

Air embolism

Mechanisms, tissue injury, decompression sickness

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Learning objectives

- Define gas embolism and distinguish two mechanisms of bubble formation.
- Explain how vascular entry, obstruction and host responses injure organs.
- Relate decompression to the bends, chokes and chronic bone necrosis.
- Explain the pressure rationale of treatment for acute decompression sickness.

An embolus is a transported intravascular mass

- May be solid, liquid or gaseous; It travels from its point of origin.
- It can lodge at a distant site where vessels become too narrow.
- Partial or complete occlusion can cause dysfunction or infarction.

Gas is a distinct form of embolic material

- A gas bubble can occupy the vascular lumen just as a solid embolus can.
- Multiple bubbles may coalesce into an obstructive frothy mass.
- Tissue injury reflects both loss of downstream perfusion and local responses.

Two mechanisms generate clinically important gas bubbles

Externally introduced air

- Open communication between vessel and ambient air
- A pressure gradient permits air to enter
- Relevant to procedures and traumatic defects

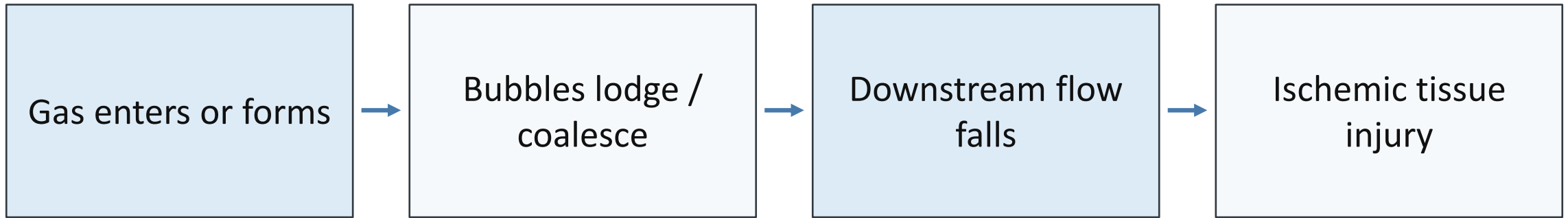
Decompression-related nitrogen

- Gas dissolves at elevated ambient pressure
- Rapid ascent brings nitrogen out of solution
- Bubbles arise in blood and body tissues

Embolic consequences depend on the vascular bed

- A lodged embolus interrupts perfusion of the tissue downstream.
- Pulmonary gas emboli can obstruct perfusion and injure alveoli.
- Cerebral bubbles can cause acute neurologic dysfunction.

Vascular obstruction is a causal sequence



Conceptual sequence derived from the textbook definition of embolism.

Schematic — conceptual sequence; Not a clinical specimen.

Gas embolism has more than one mechanism of damage

Physical obstruction

- Bubbles occupy vascular space
- Perfusion beyond the obstruction is impaired
- Potential for distal ischemia

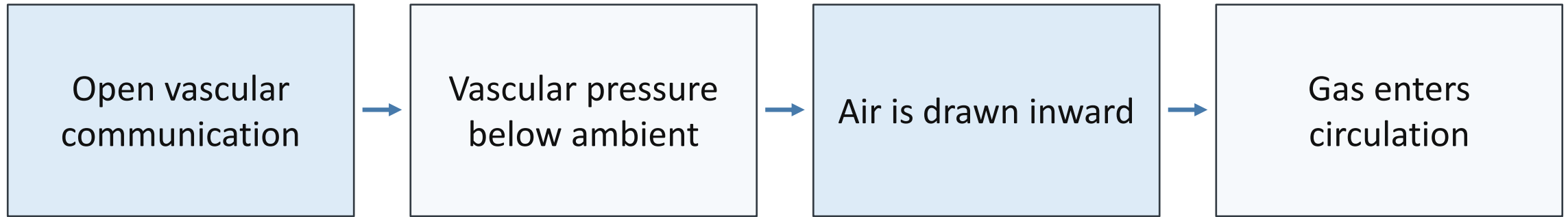
Host-mediated injury

- Pulmonary cytokine-associated inflammation
- Inflammatory and thrombogenic bubble responses
- Additional tissue damage beyond simple blockage

Exogenous air requires vascular access

- Ambient air cannot enter an intact, closed vascular compartment.
- Communication between vasculature and outside air.
- Open vessels during interventions or injury provide entry opportunities.

A negative pressure gradient entrains air



The essential entry condition is the pressure differential across an open route.

Schematic — conceptual sequence; Not a clinical specimen.

Neurosurgery in a sitting position

- A sitting position can create a gravitational pressure gradient.
- Air can thereby enter the cerebral circulation during neurosurgery.
- Position-related risk illustrates why pressure, not simply a wound, matters.

Endovascular and interventional procedures can introduce air

- Instrumentation can expose the circulation to gas.
- Both endovascular and other interventional procedures as relevant settings.
- The gas arrives by direct introduction, not by nitrogen leaving solution.

Mechanical ventilation is another recognized setting

- Mechanical ventilation as a context for air introduction.
- A ventilation-associated mechanism differs from high-pressure nitrogen dissolution.
- The passage does not provide a detailed ventilation injury taxonomy.

Obstetric and laparoscopic procedural contexts

- Obstetric and laparoscopic procedures as contexts for large-volume introduction.
- Both are procedural routes of external gas entry, distinct from decompression.
- Specific entry mechanics are not detailed in the passage.

Chest wall injury can provide an entry route

- Traumatic injury can connect the vasculature with outside air.
- The direction of flow depends on the pressure relationship.
- If gas enters, the clinical effects depend on where the bubbles circulate.

Recognize the reported entry contexts

Context	Relevant mechanism
Sitting neurosurgery	Gravitational pressure gradient
Endovascular / interventional procedures	Potential gas introduction
Mechanical ventilation	Recognized exposure context
Obstetric / laparoscopic procedures	Potential high-volume introduction
Chest wall injury	Traumatic communication

Entry and bubble generation must not be conflated

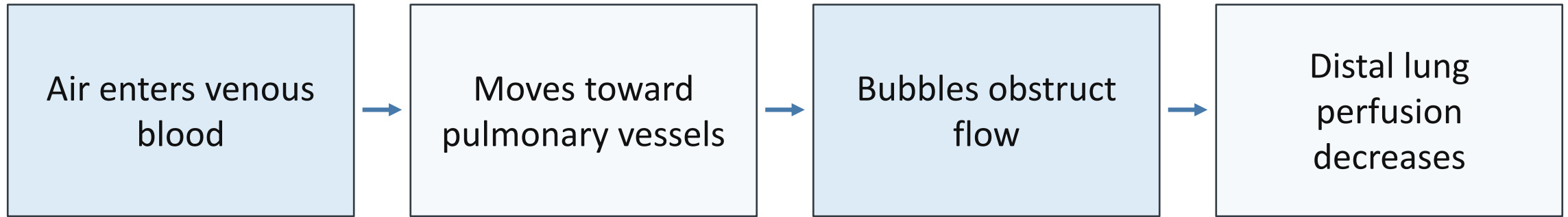
Air introduced from outside

- Vascular communication
- Negative pressure gradient
- Procedure or injury-associated context

Nitrogen liberated from solution

- Prior high-pressure gas exposure
- Rapid decrease in ambient pressure
- Tissue and blood bubble formation

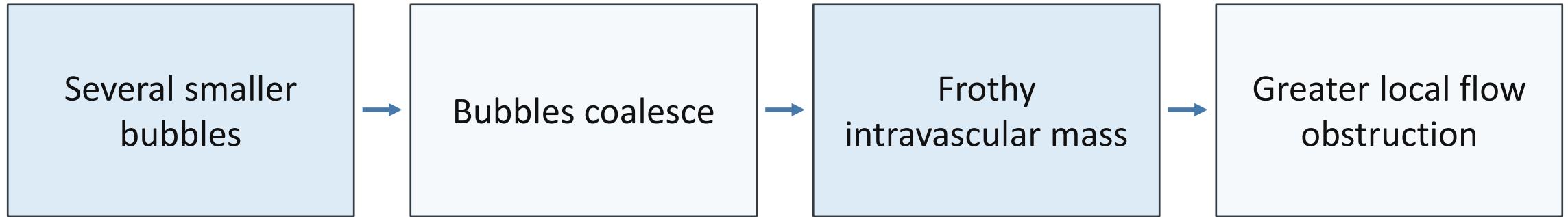
Venous gas can affect pulmonary perfusion



Schematic circulation pathway; Organ-level consequences are.

Schematic — conceptual sequence; Not a clinical specimen.

Why coalescence matters



Schematic — conceptual sequence; Not a clinical specimen.

Pulmonary effects: The >100 mL statement

- Generally more than 100 mL is needed for clinical effects in the pulmonary circulation.
- This is a descriptive textbook statement—not a universally safe exposure limit.
- The statement pertains to pulmonary circulation, not all arterial or cerebral events.

Rapidly introduced air can be fatal

- 300–500 mL introduced over several seconds may be fatal.
- The quoted figures refer to a rapid-entry scenario.
- Do not apply the figure to a different vascular bed as a clinical rule.

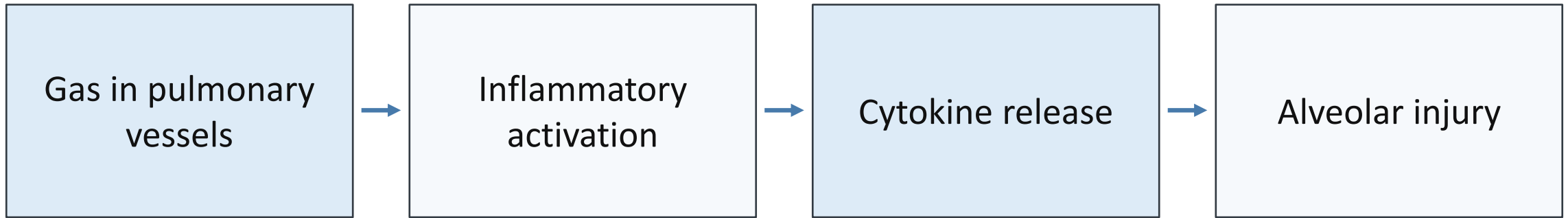
Pulmonary injury starts with perfusion obstruction

- Air in pulmonary vasculature blocks flow to downstream regions.
- A downstream region loses normal access to the pulmonary circulation.
- Mechanical blockage is one—but not the only—injury mechanism.

The pulmonary host response is inflammatory

- Gas entry produces an intense inflammatory response in the pulmonary vasculature.
- The reaction includes cytokine release.
- The lung injury can extend beyond the immediate site of mechanical blockage.

Alveolar injury follows the vascular response



The tissue consequence is linked to both vascular and inflammatory pathology.

Schematic — conceptual sequence; Not a clinical specimen.

Two parallel pathways to pulmonary damage

Obstruction pathway

- Intravascular gas
- Reduced distal perfusion
- Ischemic consequences

Inflammatory pathway

- Pulmonary vascular gas
- Cytokine-associated response
- Alveolar damage

Cerebral gas bubbles cause neurologic dysfunction

- Gas bubbles in the central nervous system can cause multiple neurologic deficits.
- Memory loss, ataxia, visual disturbances and sudden coma.
- Clinical presentation depends on involvement of the nervous system.

Neurologic manifestations span several functions

Memory loss

- A named cerebral manifestation
- Highlights dysfunction beyond pulmonary compromise

Ataxia

- A named disturbance of coordination
- Points to neurological involvement

Match manifestations to the affected compartment

Context	Relevant mechanism
Pulmonary vascular gas	Distal perfusion obstruction; inflammation; alveolar injury
Cerebral vascular gas	Memory loss; ataxia; visual changes; coma
Periarticular / muscular gas	Painful decompression syndrome — the bends
Chronic skeletal gas effects	Foci of ischemic bone necrosis

Decompression sickness is a gas embolic disorder

- It follows a sudden decrease in atmospheric pressure.
- The gas source is predominantly nitrogen previously dissolved in blood and tissues.
- Bubble formation results from rapid depressurization, not necessarily an open wound.

Pressure transitions identify risk environments

- Scuba and deep-sea diving, and underwater construction work, involve pressurized exposure.
- Rapid ascent in unpressurized aircraft is also a recognized setting.
- Precautions against abrupt decompression matter during space travel.

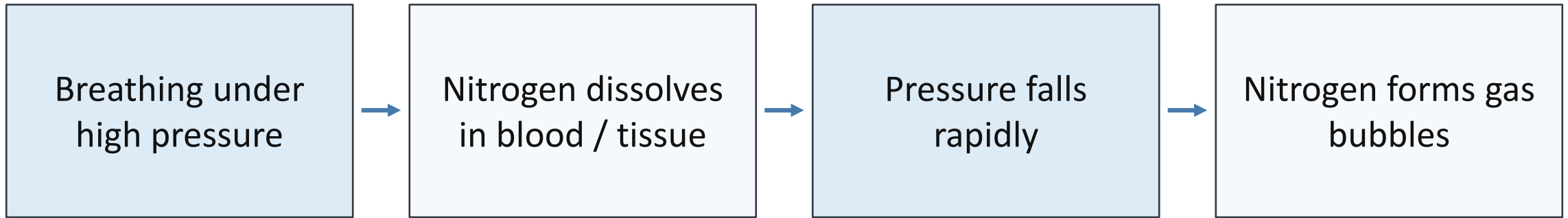
High-pressure breathing increases dissolved gas

- During a deep-sea dive, air is breathed under high pressure.
- More gas dissolves into blood and tissues under these conditions.
- Nitrogen is the gas emphasized by Robbins.

Blood and tissue act as dissolved-gas reservoirs

- While pressure is elevated, nitrogen can be present in dissolved form.
- The reservoir includes both circulating blood and body tissues.
- Dissolved nitrogen is not itself the same as an obstructing bubble.

Rapid ascent changes the dissolved-gas state



Conceptual explanation of rapid ascent given.

Schematic — conceptual sequence; Not a clinical specimen.

Bubbles can form outside the vasculature

- Nitrogen emerging from solution in both tissues and blood.
- Periarticular bubbles help explain painful musculoskeletal disease.
- Intravascular bubbles can also obstruct perfusion and provoke host responses.

A decompression bubble is not entrained outside air

Externally introduced embolism

- Gas crosses an open boundary
- Pressure gradient draws air inside
- Examples: Procedure, chest wall injury

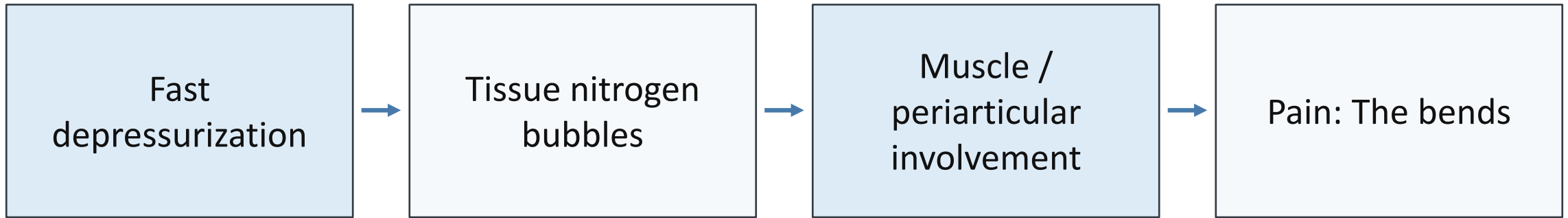
Decompression embolism

- Dissolved nitrogen becomes gaseous
- Atmospheric pressure falls too quickly
- Examples: Ascent / decompression

The bends: A tissue-based presentation

- Rapid bubble formation occurs in skeletal muscles and supporting tissues.
- Periarticular locations are particularly important.
- The consequence is the painful condition called the bends.

Why the joints and muscles hurt



Musculoskeletal pain arises from bubbles forming in and around these tissues.

Schematic — conceptual sequence; Not a clinical specimen.

Bubble-related inflammation and thrombogenicity

Inflammatory response

- Host reaction to intravascular gas
- Adds tissue-level injury

Thrombogenic response

- Host response favoring clot-associated phenomena
- A second mechanism beyond bubble size

The chokes: Respiratory injury during decompression

- Bubbles may form in pulmonary vessels during rapid decompression.
- Associated pulmonary effects include edema, hemorrhage and focal atelectasis or emphysema.
- The resulting respiratory distress is termed the chokes.

The chokes: Correlate pathology with symptoms

- Edema represents a pulmonary tissue response to gas emboli.
- Hemorrhage is another described lung abnormality.
- Focal atelectasis or emphysema contributes to the pathology associated with respiratory distress.

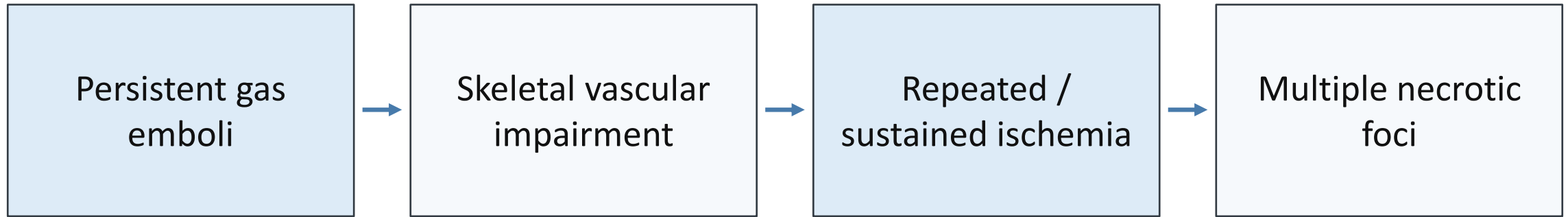
Bends and chokes represent different compartments

Syndrome	Predominant site	Main consequence
Bends	Skeletal muscles / periarticular tissues	Pain
Chokes	Pulmonary vasculature and lung	Respiratory distress
Shared trigger	Rapid fall in atmospheric pressure	Nitrogen bubble formation

Caisson disease: Chronic decompression injury

- A chronic form of decompression sickness is called caisson disease.
- The name derives from pressurized vessels used in bridge construction.
- Workers experienced both acute and chronic decompression problems.

Persistent skeletal gas leads to ischemic necrosis



Skeletal emboli with multiple foci of ischemic necrosis.

Schematic — conceptual sequence; Not a clinical specimen.

Typical skeletal sites in caisson disease

Femoral heads

A common site of
ischemic necrosis

Tibia

Included among the
favored sites

Humeri

Another specifically
named location

Acute and chronic decompression consequences differ

Acute decompression sickness

- Bubble formation after rapid pressure fall
- Painful bends or respiratory chokes
- Recompression therapy described

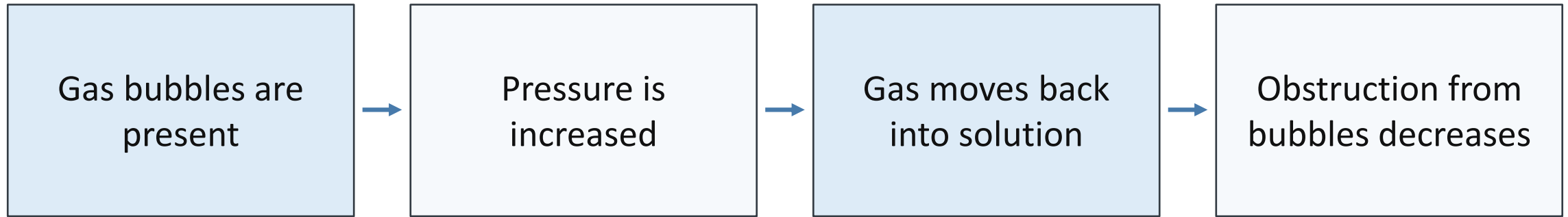
Chronic caisson disease

- Persistence of skeletal gas emboli
- Multiple ischemic necrotic foci
- Common sites include femoral head, tibia, humerus

Acute decompression sickness: Recompression

- Treatment using a sufficiently pressurized chamber.
- The high pressure forces gas bubbles back into solution.
- This is the stated mechanism of treatment for acute decompression sickness.

Why increased pressure reduces visible bubbles



A mechanism schematic; No pressure regimen is specified in the source.

Schematic — conceptual sequence; Not a clinical specimen.

Slow decompression completes the process

- Pressure is subsequently reduced gradually.
- This permits gas resorption and exhalation.
- Slower decompression helps prevent bubbles from reforming.

Compare introduced air and decompression disease

Feature	Introduced air	Decompression
Mechanism	Direct vascular entry	Nitrogen leaves solution
Relevant pressure	Air–vessel gradient	Fall in ambient pressure
Examples	Surgery, procedures, chest trauma	Diving, caisson work, rapid ascent
Major injury	Vascular obstruction / inflammation	Blood and tissue bubbles; organ injury

Air embolism in the broader embolic differential

Type	Material	Textbook context
Gas embolus	Gas bubbles	Entry or decompression
Fat embolus	Fat globules $\pm$ marrow	Typically skeletal injury
Thromboembolus	Dislodged clot	Thrombosis with embolization

Illustrative case: Seated neurosurgical procedure

During a procedure in the sitting position, gas is suspected to have entered a cerebral vessel.

- Which two conditions permit external air to enter?
- What makes body position relevant?

Interpretation

Open vascular communication plus a negative pressure gradient; A gravitational gradient can favor entrainment.

Illustrative case: Pulmonary gas after a procedure

A substantial volume of intravascular air is introduced rapidly during a procedure.

- Which two lung-injury pathways should be distinguished?
- What quantitative caution applies?

Interpretation

Downstream perfusion obstruction and cytokine-associated alveolar injury; The source's volume statements are contextual, not safety cutoffs.

Illustrative case: Rapid ascent after a deep dive

A diver ascends quickly after breathing compressed air at depth and subsequently develops painful periarticular symptoms.

- What is the source of the gas bubbles?
- What syndrome best fits the tissue location?

Interpretation

Nitrogen coming out of solution in tissues and blood; The bends.

Illustrative case: Respiratory distress on ascent

After rapid depressurization, a diver develops respiratory distress.

- Which named decompression syndrome is relevant?
- What lung findings does Robbins associate with it?

Interpretation

The chokes; Edema, hemorrhage and focal atelectasis or emphysema.

Illustrative case: Chronic skeletal consequences

A worker with repeated exposure to pressurized environments is evaluated for a chronic skeletal complication.

- What chronic condition is described?
- Which skeletal locations should be considered?

Interpretation

Caisson disease with multiple ischemic necrotic foci; Femoral heads, tibia and humeri are named sites.

Interpretive pitfall: Not all gas is entrained air

- Outside air entering an open vessel is one route to gas embolism.
- In decompression sickness, nitrogen is liberated from previously dissolved gas.
- A correct mechanistic explanation must identify which route applies.

Interpretive pitfall: Bubbles are not purely mechanical

- Mechanical blockage can interrupt downstream perfusion.
- Pulmonary cytokine release can injure alveoli.
- Even small bubbles may stimulate inflammatory and thrombogenic reactions.

Integrated clinicopathologic synthesis

Source	Mechanism	Consequence
Outside-air entrainment or rapid decompression	Gas bubbles: Obstruction and host response	Pulmonary, neurologic, musculoskeletal or skeletal injury

Reference

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).
- Sections used: Embolism; Air Embolism; adjacent descriptions of fat and thromboembolic material.
- Native diagrams are explanatory schematics; illustrative cases are not clinical records.