

VOLUME 2 • CHAPTER 32 • PART 2

Obstruction and Obstructive Disease

Upper airway obstruction, ARDS, emphysema, chronic bronchitis, and asthma

Five diseases that narrow, fill, or destroy the airway — and three of them are the ones you will meet most.
45 minutes • Aligned to Region SOPs

Five diseases — and the mechanism that separates them

1 Upper airway obstruction

Something is IN the pipe. The fastest killer here, and the only one where the intervention is mechanical rather than pharmacological.

2 ARDS

The membrane has THICKENED with fluid, so gas cannot cross. Where positive pressure first earns its keep.

3 Emphysema

The alveolar walls are DESTROYED, so there is less surface to cross. The only one of the five where the lost tissue does not come back.

4 Chronic bronchitis

The airway is inflamed and full of MUCUS, so less air arrives. The alveoli themselves are largely spared.

5 Asthma

The muscle around the bronchiole has CONTRACTED and the mucosa is swollen. The most reversible disease in the chapter.

Two of these are DIFFUSION failures. Three are VENTILATION failures.

ARDS and emphysema break the membrane — gas cannot cross. Obstruction, chronic bronchitis and asthma stop air arriving in the first place.

That split predicts which ones improve on oxygen alone, and which ones need air actually moved. It is the Part 1 framework doing work on day one of the diseases.

The framing sentence for both disease sessions

“Specific respiratory diseases are treated on the basis of the underlying cause. It is more important to stress the priorities of care for airway and breathing rather than attempting to diagnose the respiratory problem.”

Nobody expects a field diagnosis. What is expected is recognising WHICH PROCESS is failing and applying the right lever — which is exactly what Part 1 built.

Upper Airway Obstruction

The fastest killer in the chapter — and the one where the answer is your hands, not a drug.

The tongue · Foreign body · Laryngeal edema

Three natures, three different answers

1 The tongue — fixed by POSITION

The most common cause of upper airway obstruction, and the reason every basic maneuver in Chapter 22 existed. Head-tilt/chin-lift or jaw thrust, plus an adjunct.

2 A foreign body — fixed by REMOVAL

Conscious and able to speak: encourage the COUGH, which moves more air than your thrusts will. Severe obstruction or poor exchange: rapid abdominal thrusts.

3 Laryngeal edema — fixed by DRUGS or a KNIFE

Supplemental oxygen and RAPID airway management. Neither suction nor positioning touches this one, and the window for placing a tube is closing while you watch.

The unconscious patient — four steps, and two details people get wrong

Unresponsive? Start CPR

Regardless of pulse. Compressions generate intrathoracic pressure that can dislodge the object.

Look before each breath

Each time you open the airway during CPR, LOOK for an object and remove it if you see one. You do not sweep blindly.

Then visualise it

If obstruction persists and you cannot ventilate: laryngoscope, see the object, remove it with Magill forceps.

Then ventilate

Once the obstruction is removed, begin ventilation and administer supplemental oxygen.

Assessment varies with the CAUSE and the HISTORY of the event — in obstruction, the story usually makes the diagnosis before the exam does.

REGION SOP Your ladder: let them clear it → back blows and thrusts → CPR regardless of pulses if unresponsive → video laryngoscopy and Magill forceps → if past the cords, intubate and advance the object into the right mainstem, then withdraw and ventilate the left lung → suction ≤ 10 sec → cricothyrotomy (needle < 10 yrs, surgical ≥ 10).

ARDS

Noncardiogenic pulmonary edema — fluid in the interstitium, from almost any insult you can name.

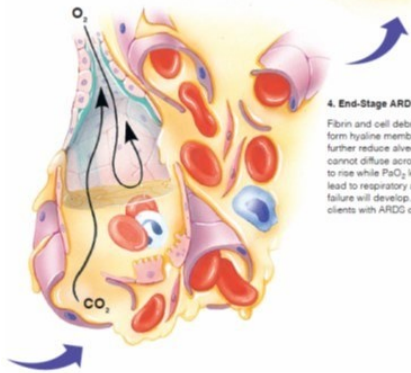
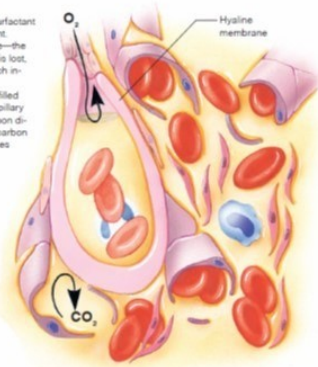
Pathophysiology · A very long cause list · Positive pressure

A common final pathway, not a specific disease

3. Alveolar Collapse

Protein-rich fluid accumulates in the alveoli, inactivating surfactant and damaging type II alveolar cells that produce surfactant. (Surfactant is important in maintaining alveolar compliance—the ability of tissue to stretch or distend.) As active surfactant is lost, the alveoli stiffen and collapse, leading to atelectasis, which increases breathing effort.

Decreased alveolar compliance, atelectasis, and fluid-filled alveoli interfere with gas exchange across the alveolar-capillary membrane. Blood oxygen (PaO_2) levels fall. Because carbon dioxide diffuses more readily than oxygen, however, blood carbon dioxide (PaCO_2) levels also fall initially as tachypnea causes more CO_2 to be expired.



4. End-Stage ARDS

Fibrin and cell debris from necrotic cells combine to form hyaline membranes, which line the interior of the alveoli and further reduce alveolar compliance and gas exchange. Because CO_2 cannot diffuse across hyaline membranes, PaCO_2 levels now begin to rise while PaO_2 levels continue to fall. Rising PaCO_2 levels can lead to respiratory acidosis. Without respiratory support, respiratory failure will develop. Even with aggressive treatment, almost 50% of clients with ARDS die.

1 Noncardiogenic — that is the whole name

The lungs are full of fluid and the heart is not the reason. Cardiogenic edema is a PUMP problem; ARDS is a PERMEABILITY problem.

2 Three mechanisms, one result

Increased pulmonary capillary PERMEABILITY • DESTRUCTION of the capillary lining • increased OSMOTIC FORCE drawing fluid in. All three produce interstitial edema.

3 Which is Part 1's thickened membrane

“Accumulation of fluid and inflammatory cells in the interstitial space” was named as the most common cause of impaired diffusion. ARDS is that mechanism at its most severe.

4 Assessment follows the CAUSE

Specific symptoms relate to the underlying cause — there is no ARDS presentation of its own. You see the cause, plus progressive hypoxia that responds poorly to oxygen alone.

Management, in two sentences

Treat the UNDERLYING CAUSE — that is the hallmark. And use POSITIVE PRESSURE for any ARDS patient in respiratory failure: PEEP and CPAP maintain alveolar patency.

Figure 32-30 The progression of ARDS.

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

Why the cause list is so long — and what to do about it

Infection and inflammation

Sepsis • pneumonia • pancreatitis

Something entered the lung

Aspiration • near drowning • inhalation injury • oxygen toxicity

Trauma and its consequences

Pulmonary injury • burns • head injury • emboli • tumour destruction

Drugs and environment

Aspirin • opiates • high altitude • hypothermia

Procedures and global insults

Cardiopulmonary bypass • hemodialysis • hypoxia • hypotension • cardiac arrest

The length of that list IS the message

ARDS is not a disease you diagnose — it is where a great many different insults end up. Notice that pneumonia, inhalation injury and near drowning all appear here AND appear again in Part 3 as diseases in their own right. That is not duplication: a severe enough version of any of them becomes this.



Figure 32-29 Supporting oxygenation and ventilation.

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

Paramedic Education • Ch 32 Pulmonology • Part 2

What recognition actually looks like in the field

ARDS is not a prehospital diagnosis and it does not need to be. What matters is noticing **HYPOXIA DISPROPORTIONATE TO THE APPARENT CAUSE** — a patient whose saturation is not improving the way it should on high-concentration oxygen, with one of these insults in the recent history.

That pattern is a diffusion failure, and **PRESSURE** will help more than flow. Treat what you can see, and support the rest.

Obstructive Lung Disease

Asthma, emphysema and chronic bronchitis — described as the most common lung diseases in prehospital care.

Emphysema · Chronic bronchitis · Asthma · Status asthmaticus

Destruction of the alveolar walls — and it does not come back



Figure 32-32 The emphysema patient.

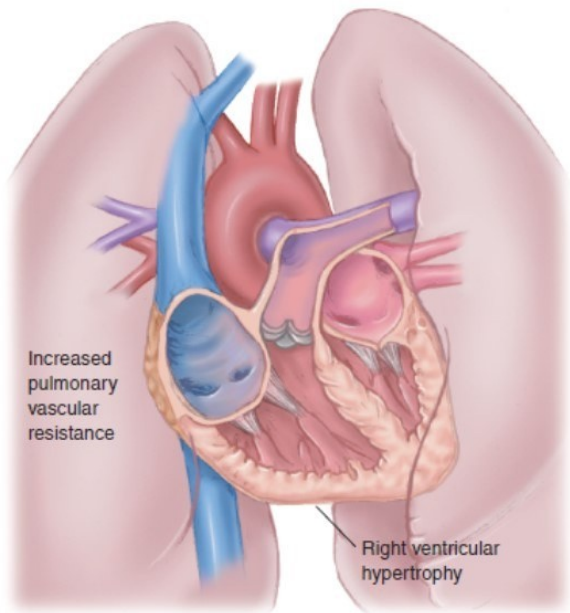


Figure 32-31 Cor pulmonale — right ventricular hypertrophy.

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

1 Diffusion fails

Destruction of the alveolar walls distal to the terminal bronchioles, LESSENING THE AREA available for gas exchange.

2 Perfusion fails

Pulmonary capillaries DECREASE IN NUMBER → pulmonary hypertension → right heart failure — cor pulmonale.

3 And ventilation fails

WEAKENED small bronchioles with no cartilage collapse on exhalation — which is why they cannot get air OUT.

4 So it breaks all three processes

Part 1's multi-process patient — and why the goal is RELIEF, not cure.

The classic finding	And the mechanism behind it
Recent weight loss	The work of breathing burns calories all day, every day
Barrel chest	Air trapped and never fully exhaled, remodelling the thorax over years
PINK hue to the skin	Chronic hypoxia drives erythropoietin → POLYCYTHEMIA. A compensation — and why they are “pink puffers,” not cyanotic
Pursed lips	The patient generating their own CPAP, involuntarily, to splint collapsing small airways open. The body invented PEEP first
Clubbed fingers	Years of hypoxemia — so it tells you about the BASELINE, not about today

Mucus, not destruction — and the alveoli are largely spared

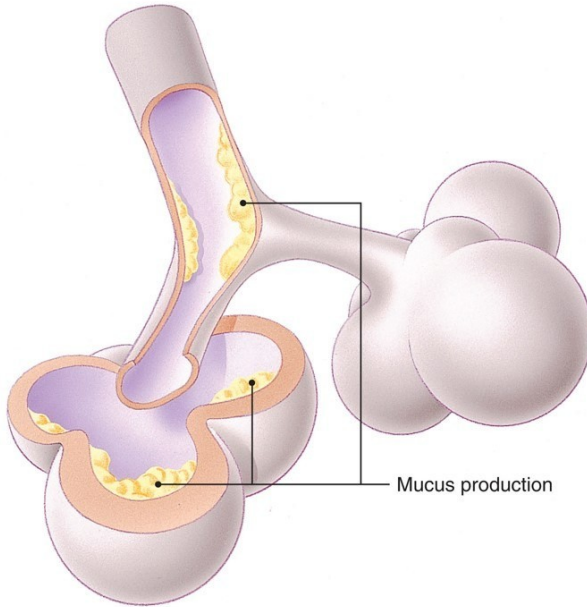


Figure 32-33 Mucus production in chronic bronchitis.

The one management step that surprises people

FLUID ADMINISTRATION is suggested with signs of dehydration. The rationale: thick secretions are far harder to clear in a dry patient.

Otherwise — relieve hypoxia, reverse bronchoconstriction, bronchodilators by nebuliser.

“Unlike emphysema, the alveoli are NOT severely affected, and diffusion remains NORMAL.”

One sentence separates the two diseases. Emphysema destroys the gas-exchange surface; chronic bronchitis leaves it intact and blocks the road to it.

1 More goblet cells, more mucus

Permanently. Chronic inflammation plus mucus narrows the airway, so LOWERED ALVEOLAR VENTILATION reduces gas exchange — even though the alveoli work fine.

2 A different route to cor pulmonale

Raised PaCO_2 causes PULMONARY VASOCONSTRICTION → pulmonary hypertension → cor pulmonale. In emphysema capillaries are destroyed; here they are intact but constricted.

3 Blue bloaters

Heavy cigarette use — but it occurs in NONSMOKERS too. Often a history of repeated respiratory infections, since chronic mucus is a reservoir. Tend to be overweight and cyanotic.

4 And it shows on your monitor

ECG changes reflect increased right ventricular size and ventricular strain — the cor pulmonale, visible on equipment you already have attached.

Pink puffer, blue bloater — and why the distinction still matters

	EMPHYSEMA — “pink puffer”	CHRONIC BRONCHITIS — “blue bloater”
The lesion	Destruction of ALVEOLAR WALLS distal to the terminal bronchioles	Increase in the number of GOBLET CELLS — mucus, not destruction
Diffusion	IMPAIRED — there is less surface left to cross	NORMAL — the alveoli are not severely affected
Colour, and why	PINK, from polycythemia compensating for chronic hypoxia	CYANOTIC — frank hypoxemia, without the same compensation
Build	Thin, with recent WEIGHT LOSS — the work of breathing burns calories	Tends to be OVERWEIGHT
On examination	BARREL CHEST • PURSED LIPS (auto-PEEP) • clubbed fingers	Productive cough • history of repeated respiratory infections
Route to cor pulmonale	Capillaries physically DESTROYED → pulmonary hypertension	Raised PaCO ₂ → vasoCONSTRICTION → pulmonary hypertension

The honest caveat — say this out loud

These are classic textbook descriptions. MOST REAL PATIENTS HAVE FEATURES OF BOTH — both diseases have the same cause, and the same person smoked for forty years. The pure pink puffer and the pure blue bloater are teaching constructs.

So why learn it? Because it predicts what you will FIND on examination, and it predicts how the patient will RESPOND: reversible narrowing answers a bronchodilator, destroyed tissue does not. Your protocol treats them together, and that is correct — this distinction shapes your expectations, not your algorithm.

A chronic inflammatory disorder — and a two-phase reaction

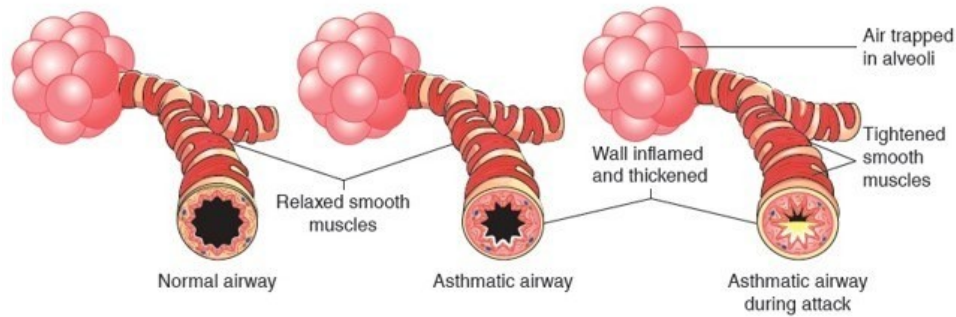


Figure 32-34 Bronchoconstriction in asthma.

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

1 CHRONIC — it is there between attacks

The airway of an asthmatic is abnormal on a good day. This is not an episodic disease that visits; it is a permanent one that flares.

2 INFLAMMATORY — spasm is only half of it

If it were pure muscle spasm a bronchodilator would fix it, and it would stay fixed. It does not.

3 Phase one — within MINUTES

HISTAMINE and other mediators → bronchoconstriction and edema. The phase your bronchodilator treats.

4 Phase two — 6 to 8 HOURS later

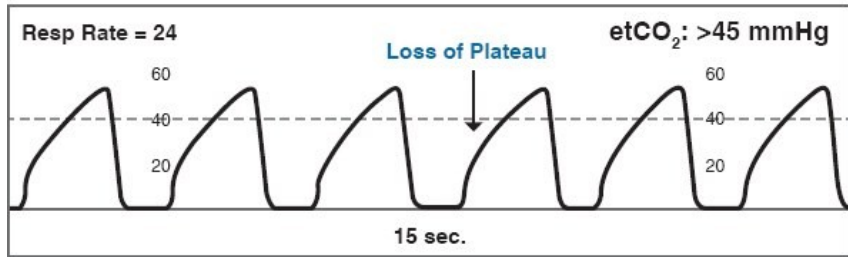
INFLAMMATION of the bronchioles as immune cells invade the mucosa. This is the phase your bronchodilator does NOT treat.

Which explains the patient who improves on your nebuliser and deteriorates after you have gone

The second phase is still coming, six to eight hours out. That is why steroids exist, why they take hours to work, and why they are given early even though nothing visible happens.

And it is the most important discharge conversation in this chapter: a patient who feels better after a nebuliser is NOT CURED — which matters most for the asthmatic who is thinking about refusing transport.

REGION SOP Triggers VARY FROM ONE INDIVIDUAL TO THE NEXT — so the history question is not “what triggers asthma” but “what triggers YOURS.” Patients almost always know, and they are rarely asked.



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

1 Dyspnea, wheezing, cough

The three commonest. COUGH matters — cough-variant asthma exists, and a patient with a persistent dry cough and no wheeze can still be asthmatic.

2 One- to two-word dyspnea

Count the words per breath. Part 1's life-threat finding, available with no equipment and no delay.

3 Pulsus paradoxus and tachycardia

A systolic drop of 10 mmHg OR MORE with inspiration.
Huge negative intrathoracic pressure swings shift the septum left and cut LV filling — a genuine marker of severity.

4 Low peak flow, falling saturation

Peak flow measures airway resistance directly but needs a cooperative patient. Saturation is a LATE finding — Part 1's flat curve.

The shark fin improves BEFORE the number does

Sloped upstroke, rising plateau — alveoli with different time constants emptying at different rates. Which makes the SHAPE the earliest objective evidence your bronchodilator is working: earlier than the breath sounds, and much earlier than the patient's own report.

Two questions and one warning

Ask what steps they have **ALREADY TAKEN** — a patient who has used their rescue inhaler eight times is a different patient from one who has used it once, even if they look identical. And ask whether this has happened before, and when it started.

The warning, carried from Part 1: a SILENT chest in an asthmatic is not improvement. Noise requires airflow.

Management — and your protocol, step by step

Tier	What defines it	What you give
MILD / MODERATE	GCS 15 · MAP > 65 · SBP > 90 · skin warm and dry	DuoNeb — ALBUTEROL 2.5 mg with IPRATROPIUM 0.5 mg, may repeat × 2
SEVERE / IMPENDING FAILURE	Altered mental status · pale and diaphoretic	DuoNeb delivered THROUGH CPAP at 5 cm PEEP, may increase to max 10 cm, DuoNeb may repeat × 2
... then	Still severe	MAGNESIUM SULFATE 2 g in 100 mL D5W IVPB over 15 min — “do not delay administration” (≥ 65, OLMC to consider 1 g)
... still deteriorating	Contact OLMC	EPINEPHRINE 1 mg/mL, 0.3 mg IM in the anterolateral thigh

Why two drugs, not one

ALBUTEROL is a beta-2 agonist — it relaxes bronchial smooth muscle directly. IPRATROPIUM is an anticholinergic — it blocks muscarinic receptors, bronchodilating and drying secretions. Two receptors, additive effect. Hence DuoNeb.

The drug rides in on the pressure

Albuterol and ipratropium may be given IN-LINE with CPAP or intubation. You do not choose between the drug and the pressure — a point people routinely get wrong by stopping the nebuliser to apply CPAP.

Magnesium cautions

NOT for renal failure or dialysis patients. May cause hypotension, which usually responds to a fluid bolus.

And be ready for fatigue

“Be prepared for the patient to fatigue and require aggressive airway management and ventilation.” Asthma deaths are fatigue deaths — have the bag ready before you need it.

REGION SOP Oxygen to maintain SpO₂ 94–99%. Obtain ECG and 12-lead, monitor capnography. And: “Do not delay transport while waiting for response.”

Status asthmaticus, and the asthmatic child

1 Status asthmaticus — defined by FAILURE

A severe, prolonged attack that cannot be broken with repeated doses of bronchodilators. The definition is functional, not numeric — you can only recognise it after you have tried.

2 Respiratory arrest is imminent

Recognise it and be prepared for endotracheal intubation. But know that intubation does not fix bronchospasm — it secures the airway of a patient who was going to lose it.

3 Pediatric asthma is the SAME disease

Pathophysiology and treatment are the same as in adults, with altered medication dosages. Say the reassuring half first.

4 And the noisy child who is NOT asthmatic

Croup is STRIDOR, not wheeze, and it is upper airway. The instinct to treat any noisy child with albuterol is strong and wrong.

Every sign that the asthmatic is losing reads as improvement to an inexperienced eye

A FALLING respiratory rate in a patient who was tachypneic · a QUIETENING chest · rising CO₂ on the capnograph · declining mental status · and the patient who STOPS FIGHTING YOU. The asthmatic who becomes calm and quiet is not getting better — and this is the single most dangerous misreading in the chapter.

REGION SOP Pediatric: ALBUTEROL 2.5 mg NEB (repeat 5 min) · DuoNeb · MAGNESIUM 25 mg/kg over 15 min, max 2 g · EPINEPHRINE 0.01 mg/kg IM, max 0.3 mg · BVM 1 breath q2–3 sec if failing.
Croup: humidified oxygen, NEBULIZED EPINEPHRINE 0.5 mg/kg (max 5 mg), and KEEP THE PATIENT CALM — DO NOT AGITATE.

What to carry out of Part 2

1 Three natures of obstruction

The tongue is fixed by POSITION. A foreign body by REMOVAL. Laryngeal edema by DRUGS or a KNIFE — and neither suction nor positioning touches the third.

2 ARDS is a final pathway

Noncardiogenic pulmonary edema — fluid in the interstitium from almost any insult. Treat the CAUSE, and support with positive pressure.

3 Emphysema breaks all three processes

Destroyed alveolar walls (diffusion), fewer capillaries (perfusion), weakened bronchioles (ventilation). The goal is RELIEF, because the tissue does not come back.

4 Chronic bronchitis spares the alveoli

“Unlike emphysema, diffusion remains normal.” Mucus and inflammation block the road to an intact gas-exchange surface.

5 Asthma has two phases

Minutes — histamine, bronchoconstriction, edema, and your bronchodilator works. Six to eight HOURS — inflammation, and it does not. Hence steroids, and hence the discharge conversation.

6 The quiet patient is losing

A silent chest, a falling rate, decreasing retractions, a child who stops fighting you. Every one reads as improvement and every one is deterioration.

These three obstructive diseases are the most common lung diseases in prehospital care.

This shift: find a COPD or asthma patient’s medication list and work out from the drugs alone which disease they have and how severe it is. Home oxygen, a controller inhaler, a nebuliser at the bedside, prednisone in the cupboard — each one is a piece of the story.

Check your understanding

- 1 What is the most common cause of upper airway obstruction, and what fixes it?
- 2 A conscious adult is choking and can speak. What do you do, and why is that better than thrusts?
- 3 Your obstructed patient goes unresponsive. What two things do people get wrong at this point?
- 4 What does the “non” in noncardiogenic mean in ARDS, and what three mechanisms produce the edema?
- 5 State the two-sentence management of ARDS.
- 6 Define emphysema, name its major cause, and give the three processes it breaks.
- 7 Why is the emphysema patient PINK rather than cyanotic?
- 8 What are pursed lips actually doing, and what is the technical name for it?
- 9 Define chronic bronchitis. Which sentence separates it most sharply from emphysema?
- 10 Trace the route from raised PaCO_2 to cor pulmonale. How does emphysema reach the same endpoint differently?
- 11 Which management step in chronic bronchitis surprises people, and what is the rationale?
- 12 Describe both phases of the asthma reaction with timing — and say what each one means for your treatment.
- 13 Define pulsus paradoxus, explain the mechanism, and say what it tells you.
- 14 Give your protocol’s two tiers for asthma/COPD, including what defines each and what you give.
- 15 Who must not receive magnesium sulfate, and what side effect should you anticipate?
- 16 Define status asthmaticus. Name four findings that look like improvement and are not.

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