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Original Scientific Paper

Effect of Hyperbaric Oxygenation on the Level of Free Fatty Acids in Experimental Spinal Cord Injury in Rabbits*

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The aim of this study was to investigate the influence of hyperbaric oxygenation treatment (HBOT), a potent inhibitor of tissue hypoxia, on the motor activity and the spinal cord tissue concentration of free palmitic (C16:0), stearic (C18:0), oleic (C18:1n-9), arachidonic (C20:4n-6) and docosahexaenoic (C22:6n-3) acids, in rabbits with spinal cord injury (SCI). SCI resulted in paraplegia and accumulation of all analyzed free fatty acids (FFAs) in the spinal cord after neurotrauma. HBOT (222.915 kPa pressure of 99.5 % oxygen for 45 min), applied immediately after injury and followed by 8 consecutive daily treatments, improved motor impairment and completely prevented the SCI-induced increase in FFAs. These data suggest that a perturbation of the membrane lipid metabolism may contribute to the functional deficit associated with SCI, and HBOT may protect the injured spinal cord tissue, at least in part, by limiting these posttraumatic membrane lipid changes.

Keywords
free fatty acids
hyperbaric oxygenation
spinal cord injury
rabbit

INTRODUCTION

Fatty acids are components of membrane phospholipids, playing an important role in maintaining the structure and function of the cell membrane. A severe impact spinal cord injury (SCI) results in a decrease in total phospholipid content of the spinal nerve tissue for up to three days following injury,[1] but no major class of phospholipids is selectively hydrolyzed.[1–4]

Such impact injury to the spinal cord is associated with a biphasic increase in spinal cord free fatty acid (FFA) levels.[1] The initial increase observed within 5 min of SCI is followed by a decline 30 min after the injury. The secondary increase, appearing one hour after the SCI, is more persistent. It peaks 24 h post trauma and declines over the following six days.[1] Due to the specific phospholipid composition of neurons, free arachidonic acid (AA, C20:4) is reported to be the primary fatty acid released as a result of injury-mediated tissue damage. Murphy and co-workers have proposed that a release of FFAs, in particular AA, may not always involve general membrane degradation but rather an up-regulation and/or overstimulation of phospholipase (PL) A2 linked membrane receptors.[5] Release of FFAs, due to the acti-

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vation of membrane PLs and lipases,[6–8] is one of the first pathophysiological events that follows primary trauma of the spinal cord, independent of the type of injury.[1–3,9,10] FFAs, and AA in particular, may lead to a secondary damage to spinal cord neurons by inducing oxidative stress or increasing the intracellular calcium level.[11] Additionally, AA plays a significant role in the cell trauma by being a precursor for prostaglandin and leukotriene synthesis.[12]

Major factors contributing to the secondary tissue damage include a release of neurochemicals, impairment of the microcirculation, increase of vascular permeability, formation of oedema and tissue hypoxia.[13,14] It seems likely that these phenomena are interrelated in a complex way and that early modification of any of these factors might influence the others.[14]

Hyperbaric oxygenation treatment (HOBT) involves intermittent inhalation of 100 % oxygen under pressure higher than 1 atm (101.325 kPa).[15] Physiologically, this produces a directly proportional increase in the plasma volume fraction of transported oxygen, which is readily available for cellular metabolism.[16] The blood oxygen concentration at sea level is 0.3 ml/dl.[17,18] Tissues at rest extract 5 to 6 ml O_2/dl blood, assuming normal perfusion.[17,19] Administering 100 % oxygen at ambient pressure increases the amount of oxygen dissolved in the blood fivefold to 1.5 ml/dl, and at 3 atm (303.975 kPa) the dissolved oxygen content is approximately 6 ml/dl,[17] more than enough to meet the resting cells requirements without any contribution from oxygen bound to haemoglobin.[20] Thus, HBOT could sustain life without circulating haemoglobin.[18]

HBOT has been tested in various animal[21–24] and clinical studies.[25–29] It is the primary therapy for arterial gas embolism, decompression sickness, exceptional blood-loss anaemia and severe carbon monoxide poisoning, and adjunctive therapy for *Clostridial myconecrosis*, compromised skin grafts and osteoradionecrosis prevention.[20] HBOT is still under discussion[30] as adjunctive treatment for several clinical indications, such as stroke and brain trauma,[31,32] refractory osteomyelitis, selected problem wounds (diabetic and pressure ulcers), radiation-induced soft tissue injury, radiation-induced hemorrhagic cystitis, necrotizing fascitis, crush injury, compartment syndromes, acute traumatic peripheral ischemia, cerebrovasular accidents, head injury, spinal cord injury, retinal artery insufficiency, optic neuritis, fracture healing and bone grafts.[20,24,33] It has been shown to diminish the infarct size in focal cerebral ischemia[34] without secondary injuries due to oxidative stress,[35] and to prevent a decline in Na^+, K^+- ATPase activity in global cerebral ischemia-exposed rats.[36]

Even though extensive clinical experience with HBOT has proven to be beneficial, the exact biochemical mechanism is not clear.

The aim of this study was to examine the influence of HBOT on the spinal cord FFA content in rabbits exposed to SCI.

EXPERIMENTAL

Animals

The study was carried out on adult *Hila* rabbits, of both sexes, weighing 2.5–3.0 kg. They were obtained 3.5 months old from the *Rabbit Heaven* farm (Sesvete, Croatia). The animals were maintained on a 12 h light-dark cycle and allowed free access to food and water. All experiments were performed between 10:00 h and 12:00 h in a silent room, at a temperature of 22–24 °C. All animals were handled according to the guidelines of the Society of Neuroscience,[37] and all animal use procedures were approved by the Faculty Ethical Committee.

The animals were randomly divided into four experimental groups (n = 6): control group SHAM (sham-operated, laminectomized animals, without spinal cord injury), control group INJURY (laminectomized animals with spinal cord injury), control group SHAM-HBOT (laminectomized, HBOT-treated animals) and experimental INJURY-HBOT group (animals with spinal cord injury, HBOT-treated) (Table I).

After the injury and/or laminectomy, rabbits were individually housed and their bladders were emptied by manual compression daily. Hind limb motor activity was controlled daily over the course of the nine post-operating days and scored according to Tarlov's[38] system. The measures were performed blindly. Hind limb function was graded using a five-point ordinal scale as follows: 1 = complete paralysis; 2 = minimal functional movement; 3 = movement of legs, does not support weight; 4 = hopping, partially impaired; 5 = normal motor function.

Rabbits were monitored for 9 postoperative days because a spinal shock lasts for 5 to 7 days.[32]

At the end of the experiment, on the ninth post-operation day, animals were killed (following the recommenda-

TABLE I. Distribution of animals to experimental groups[a]

	Control SHAM	Control INJURY	Control SHAM-HBOT	Experimental INJURY-HBOT
HBOT for 9 days			+	+
Laminectomy only	+		+	
Laminectomy + spinal cord injury		+		+

[a] n = 6 per group.

tions for euthanasia of experimental animals);[39] the spinal cord was traversed and removed from the spinal canal, and as soon as possible frozen in liquid nitrogen and prepared at –84 °C for further analysis. Spinal cord samples were taken at the level of the second lumbar vertebra (L2).

Spinal Cord Injury

Medial dorsal lumbar laminectomy in rabbits was performed under pentobarbital sodium anesthesia (30 mg/kg intravenously) using the modified technique of Albin *et al*.[40] The vertebral column was exposed in the upper lumbar region and a one-segment laminectomy was performed at the level of the second lumbar vertebra (L2). In twelve animals, laminectomy was followed by contusion of the spinal cord, provoked by a strike of 150 g cm, which resulted in a spontaneously irreversible paraplegia. Namely, a tapered weight of 12.5 g was dropped from a height of 120 mm onto the exposed spinal cord, striking it with a force of 150 g cm, and producing paraplegia detected as a significant spinal cord evoked potential waveform abnormality. After the controlled SCI, the wound was completely sutured with the muscle and skin. Each traumatized animal received 500 000 IU (100 IU ≈ 60 μg) of benzyl penicillin. During the following nine post-operation days the general status, degree of injury, and the appearance of the wound were controlled.

Hyperbaric Oxygenation

Non-traumatized rabbits (SHAM-HBOT group) were exposed to HBOT once daily for nine days after laminectomy, lasting 45 minutes. The pressure in the HBO chamber was 222.915 kPa (2.2 bar) with a constant flow of 99.5 ± 0.5 % oxygen (Montkemija, Bakar, Croatia). The INJURY-HBOT group of rabbits was submitted to HBOT immediately after the injury and for the eight subsequent days, under the same conditions (Figure 1).

Constant flushing of the chamber atmosphere with oxygen at a rate of 10 l/min and Sofnolime granules (Molecular Products, Thaxted, Essex, UK), spread in a canister on the bottom of the chamber, successfully prevented CO_2 accumulation in the chamber atmosphere. The oxygen content was monitored continuously and maintained at ≥ 98.5 % (Oxymeter^{TM}, Dräger, Lübeck, Germany). The CO_2 concentration was not allowed to rise above 0.1 %. An environmental control system maintained the inner temperature and relative humidity at 25 ± 1 °C and 50 ± 20 %, respectively. After the HBOT, the chamber was decompressed within 5 min in order to prevent pulmonary barotrauma in the animals.[41]

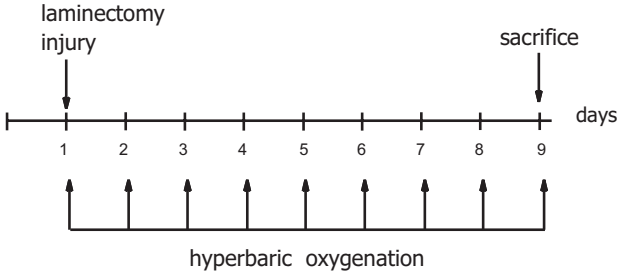

Figure 1. Schematic representation of the experimental protocol. The SHAM-HBOT and INJURY-HBOT groups of rabbits were placed into the experimental hyperbaric chamber immediately after operation (laminectomy or injury) and during eight post-operative days.

Free Fatty Acids

Total lipids were extracted from the spinal cord tissue by the modified method of Folch *et al*.[42] Frozen samples were weighed (200–400 mg) and homogenized in 10 mL of a chloroform-methanol (vol. ratio 2:1) mixture (Ultrasonic Fisher Scientific, Model 60, three times for 3 min at 5000 rpm). Nonadecanoic acid (C19:0) was added as an internal standard in all samples and the antioxidant butylated hydroxytoluene (BHT) was added at 50 mg/L in all solvents. Homogenates were left overnight at 4 °C. After filtration, 2 mL of 0.034 % $MgCl_2$ aqueous solution was added. The mixture was well mixed and left at 4 °C overnight. Water and organic phases were separated and the lower, organic phase was evaporated to dryness under N_2 gas. Total lipid extract was weighed and dissolved in 1 mL of the chloroform-methanol (2:1) mixture.

The FFAs were separated by preparative two step thin-layer chromatography on silica gel plates (Silicagel 60 F_{254}; Merck, Darmstadt, Germany, 20 × 20 cm, 0.2 mm) using the following developing solvents: petroleum ether-ether-acetic acid (vol. ratio 97:3:1) and petroleum ether-ether-acetic acid (vol ratio 80:20:1). Streaks on thin-layer plates were detected under a UV lamp and scraped off.

FFA methyl esters were prepared by methylation with a BF_3-methanol (3.0 mL, 14 % mixture; 90 min, 100 °C),[43] water was added, the mixture was extracted with petroleum ether, and quantified by gas chromatography using the C19:0 as internal standard. Identification was done by comparison with commercial fatty acid methyl ester standards (Sigma-Aldrich, Germany).

The fatty acid gas chromatography analysis was performed on the Perkin-Elmer, Model 8410 gas chromatograph with FID detector using a metal column (diameter Φ = 1/2 in, length 3 m) packed with 10 % FFAP on Chromosorb W. H P. 80–100 mesh, flow 30 mL/min.

Chloroform used for GC was SupraSolv purchased from Merck (Germany). All other reagents were of GR grade (Kemika, Croatia).

Data Analysis

The values are expressed as means ± S.E.M. of six rabbits. Analyses of variance (ANOVA) were performed at the $p \leq 0.05$ level to compare the results of different treatments. The Tukey HSD test was used *post hoc*.

RESULTS

Effect of Spinal Cord Injury

Untreated, sham-operated rabbits (SHAM) exhibited mild and temporary disturbances in moving their hind legs on

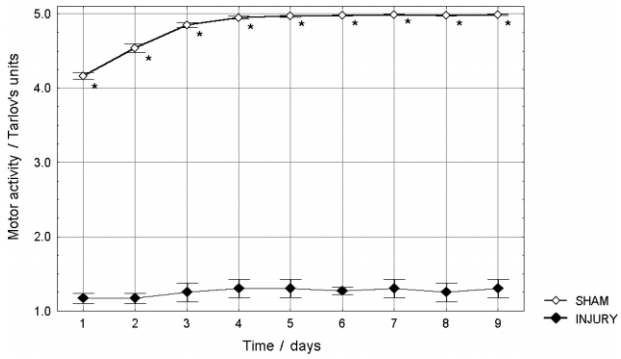

Figure 2. Motor activity (Tarlov's units) in animals of Control SHAM and Control INJURY during nine post-operative days. *$p<0.05$: significantly different Control SHAM.

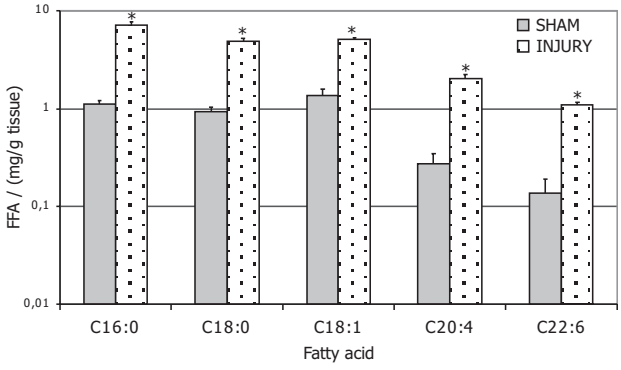

Figure 3. Effects of experimental impact injury on the level of FFAs in the rabbit spinal cord tissue. Values are expressed as mg FFA/g wet tissue, given by methyl esters of fatty acids after separation by gas chromatography, using nonadecanoic acid (C19:0) as internal standard. Only major components are shown: palmitic (C16:0), stearic (C18:0), oleic (C18:1), arachidonic (C20:4) and docosahexaenoic (C22:6) acids in laminectomized (SHAM) and injured (INJURY) rabbits. Each column represents mean ± S.E.M.; $n = 6$ for each group. *$p<0.05$: significantly different from control group SHAM.

the first and second postoperative days. These symptoms completely disappeared on the third postoperative day.

Contusion of the spinal cord provoked by a strike of 150 g cm resulted in a spontaneously irreversible paraplegia in the experimental animals. As a result, a statistically significant difference during the whole testing period was observed between untreated, sham-operated rabbits (SHAM) and untreated rabbits with spinal cord trauma (INJURY) (Figure 2).

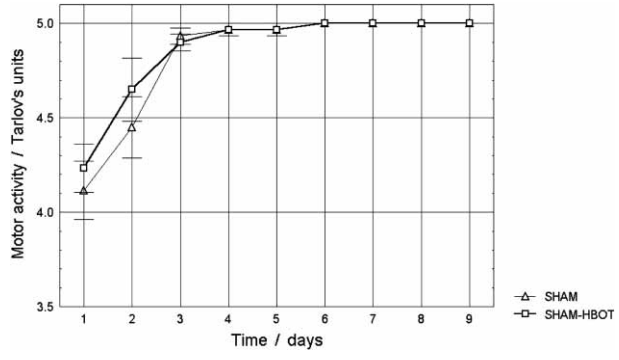

Figure 4. Motor activity (Tarlov's units) of laminectomized SHAM animals treated with HBOT during nine post-operative days. *$p<0.05$: significantly different from Control SHAM.

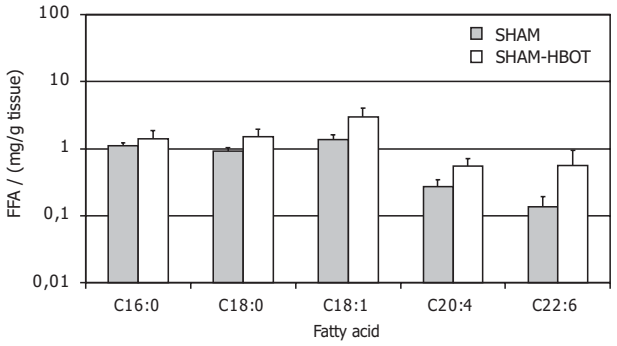

Figure 5. Levels of free palmitic (C16:0), stearic (C18:0), oleic (C18:1), arachidonic (C20:4) and docosahexaenoic (C22:6) acids (mg/g tissue) in the control group SHAM and the group treated with hyperbaric oxygen (SHAM-HBOT) (223 kPa, 45 min) 9 days after laminectomy. Each column represents mean ± S.E.M.; $n = 6$ for each group. *$p<0.05$: significantly different from Control SHAM.

Figure 3 shows the effect of spinal cord injury on the level of FFAs in spinal cord tissue in untreated rabbits. In comparison with sham-operated animals (SHAM), spinal cord injury (INJURY) was associated with a statistically significant increase in all examined FFAs: free palmitic (C16:0), stearic (C18:0), oleic (C18:1n-9), arachidonic (C20:4n-6) and docosahexaenoic acids (C22:6n-3).

Effect of HBOT

Treatment with HBOT had no influence on motor activity (Figure 4) or the FFA levels in the animals with laminectomy only (SHAM-HBOT *vs.* SHAM) (Figure 5).

Figure 6 presents the motor activity of untreated and HBOT rabbits with spinal cord injury (INJURY-HBOT *vs.* INJURY). ANOVA revealed a significant beneficial effect of HBOT and *post-hoc* analysis showed that mo-

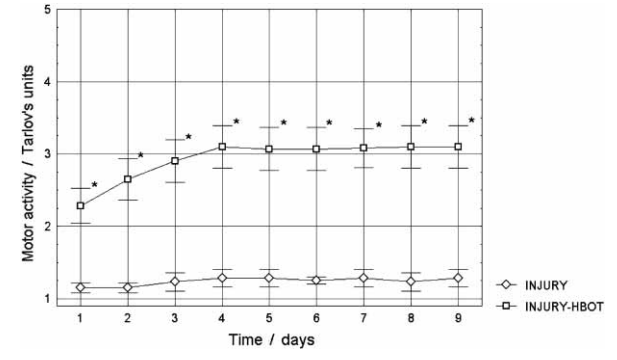

Figure 6. Motor activity (Tarlov's units) of injured animals treated with HBOT during nine post-operative days. (INJURY *vs*. INJURY-HBOT). *$p<0.05$: significantly different from control group (INJURY).

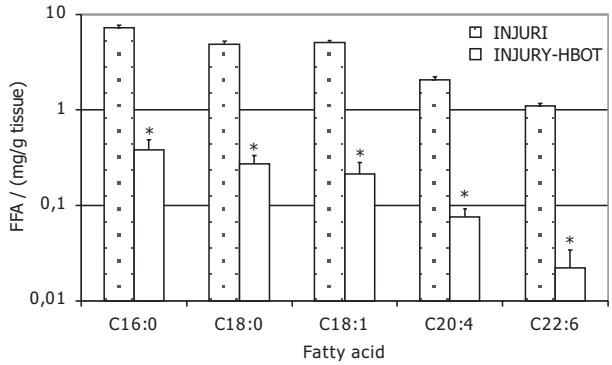

Figure 7. The effect of HBOT on the levels of free palmitic (C16:0), stearic (C18:0), oleic (C18:1), arachidonic (C20:4) and docosahexaenoic (C22:6) acids 9 days after the experimental spinal cord injury in rabbits (INJURY-HBOT). Each column represents mean ± S.E.M.; $n = 6$ for each group; *$p \leq 0.05$; significantly different from control group (INJURY).

tor activity was improved in all groups receiving HBOT compared to the untreated group with SCI.

HBOT resulted in a decrease in free palmitic [$F(1,9) = 138.33$; $p < 0.001$], stearic [$F(1,9) = 139.04$; $p < 0.001$], oleic [$F(1,9) = 341.49$; $p < 0.001$], arachidonic [$F(1,9) = 95.15$; $p < 0.001$] and docosahexaenoic [$F(1,9) = 216.63$; $p < 0.001$] acids in the rabbit spinal cord tissue samples (Figure 7). F is the variance ratio (defined as the ratio of two independent estimates of variance).

DISCUSSION

In this study, as expected, SCI resulted in motor disturbances and accumulation of FFAs at the injury site. The data correlates well with previously published reports.[1–3,5,44–46]

Loss or decrease of motor activity is the most prominent and the most functionally relevant result of spinal cord injury.[40] Motor disturbances observed in the rabbits that experienced trauma in response to the force of 150 g cm were not a result of the laminectomy procedure. Symptoms of the injured animals differed significantly from the mild and temporary symptoms observed in the group that was only laminectomized.

Following impact trauma, the spinal cord undergoes a progressive series of auto-destructive pathological changes.[2,3,47] A significant loss of motor function occurs in severe cases as a result of hemorrhagic necrosis of the central grey matter, which begins within minutes of injury. A very likely site of posttraumatic molecular damage is the cell membrane that undergoes marked alterations of integrity and function.[2,3] Changes in lipid metabolism may play an important role in delayed secondary tissue damage.[1]

Demediuk and co-workers, who followed a seven-day post-injury period, discovered that traumatic SCI results in early, transient, post-injury membrane phospholipid hydrolysis. In the second wave, more delayed and sustained lipid hydrolysis occurs, the magnitude of which is related to the severity of injury.[1] Under our experimental conditions, samples were taken 9 days after injury and we still observed a 6-fold increase in free AA.[48] This is in line with the gradual spontaneous wound-healing process that follows spinal cord injury and has been reported by other authors.[1,16]

Under our experimental conditions, HBOT did not alter spinal cord FFA levels of laminectomized animals. On the contrary, when the rabbits with SCI were exposed to HBOT immediately after injury and for eight consecutive days, the treatment significantly prevented SCI-induced increases in all measured FFAs. To our knowledge this is the first time that influence of HBOT on the FFA level and cell membrane integrity has been reported in a SCI model.

Our findings are in agreement with some earlier studies describing a beneficial effect of HBOT in SCI models. HBOT has been reported to improve neurological recovery following SCI.[49] Potential mechanisms of HBOT are: tissue protection (ameliorates the hypoxic state induced by oedema and circulatory compromise after SCI, reduced tissue oedema, preserves intracellular adenosine triphosphate, increases the flexibility of red blood cells, terminates lipid peroxidation, prevents adherence of PMNs (polymorphonuclear neutrophiles) to damaged endothelial cells), wound repair and healing promotion (enhances neovascularization, stimulates fibroblast growth, increases collagen deposition), as well as osteogenesis (synergistic effect with the osteoinduction effects of rhBM P-2, enhances osteoclastic activity).[50,51]

Narayana and co-workers performed magnetic resonance imaging (MRI) in order to assess the efficacy of HBOT in experimental SCI in the rat.[22] A moderately severe injury was chosen for these studies. Improvement in neurological recovery (based on the Tarlov scale)[38] was observed following HBOT treatment over a period of 72 h. Based on MRI data, HBOT appears to arrest the spread of haemorrhage and resolves edema.[22] Furthermore, Higgins and co-workers conducted a study of acute effects of HBOT on long-tract function following spinal cord trauma. Their observations suggested that HBOT can preserve marginally injured neuronal elements of the spinal cord long tracts during the early phases of traumatic SCI. These protective effects may be based on the reversal of focal tissue hypoxia, or reduction of tissue edema.[52] More recently, Murakami and co-workers[23] reported that shortly after ischemic injury HBOT has protective effects against ischemic spinal cord damage, though HBOT does not change the prognosis. This was the first study that demonstrated the influence of HBOT on delayed neuronal cell death in the spinal motor neurons. Additionally, these authors have shown that if HBOT is started 6 h after ischemic insults, it does not exhibit any protective effects on the spinal motor neu-

rons.[23] An earlier start of treatment is therefore necessary. Later, Huang and co-workers discovered that multiple HBOT in rats with SCI for up to 6 h after injury with further serial HBOT administration is superior to a single HBOT.[49] Namely, the group of animals receiving a single HBOT intervention beginning at 30 min and 3 h, or serial HBOT starting at 6 h following the injury had a significantly better neurological recovery than animals with SCI only.[49] In our study, animals were treated immediately after the injury and for 8 consecutive days, providing a combination of the two most successful HBOT regimens so far described[49] in order to achieve a tissue protective effect.

Delayed neuronal death observed by Murakami and co-workers could be associated with the decrease in FFAs observed in our experiment after treatment with HBOT since neuronal cell integrity is essential for its function.[23] SCI in our model seems to be a non-specific traumatic injury causing a non-specific liberation of FFAs and HBOT is able to prevent this non-specific membrane phospholipid cleavage and the resulting disturbances in cell homeostasis. Further detailed time-dependent changes should be studied, and then linked to particular biochemical changes.

The period after injury can be divided into three different therapeutic windows: biochemical and vascular events, starting at the time of injury and continuing for up to 48 h; the influence of inflammatory cells, starting within hours of injury and peaking four days after injury; and axonal regeneration and lesion repair starting one week after injury.[53]

HBOT seems to act throughout all three therapeutic windows. In the first phase, it induces vasoconstriction (preventing oedema formation) and in parallel it provides sufficient quantities of oxygen to the tissue (addressing problem of tissue hypoxia and resulting reperfusion injury), compensating for vascular insufficiency. Hence, the cascade of biochemical events started by hypoxia, Ca^{2+} influx and liberation of FFAs might be stopped, at least partially at the very beginning.

Following an ischemic interval, secondary injury is largely mediated by the inappropriate activation of leukocytes. Neutrophils have been implicated in reperfusion injury, which can worsen primary injury.[17] Adhering to the walls of ischemic vessels, they release proteases and produce free radicals, causing pathological vasoconstriction and extensive tissue destruction.[54] Hyperbaric oxygen inhibits neutrophil activation, adherence and postischemic vasoconstriction in ischemic rat tissue.[55,56]

Further, it has been recognized that much of the posttraumatic degeneration of the spinal cord following injury is caused by biochemical events involving reactive oxygen-induced lipid peroxidation.[57] HBOT could be also beneficial in this phase and antioxidant systems may be involved in its mode of action, despite the apparent contradiction in this statement.[58] As a matter of fact, decreased levels of the peroxidation product malondialdehyde, as well as increased activities of antioxidant enzymes, have been reported in some animal models after HBOT. Thus, Gulec and co-workers observed that malondialdehyde levels in erythrocytes, plasma and intestinal tissue were decreased and the levels of glutathione peroxidase (GPX) and superoxide dismutase (SOD) were significantly increased in rats with acute distal colitis treated with HBOT. Furthermore, HBOT was accompanied by a significant decrease in colonic weight, PGE_2 generation, myeloperoxidase, and NOS (nitric oxide synthase) activities in studies performed by another group.[59]

Contrary to these findings, prolonged exposure to oxygen under increased atmospheric pressure is known to produce significant toxic effects in a variety of body tissues.[60] However, the onset of such overt toxic manifestations can be delayed when HBOT is administered intermittently.[60] A number of studies suggest that pronounced levels of free radicals are only observed at high pressures (>3 atm (303.975 kPa)) or after long HBOT exposure.[61] Also, it has been reported that exposing rats to 4 atm (405.300 kPa) of oxygen for 90 min was associated with an increased level of lipid peroxidation products and that it altered enzymatic GPX in brain[49] and 100 % oxygen at 5 atm (506.625 kPa) produced seizures.[62] Free radicals were also observed when rats were exposed to HBOT for 48 hours.[63] In contrast, Dennong *et al*.[64] showed that HBOT (2.5 atm (253.313 kPa) 3 x 30 min) did not generate detectable free radical levels in humans, and Mink and Dutka[62] demonstrated that HBOT at low pressure (2.8 atm (283.710 kPa) for 75 min) was not associated with an increase in lipid peroxidation in a global cerebral ischemic model in rabbits despite increased amounts of free radicals in brain.[49] Nylander and co-workers reported that lipid peroxidation did not occur with the pressure and exposure time commonly employed in routine HBOT.[65]

Since we used 2.2 atm (222.915 kPa) for 45 min intermittently during our procedure and the motor score was significantly improved in HBOT rabbits *vs.* non-treated rabbits, we believe that it is more likely that the decrease in FFAs in our experiment is a result of decreased liberation from cell membranes and not of their greater oxidation into hydroxyl acids and isoprostanes. In this experiment, the decrease in FFAs observed could be a combined result of several elements of HBOT, such as prevention of the hypoxia-induced cascade of membrane degradation and free radical formation, inactivation of neutrophils and potentiation of antioxidant tissue mechanisms. We have observed similar dose-dependent decrease in FFAs after indomethacin treatment in this model of SCI.[66]

CONCLUSIONS

Under our experimental conditions, experimental SCI induced a significant increase in all the measured FFAs: palmitic, stearic, oleic, arachidonic and docosahexaenoic

acids. HBOT applied immediately after injury and followed by 8 consecutive daily treatments prevented the SCI-induced increase in spinal cord FFAs.

These data suggest that a perturbation of membrane lipid metabolism may contribute to the tissue necrosis and functional deficit associated with a SCI, and HBOT may protect the injured spinal cord tissue, at least in part, by limiting these posttraumatic membrane lipid changes.

Additionally, these results suggest that HBOT attenuates hypoxia-induced changes in the spinal cord tissue after injury and could be an adjunct in its treatment.

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SAŽETAK

Utjecaj hiperbarične oksigenacije na razinu slobodnih masnih kiselina u eksperimentalnome modelu kontuzije leđne moždine kunića

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Cilj ovoga rada bio je istražiti utjecaj hiperbarične oksigenacije, potentnoga sredstva korekcije hipoksije, na motoričku aktivnost kunića s povredom leđne moždine i koncentraciju slobodnih masnih kiselina (FFA, free fatty acid) u uzorcima tkiva povrijeđene moždine, i to palmitinske (C16:0), stearinske (C18:0), oleinske (C18:1n-9), arahidonske (C20:4n-6) i dokozaheksaenske (C22:6n-3). Rezultat eksperimentalne povrede leđne moždine je teški motorički deficit u kunića, ali i akumulacija svih analiziranih FFA. Hiperbarična oksigenacija (HBOT), terapija udisanjem 100 % kisika u uvjetima povišenog tlaka (222.915 kPa kroz 45 minuta) neposredno nakon povrede te još 8 uzastopnih dana, popravila je motoričko oštećenje i u potpunosti zaustavila porast koncentracije FFA uzrokovan povredom. Ovi podaci ukazuju da poremećaj u metabolizmu membranskih lipida može doprinijeti funkcionalnome deficitu, koji je posljedica povrede leđne moždine, kao i da HBOT može zaštititi povrijeđeno tkivo, ograničavajući posttraumatske promjene membranskih lipida.