Adjunctive hyperbaric oxygen therapy contributes healing in electrical ınjury: a case report of high voltage electrical injury

Elektrik yaralanmalarında yardımcı tedavi olarak hiperbarik oksijen: yüksek voltaj elektrik yanığı olgusunda hiperbarik oksijen (HBO) kullanımının iyileşmeye katkısı

Maide CİMŞİT[1], Şamil AKTAŞ[1]

Elektrik yanıklarında kurtarılabilen doku oranını arttırmak ve klinik sonuçları arzu edilen düzeylere çıkarabilmek için yeni tedavi yaklaşımlarına ve protokollere ihtiyaç bulunmaktadır. İlişkin literatürün gözden geçirilmesi ve referansların taranması sonucu elektrik yanıklarında termal hasarı önlemek amacıyla hiperbarik oksijen tedavisinin kullanımına ait bir yayına rastlanmamıştır. Bununla birlikte hiperbarik oksijen tedavisi, elektrik yaralanmalarının kliniğini oluşturan termal yanık, ezilme (crush) yaralanması, nekrotizan yumuşak doku infeksiyonları, sorunlu yaralar ile deri greft ve flepleri gibi bir çok durumda endikedir.

Geri dönüşsüz hasar oluşmadan önce uygulanan hiperbarik oksijen tedavisinin yararlı olacağını göstermek üzere yüksek voltajlı elektrik çarpmasından sonra yardımcı tedavi olarak ilk hafta günde iki kez ve sonraki altı gün günde bir kez olmak üzere 2,4 ATA'lık basınçta 90 dakikalık toplam 20 seans hiperbarik oksijen tedavisine alınmış 11 yaşındaki bir olgu sunulmuştur

Tedaviden en fazla yararın beklendiği erken dönem (tedavi penceresi) geçildikten sonra oldukça gecikmeli bir evrede başlanmış olsa bile hiperbarik oksijen tedavisi ile nekroz, infeksiyon ve doku kaybına karşı savaşımda önemli faydalar sağlanmıştır..

Yara iyileşmesine katkıda bulunduğu için hiperbarik oksijen tedavisinden elektrik yanıklarında yardımcı tedavi ajanı olarak yararlanılabileceği ileri sürülmüştür. Olumlu sonuçların elde edilebilmesi için tedavinin ilk 24 saat içinde başlatılması daha uygundur.

Anahtar sözcükler: Elektrik yanığı, tedavi, hiperbarik oksijen,

In electrical injuries, new treatment modalities and guidelines are needed for improving clinical outcome and the survival of damaged tissue. Although there is no published study about hyperbaric oxygen (HBO) therapy for electrical injury in the literature, it is indicated in conditions, which may contribute to the clinical presentation of electrical injury such as thermal burns, crush injuries, necrotizing soft tissue infections, problematic wounds and compromised skin grafts and flaps.

An 11-year-old child with high voltage electrical injury treated with adjunctive hyperbaric oxygen for 90 minutes twice a day at 2,4 ATA for one week, then once a day for six days for a total of 20 sessions was presented to demonstrate the beneficial effects of hyperbaric oxygen therapy initiated before irreversible damage had taken place.

Although hyperbaric oxygen therapy was initiated rather late, when the most effective window for intervention had already past, HBO was effective in fighting against necrosis, infection and tissue loss.

Adjunctive HBO therapy is suggested for electrical injuries for its contribution to healing. In order to see the favourable effects of HBO, it is better to start the treatment within the first 24 hours following injury.

Key words: Hyperbaric oxygen, electrical injury

[1]İstanbul Üniversitesi İstanbul Tıp Fakültesi Sualtı Hekimliği ve Hiperbarik Tıp Anabilim Dalı

[1]Istanbul University, Istanbul Medical Faculty of Medicine, Department of Underwater and Hyperbaric Medicine

İLETİŞİM *(Correspondence)*: Maide Cimşit, İstanbul Tıp Fükültesi Sualtı hekimliği ve Hiperbarik AD 34390 Çapa-İstanbul
Faks: 0212 531 18 17 e-mail: cimsit@mail.com

Electrical injuries are not uncommon and account for 3-8% of the total admissions to burn units.[1-3] It is associated with a mortality rate of 0,5-2,5 per 100 000 persons/ year.[4,5]

Low voltage (<1000 V) injuries are commonly seen in children less than six years of age, whereas high voltage (>1000 V) injuries occur in older children and adolescents. [6]

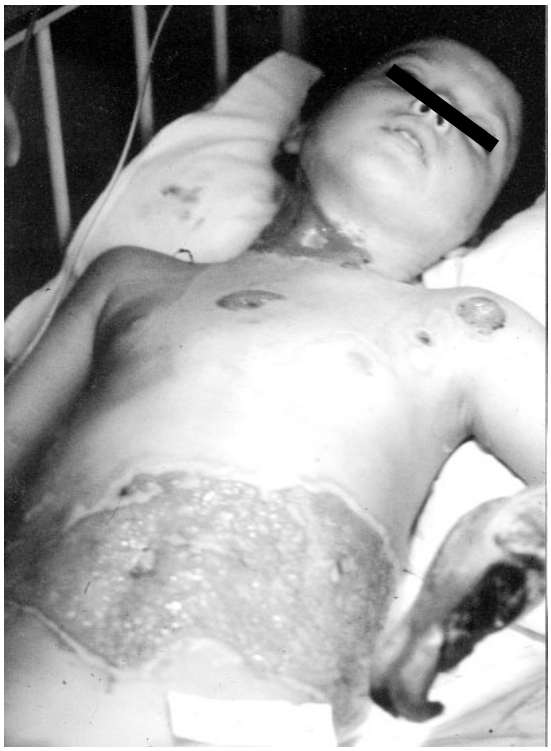
Although survival following electrical injuries has improved as a result of increased vital physiologic support, little progress has been made in improving the survival of damaged tissue.[7] Major limb amputation rates have been reported to be as high as 71%.[8]

The underlying pathophysiology of electrical injuries in living tissues is not fully understood[9]. Because of little progress in understanding of the pathophysiology of tissue injury, the clinical outcome has not improved substantially. Improvement of clinical outcome is expected due to new treatment modalities and guidelines based on more relevant pathophysiology.

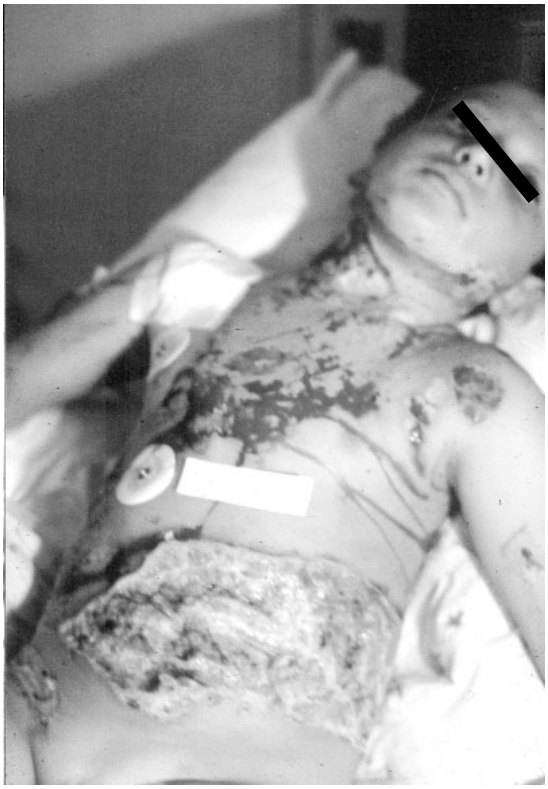
Hyperbaric oxygen (HBO) therapy, in which a patient breathes 100% oxygen intermittently while inside a treatment chamber at a pressure higher then sea level pressure (i.e., >1 atmosphere absolute), is used widely as an adjunctive therapy to resolve certain recalcitrant, expensive, or otherwise hopeless medical problems. The Hyperbaric Oxygen Committee of the Underwater and Hyperbaric Medical Society (UHMS) approved 13 indications for HBO in their current report revised in 1999.[10] On the other hand, European Committee of Hyperbaric Medicine (ECHM) approves 17 indications.[11] There is no published experimental or clinical study about the use of HBO in electrical injury in the literature, as far as we are aware of.

The primary rationale for using HBO is that it increases tissue oxygen tension in hypoxic tissue to levels, which make where it is possible for the host responses to function. With HBO at 2 atmosphere absolute (ATA) pressures, arterial blood oxygen content is increased by 125%, while plasma and tissue oxygen tension increase 10-fold. This results in a three-fold increase in oxygen diffusion through tissue fluids, partially compensating for the detrimental effect of edema on oxygen availability to cells.[10]

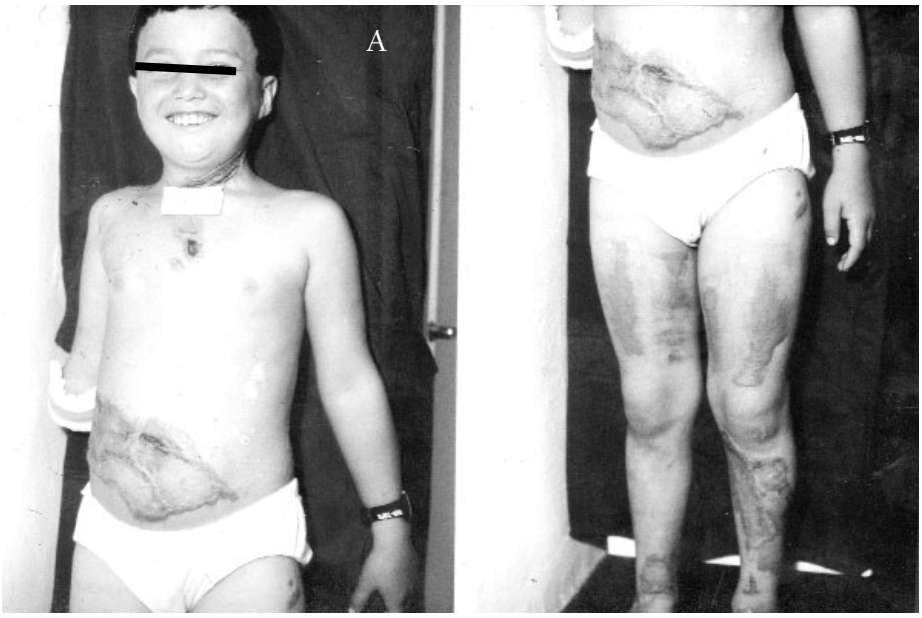
Edema reduction due to vasoconstriction is a secondary effect of tissue hyperoxygenation. Va-



Picture 1. Case TA. On the second day of HBO therapy.



Picture 2. At the end of the HBO therapy, before left forearm amputation and skin grafting.



Picture 3a and 3b. Case TA. On admittance for control.

soconstriction does not diminish local oxygen delivery, since it is maintained by hyperoxygenation. This anti -edematous effect of HBO is highly significant.[12]

Tissue oxygen tension influences the rate of collagen deposition, angiogenesis, and bacterial clearance in wounds. Fibroblastic proliferation, as well as synthesis, expression, and stabilization of collagen are processes that use utilize molecular oxygen. Neovascularization and epithelization are also promoted by HBO therapy in compromised tissue.[10, 13, 14]

CASE REPORT

An 11-year-old child who entered the emergency room with an electrical injury was examined in our clinic on the fifth day. There were 2nd and 3rd degree burns due to high voltage current which passed over 35% of his body from his left hand to his left foot. The fourth and fifth fingers of the hand were necrosed, and the others were contracted. There were also infected burn fields on the neck, anterior thorax and abdomen, and both arms and legs (Fig 1). The clinical examination was normal except for the burned areas and there was no cardiac arrest in the history.

At admittance, a three-fold increase in SGOT, a slight increase in SGPT, and a six-fold increase in creatine phosphokinase and lactate dehydrogenase levels were detected. No hemoglobin or myoglobin was present in the urine.

Before the start of HBO therapy, the urine output was maintained at 2500-3000 cc/day. Three minimal debridements and the 4th and 5th fingers and half of the 5th metacarp of the left hand were amputated. Staphylococcus aureus and Pseudomonas aeruginosa were found in the cultures taken from the wound sites. Antibiotherapy was administered daily as ciprofloxacin 200 mg bid, amikacin 500 mg qd and vancomycin 250 mg qid.

HBO therapy was applied for 90 minutes twice daily at 2,4 ATA for one week, then once a day for six days, to reach a total of 20 sessions.

Laboratory parametres normalized on the 10th day of admittance. Another debridement of necrotic tissues was performed. All wounds except the distal left arm were well granulated and free of infection (Fig 2). The left forearm of the patient was amputed from the proximal level, where the current entered the body.. Split thickness skin grafts were successfully applied to the other parts of the injury (Fig 3a, b).

DISCUSSION

High voltage electrical trauma results in cell damage by two different and also summative mechanisms: 1) thermal tissue damage, which occurs

as a result of the joule heating of tissue to supraphysiological temperatures,[15] and 2) electroporative damage which results from cell membrane breakdown by strong electrical forces. [16]

The pattern and extent of tissue injury is variable in electrical trauma. It can range from a minimal skin contact burn to amputation of extremities. Musculoskeletal and cutaneous injuries are the most common findings in electrical traumas.[17]

Electricity can lead to partial-thickness skin burns, full-thickness burns, or more extensive deeper tissue burns. Superficial injuries are usually caused by flash burns or splash burns. The high temperature can result in severe burns or ignition of clothing and surrounding materials.[18]

Electrical injury is usually presented with charred skin craters at the contact sites and an adjacent area of inflamed, edematous skin.[19] Edema also occurs in subcutaneous tissue and muscle. Although the edema is usually greatest around the contact sites, it extends for a considerable distance.[20] Within minutes to hours of injury, edema increases because of altered vascular permeability and release of intracellular osmotically active peptides. Edema worsens over the following 24-48 hours.[21]

When the pressure within the muscle compartment, which is caused by edema, exceeds approximately 30 mmHg, muscle perfusion pressure rises far above normal levels and ischemia occurs.[20] Compartment syndrome causes muscle necrosis and compression neuropathies. It has also been shown that heat or current-induced vessel damage results in aneurismal dilatation, tissue haemorrhage, vascular thrombosis and devascularization.[21, 22]

According to the Underwater and Hyperbaric Medical Society, Hyperbaric Oxygen Committee and ECHM, a thermal burn is one of the approved indications of HBO therapy. The main goals of burn therapy are minimizing edema, preserving marginally viable tissue, reinforcement of host defences and promotion of wound closure. Adjunctive hyperbaric oxygen therapy can cover these problems by maintaining microvascular integrity, minimizing oedema and providing the vital substrates necessary to maintain viability.

Ischemia-reperfusion injury is also important in the pathophysiology of electrical injuries.[23] Hyperbaric oxygenation appears to protect tissues from reperfusion injury. HBO inhibits the adherence of neutrophils to the endothelial wall of blood vessels and prevents the damage and extravascular extravasation and progressive edema. HBO antagonizes lipid peroxidation of the cell membranes.[24, 25] In addition, HBO provides sufficient additional oxygen for reperfused tissues to generate oxygen radical scavengers.[26-27]

Resembling the crush injury, progressive tissue necrosis is at greater extent than originally apparent trauma in electrical injuries. However, 'progressive necrosis' is still a controversial concept [3, 28,33] Delayed wound closure in the extremity leads to scar contracture, stiff joints and significant functional limitation. Therefore primary amputation is mostly applied at the first indication of debridement if the extremities are obviously mummified. The debrided wound can be left open for 24-48 hours until a second wound exploration and debridement can be performed. The tissue damage can be very patchy, irregular and scattered.[9] Therefore initially it might be very difficult to discriminate between viable and necrotic tissues. Recurrent exploration and debridement are often required.[6] The most widely practised surgical approach is to reinspect the wound and debride obviously necrotic tissue every 48 hours.[18]

In electrical injuries completely necrotic tissues are surrounded with a poorly oxygenated intermediate area and an outer zone where there is well-oxygenated healthy tissue. The main reason for using HBO therapy is to prevent or restrict tissue loss. For the already necrotic zone, the use of oxygen only hastens the development of demarcation zone. Oxygen therapy has no beneficial effect on necrotic tissue. For the intermediate area, on the other hand, the proper use of HBO can dramatically change the prognosis. This is an area where the favourable effects of HBO are seen. Anti-hypoxic, anti-edematous, and antibacterial effects of HBO, together with the promoting effect on wound healing reduces reduce tissue loss and morbidity.[10,34]

Infection is a major problem in electrical injury. If a skin burn is present, it potentiates cross-contamination from wound to wound. Wound treatment is much more difficult as topical antibiotics will not be able to reach the area of infection. During the initial 2-3 days, the principal causative organism is *Staphylacoccus* aureus. Later, organisms, such as *Pseudomonas* aeruginosa,

which prefers low-oxygen environment, are commonly encountered.[21] Also anaerobes such as Clostridiums may cause tetanus, gas gangrene and necrotizing soft tissue infections.

Hyperbaric oxygen has a direct antibacterial effect on anaerobes and microaerophylic aerobes, and an indirect effect on aerobic microorganisms. HBO facilitates oxidative killing of microorganisms by leukocytes, which is impaired in ischemic tissue.[35-37] It has also shown that HBO has a synergistic effect with some antibiotics such as aminoglycosides, quinolones, vancomycin and clindamycin.[38]

In order to observe these effects, HBO therapy must be initiated before the self-perpetuating problems of edema and ischemia lead to irreversible changes. The effects of inflammatory mediators that provide microvascular ischaemic cell death begin immediately after the injury and the most of the ensued ischemia would be expected to be complete by 72-96 hours.[39] If indicated surgery is delayed, HBO therapy can be administered before the surgery. The early application of HBO therapy is essential for maximum efficacy.[10]

In the case presented, HBO therapy was started on the 5th day of the injury when the most effective window for intervention has already passed. Nevertheless HBO was effective in fighting against necrosis, infection and tissue loss. The benefits could have been much more greater if HBO therapy started earlier, preferably within the first 24 hours following injury. We suggest that adjunctive HBO therapy promotes healing of electrical injuries.

REFERENCES

1. Hunt JL, Sato R, Baxter CR. Acute electric burns. Current diagnostic and therapeutic approaches to management. Arch Surg. 1980; 115: 434-438.
2. Haberal MA. An eleven-year survey of electrical burn injuries. J Burn Care Rehabil. 1995; 16: 43-48.
3. Luce EA. Electrical burns. Clin Plast Surg. 2000; 27: 133-143.
4. Ore T, Casini V. Electrical fatalities among U.S. construction workers. J Occup Environ Med. 1996; 38: 587-592.
5. Wright RK. Davis JH. The investigation of electrical deaths: a report of 220 fatalities. J Forensic Sci. 1980; 25: 514-521.
6. Jain S, Bandi V. Electrical and lightning injuries. Crit Care Clin. 1999; 15: 319-331.
7. Lee RC. Preface. In: Lee RC, Cravalho EG and Burke JF, eds. Electrical Trauma: The Pathophysiology, Manifestation and Clinical Manifestation. 1st ed. Cambridge: Cambridge University Press; 1992: xiii.
8. Rouse RG, Dimick AR. The treatment of electrical injury compared to burn injury: A review of pathophysiology and comparison of patient management protocols. J Trauma. 1978; 18: 43-47.
9. DeBono R.A. histological analysis of a high voltage electric current injury to an upper limb. Burns. 1999; 25: 541-547.
10. Hampson NB. Hyperbaric Oxygen Therapy: 1999 Committee Report. Kessington, MD: Undersea and Hyperbaric Medical Society, 1999.
11. ECHM, European Consensus Conference on Hyperbaric Medicine: Reports and Recommendations. Wattel F, Mathieu D, eds, .Medical University of Lille, 1994.
12. Nylander G, Lewis D, Nordstrom H, et al. Reduction of post-ischemic edema with hyperbaric oxygen. Plast Reconstr Surg. 1985; 76: 596-603.
13. Hunt TK, Pai MP. The effect of varying ambient oxygen tensions on wound metabolism and collagen synthesis. Surg Gynecol Obstet. 1972; 135: 561-567.
14. Manson PN, Im MJ, Myers RA. Improved capillaries by hyperbaric oxygen in skin flaps. Surg Forum. 1980; 31: 564-567.
15. DiVincenti FC, Moncrief JA, Pruitt BA. Electrical injuries: a review of 65 cases. J Trauma. 1969; 9: 497-507.
16. Lee RC, Kolodney MS. Electrical injury mechanisms: electrical breakdown of cell membranes. Plast Reconstr Surg. 1987; 80: 672-679.
17. Lederer W., Liedermann FJ, Cerchiari E, et al. Electricity-associated injuries I: outdoor management of current-induced casualties. Resuscitation. 1999; 43: 69-77.
18. Güloğlu R. Yanık. In: Kalaycı G, Acarlı K, Demirkol K, Ertekin C, Mercan S, Özmen V, Sökücü N, Tezelman S, eds, Genel Cerrahi. Nobel Tıp Kitabevleri; 2002: 283-296.
19. Lee RC. Pathophysiology and clinical management of electrical injury. In: Lee RC, Cravalho EG and Burke JF, eds. Electrical Trauma: The Pathophysiology, Manifestation and Clinical Manifestation. 1st ed. Cambridge: Cambridge University Press; 1992: 33-79.
20. Hunt JL. Soft tissue patterns in acute electric burns. In: Lee RC, Cravalho EG and Burke JF, eds. Electrical Trauma: The Pathophysiology, Manifestation and Clinical Manifestation. 1st ed. Cambridge: Cambridge University Press; 1992: 83-104.
21. Demling RH. Pathophysiology and clinical management. In: Lee RC, Cravalho EG and Burke JF, eds. Electrical Trauma: The Pathophysiology, Manifestation and Clinical Manifestation. 1st ed. Cambridge: Cambridge University Press; 1992: 122-132.

22. Stueland DT, Stamas P Jr, Welter TM, et al. Bilateral humeral fractures from electrically induced muscular spasm. J Emerg Med. 1989; 7: 457-459.

23. Hussmann J, Zamboni WA, Russell RC, et al. A model for recording the microcirculatory changes associated with standardized electrical injury of skeletal muscle. J Surg Res. 1995; 59: 725-732.

24. Zamboni WA, Roth AC, Russell RC, et al. The effect of acute hyperbaric oxygen therapy on axial pattern skin flap survival when administered during and after total ischemia. J Reconstr Microsurg. 1989; 5: 343-347.

25. Thom SR. Functional inhibition of neutrophil B2 integrins by hyperbaric oxygen in carbon monoxide mediated brain injury. Toxicol Appl Pharmacol. 1993; 123: 248-256.

26. Thom SR, Elbuken ME. Oxygen dependent antagonism of lipid peroxidation. Free Radic Biol Med. 1991; 10: 413-426.

27. Ferrari R, Ceconi C, Curello S, et al. Oxygen-mediated myocardial damage during ischaemia and reperfusion: role of the cellular defenses against oxygen toxicity. J Mol Cell Cardiol. 1985; 17: 937-945.

28. Garcia-Sanchez V, Morell PG. Electric burns: high- and low-tension injuries. Burns. 1999; 25: 357-360.

29. Robson MC, Murphy RC, Heggers JP. A new explanation for the progressive tissue loss in electrical injuries. Plast Reconstr Surg. 1984; 73: 431-437.

30. Zelt RG, Daniel RK, Ballard PA, et al. High-voltage electrical injury: chronic wound evaluation. Plast Reconstr Surg. 1988; 82: 1027-1041.

31. Parshley PF, Kilgore J, Pulito JF, et al. Aggressive approach to the extremity damaged by electric current. Am J Surg. 1985; 150: 78-82.

32. Ferreiro I, Melendez J, Regalado J, et al. Factors influencing the sequelae of high tension electrical injuries. Burns. 1998; 24: 649-653.

33. Luce EA. The spectrum of electrical injuries. In: Lee RC, Cravalho EG and Burke JF, eds. Electrical Trauma: The Pathophysiology, Manifestation and Clinical Manifestation. 1st ed. Cambridge: Cambridge University Press; 1992: 105-121.

34. Cimsit M. The role of hyperbaric oxygen therapy (HBO) in crush injuries. Ulus Travm Derg. 2002; 8(suppl): 161-163.

35. Mader JT, Adams KR, Sutton TE. Infectious diseases: pathophysiology and mechanisms of hyperbaric oxygen. J Hyper Med. 1987; 2: 133-140.

36. Knighton DR, Fiegel VD, Halverson T, et al. Oxygen as an antibiotic: the effects of inspired oxygen on bacterial clearance. Arch Surg. 1990; 125: 97-100.

37. Hohn DC, MacKay RD, Halliday B, et al. The effect of oxygen tension on microbicidial function of leucocytes in wounds and in vitro. Surg Forum. 1976; 27: 18-20.

38. Marzella L, Vezzani G. Effects of hyperbaric oxygen on activity of antibacterial agents. In: Oriani G, Marroni A, Wattel F, eds. Handbook on Hyperbaric Medicine. Milano: Springer-Verlag; 1996: 699-713.

39. Shaw JM, Robson MC. Electrical injuries. In: Herndon DN, ed. Total Burn Care. London: WB Saunders; 1996:401-407.