Hyperbaric oxygen (HBO) therapy is successfully used in treating anaerobic infection that results in gas gangrene as well as severe aerobic infections such as necrotizing fasciitis and chronic refractory osteomyelitis. In addition, it reduces morbidity and mortality resulting from carbon monoxide intoxication. Many protocols using HBO to promote tissue and bone healing by increasing oxygenation have been investigated and therapeutic indications are better defined for using HBO in selected orthopedic situations.

**Physiopathology of Hyperbaric Oxygen Treatment**

The use of hyperbaric oxygen treatment rests on the principle of the solubility of gases in liquids. Oxygen is transported towards tissues by blood flow in dependent or dissolved form. In the event of, in particular, hypoxic tissue suffering, tissue hyperoxygenation is possible by increasing the dissolved quantity of oxygen per oxygen inhalation under pressures higher than the atmospheric pressure.

**Oxygen Transport to Tissues**

**In Normobaric Oxygen (20% oxygen)**

Alveolar stage: The diffusion of oxygen between alveoli and capillaries of the lungs is carried out according to a low gradient of pressure between alveoli and capillaries.

Blood stage: In blood, oxygen is transported in two forms: the major part is combined with hemoglobin (98.5%), and the other part, proportionally weaker (1.5%), is dissolved in plasma. The quantity \( q \) of gas that dissolves is directly proportional to the arterial partial oxygen pressure \( \text{PaO}_2 \), and to the coefficient of solubility \( \alpha \) in blood according to the law of Henry \( Q = \alpha \text{PaO}_2 \), which is 0.3 ml/100 ml of plasma. This intermediate dissolved form is essential to allow the fixing of oxygen on hemoglobin. In addition, the degree of saturation of hemoglobin depends on \( \text{PaO}_2 \) and varies according to a non-linear curve of dissociation. The maximum oxygen capacity fixed can reach a level of 20.1 ml/100 ml of blood, that is to say, a total quantity of oxygen transported of 20.4 ml/100 ml.

**In Hyperbaric Oxygen (100% oxygen)**

The increase in the arterial partial oxygen pressure has several consequences.

The increase in the quantity of oxygen dissolved can be enough to meet the tissue needs (6 ml/100 ml) with 3 atmospheres (3 ATA). The affinity to hemoglobin for oxygen is decreased.

A systemic arterial hypertension related to a peripheral vasoconstriction and a bradycardia reflex results, related to a vasoconstriction reflex to tissue hypoxia and not to a general
reaction which would involve a fall of the oxygen delivered with fabrics. The quantity of oxygen exchanged is, in fact, increased by pericapillary diffusion.

A persistent increase in the deformability of red cells.

**Effects of HBO on Tissue and Cell Growth**

HBO increases dissolved oxygen in the blood and results in a high partial pressure of oxygen (\(\text{PaO}_2\)). An increase of \(\text{PaO}_2\) maintains tissue oxygenation in the absence of hemoglobin [1] and also has an antiedematous effect. In fact, one finds in the microcirculatory sector a reduction in the blood flow of about 20% by the phenomenon of vasoconstriction reflex, which decreases the capillary flow of transudation and thus the formation of edemas. In addition, it stimulates fibroblast growth, increases collagen and intracellular adenosine triphosphate synthesis, and promotes more rapid growth of capillaries (angioneogenesis) [2,3] and osteoblastic and osteoclastic activity [4]. The stimulation of osteogenesis by HBO has been reported in animal experiments and clinical cases.

**Effects of HBO on Wound Healing**

The cicatrization of a wound is a complex phenomenon which brings into play several cellular types in a process of detersion, angioneogenesis, and cellular repair. After a first inflammatory phase with polymuclear surges of neutrophiles and macrophages, a second phase of granulation with conjunctive proliferation of neocapillaries, fibroblasts, and collagen synthesis begins. A last phase of epithelialization finishes the process. It has for a long time been recognized that delays or failures of cicatrization are primarily due to the existence of an infection or a local ischemia. The hypoxic area involves a reduction or even a stop of the cellular proliferation and collagen synthesis; from there a stop of neocapillary formation, just as a significant reduction in the macrophagic activity and bactericidal capacity of the polymuclear cells. In addition, a sensitivity particular to the infections of ischemic tissues deteriorates the local conditions.

An additional oxygen contribution, such as carried by hyperbaric oxygen treatment, involves a series of eutrophic and healing effects [2]:

- Increase in the distance of pericapillary diffusion of oxygen.
- Angiogenesis stimulation by accelerating growth of neocapillaries.
- Fibroblastic proliferation by increasing local oxygen pressure.
- Quantitative and qualitative increase (hydroxylation of the proline) of collagen synthesis.
- Epithelialization by increasing mitotic index, and mobility of the epithelial cells.

**Effects of HBO on Bone Healing**

The process of osseous repair is comparable with conjunctive repair. As soon as the fracture hematoma is formed an inflammatory reaction with detersion involves the osteolytic aspects of the distal ends of the fracture. Cartilage and osseous cellular differentiation occurs after a proliferative phase within young conjunctive tissue of granulation. It is performed, in fact, by a metamorphosis of the local fibroblasts which leads to two types of ossification; on one hand, enchondral, central, caused by the development of a cartilage matrix, and on the other hand, periostal, which produce an osseous structure directly. The two processes lead initially to osteoid plates, which will be the object of a secondary replanning leading to complete repair.

Hyperbaric oxygen treatment by the increase of the partial pressure of oxygen promotes the capillary proliferation to reappear, just as it restores normal capacities of synthesis and proliferation of osteoblasts. Osteogenesis and collagen synthesis are decreased by hypoxia, and a moderate increase of oxygen supply (80 mm Hg) stimulates their appearance [5]. The osteoclastic activity begins again, and allows resorption of the infected and necrosed osseous residues [4].