWN respiratory disease? And a full medication history, which the text calls essential.

2 Medications are unusually high-yield here

Home oxygen means chronic hypoxemia. Rescue plus controller inhaler means asthma. Prednisone in the house means this is not the first bad episode. Furosemide points at the heart, not the lungs.

3 And the question nobody asks

Have they been TAKING them? Non-adherence and a lapsed prescription are among the commonest causes of the exacerbation in front of you.

4 Four steps to the exam

INSPECTION · PALPATION · PERCUSSION of the chest wall · AUSCULTATION. Percussion gets skipped in the field — hyperresonance suggests pneumothorax, dullness suggests consolidation, but it needs quiet you rarely have.

Breath sounds — normal, and the six abnormal ones

Sound	Where it comes from	What it means
Snoring	Upper airway, soft tissue — usually the tongue	Partial obstruction. Fixed by POSITION, not by drugs
Stridor	Larynx or upper trachea — a narrowed rigid airway	Edema, foreign body, croup, epiglottitis, anaphylaxis. This one means AIRWAY EMERGENCY
Wheezing	Lower airway narrowing — bronchospasm, edema, secretions	Asthma and COPD — and heart failure, which also wheezes
Rhonchi	Secretions in the larger airways	Often CLEARS OR CHANGES with a cough — a useful bedside discriminator
Crackles	Fluid in alveoli, or collapsed alveoli popping open	Pulmonary edema, pneumonia, ARDS
Pleural friction rub	Inflamed pleural surfaces grating together	Leathery, and it accompanies pleuritic pain. Pneumonia and pulmonary embolism

Normal sounds — three of them

BRONCHIAL over the trachea, loud and harsh.
VESICULAR at the periphery, soft. BRONCHOVESICULAR between. Hearing bronchial sounds where you expect vesicular means CONSOLIDATED lung is transmitting — a pneumonia finding.

Diminished beats loud, and not in a good way

Absent or quiet sounds are worse than loud ones.
Noise requires airflow. This is the life-threat list restated, and it is worth hearing twice in one session.

And a CLEAR chest is a finding

Not the absence of one. A clear chest in a genuinely hypoxic patient points at PERFUSION — which is the case study, and which is why the framework earns its keep.

Three diagnostics — and what each one cannot tell you



Figure 32-21 Pulse oximetry.



Figure 32-26 Capnography.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 Pulse oximetry — fast, and blind to one thing

A rapid and accurate means of assessing saturation. But most oximeters CANNOT distinguish hemoglobin saturated with oxygen from hemoglobin saturated with CARBON MONOXIDE — which is why CO gets its own section in Part 3.

2 Peak flow — resistance, measured

Peak expiratory flow rate: how fast the patient can blow air out, so a direct measure of AIRWAY RESISTANCE. Technique: full inhalation, tight seal, one hard fast blast, best of three — compared against their own personal best.

3 And its limitation is the problem

It requires a COOPERATIVE patient who understands the device. So the sicker they are, the less likely you get a usable number — most useful in mild-to-moderate asthma, and useless in the patient you are most worried about.

4 Capnography — now standard of care

Continuous waveform capnography is becoming standard for breathing and non-breathing patients being ventilated. Essentially every protocol you have says “monitor capnography.”

Read the capnography lists against the framework and the mechanism falls out

DECREASED CO₂: shock · cardiac arrest · pulmonary embolism · bronchospasm · incomplete airway obstruction. INCREASED CO₂: hypoventilation · respiratory depression · hyperthermia.

Shock, arrest and PE are all PERFUSION failures — less blood delivering CO₂ to the lungs means less CO₂ exhaled. That is exactly why the woman in your case study has an ETCO₂ of 26 while breathing 30 times a minute.

REGION SOP Two of your own protocols carry the same pearl: “When assessing for cause of respiratory distress, CHF tends to be associated with LOWER levels of Capnography compared to COPD.” That is a discriminator you can apply on scene, in a patient who wheezes and could be either.

Each of these three answers a narrow question well and a wider one badly.

Management principles — and oxygen as a drug

1 The airway always has the first priority

Before diagnosis, before drugs, before everything. If it is compromised it gets corrected before you move on — which is why the primary assessment stops there.

2 Hypoxia, or the possibility of it, gets oxygen

Any patient with hypoxia receives oxygen. Any patient whose illness or injury SUGGESTS the possibility of hypoxia receives oxygen until pulse oximetry is available.

3 Titrate — including in COPD

“Administer ENOUGH oxygen to maintain an adequate SpO₂.” Not withhold. The hypoxic drive folklore — give a COPD patient less oxygen — has killed people. Titrate to a target; never restrict flow from a hypoxic patient.

4 Strive for NORMOXIA

Avoiding both hypoxia AND hyperoxia. Excess oxygen causes vasoconstriction and generates reactive oxygen species, and in post-arrest, stroke and MI it is associated with worse outcomes. It is not free.

“Oxygen should be treated like any other drug.”

Which implies a dose, a target, an indication, and side effects — and that giving more than needed is a medication error. Nobody would give three times the dose of a cardiac drug because the patient looked unwell. The same discipline applies here.

REGION SOP Routine Medical Care targets SpO₂ > 94%. Asthma/COPD targets 94–99%. Post-arrest: target 94–99% and “do not hyperoxygenate.” The textbook says typically ≥ 96% — both are titration instructions to a range, and both mean the same thing operationally.

What to carry out of Part 1

1 Three processes, three failures

VENTILATION moves air. DIFFUSION crosses the membrane. PERFUSION brings the blood. Every disease in this chapter breaks one or more, and the treatment follows from which.

2 Resistance and compliance split the room

A resistance problem struggles to get air OUT and wheezes. A compliance problem struggles to get air IN and breathes fast and shallow. Available at the bedside with no equipment.

3 A clear chest is a finding

Not the absence of one. Clear lungs with real hypoxia points at PERFUSION — and that patient responds least well to oxygen alone.

4 Nine findings mean dying

Altered mental status • severe central cyanosis • absent breath sounds • stridor • one-to-two-word dyspnea • HR ≥ 130 • pallor and diaphoresis • retractions • accessory muscle use. Seven need no equipment.

5 Every monitor is blind to something

The oximeter cannot see carbon monoxide. Peak flow needs a cooperative patient. Capnography reports CO₂ — which is as much about perfusion as ventilation.

6 Oxygen is a drug

It has a dose, a target, an indication and side effects. Titrate for normoxia — and never withhold it from a hypoxic patient to protect a drive.

The airway always has the first priority. Everything else is a question of which process broke.

This week: for every patient you see — respiratory complaint or not — decide from the doorway which of the three processes is working and which is not. Eight seconds, and it builds the pattern recognition the whole chapter depends on.

REVIEW

Check your understanding

- 1 Name the three processes that must work, and say in one line what each one does.
- 2 Distinguish intrinsic from extrinsic risk factors, name the most important of each, and say how the distinction changes your history.
- 3 What do the turbinates do, and by what mechanism?
- 4 What is wrapped around a bronchiole, how wide is it, and why does that one sentence explain asthma?
- 5 What three things make up the respiratory membrane, and name three diseases that work by altering it.
- 6 Contrast the pulmonary and bronchial circulations. Why does a pulmonary embolism usually NOT infarct the lung?
- 7 List the five structures that must be intact for ventilation to occur.
- 8 Define airway resistance and lung compliance. Which diseases break each, and what does each patient look like?
- 9 Diffusion is passive and free. Name the only three ways to break it.
- 10 Quote the sentence about a lung segment without blood flow — and say what it predicts about oxygen therapy in PE.
- 11 Which abnormal respiratory pattern is a CORRECTIVE measure, what is it correcting, and why must you not slow it?
- 12 List the nine findings suggesting a life-threatening respiratory problem. How many need no equipment?
- 13 Which breath sound finding is more ominous than loud wheezing, and why?
- 14 What can a standard pulse oximeter not distinguish, and what is the clinical consequence?
- 15 Name the five conditions associated with DECREASED capnography, and explain the mechanism in three of them.
- 16 What does “treat oxygen like any other drug” actually require of you?

Answer these before the session and again after — that gap is the measure of what you learned today.