BENDS

Authored by

mohammad looti

November 9, 2025

BENDS (Decompression Sickness)

Primary Disciplinary Field(s): Diving Medicine, Hyperbaric Medicine, Physiology

1. Core Definition and Nomenclature

The term **Bends** is the common, colloquial designation for a spectrum of pathological symptoms resulting from **decompression sickness** (DCS). Medically, DCS is a disorder arising from the inadequate elimination of inert gases--primarily nitrogen--that have dissolved in the body tissues and fluids during exposure to elevated ambient pressure. When the surrounding pressure is suddenly or rapidly reduced, these dissolved gases come out of solution, forming microbubbles within the blood and tissues, a process governed by the physical principles described in **Henry's Law**. These gas bubbles cause both mechanical trauma and vascular obstruction, leading to the diverse clinical manifestations characterized by the Bends.

The condition is fundamentally classified as an environmental injury and falls under the purview of diving and hyperbaric medicine. Synonyms for this disorder include **aeroembolism** and **caisson disease**, the latter term specifically linking the ailment to the high-pressure environments found in underwater construction foundations. While the term Bends often strictly refers to the manifestation of pain in the muscles and joints--the classic and most common presentation--it is frequently used interchangeably with the broader category of Decompression Sickness, which encompasses more severe neurological and pulmonary symptoms. The severity of the condition dictates immediate medical intervention, usually involving therapeutic recompression.

2. Pathophysiology: Nitrogen Bubble Formation

The genesis of the Bends lies in the basic physics of gas dissolution in liquids, particularly under varying pressure gradients. During a dive or prolonged exposure to a hyperbaric environment (such as in a pressurized tunnel or caisson), the partial pressure of inert gases--typically nitrogen when breathing air--increases significantly. According to **Dalton's Law of Partial Pressures**, the body absorbs these gases until the tension in the tissues equilibrates with the surrounding ambient pressure. This process, known as saturation, is slower in some tissues (e.g., fat and cartilage) than in others (e.g., blood and lungs).

The critical physiological failure occurs when the external pressure is decreased too quickly, as happens during a rapid ascent. The inert gas, which is highly soluble under pressure, suddenly becomes less soluble at the lower pressure. If the decompression rate exceeds the rate at which the body can safely off-gas the nitrogen via the lungs, the gas rapidly nucleates and forms bubbles. These bubbles begin in supersaturated tissues and can grow large enough to cause significant cellular damage or aggregate to form **gas emboli**. These emboli then obstruct venous and, less

commonly, arterial circulation, leading to localized ischemia and inflammatory responses.

The size, location, and total volume of these nitrogen bubbles determine the clinical presentation of the Bends. Bubbles forming near joints and muscle fascia lead to the characteristic severe musculoskeletal pain, while those entering the central nervous system or blocking pulmonary circulation result in life-threatening Type II DCS. The inflammatory reaction triggered by the presence of these bubbles further exacerbates tissue damage, contributing to symptoms like edema and neurological deficits.

3. Clinical Manifestations (Symptoms of the Bends)

Decompression sickness is clinically divided into two major categories: Type I (mild) and Type II (severe). The symptoms described in the source material--severe pain, cramps, and difficulty breathing--represent both common Type I and serious Type II presentations. The onset of symptoms typically occurs within minutes to hours following decompression, although delayed onset up to 24 hours is also possible, particularly following marginal decompression profiles.

Type I DCS, often referred to simply as the Bends, primarily affects the musculoskeletal system and skin. The hallmark symptom is deep, aching pain, most frequently localized in the large joints (shoulders, elbows, hips, knees). This pain is often agonizing and resistant to common analgesics, compelling the affected individual to adopt a curled or "bent" posture, hence the derivation of the name **Bends**. Cutaneous manifestations may include mottling, itching (pruritus), and a rash, often referred to as skin bends. While Type I DCS is not immediately life-threatening, it requires prompt treatment to prevent chronic injury.

Type II DCS involves serious systemic compromise, affecting the neurological, pulmonary, or circulatory systems. **Neurological DCS** symptoms range from mild paresthesia and numbness to severe paralysis, confusion, and unconsciousness, resulting from bubbles lodging in the spinal cord or brain. **Pulmonary DCS**, often called "the Chokes," is characterized by substernal chest pain, a persistent dry cough, and the severe difficulty breathing (dyspnea) mentioned in the definition, resulting from massive gas embolization in the small vessels of the lungs. Without immediate recompression therapy, Type II DCS can lead to permanent disability or death.

4. Risk Factors and Predisposing Conditions

Several factors can significantly increase an individual's susceptibility to developing the Bends following decompression. The primary determinant is the **decompression profile** itself, meaning the depth and duration of the exposure. Deeper and longer dives necessitate greater inert gas absorption and thus require more careful, slower ascent schedules to facilitate safe off-gassing. Failure to strictly adhere to established decompression tables or the improper use of dive computers dramatically elevates risk.

Physiological factors also play a critical role. Variables such as age, poor physical fitness, and obesity increase risk because adipose tissue (fat) absorbs and retains nitrogen much more readily than lean tissue, making safe off-gassing more challenging. Dehydration reduces plasma volume and hinders nitrogen transport and elimination through the bloodstream, thereby increasing the risk of bubble formation. Furthermore, certain medical conditions, notably a **Patent Foramen Ovale** (PFO)--a small opening between the atria of the heart present in many adults--allows venous bubbles to bypass the pulmonary filter and enter the arterial circulation, significantly increasing the likelihood of catastrophic arterial gas embolism (AGE) or neurological DCS.

5. Historical Context: Caisson Disease

The understanding of the Bends emerged not from recreational diving, but from 19th-century industrial engineering. The condition was initially termed **Caisson Disease** because of its prevalence among workers constructing bridge foundations and underwater tunnels using pressurized caissons. These structures maintained high air pressure to prevent water intrusion, subjecting workers to prolonged hyperbaric exposure. A particularly high-profile outbreak occurred during the construction of the Brooklyn Bridge in the 1870s, where workers frequently experienced severe joint pain upon returning to the surface.

Early scientific investigation was driven by the catastrophic morbidity and mortality associated with these projects. French physiologist **Paul Bert** (1878) provided the foundational understanding, demonstrating experimentally that the symptoms were caused by nitrogen bubbles and that treatment required recompression to force the gas back into solution, followed by slow, controlled decompression. This discovery provided the theoretical basis for modern hyperbaric treatment.

Further critical advancements came from British physiologist **John Scott Haldane** in the early 20th century. Haldane, working for the British Admiralty, developed the first scientifically derived staged decompression tables, establishing the principle of limiting the ratio of pressure decrease in any given time. His tables revolutionized diving safety and laid the groundwork for all subsequent decompression models used today in both naval and recreational diving.

6. Prevention Strategies

Prevention of the Bends is paramount in hyperbaric operations and relies on meticulous adherence to established decompression protocols designed to manage the rate of inert gas off-gassing. The primary prophylactic tool is the **Decompression Table** or, more commonly today, the use of sophisticated electronic **dive computers** that continuously calculate tissue nitrogen loading based on depth and time. These tools ensure that the ascent rate is slow enough to prevent critical supersaturation.

No-Decompression Limits: Divers are trained to stay within depth and time limits that

theoretically allow direct ascent to the surface without requiring formal decompression stops, relying instead on a slow controlled ascent rate (usually 30 feet per minute or less).

Staged Decompression: For dives exceeding no-decompression limits, mandatory stops at specific shallow depths are required to allow nitrogen partial pressures in the tissues to drop safely before further ascent.

Safety Stops: Even on no-decompression dives, a voluntary 3-5 minute stop at 15-20 feet (5-6 meters) is standard practice to further reduce the risk of subclinical bubble formation, providing a crucial margin of error.

Gas Mixture Management: The use of alternative breathing gases, such as **Nitrox** (Enriched Air Nitrox), which contains a lower percentage of nitrogen than standard air, significantly reduces the nitrogen load absorbed by the body, thus extending no-decompression limits and lowering the overall risk of the Bends.

7. Treatment Protocols

The definitive treatment for the Bends, regardless of severity (Type I or Type II), is immediate **Hyperbaric Oxygen Therapy** (HBOT), often administered in a recompression chamber. The principle of this treatment directly reverses the pathophysiology of the condition. The affected individual is placed in a chamber and repressurized, sometimes to a pressure equivalent of 60 feet of seawater or deeper, which physically resuspend the nitrogen bubbles back into the solution within the blood and tissues.

Simultaneously, the patient breathes 100% **oxygen**, a crucial step in the therapeutic process. The high partial pressure of oxygen creates a very steep gradient for nitrogen. Because the body is being flooded with oxygen, the inert nitrogen gas is rapidly "washed out" of the tissues and eliminated through the lungs, shrinking the remaining bubbles. This process reverses the ischemic damage caused by the emboli and promotes the reoxygenation of deprived tissues.

Treatment protocols, such as the widely used U.S. Navy Treatment Tables, dictate the specific pressure profile and duration of oxygen exposure. Prognosis is heavily dependent on the time elapsed between the onset of symptoms and the initiation of recompression. Delaying treatment significantly increases the risk of permanent neurological or musculoskeletal damage. Specialized medical personnel trained in diving and hyperbaric medicine are essential for the safe and effective administration of this life-saving therapy.

Further Reading

Decompression Sickness (Wikipedia)
Diver's Alert Network (DAN) on DCS
Hyperbaric Medicine (Wikipedia)