VOLUME 1 · CHAPTER 22 · PART 1

Airway Management and Ventilation

Anatomy, Physiology, and Respiratory Assessment

The first priority of every patient contact — and the one you have four minutes to get right. Aligned to Region SOPs

THE CHAPTER

275 minutes, four parts — and where today sits

1 Foundations — today

Anatomy, physiology, respiratory problems, and assessment. 65 minutes. Nothing invasive.

2 Basic management

Positioning, oxygenation, manual maneuvers, basic adjuncts, and ventilation — everything achievable without a laryngoscope.

3 Advanced management

Extraglottic devices, endotracheal intubation, and special intubation considerations.

4 When it goes wrong

The difficult airway, medication-assisted intubation, cricothyrotomy, suctioning, gastric decompression, and transport ventilators.

Today — minute by minute

5 Case Study

Read now, solved at the end.

5 Introduction

Four minutes, and one governing idea.

15 Anatomy

Upper airway, lower airway, pediatric differences.

15 Physiology

Ventilation, diffusion, regulation, volumes.

5 Respiratory Problems

Obstruction and inadequate ventilation.

20 Assessment

Primary, secondary, and the monitoring technologies.

WHERE THIS FITS

Standard, competency, and your objectives

THE STANDARD

Airway Management, Respiration, and Artificial Ventilation

This is its own standard, separate from Assessment and from Medicine — because these decisions are made before the diagnosis is known. You do not need to know why the patient stopped breathing in order to know that they have.

THE COMPETENCY

Integrates complex knowledge of anatomy, physiology and pathophysiology into the assessment to develop and implement a treatment plan.

The verb is INTEGRATES, not recalls. Recall is the EMT expectation. Integration means holding this patient's anatomy, their physiology, your protocol, and your equipment in mind at once and producing a plan — then changing it when it fails.

1 Perform it

Execute the maneuver correctly on a manikin, in order, without prompting. This is what most programs teach.

2 Choose it

Select the right maneuver for this patient, this anatomy, this mechanism. This is what most programs test.

3 Abandon it

Recognise that the current plan is failing and change it BEFORE the patient decompensates. This is where airway patients are actually saved — and it is built entirely on today.

CASE STUDY

Read now. We solve it at the end.

Dispatch
Difficulty breathing, 0240 hours, private residence.

On arrival
A 61-year-old woman is sitting upright at the edge of her bed, tripoding. She answers you in single words. Her skin is cool and grey. Respirations are 40 and shallow, with visible accessory muscle use and intercostal retractions. Breath sounds are present bilaterally but very quiet, with no wheeze. HR 128, BP 154/88, SpO_2 91% on room air. Her husband says she has “bad lungs” and has been getting worse for three days.

Her airway is open. She is moving air. She is also dying.

Hold these questions:

1. Her airway is patent and she is breathing 40 times a minute. Why is that not reassuring?
2. What is her approximate minute volume — and how much of it is reaching an alveolus?
3. Her SpO_2 is 91%. What does that number tell you, and what does it not tell you?
4. What would a nonrebreather mask fix here, and what would it leave untouched?

Four minutes, and one governing idea

1 Four minutes

That is the window before brain injury or death when airway maintenance and ventilation fail. Every other priority in medicine is negotiable against a longer clock than this one.

2 It is a judgment, not a flowchart

Approach airway and ventilation problems globally, considering the whole picture rather than following predetermined steps. The steps are how you execute a decision — they are not the decision.

3 Oxygenation ≠ ventilation

Oxygenation is getting O_2 onto hemoglobin. Ventilation is moving air, which is really about clearing CO_2. Separate problems, separate solutions, separate monitors.

4 The basics carry the chapter

A correctly performed jaw thrust with a good mask seal beats a misplaced endotracheal tube every time. Without the basic skills, the advanced procedures are worthless.

Oxygen fixes oxygenation. Only moving air fixes ventilation.

A nonrebreather on a patient whose problem is carbon dioxide changes the number on the pulse oximeter and nothing else. Merging these two ideas is the most common conceptual error in the whole chapter — and the one with the highest body count.

Anatomy of the Respiratory System

From the nares to the alveolus — and the handful of structures you will be looking for with a laryngoscope in your hand.

Upper airway · Lower airway · Pediatric differences

ANATOMY

The upper airway — mouth and nose to the larynx

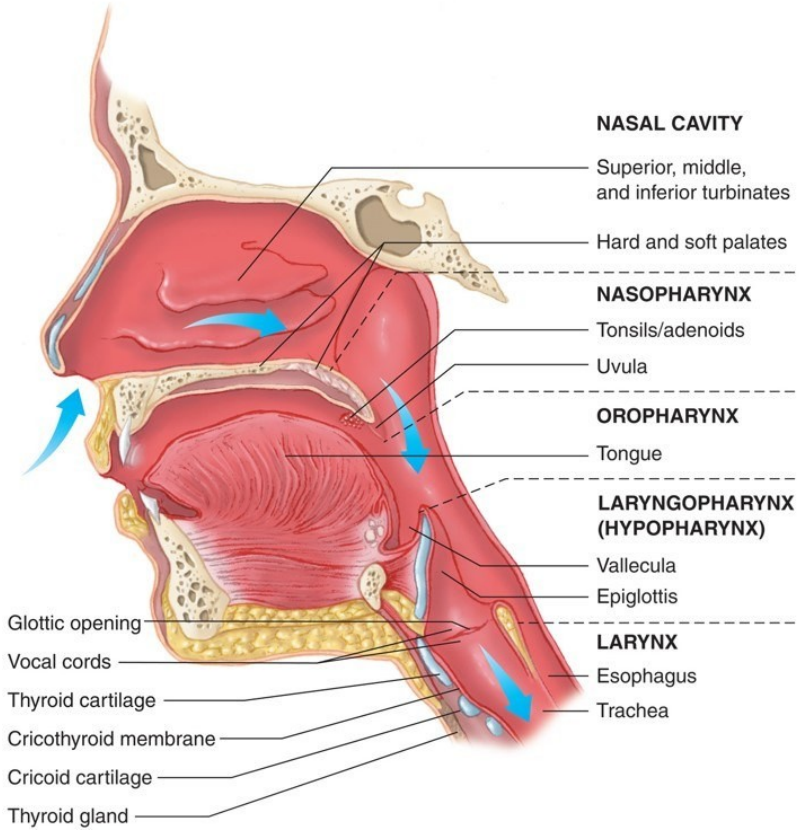

Figure 22-1 Anatomy of the upper airway.

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

The nose — filter, warm, humidify

Nasal hairs filter at the nares; turbulence deposits particles onto the mucosa; a RICH blood supply warms and humidifies immediately. That rich supply is exactly why nasal airways and nasal intubation bleed.

What else lives there

Sinuses, eustachian tubes, and lacrimal ducts open into the nasal cavity — which is why a nasal airway is contraindicated with significant facial injury or suspected basilar skull fracture.

The mouth — and the tongue

Cheeks, hard and soft palates, and tongue form the oral cavity. The TONGUE is the most common airway obstruction, and every basic maneuver in Part 2 exists to move it.

The hyoid bone

Just beneath the chin. The only bone in the axial skeleton that articulates with no other bone — and the landmark that defines where the oropharynx ends.

The pharynx, in three parts

Nasopharynx, oropharynx (soft palate to hyoid), and laryngopharynx (hyoid to esophagus posteriorly, larynx anteriorly). The laryngopharynx is the one that matters most in airway management.

A dual-purpose tube

The mouth and pharynx serve respiration AND digestion. Every protective mechanism on the next slide exists to manage that compromise — and every one of them can be defeated.

ANATOMY

The larynx — and the landmarks you will hunt for

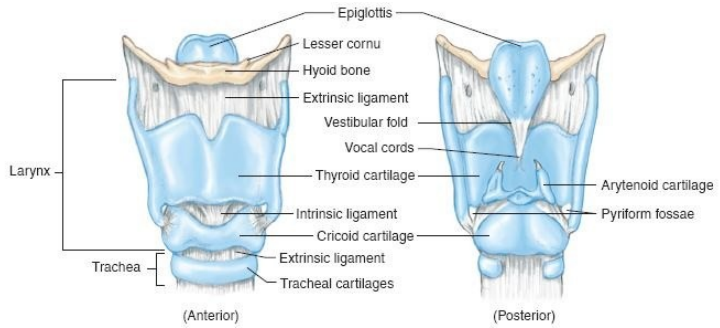

Figure 22-2 Anatomy of the larynx, anterior and posterior.

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

REGION SOP Assess for a positive gag reflex before proceeding. An intact gag reflex is a listed contraindication to the i-gel — its presence routes you into the Drug Assisted Intubation protocol instead.

1 Epiglottis

Leaf-shaped cartilage; closes the airway during swallowing. THE landmark: a straight blade goes under it and lifts it, a curved blade stops short of it. Find this and you will usually find the cords.

2 Vallecula

The fold between the base of the tongue and the epiglottis, just anterior and superior to it. This is where the tip of a Macintosh blade goes. Demonstrate it on the model — do not just name it.

3 Glottic opening and cords

Housed by the thyroid cartilage. The true vocal cords lie within the laryngeal cavity, and the glottis is the NARROWEST part of the adult trachea — which reverses in children.

4 Cricoid cartilage

Beneath the thyroid cartilage, in front of the esophagus. Considered the first tracheal ring and the only COMPLETE ring in the airway.

5 Cricothyroid membrane

The soft depression between thyroid and cricoid. Find it on yourself, then on a partner — it is where the scalpel goes in Part 4.

6 The gag reflex

Prevents foreign material entering the trachea. Its presence or absence is the gating decision for an OPA and for the i-gel — and your SOP asks you to assess it explicitly.

ANATOMY

The lower airway — larynx to alveolus

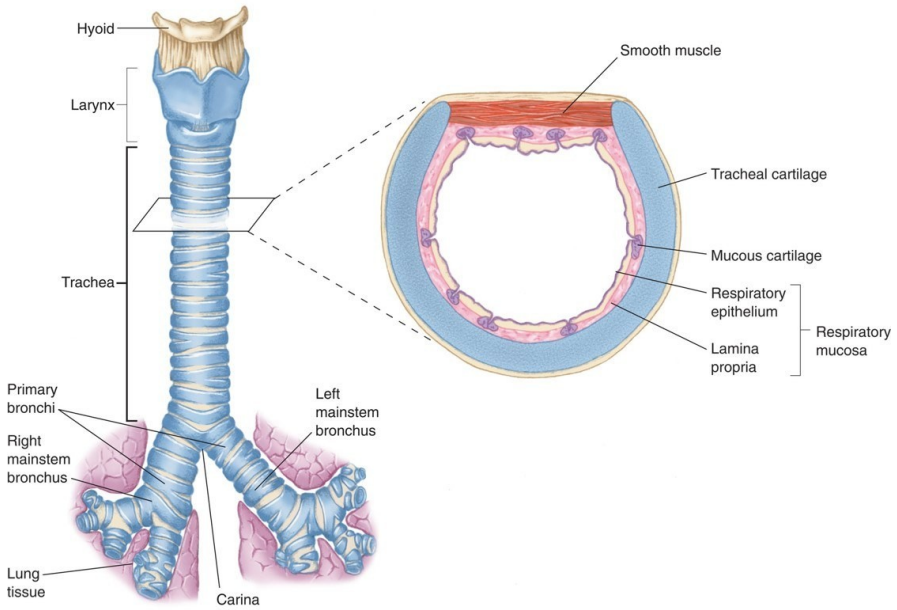

Figure 22-3 The lower airway, with tracheal cross section.

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

Unilateral breath sounds on the LEFT after intubation: the tube is in the right mainstem. Withdraw it — the reflex to advance is the wrong one, and it is common.

Trachea

C-shaped cartilage rings with the open side facing posteriorly, against the esophagus. Respiratory epithelium and mucus trap what the upper airway missed — all of which intubation bypasses.

The carina, and an asymmetry that matters

The right mainstem is almost STRAIGHT; the left angles away. So: a tube advanced too far goes right, an aspirated object goes right, and absent left breath sounds after intubation means pull back, not push.

Bronchi to bronchioles

Mainstem bronchi enter at the hilum, divide into secondary and tertiary bronchi, then bronchioles. The respiratory bronchioles terminate at the alveoli.

Lobes

Right lung: upper, middle, lower. Left lung: upper and lower only — the heart takes the space. This is what you are listening under during auscultation.

Pleura — and why it hurts

Visceral pleura envelops the lung and has NO nerve fibres. Parietal pleura lines the thoracic cavity and is rich with them. A lung can be badly injured without pain until the parietal surface is involved.

The pleural space

A POTENTIAL space with a small amount of lubricating fluid. It becomes a real space only in pneumothorax and hemothorax — which is the whole basis for needle decompression.

ANATOMY

The alveolus — where the entire point of all this happens

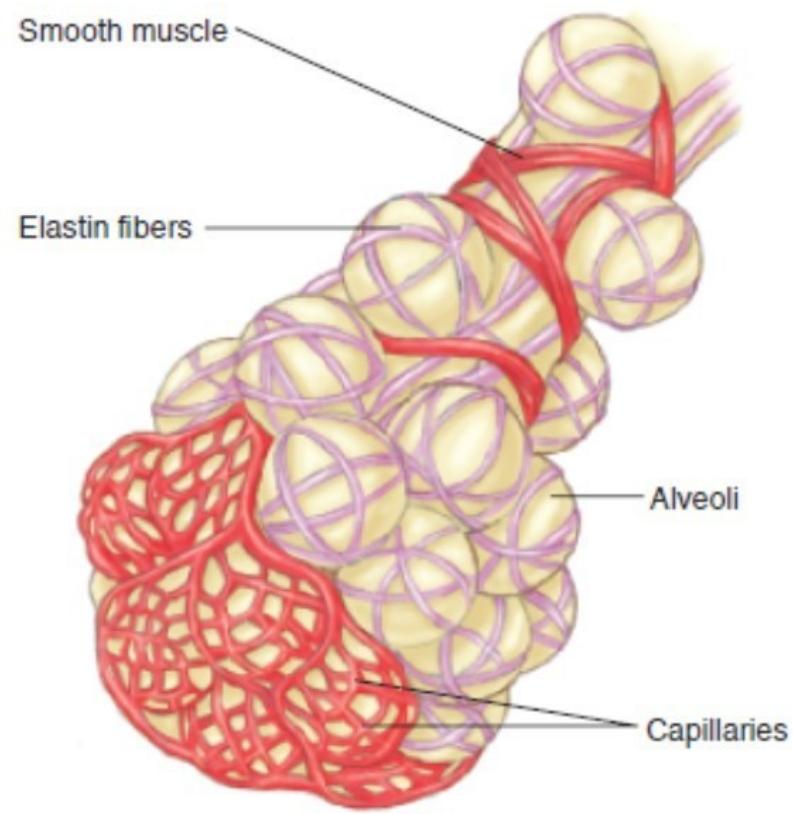

Figure 22-4 Anatomy of the alveoli.

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

1 One or two cells thick

Thinness IS the function. Diffusion distance determines how fast gas moves, so anything that thickens the membrane — pulmonary edema, pneumonia, fibrosis — impairs oxygenation directly and mechanically.

2 More than 40 square metres

Roughly the floor area of a small studio apartment, folded into a chest cavity. That surface area is the entire engineering achievement of the lung.

3 Surfactant

Decreases surface tension so alveoli resist collapse and expand more easily. It explains neonatal RDS, it explains atelectasis in a patient not breathing deeply, and it is why PEEP and CPAP work — keeping alveoli open is cheaper than reopening them.

4 Ventilation must meet perfusion

Air and blood have to arrive at the SAME alveolus. An alveolus with no blood flow, or blood flowing past a collapsed alveolus, both produce a patient who appears to be breathing and is not exchanging.

ANATOMY

The pediatric airway — same design, different tolerances

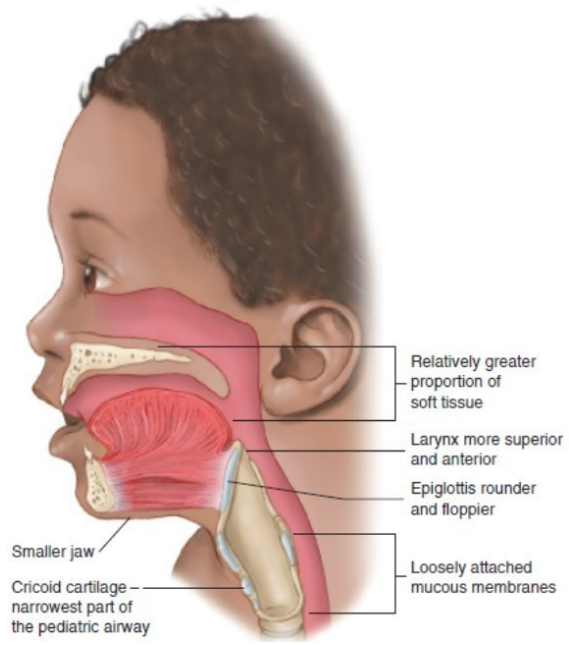

Figure 22-5 The pediatric airway.

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

The anatomic difference	What it does to you
Smaller jaw, relatively larger tongue	Obstructs far more readily. Positioning and a correctly sized adjunct matter more than they do in an adult
Rounder, floppier epiglottis	Harder to control indirectly — which is why a STRAIGHT blade is usually preferred in children
Larynx more superior and anterior, funnel-shaped	The view is genuinely different — expect adult geometry and you will look too low
Cricoid cartilage is narrowest, not the glottis	A tube that passes the cords can still be too large. Cricoid pressure can worsen the situation rather than help
Softer, more pliable ribs and thoracic cage	Significant intrathoracic injury with no rib fractures — and less force available for an effective cough
Relatively large occiput	The head flexes when supine on a flat surface. Padding goes under the SHOULDERS, not the head

Higher basal metabolism plus a smaller functional residual capacity means children desaturate dramatically faster. Preoxygenation buys far less time than it does in an adult — and infants have greater vagal tone, so laryngoscopy produces bradycardia readily. Watch the monitor during the attempt, not just the tube.

REGION SOP The pediatric drug-assisted airway procedure specifies padding under the SHOULDERS during BVM ventilation — that instruction exists because of the occiput.

Physiology of the Respiratory System

Why air moves, how gas crosses, what tells you to breathe, and the volumes that turn a respiratory rate into a meaningful number.

Ventilation · Diffusion · Regulation · Volumes

PHYSIOLOGY

Respiration and ventilation are not the same word

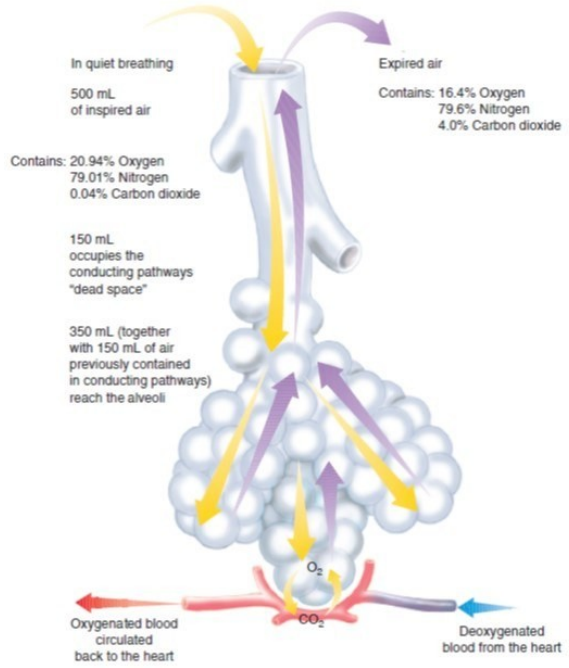

Figure 22-6 Respiratory gases exchanged between alveoli and red blood cells.

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

Respiration

The EXCHANGE of gases. Pulmonary (external) respiration happens between alveoli and pulmonary capillary blood. Cellular (internal) respiration happens between red cells and the tissues — and that is where CO_2 is produced.

Ventilation

The MECHANICAL movement of air in and out. A patient can ventilate beautifully and respire terribly: carbon monoxide moves air, raises the chest, reads a high saturation, and delivers no oxygen to a cell.

Inspiration is active

Brain → phrenic nerve (C3, C4, C5) → diaphragm contracts and descends. Intercostals draw the ribs up and out. The cavity enlarges, pressure falls BELOW atmospheric, and air is invited in. The lung does not suck.

Expiration is passive

Muscles relax, the cavity shrinks, pressure rises, and air leaves. It costs nothing. This asymmetry is why a fatiguing patient fails on inspiration — and why obstructive patients, who must WORK to exhale, tire so profoundly.

At full inflation, stretch receptors signal the vagus to inhibit inspiration — a genuine safety stop. A bag-valve mask bypasses it entirely, which is the origin of every barotrauma argument in Part 2.

PHYSIOLOGY

Pulmonary circulation, partial pressures, and diffusion

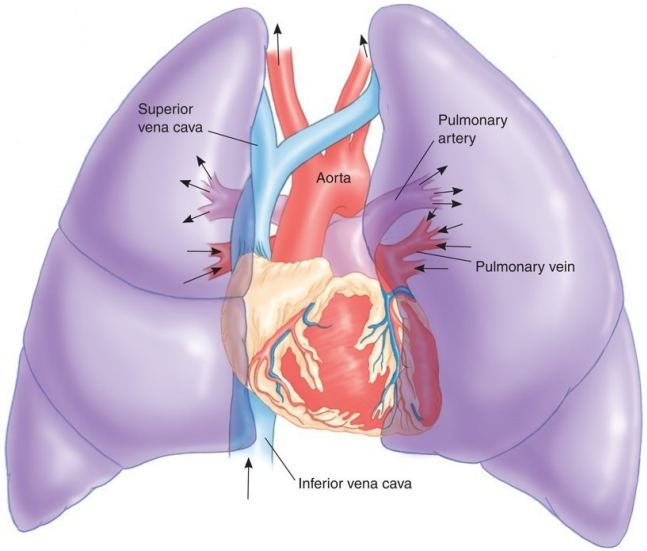

Figure 22-7 Pulmonary circulation.

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

Two ways to mismatch

Ventilation without perfusion is DEAD SPACE — pulmonary embolism is the pure form. Perfusion without ventilation is SHUNT — atelectasis and consolidation. Both cause hypoxemia; neither is fixed by turning up the oxygen alone.

1 The circuit — and the naming trap

Tissues take O_2 and give up CO_2 → venous return to the right heart → out the pulmonary ARTERY (deoxygenated) → lungs → back via the pulmonary VEIN (oxygenated) → left heart → systemic. This is the ONE place where artery and vein carry the opposite of what you expect.

2 Partial pressure

The pressure exerted by one gas in a mixture. Oxygen is 21% of an atmosphere of 760 torr, so inspired $PO_2 = 0.21 \times 760 = 159.6$ torr. Altitude does not change the percentage — it changes the pressure.

3 Diffusion

Gas moves from higher partial pressure to lower, seeking equilibrium. It is passive and free. So the only ways to break it are: change the gradient, thicken the membrane, or take away the blood flow.

4 Which gives you the whole differential

Alveolar and arterial pressures are essentially equal in a NORMAL lung. When they diverge, that divergence is itself the diagnosis — and it points at one of those three failures.

PHYSIOLOGY

Oxygen in the blood — and the curve behind the oximeter

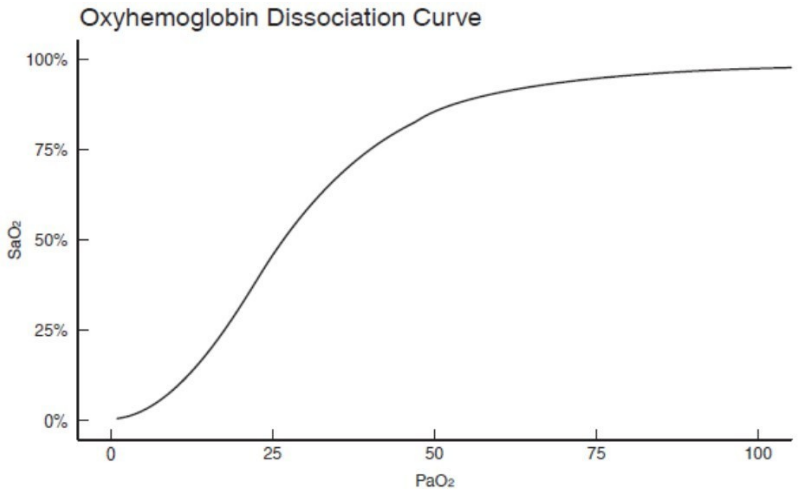

Figure 22-8 The oxyhemoglobin dissociation curve.

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

The shape IS the message

Above roughly 90% the curve is FLAT: large falls in PaO_2 barely move the saturation, so the oximeter looks reassuring while the patient deteriorates. Below 90% it turns STEEP: small further falls produce large drops, and the patient falls down the curve fast.

So a saturation going 99 → 94 is a far bigger physiologic change than it reads. And 90% is standing at the edge of a cliff. Act on the slope and the trend, not the number.

What lowers blood oxygen	And what actually fixes it
Decreased hemoglobin — anemia, hemorrhage	More oxygen does very little. Fix the hemoglobin
Inadequate alveolar ventilation — low inspired O_2, muscle paralysis, emphysema, asthma, pneumothorax	INCREASE VENTILATION. Positive pressure, not a mask
Decreased diffusion — pulmonary edema, pneumonia, COPD	Supplemental oxygen and pressure (CPAP), plus drugs for the underlying cause
V/Q mismatch — collapsed alveoli, as in atelectasis	Recruit the alveoli. PEEP and CPAP, not flow

97% of oxygen is bound to hemoglobin. Only one of these four fixes is “put on a mask.”

PHYSIOLOGY

Carbon dioxide — the gas that actually drives you

1 How it travels

About 70% as bicarbonate ion (HCO_3^-), about 23% bound to hemoglobin, and under 7% dissolved in plasma. That bicarbonate figure is the hinge between respiratory and metabolic acid–base.

2 Production rises with

Fever, muscle exertion, shivering, and metabolic processes producing acids. Shivering is the one people miss — in the hypothermic or post-arrest patient it is a large, unrecognised CO_2 load.

3 Elimination falls with

Anything that reduces alveolar ventilation: respiratory depression by drugs, airway obstruction, respiratory muscle impairment, and obstructive disease such as asthma and emphysema.

4 Hyperventilation is about CO_2, not rate

It lowers CO_2 through increased rate OR increased depth. Which means a patient can breathe 40 times a minute and be HYPOventilating — exactly like the woman in your case study.

This slide is the reason capnography is a VENTILATION monitor

A pulse oximeter answers “is oxygen bound to hemoglobin?” A capnograph answers “is air actually moving?” They are different questions with different answers, and a patient can pass one while failing the other. Everything on this slide is what the capnograph is watching.

REGION SOP Your head and spinal injury protocols target a CAPNOGRAPHY VALUE of 35–40 mmHg for signs of raised ICP — not a respiratory rate. The number is what matters to the brain; the rate is only how you get there.

PHYSIOLOGY

What tells you to breathe

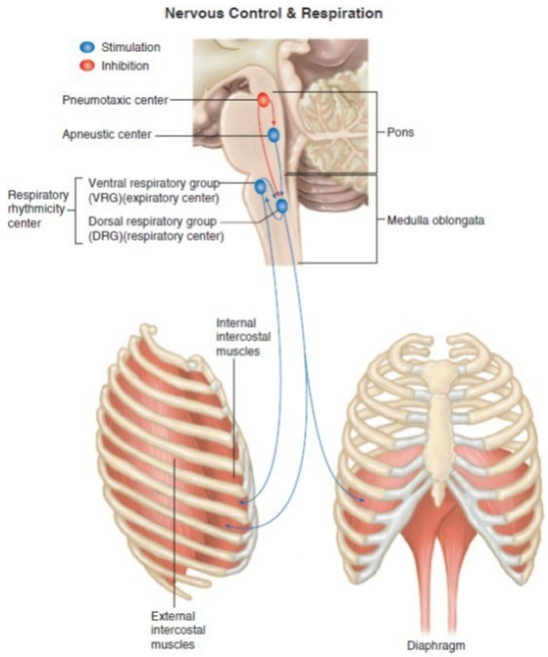

Figure 22-9 Nervous control of respiration.

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

Normal rates

Adults 12–20 · Children 18–24 · Infants 40–60

The medulla

The respiratory centre. Stretch receptors feed back into it to INHIBIT respiration at full inflation.

The chemoreceptors

In the medulla, the carotid bodies and the aortic arch. Stimulated by decreased PaO_2, increased $PaCO_2$, and decreased pH.

CO_2 is primary

Sensed centrally, largely via CSF pH. This is your normal, minute-to-minute drive to breathe.

O_2 is secondary

The hypoxic drive, sensed peripherally. A backup system — and the source of a great deal of prehospital folklore.

Hypoxic drive — what the guidance actually says

"Titrate oxygen to maintain a slightly lower pulse oximeter reading; the flow of oxygen needed to do that does not matter."

Read that twice. The instruction is to titrate to a TARGET — and explicitly NOT to restrict flow. There is no patient in whom the right action is to let a saturation fall in order to protect a drive. The folklore version of this concept has killed people.

REGION SOP Rate is a weak vital sign on its own. Fever, emotion, pain, hypoxia, acidosis, stimulants, depressants and sleep all move it — and at least four of those have nothing to do with the lungs.

PHYSIOLOGY

Lung volumes — what turns a respiratory rate into a real number

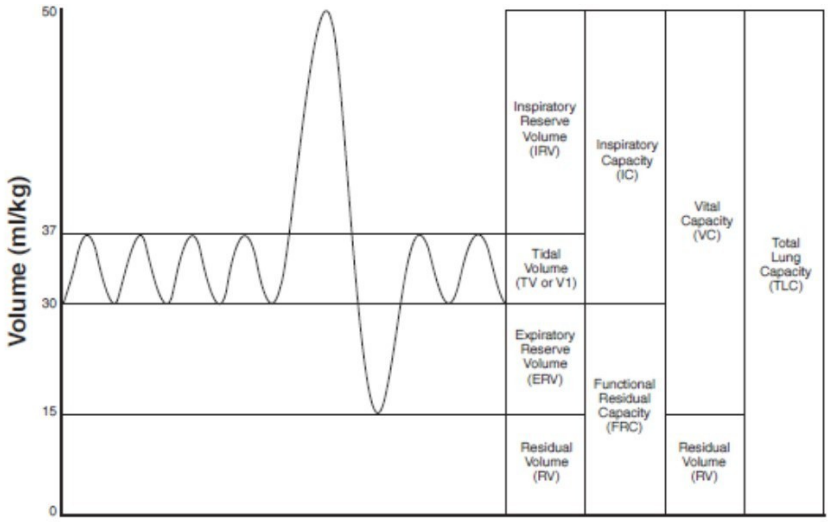

Figure 22-10 Respiratory volumes and capacities.

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

Volume	Adult value	Why you care
Tidal volume	~500 mL (5–7 mL/kg)	The size of one normal breath
Dead space volume	~150 mL	Gas that never reaches an alveolus
Alveolar volume	~350 mL	The part that actually exchanges
Minute volume	Tidal × rate	What you can measure at the bedside
Alveolar minute volume	Alveolar × rate	What the patient actually gets
Functional residual capacity	Volume left after a normal exhale	Your oxygen reservoir during apnea — what preoxygenation fills

Two patients. Identical minute volume. Very different patients.

A: 500 mL × 12 = 6,000 mL/min. Alveolar: 350 × 12 = 4,200 mL.
B: 200 mL × 30 = 6,000 mL/min. Alveolar: 50 × 30 = 1,500 mL.

Same minute volume. Barely a third of the effective ventilation — because dead space is subtracted from EVERY breath, so a fast shallow pattern pays that tax over and over.

Fast and shallow is not compensation. It is the most efficient way to move air without ventilating.

5 MINUTES

Respiratory problems — two failures, not one

1 Obstruction — the loud failure

Interference with air movement through the upper airway; may be partial or complete. Causes: the tongue, foreign bodies, teeth, spasm, trauma, edema, vomitus, and blood. An immediate threat to life and a true emergency.

2 Inadequate ventilation — the quiet failure

Insufficient minute volume compromising both oxygen intake and CO_2 removal. Nothing to pull out, no noise, and an airway that looks entirely fine. This is the one that gets missed.

Sort obstructions by what fixes them — not by what they are

Position fixes it

The tongue. Head-tilt/chin-lift or jaw thrust, plus an adjunct. This is the most common obstruction there is.

Suction fixes it

Vomitus, blood, secretions. Limited to 10 seconds, and the patient is oxygenated before and after.

Removal fixes it

Foreign bodies and teeth. Thrusts, direct visualisation, Magill forceps — or bypass it into a mainstem bronchus.

Only drugs or a knife fix it

Edema and spasm. Neither suction nor positioning touches these, and this is exactly where providers waste the time they do not have.

Partial obstruction makes noise. COMPLETE obstruction is silent. A previously stridulous patient who has gone quiet has got worse, not better.

REGION SOP The Airway Obstruction protocol escalates along exactly these lines: let the patient clear it → back blows and thrusts → direct visualisation with video laryngoscopy and Magill forceps → if past the cords, intubate and deliberately advance the object into the right mainstem, then withdraw and ventilate the left lung → suction ≤ 10 seconds → cricothyrotomy.

Respiratory System Assessment

The largest block in Part 1 — and the only one you will use on every single patient, including the ones with no airway complaint.

Primary · Secondary · Auscultation · Monitoring technology

ASSESSMENT

Primary assessment — the sixty-second version

1 Airway — is it patent?

Look and listen. Snoring, gurgling and stridor are all partial obstruction with a name. Silence in a patient who should be making noise is worse, not better.

2 Breathing — is it ADEQUATE?

Not “is it present.” Adequacy has four properties: rate, depth, effort, and effectiveness. A patient can be breathing 40 times a minute and be inadequate on three of the four.

3 Look at the chest wall

Asymmetrical movement — paradoxical breathing — means a flail segment or a serious ventilatory problem. It is visible from the doorway if you are looking, and most people are not.

4 Then act, immediately

Not breathing or suspected airway problem → open it, head-tilt/chin-lift or jaw thrust. Breathing adequate → supplemental oxygen, move to circulation. Breathing inadequate or absent → begin artificial ventilation.

Chest rise is continuous feedback, not a one-time check

Whenever you assist ventilation with a device, place an adjunct, or intubate, watch the chest rise and fall to confirm it is working. It costs nothing, it needs no equipment, and it is the fastest way to catch a problem that every monitor in this chapter will confirm more slowly.

ASSESSMENT

Secondary assessment — inspection and respiratory patterns

Pattern	What it looks like	What it usually means
Kussmaul	Deep, rapid, laboured — sustained	Metabolic acidosis being compensated. Classically DKA. The patient is blowing off CO_2 on purpose
Cheyne–Stokes	Crescendo–decrescendo depth with apneic pauses	Brainstem injury, raised ICP, or severe heart failure
Biot (ataxic)	Irregular, unpredictable, with irregular pauses	Significant brainstem injury or raised ICP. More ominous than Cheyne–Stokes
Central neurogenic hyperventilation	Sustained, deep, rapid — no pauses	Midbrain or upper pons injury
Agonal	Slow, gasping, ineffective	This is NOT breathing. This patient is in arrest — and both lay callers and new providers describe it as breathing

Inspection comes first

Skin colour, dyspnoea, rate, and accessory muscle use — all available before you touch the patient.

Mental status IS a respiratory sign

Confusion, agitation and combativeness are hypoxia or hypercarbia until proven otherwise. And a patient who suddenly calms has often not improved.

Modified forms of respiration

Coughing, sneezing, hiccoughing, sighing, and grunting. GRUNTING is auto-PEEP — exhaling against a partly closed glottis to hold alveoli open. It signals real distress.

ASSESSMENT

Auscultation and palpation — the two free skills

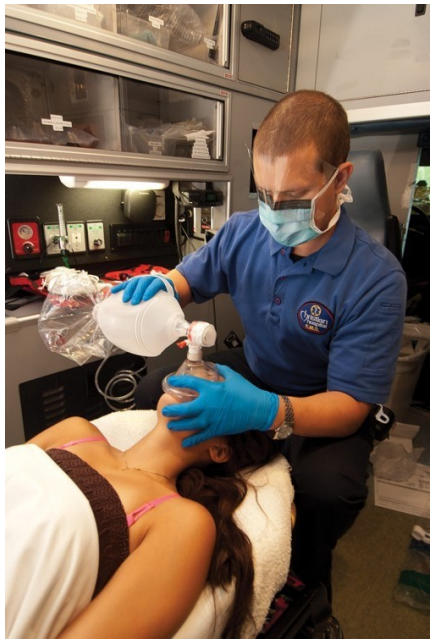

Figure 22-15 Assessing ventilation.

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

1 Start at the mouth and nose

Listen for adequate air movement before you reach for the stethoscope. This step gets skipped and it is the fastest information on scene.

2 Then six fields, compared

Left and right apex, left and right base, and the thoracic back. Listen to the SAME location on both sides before changing location — the comparison is the diagnostic act.

3 Sort the sounds by location

Snoring and gurgling: upper airway — position or suction. Stridor: laryngeal — edema or foreign body. Wheeze: lower airway narrowing. Crackles: fluid or alveoli reopening. Rhonchi: secretions in large airways.

4 Then put a hand on the bag

If you are ventilating, gauge compliance. Rising resistance means obstruction, tension pneumothorax, bronchospasm, or a displaced tube.

Quiet, diminished or absent breath sounds are an OMINOUS finding

Noise requires airflow. The severe asthmatic who stops wheezing has not improved — they have stopped moving enough air to make a sound, and they are close to arrest. The same is true of the stridulous patient who goes quiet. Louder is often better; silence is the emergency.

ASSESSMENT

Pulse oximetry — what it measures, and the four ways it lies

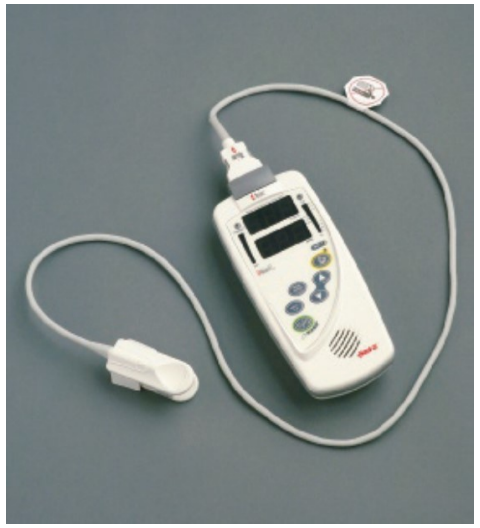

Figure 22-17 Pulse oximeter.

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

What it actually reports

The percentage of hemoglobin binding sites that are OCCUPIED. Not delivery. Not ventilation. Not use. Normal is 96–99% at sea level.

1 It cannot tell WHAT is bound

Two wavelengths distinguish two things. Carboxyhemoglobin reads as oxyhemoglobin — a CO patient can read 100% while suffocating. Methemoglobin pulls the reading toward the mid-80s regardless of truth.

2 It needs a pulse

Hypoperfusion, hypothermia, vasoconstriction and hypotension degrade or destroy the signal. In shock, the ABSENCE of a reading is itself a finding worth reporting.

3 It is a late warning

The flat top of the oxyhemoglobin curve means the number stays reassuring while PaO_2 falls a long way. Hold your breath and watch how long it takes to move — that lag is the whole lesson.

4 And it is easily fooled

Motion, nail polish, dirt, bright ambient light, and a badly placed probe. Cheap failures, but they produce confident numbers.

A normal saturation says NOTHING about ventilation. A patient on high-flow oxygen can sit at 100% while their CO_2 climbs into narcosis — which is the everyday opioid overdose, and the entire argument for the next slide.

REGION SOP "Pulse oximetry may give falsely elevated readings in patients with methemoglobin or CO / CN exposure." Your asthma/COPD protocol targets SpO_2 94–99% — a titration instruction, not a maximise-the-flow one.

Pulse CO-oximetry — more wavelengths, more answers

Figure 22-18 Pulse CO-oximetry — SpCO, SpMet.

Figure 22-19 Total hemoglobin (SpHb).

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

SpCO	What the patient looks like
< 5%	Normal for a non-smoker
5–9%	Minor headache — and may be NORMAL for a smoker
10–19%	Headache, shortness of breath
20–29%	Headache, nausea, dizziness, fatigue
30–39%	Severe headache, vomiting, vertigo, altered LOC
40–49%	Confusion, syncope, tachycardia
50–59%	Seizures, shock, apnea
> 59%	Coma, death, cardiac dysrhythmias

Two wavelengths, two answers. More wavelengths, more answers.

The conventional oximeter is not merely unhelpful in carbon monoxide poisoning — it is actively misleading, reporting a normal or high number in a patient being asphyxiated. CO-oximetry is the only prehospital tool that catches it.

A smoker's baseline sits where anyone else would be abnormal — so a smoker with a genuine exposure starts from a higher floor and reaches dangerous values sooner. And in a fire, consider CYANIDE: oxygen arrives normally and the cell cannot use it. Neither oximeter sees that one.

REGION SOP CO / smoke inhalation: remove from the environment, OXYGEN 100% by NRB or support respirations with a BVM, monitor capnography, and hold a high index of suspicion with a LOW intubation threshold when airway involvement is possible.

ASSESSMENT

Capnography — the vocabulary, then the devices

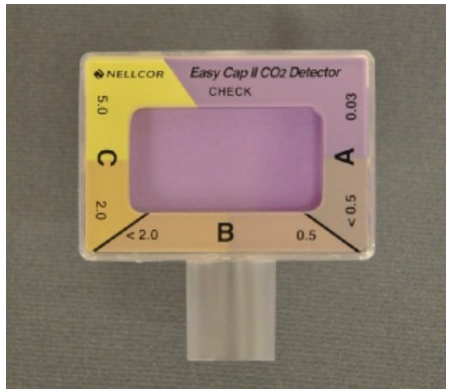

Figure 22-21 Colorimetric $ETCO_2$ detector.

Figure 22-23 Capnography via nasal cannula.

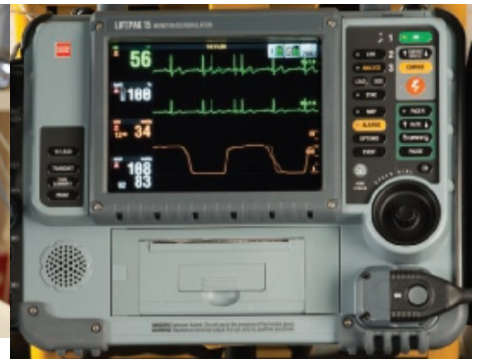

Figure 22-25 Waveform capnography.

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

Term	What it means
Capnometry	The measurement of exhaled CO_2
Capnography	The graphic recording or display
Capnograph	The device itself
Capnogram	The waveform it draws
$ETCO_2$ / $PETCO_2$	The end-tidal value — what you read
$PaCO_2$	The ARTERIAL value — from a blood gas
End-tidal gradient	The difference between the two, which widens with dead space, PE and poor perfusion

Mainstream

Sensor sits in the airway circuit. No lag, fast response — but heavier at the tube and easier to damage.

Sidestream

Draws a sample down a line to the monitor. Lighter, and usable on NON-intubated patients via nasal cannula — with a short lag, and a line that can occlude.

Colorimetric

pH-sensitive paper with a colour scale. QUALITATIVE only — presence, not amount. No number, no trend, no waveform. A backup, not a monitor.

Water in the line

The single most common reason a good waveform disappears. Check the line before you doubt the patient.

REGION SOP Capnography is the stated GOLD STANDARD for confirming placement after intubation, after i-gel insertion, and after both needle and surgical cricothyrotomy — and continuous waveform monitoring is required for any patient receiving sedation or pain medication.

ASSESSMENT

The normal capnogram — four phases

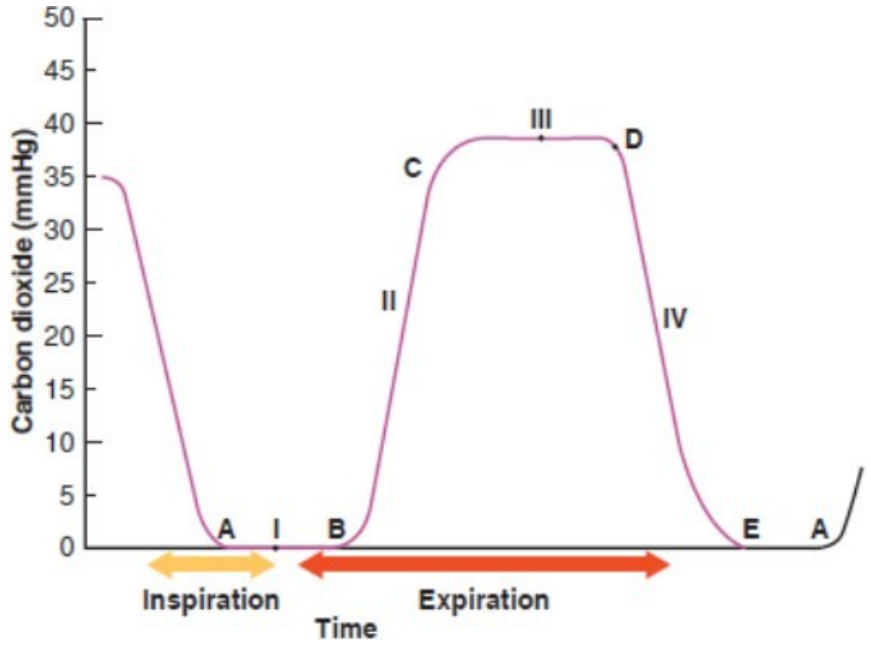

Figure 22-26 The normal capnogram, with phases I–IV.

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

I Respiratory baseline

End of inhalation. Should sit at ZERO — inhaled gas contains essentially no CO_2. Any elevation here means rebreathing.

II Respiratory upstroke

Beginning of exhalation, as dead space gas is replaced by alveolar gas. Should be STEEP. Sloping means obstruction to flow.

III Respiratory plateau

Full exhalation — and the point at which $ETCO_2$ is measured. Should be flat and slightly rising. Normal value 35–45 mmHg in an adult.

IV Inspiratory phase

Beginning of inhalation. Should fall sharply back to zero. A phase IV that does not reach zero means rebreathing or a faulty valve.

Four questions that let you read any capnogram you have never seen before

Is there a waveform at all? What is its SHAPE? What is its HEIGHT — the number? What is its TREND over time?

Height without shape is nearly worthless. And in an arrest: a waveform of any size with a normal shape means the tube is in the trachea. A low number reflects poor pulmonary blood flow — not a bad tube. Misreading that pulls correctly placed tubes.

ASSESSMENT

Reading the waveform — six patterns worth recognising

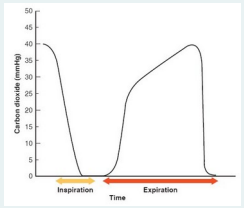

Figure 22-28 · Shark fin — obstruction

Sloped upstroke, rising plateau. Alveoli emptying at different rates: asthma, COPD, bronchospasm. The SHAPE improves before the number does, so it tells you the bronchodilator is working before the breath sounds do.

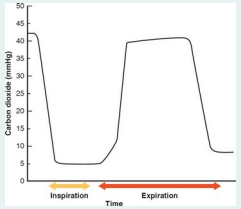

Figure 22-29 · Elevated baseline — rebreathing

Phase I fails to reach zero. Inadequate expiratory time, faulty exhalation valve, or exhausted absorber. In the field it usually means you are ventilating too fast for the patient to fully exhale.

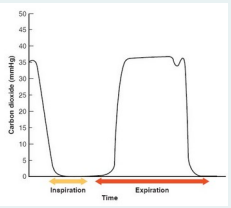

Figure 22-30 · Curare cleft

A notch in the plateau — a spontaneous breath breaking through as neuromuscular blockade wears off. Meaning: your paralysed patient is waking up. If they are paralysed without adequate sedation, that is urgent.

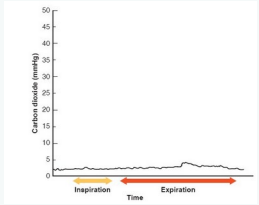

Figure 22-31 · No waveform — the emergency

Esophageal intubation, disconnection, complete obstruction, or arrest with no circulation. A flat line means find out which of those it is, right now. The most important single pattern in this chapter.

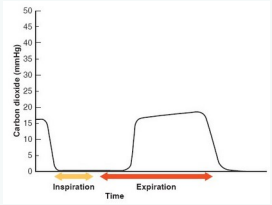

Figure 22-33 · Persistently low $ETCO_2$

V/Q mismatch, hyperventilation, low cardiac output, pulmonary embolism. In arrest, a RISING value is the earliest sign of ROSC — often before a palpable pulse.

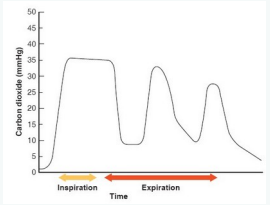

Figure 22-32 · Leak or variable waveform

Irregular, variable amplitude. Cuff leak, partially displaced tube, or a leak in the circuit. Check the equipment before you re-diagnose the patient.

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

ASSESSMENT

Rate and depth on the waveform — and what the SOP does with it

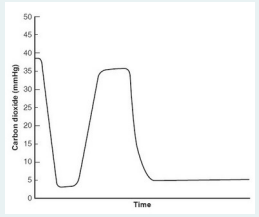

Figure 22-34 · Apnea

A flat line after normal waveforms. The patient has stopped breathing — and the capnograph shows it within ONE missed breath, where the pulse oximeter takes a minute and only after they are already hypoxic.

Figure 22-35 · Hyperventilation

Falling $ETCO_2$ with a normal shape at an increased rate. Diagnosed by the NUMBER and the TREND, with the shape intact — the reverse of the obstructive patterns.

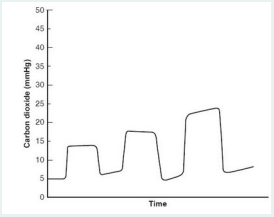

Figure 22-36 · Hypoventilation

Rising $ETCO_2$. Depressed drive, fatigue, or inadequate assisted ventilation. Again: number and trend, shape intact.

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

Why every sedated patient gets a waveform

Give fentanyl, midazolam or ketamine and the patient stops breathing: the capnograph shows it immediately, the pulse oximeter about a minute later — after they are already hypoxic.

The oximeter tells you they became hypoxic. The capnograph tells you they stopped breathing. Only one of those gives you time.

And why the head injury target is a NUMBER, not a rate

Aggressive hyperventilation constricts cerebral vessels and worsens ischemia. So the goal is a narrow window — and the only way to hit a narrow window is to measure it rather than guess at a rate.

REGION SOP Signs of raised ICP: ventilate 1 breath every 2–3 seconds to a CAPNOGRAPHY value of 35–40 mmHg. Separately — when sorting out the cause of respiratory distress, CHF tends to be associated with LOWER capnography values than COPD.

SUMMARY

What to carry out of Part 1

1 Patency and adequacy are different questions

An open airway is not an adequately ventilated patient. The case study is a patent airway, a good respiratory rate, and a dying patient — all at once.

2 Oxygenation ≠ ventilation

Oxygen fixes oxygenation. Only moving air fixes ventilation. Merging these two ideas is the most expensive conceptual error in the chapter.

3 Dead space is charged on every breath

Which is why fast and shallow moves air without ventilating. Minute volume is what you can measure; ALVEOLAR minute volume is what the patient gets.

4 The oximeter is a late warning

The flat top of the curve keeps the number reassuring while PaO_2 falls. And it cannot tell you WHAT is bound — which is why CO poisoning reads normal.

5 Capnography is the ventilation monitor

It shows apnea within one breath, tells you a bronchodilator is working before the breath sounds do, and is your system's gold standard for every tube you place.

6 Silence is the emergency

Quiet, diminished or absent breath sounds are an ominous finding. Noise requires airflow — the asthmatic who stops wheezing has got worse, not better.

Four minutes. Everything in this chapter is an argument about how to spend them.

This week: assess ten people with NO respiratory complaint. Rate, effort, pattern, six fields. Recognising abnormal requires having heard a great deal of normal — and that is the one thing you cannot cram.

CASE REVIEW

Back to your 61-year-old at 0240

Do the arithmetic

40 breaths at roughly 200 mL is a minute volume of 8,000 mL — which reads as more than adequate. But dead space of ~150 mL is charged on EVERY breath, so alveolar volume is about 50 mL. Alveolar minute volume: 50 × 40 = 2,000 mL, against a normal of ~4,200.

Why she chose this pattern

Fast and shallow is what a fatiguing obstructive patient does. Each breath costs work, and the cheapest way to reduce the cost of one breath is to make it smaller. Rational — and self-defeating, because the dead space tax is fixed per breath.

What 91% actually means

She is standing on the shoulder of the oxyhemoglobin curve. The flat part is already spent. From here, small further deterioration produces large drops — and it will happen faster than the number suggests.

And the nonrebreather?

It would raise her saturation and change nothing about her CO_2, her work of breathing, or her fatigue. It treats the number. She needs PRESSURE and support — which is Part 2, and CPAP with an in-line bronchodilator under your asthma/COPD protocol.

She was never an airway problem. She was a ventilation problem the whole time — and every monitor that would have told you so measures carbon dioxide, not oxygen.

REVIEW

Check your understanding

1. Name the three regions of the pharynx and their boundaries. Which is most important in airway management, and why?
2. Name the three laryngeal structures you must be able to locate, and state what each one is for.
3. What is the narrowest part of the airway in an adult? In a child? Give one consequence of each.
4. Why do a misplaced tube and an aspirated foreign body both end up on the same side?
5. Which phase of breathing costs energy, and why does that explain fatigue in obstructive disease?
6. Distinguish respiration from ventilation, and give a patient who does one well and the other badly.
7. Explain the clinical meaning of the SHAPE of the oxyhemoglobin dissociation curve above and below 90%.
8. Define tidal, dead space and alveolar volume. Compute alveolar minute volume at 200 mL and a rate of 40.
9. Why can a pulse oximeter read 100% in a dying carbon monoxide patient — and what detects it instead?
10. Why are quiet or absent breath sounds a worse finding than loud wheezing?
11. Describe the shark fin capnogram and explain what makes it more useful than the $ETCO_2$ number alone.
12. Your patient receives ketamine and stops breathing. Which monitor tells you first, and how much sooner?

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