Annales Chirurgiae et Gynaecologiae 90: Suppl 215: 5–7, 2001

NEW ASPECTS OF HYPERBARIC OXYGEN THERAPY IN IMPROVING TISSUE SALVAGE AFTER ACUTE MUSCULO-SKELETAL TRAUMA

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Key words: Hyperbaric oxygen; wound healing; trauma

According to definition, both muscle and bone must be involved in acute musculo-skeletal trauma, but injury to other tissues such as skin, major blood vessels, connective and nerve tissues can also be involved. The injury may be so severe that either the viability of the tissue is in question, or, if the tissue survives, functional deficit is possible. Like in crush injuries the injury gradient is important in acute severe musculo-skeletal trauma (24, 25). In these cases a transition of tissue-injury severity from irreversibly damaged to minimally traumatized to normal exists. A "gray zone" between the irreversibly damaged and minimally traumatized tissues may or may not survive. Improving survival of this gray-zone tissue is crucial in the management of acute musculoskeletal trauma (27). This also serves a target for hyperbaric oxygen therapy (HBOT).

Physical damage and ischemia may contribute to each other in such a way that a vicious circle effect results (8, 25). Physically damaged tissues are contused or in discontinuity. The leakage of fluid through injured capillaries and cell walls results in tissue edema while disruption in cell activities causes loss of function. Edema results in progressive ischemia and hypoxia since the diffusion distance from the capillary to the cell is increased. Local tissue oxygen tensions should be 30–60 mmHg for effective wound repair and leukocyte oxidative killing mechanisms to occur (11, 18, 22). If critical ischemia is not corrected but continues, the tissues die. In closed injuries of the extremities increasing intracompartmental pressure becomes greater than the capillary perfusion pressure. Tissues within compartment become ischemic, cease to function, and die.

In soft tissue trauma, at the site where microcirculation is disrupted, plasma will leak into the injured tissue and cause further edema. At this level, oxygen supply to the cells will be only that resulting from the physically dissolved oxygen in the plasma. Under ordinary circumstances, this fraction of oxygen is insufficient for tissue needs.

TREATMENT OF ACUTE MUSCULO-SKELETAL TRAUMA

Acute musculo-skeletal trauma must be recognized without a delay and treated aggressively to prevent or minimize irreversible damage to injured tissues. Appropriate interventions must be started at the scene of the accident and continued until the problem is resolved (27). Splinting, control of bleeding, application of compression dressings to open wounds, and prevention of dependent edema are directed at preventing additional injury. Most important, however, is to employ basic and advanced life-support measures if required to keep the patient alive.

The emergency department physician is expected to perform a thorough physical examination, ascertain the severity of the injury, initiate appropriate x-ray and laboratory studies, and contact the specialists required for definitive care of the injury. When an open wound is a component of the musculo-skeletal trauma it must be inspected, cultured, irrigated, and covered with sterile dressings. Tetanus prophylaxis is taken care of. Antibiotics are administered intravenously. Broad spectrum antibiotic coverage is recommended. Attention to maintain blood volume is crucial.

Definitive treatment and follow-up care will be given by the specialist surgeon. The classification system for musculo-skeletal trauma should be used as a guideline (7). Additional studies such as intracompartmental tissue pressure measurements and angiography may be carried out to help the specialist determine the definitive treatment. Surgical intervention such as debridement, fasciotomy, and stabilization of fractures is required. Vascular reconstructive surgery can deal effectively with large vessel trauma. Further, attention to the patient's hemodynamics and nutritional status is essential. The volume and oxygen carrying capacity of the circulating blood must be maintained normal. Hyperbaric oxygen therapy should be employed to optimize tissue repair and achieve maximal tissue salvage (25, 27).

THE ROLE OF HBOT IN ACUTE MUSCULO-SKELETAL TRAUMA

Hyperbaric oxygen therapy can be used as an adjunct for the treatment of acute ischemic musculo-skeletal trauma. Hyperbaric oxygen is administered in either a multiplace or monoplace hyperbaric chamber. The multiplace chamber is pressurized with air and the patient breathes oxygen through a mask or intubation tube. The monoplace chamber is pressurized with oxygen and the patient breathes pure oxygen directly. Normally, pressures of 2–2.5 ATA are used.

Under HBO hemoglobin in the circulating blood becomes fully oxygenated and in addition, oxygen physically dissolves in plasma in direct proportion to the partial pressure of oxygen. Under HBO, enough oxygen can become physically dissolved in plasma to meet tissue oxygen requirements without support from hemoglobin-borne oxygen (3). At 2–2.5 ATA oxygen pressure physically dissolved oxygen in plasma increases over tenfold. This primary effect of HBO generates a favorable gradient for oxygen diffusion from functioning capillaries to ischemic tissue sites. This increment in oxygen supply may be the factor that allows the compromised tissues to survive, and so tissue viability and function are maintained (27).

Vasoconstriction is the second fundamental effect of HBO in the treatment of acute musculo-skeletal trauma (25). Vasoconstriction as such would appear to be undesirable in ischemic conditions because it reduces blood flow. However, the hyperoxygenation effect of HBO adequately compensates for the decreased flow so that the net effect is no reduction in tissue oxygenation. Inhibition of edema also has a beneficial effect on blood flow in the microcirculation (19).

The third effect of HBOT important in acute musculo-skeletal trauma is its role in wound and bone healing and prevention of infection in compromised tissues. In soft tissue wounds the most important effects of HBOT are the stimulation of fibroblast proliferation and differentiation, increased collagen deposition, neovascularization of ischemic tissue as well as enhanced leukocyte microbial killing (1, 16, 17). On the other hand, the supply of oxygen is a fundamental and, to a great extent, limiting factor in the healing of fractures (5) and recovery from osteomyelitis (13, 14). Variations in oxygen supply could determine the type of tissue that differentiated in a culture of multipotent mesenchymal cells. Hyperoxia caused the differentiation to osseus tissue, whereas hypoxia resulted in cartilage formation (2). In animal experiments HBOT has been found to stimulate the healing of fractures (21, 30).

HBO has direct effects on microorganisms. This is an additional mechanism that has relevance to the treatment of acute musculo-skeletal trauma and prevention of posttraumatic sequealae (10). In controlled studies in animal models HBO alone could eradicate Staphylococcus aureus in infected bone (12).

Hyperbaric oxygen also seems to protect tissues from reperfusion injury often involved in acute musculo-skeletal trauma (20, 23, 28, 29, 31). The mechanism of action is not known at present. However, the key to successful outcome with HBOT in the reperfusion injury appears to be its timely application (25).

Theoretically, the beneficial effect of HBOT in acute ischemic musculo-skeletal trauma is manyfold. HBOT improves the survivability of injured limbs and tissues. It accelerates the demarcation process and reduces the chances of infection developing in the transition zone between the injured and nonviable tissues. Further, HBOT enhances and supports repair mechanisms of soft and hard tissue lesions involved in acute musculo-skeletal trauma.

THE CLINICAL USE OF HBOT IN ACUTE MUSCULO-SKELETAL TRAUMA

Clinical experience with hyperbaric oxygen in acute musculo-skeletal trauma is relatively limited. The complexity and diversity of these injuries make double-blind controlled clinical trials nearly impossible. Most clinical reports describe the benefits of HBOT in very subjective terms. In reports dealing with crush injuries, the authors state that HBOT is beneficial because an injury of similar magnitude treated without HBO would have resulted in amputation (25). Recent reviews indicate that the more frequent the HBO treatments the higher the likelihood of success. HBOT has also reduced edema and congestion, especially in its applications for reimplantations (4, 6) and the skeletal muscle compartment syndrome (25).

In cases with severe ischemia HBO therapy should be instituted as soon as possible, because anoxic tissues quickly die. Muscle and peripheral nerve tissues die after four to six hours of ischemia while bone cells die after 12 to 18 hours. There is a question of whether debridement of the wounds and stabilization of the fractures should be instituted before the first hyperbaric oxygen treatment. It is recommended that surgical intervention take place before the first hyperbaric oxygen treatment. However, if the operating room is not immediately available, the patient should receive hyperbaric oxygen at pressures of 2.0 to 2.5 ATA for 60–90 minutes. If the operating room becomes available, the HBOT is interrupted and resumed after surgery is completed (25).

Hyperbaric oxygen treatment schedules for acute musculo-skeletal trauma vary according to what the desired effect of HBO is. If HBOT is used to promote healing of injured tissues, pulses of HBO once or twice a day appear to be sufficient. However, if tissue viability is threatened due to acute ischemia, HBOT should be given frequently enough to maintain oxygenation adequate for tissue survival. Serial measurements of local oxygen tension in injured tissues give a guideline to the frequency of hyperbaric oxygen treatments. Hyperoxia of muscle, bone and subcutaneous tissues may persist significant periods of time after a 60–90-minute exposure to oxygen at 2.0–2.5 ATA. It is recommended that hyperbaric oxygen should be given at four to six hour intervals until the ischemic condition is resolved, usually in 24 to 48 hours. If improvement occurs, the frequency of the HBO treatments is decreased over four to six

days. By that time the ischemic tissue should have stabilized, that is, tissue perfusion is restored and edema reduced. When treating patients under HBO with accelerated protocols, the possibility of toxic effects of oxygen should be minimized (25, 27).

In the future, the use of hyperbaric oxygen therapy in the acute musculo-skeletal trauma will be based on objective criteria rather than clinical diagnoses. Methods are now available to assess tissue oxygenation during HBO exposures. For instance, those injured tissues which increase their transcutaneous oxygen tensions significantly during an HBO exposure are highly likely to heal and survive (9, 15). Further, the measurement of interstitial fluid pressure in the compartment syndrome provides an objective parameter for starting and quantifying the benefits of HBOT (26).

CONCLUSION

Hyperbaric oxygen therapy is an ideal adjunct in the treatment of acute musculo-skeletal trauma in which tissue ischemia is involved. The immediate beneficial action site of hyperbaric oxygen therapy is the "gray zone" in the gradient of tissue injury. The primary goal of HBOT in this area is maintenance of tissue viability and function during the initial ischemia and hypoxia. A secondary goal is promotion of healing of both soft and bone tissues. Although HBOT will not revitalize dead tissue it speeds up demarcation and prevents infection of the injured tissue.

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