24th Symposium

1st RIJEKA SYMPOSIUM ON HYPERBARIC OXYGEN THERAPY

21st April 2018.
9,00 am
Clinical Hospital Center Rijeka, Location Sušak, Lecture hall
Tome Strižića 3, Rijeka
Organizers

THE CROATIAN ACADEMY OF SCIENCES AND ARTS  
The Department of Biomedical Sciences in Rijeka

THE CLINICAL HOSPITAL CENTER RIJEKA  
Center for Underwater and Hyperbaric Medicine

UNIVERSITY OF RIJEKA - MEDICAL FACULTY

THE CROATIAN MEDICAL ASSOCIATION – Branch office Rijeka

Scientific Committee

Daniel Rukavina, president
Igor Barković, Mario Franolić, Jacek Kot

Organizing Committee

Mario Franolić, president
Boris Reinić, Igor Barković, Srđan Novak

Registration: 8,00 – 9,00 am

Free admission. Participants who want a certificate from the Croatian Medical Chamber need to register.
Refreshments during breaks and lunch are with no charge.

Information

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Introduction

Daniel Rukavina, M.D., PhD., Professor emeritus, Head of the Department of Biomedical Sciences in Rijeka, Croatian Academy of Sciences and Arts, Rijeka, Croatia

Mario Franolić, M.D., Clinical Hospital Center Rijeka, Rijeka, Croatia, President of the Organizing Committee

Welcome addresses

Davor Štimac, M.D., PhD., Professor, Head of the Clinical Hospital Center Rijeka, Rijeka, Croatia

Tomislav Rukavina, M.D., PhD., Dean, Medical Faculty University of Rijeka, Rijeka, Croatia

Đulija Malatestinić, M.D., PhD. Head of Department for Health Care Primorje-Gorski kotar County, Rijeka, Croatia

Alen Ružić, M.D., PhD, Associate Professor, Vice - Rector, University of Rijeka, Rijeka, Croatia

Vojko Obersnel, Mayor, City of Rijeka, Rijeka, Croatia

9,30 – 11,15 h

I. INTRODUCTORY LECTURES

Chairmen: Žarko Finderle and Mario Franolić

Boris Reinić, M.D., Clinical Hospital Center Rijeka, Rijeka, Croatia

Introduction and presentation of Center for Underwater and Hyperbaric Medicine

Igor Barković, M.D., PhD, Clinical Hospital Center Rijeka and Medical Faculty
University of Rijeka, Rijeka, Croatia

Introduction to Hyperbaric and Underwater Medicine

Žarko Finderle, M.D., PhD, Professor, University Medical Center Ljubljana, Ljubljana, Slovenia

Effects of hyperbaric oxygen on the cardiovascular system with special reference to the endothelium

Coffee break: 11,15 – 11,30
II. UNDERWATER MEDICINE

Chairmen: Pasquale Longobardi and Igor Barković

Pasquale Longobardi, M.D., Ravenna Hyperbaric Centre, Ravenna; Institute for Life Sciences- Scuola Superiore Sant’Anna, Pisa, Italy
Medical assistance at working diving site

Jacek Kot, M.D., PhD., Assistant Professor, National Center for Hyperbaric Medicine, Medical University of Gdansk, Gdansk, Poland
Extreme recreational diving

Lunch with a panel of speakers: 13,00 – 13,45

III. HYPERBARIC OXYGEN IN CLINICAL PRACTICE

Chairmen: Jacek Kot and Boris Reinić

Jacek Kot, M.D., PhD., Assistant Professor, National Center for Hyperbaric Medicine, Medical University of Gdansk, Gdansk, Poland
Severe Infections treated with Hyperbaric oxygen therapy

Marko Milošević, M.D., Clinical Hospital Center Rijeka, Rijeka, Croatia
Intensive Care in Hyperbaric Chamber

Coffee break: 15,00 – 15,15

Pasquale Longobardi, M.D., Ravenna Hyperbaric Centre, Ravenna, Italy
Hyperbaric oxygen therapy in the treatment of orthopedic trauma due to sports injuries

Mario Franolić, M.D., Clinical Hospital Center Rijeka, Rijeka, Croatia
Evaluation and Prognostic Values of Microcirculation Measurement with Transcutaneous Oxymetry (TCpO2) and Laser Doppler Flowmetry (LDF)

Hrvoje Stipančević, M.D., Department for Maritime, Underwater, and Hyperbaric Medicine, Naval Medical Institute Split, Split, Croatia
Hyperbaric oxygen therapy as a treatment for avascular necrosis of the femoral head – case presentation

IV. ROUND TABLE DISCUSSION

Moderators: Jacek Kot and Mario Franolić
**Introduction and presentation of Center for Underwater and Hyperbaric Medicine**

Boris Reinić
Clinical Hospital Centar Rijeka, Rijeka, Croatia

After decades of long efforts of hypebaric medicine enthusiasts, than have finally become successful. In 2014., within the project for a new hospital in Rijeka, construction of the future Center for underwater and hyperbaric medicine had begun. Construction work, furnishing and equipping was finalized in 2016.

The space in which the chamber is placed is 120 square meters, and the layout of remaining spaces allows a fast flow of patients, easier patient groups rotations and effective ambulatory and clinic work. Beside a general practice clinic, there is also a surgical clinic equipped in a manner that allows us performing smaller surgical procedures in local and regional anesthesia.

Multiplace hyperbaric chamber STARMED-230 was manufactured by the German manufacturer HAUX. It has 8 sitting or 2 lying places for patients, 1 place for medical personnel and 2 places in the pre-chamber. Upper limit for working pressures is 6 ATA (absolute atmosphere). Also, inside the chamber there is a mechanical ventilator with several modes of ventilation, vital functions monitor, defibrillator, perfusors, aspirator and other medical equipment certified for use in conditions of elevated atmospheric pressure which allows us treating patients who require intensive medical treatment (ICU patients).

For a safe and effective functioning of all parts of our Center it is necessary to have a wide variety of professional personnel. Physician structure is based on the principle of multidisciplinary approach. According to this, anesthesiologists, intensive care and emergency medicine physicians, pneumologist, and a traumatologist are involved in our daily work. Our medical technicians and nurses are all highly educated and experienced in anesthesia, ICU, emergency medicine and surgery. Technical part of our Center is being managed by an electrical engineer who is also in charge of all safety measures.

A great benefit of being a part of the University hospital Rijeka, is that this allows us a constant access to all diagnostic tools and therapeutic possibilities, such as medical institution can provide. All employees have been through basic diving training and various forms of education in the field of underwater and hyperbaric medicine.

Patient care in the Center started in December, 2016. In the first year of our work there were approximately 6000 treatments. The majorit of these patients
were treated or had chronic wounds, circulatory insufficiency and osteomyelitis. Emergency treatments in the hyperbaric chamber were due to decompression sickness in divers, carbon monoxide poisoning and crush injuries; all of those were successfully treated.

Also, in our first year of work we accomplished our basic goals, achieved high level of medical care, high safety standards and a high level of patient satisfaction. Further development is directed to scientific research, cooperation with other hyperbaric medicine centers, participation in international projects and implementation into Faculty of Medicine Rijeka curriculum.

To conclude, let others speak, in this instance Francois Burman (DAN director): „Your facility is really excellent; yes, technically very good, but the commitment from the staff is what makes it really top-class”.

Introduction to Hyperbaric and Underwater Medicine
Igor Barković
Clinical Hospital Center Rijeka, Rijeka, Croatia

During underwater activities human body is exposed to extreme change of pressure. A basic knowledge of physics and physiology is essential to understand most of the medical problems encountered. Diving Medicine is a specialized area of medical science dealing with the biological effects of the undersea environment on health and safety.

Hyperbaric Medicine deals with administration of oxygen under pressure as a medical treatment and is another specialized area of medical science that overlaps with Diving Medicine. Both share same basic physical and physiological lows and principles. Underwater heat exchange, sounds, light, colours, sensations change. Under pressure gas spaces compresses and physical gas laws become very important, partial pressures of gases rise, solubility of gases increase and gases become metabolically active, toxic or even can be used as medicine. Inhaling 100% oxygen under pressure greatly increase amount of oxygen dissolved in plasma. Great amount of oxygen in plasma is used as medicine and promotes angiogenesis and wound healing, kills certain anaerobes, prevents growth of some microorganisms shows antibiotic synergy, prevents production of clostridial alpha toxin, restores neutrophil mediated bacterial killing, reduces leucocyte adhesion in reperfusion injury, reduces lipid peroxidation.

The patient can be administered with systemic oxygen via two hyperbaric chambers: multiplace or monoplace.
Effects of hyperbaric oxygen on the cardiovascular system
with special reference to the endothelium
Žarko Finderle
Faculty of Medicine, University of Ljubljana, Ljubljana, Slovenia

Hyperbaric oxygen therapy (HBOT) has many consequences on the cardiovascular system. Some of them are beneficial, the others may pose additional risk, especially to the elderly patients and patients with cardiac disease. Reduced ventricular contractility, electrical disturbances and even arrhythmias have been reported. Underlying causes could be decreased excitability of cardiomyocytes, excitation – contraction coupling disturbances and bradycardia associated with increased heart distension. Bradycardia seems to be the most important factor in cardiac output decrease. The most probable cause of sinus bradycardia is increased parasympathetic tone. This acts through the stimulation of vagal centres (nucleus solitarius and dorsal motor nucleus). The common pathway of increased vagal tone to the heart is mediated by arterial baroreceptors located in the wall of carotid arteries and aortic arch. The important role of arterial baroreceptors was confirmed by the fact that, if afferent pathways from baroreceptors to the brain were abolished, bradycardia was significantly diminished or even replaced by a slight tachycardia. These baroreceptors are stimulated by arterial blood pressure increase. Generalised vasoconstriction in healthy vasculature is a well-known effect of HBOT and has a favourable effect on tissue oedema reduction with no concomitant reduction of oxygen delivery to tissues. As a matter of fact, tissue oxygenation is even improved due to the increase in amount of oxygen dissolved in plasma and consequently greater diffusion distance.

The underlying mechanism of hyperoxic vasoconstriction is a reduction of nitric oxide (NO) production in endothelial cells. Indeed, flow mediated vascular dilatation, which is attributed to increased production of NO by the endothelium in systemic vasculature during greater shear stress, was reported to diminish immediately after HBOT as well as after dives, especially after wet dives. On the other hand, repeated hyperoxic exposures may cause upregulation of endothelial NO synthase (eNOS) and neuronal NO synthase (nNOS), as was shown in mice and in experiments on cultured cerebral endothelial cells (upregulation of eNOS several hours after the exposure).

Endothelium also plays an important role in angiogenesis. Endothelial cells migrate into the wound, this being induced by vascular endothelial growth factor (VEGF) from macrophages. Studies have demonstrated that HBOT increases VEGF production already by increasing oxygen tension in and around wounds. VEGF upregulation was found also in endothelial cells gathered from human umbilical vein, the mechanism involving a transcription factor AP-1.
HBOT is probably involved also in the production of hypoxia-inducible factor (HIF), which, in turn, increases VEGF production.

Besides VEGF, HBOT promotes production of other growth factors involved in angiogenesis (fibroblast growth factor, platelet-derived growth factor), possibly through NO production. HBOT causes gene expression of angiogenin, being an angiogenesis and NO-production promotor. HBOT also increases vasculogenesis by increasing NO production in the bone marrow and consequently production of bone marrow-derived endothelial precursor cells.

Medical assistance at working diving site

Pasquale Longobardi\textsuperscript{1,2}

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Working divers have strenuous work, sometimes in remote locations far from effective medical support. They must be able to react to emergencies for themselves and their team mates. They must not have medical conditions that could get worse by diving or that could affect their ability to dive safely. They must be realistic and acknowledge their obligations to themselves, their team mates and employer. They must maintain an adequate level of fitness. According to the recommendations of the DMAC Workshop “Improving Diver Safety” held in Aberdeen 2014, attention should be paid to the question whether divers and the conditions at dive site were fit. The ‘As Low As Reasonably Practicable’ (ALARP) and ‘As Low As Reasonably Achievable’ (ALARA) principles should be applied (they are embedded in UK legislation). The Diving Contractor’s medical adviser or medical support organization is an important contribution to project planning (which involves the safe design of the project, appropriate consideration of the working environment and aspects of emergency preparedness and response). Oil Gas Producers (OGP) Association points out that safety depends on a valid Risk Assessments, but something could go wrong so, the Diving Medicine Specialist as Consultant is mandatory. Based on the Hazard Identification Process (HazId) for each dive site, the more appropriate management of illness or injury (≥ in saturation) could be: only the Diver Medic Technician (DMT), the availability of ship or rig medics, Company doctor available via telemedicine; On-call emergency medical team (near the dive site); Medical staff at working diving site.
Offshore Hazards could be grouped into

1) **Worksites activities related injuries (90%)**: Trauma and Non-diving related medical incidents.

2) **In-water Diving accident**: Dangerous marine animals, Technical or medical emergency, Entrapment, In-water contamination, Loss of consciousness

3) **Bell**: loss of consciousness; uncontrolled decompression of a Submersible Decompression Chamber (SDC) during diving operations; Loss of the Bell.

4) **Decompression chamber**: Contaminated environment; Non-diving related medical incidents; Fire; Accelerated emergency decompression from saturation.

The Diving Physician Specialist have to know and manage very well the 25 notes and the 2 workshops published by the Diving Medical Advisory Committee (DMAC, www.dmac-diving.org) and the MEDEVAC planned by the Dive Contractor in case of emergency Medical Evacuation of the sick or wounded diver.

The Medical Examination and assessment of commercial divers have to performed according the National regulations. The HSE (UK) MA1 procedure (revised in 2015) is a good reference as there is an increased focus on the BMI and weight in order to prevent cardiovascular accidents in commercial divers aged 40 or more. It’s an incentive to maintain fit. A diver is considered unfit if the BMI is more than 35, the waistline is more than 102 cm, the maximal Oxygen Uptake (VO2 max) less than 40 ml/min/kg. However, seeing as it is arguable that saturation diving is more strenuous than surface decompression diving, could be controversial if the saturation ban is required when the BMI is more than 30, waistline is more than 102 cm (male) but the maximal oxygen uptake (VO2 max) remains between 40-44 ml/minute/Kg.

The European Diving Medicine Databank (EDMD, http://www.edmd.eu/) shows the list of the Medical Examiners of Divers (MED) in Europe, approved by the DMAC/EDTmcmed National Coordinators on the basis of a logbook that analyzes knowledges and skills of each doctor.

From 2009 to 2016 the group Dive_Doctor (Italy) has provided 4,938 days of medical assistance at working diving sites, 691 days of telemedicine (only in 2016) and 1.486.450 € have been paid to Diving Physician Specialists. There are presented the statistics of the Medical assistance at following dive sites: Concordia Parbuckling Project (Italy), Azeri-Chirag-Deepwater Gunashli Project (Azerbaijan), Mosul Dam Project (Iraq), Ismailia road tunnel (Egypt).
Extreme recreational diving
Jacek Kot\textsuperscript{1,2}

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\textsuperscript{2}Medical University of Gdansk, Gdansk, Poland

In recreational diving, the risk of death is low when concerning dives up to 30 meters of depth, with compressed air in an open circuit, always with a qualified supervisor (certified buddy) and in comfort conditions (temperature, waves, visibility, currents, flora & fauna). This risk can be even 10 times higher when taking into account technical divers using closed circuit rebreathers. We can also observe the increasing number of divers doing the extreme diving for recreational purposes. This includes diving in remote locations (caves, high altitudes, North Pole) with problems of medical consultations and evacuations and diving to extreme depths with physiological problems typical for commercial divers. In fact, clear differences between professional and recreational deep diving are disappearing, at least when taking into accounts types of breathing mixtures (oxygen, nitrox, heliox and trimix) and range of dive parameters (depth and time). Training of recreational deep divers is conducted at depths of 120 - 150 meters and some divers dive to 180-200 meters using the same diving techniques. Extremely deep recreational divers go to depths over 200 meters, where physical and chemical properties of breathing gases create some physiological restrictions already known from the professional deep diving. One risk is carbon dioxide retention due to limitation of lung ventilation caused by high density of breathing gas mixture at great depths. This effect can be amplified by introduction of additional work of breathing if there is significant external resistance caused by a breathing device. The other risk for deep divers is the High Pressure Neurological Syndrome (HPNS) caused by direct compression effect, presumably on the lipid component of cell membranes of the central nervous system. The scope of modern diving medicine for recreational divers should be expanded also by these problems which previously were assigned exclusively to professional and military divers.
Severe Infections treated with Hyperbaric oxygen therapy

Jacek Kot¹,²

¹National Center for Hyperbaric Medicine, Gdansk, Poland
²Medical University of Gdansk, Gdansk, Poland

In Necrotizing Soft Tissue Infections (NSTI) necrosis is usually related with anaerobic bacteria (but can be mixed) which gives specific view, smell, and exudate. Initially it is very painful, then can be painless. It involves skin, subcutaneous layer, fascia and muscles. Most often it spreads by deep fascia (Necrotising Fasciitis). If Clostridium perfringens is present, the clostridial myonecrosis develops with many enzymes released from bacteria giving general symptoms of encephalopathy, cardiomyopathy and renal failure. In microbiological tests, the most often detected anaerobic bacteria includes Peptostreptococci spp and Bacterioides spp, the most characteristic anaerobes include Clostridium perfringes and the most often detected aerobes include Streptococcus sp and Staphylococcus sp. In most cases, there are some host-related factors like diabetes mellitus, arteriosclerosis obliterans or immunosuppression. HBOT in NSTI preserves critical ischemic areas, decreases bacterial toxin production, has bacteriostatic or bactericidal effect on anaerobes, restores bacterial killing by PMN, potentiates the efficacy of certain antibiotics, stimulates neo-angiogenesis, minimizes tissue oedema, modulates the systemic inflammatory reaction and acts synergistically with specific antibiotics (eg. Linezolid, Vancomycin, Teicoplanin or Imipenem). The best results of treatment are observed in those cases when the multi-modal approach is used. This includes Intensive Care with antibiotics, surgical approach with necrectomies, fasciotomies or amputations (when unavoidable) with leaving wounds open and HBOT started as soon as possible using high dose of oxygen (2.5 – 2.8 ATA for 60-90 minutes every 8 or 12 hours) with continuation of intensive care also during the HBO session. Taking into account clinical burden with synchronisation of those actions, it can be concluded that modern treatment of NSTI with HBOT is as challenging task for clinical team.
Hyperbaric oxygen therapy (HBOT) is a systemic, intermittent administration of oxygen in conditions when the ambient pressure is higher than 1 ATA (absolute atmosphere). Therapeutic effects of hyperbaric oxygen are seen at pressures higher than 1.4 ATA. Breathing oxygen at higher pressures increases the fraction of oxygen dissolved in plasma and as such demonstrates the beneficial effect on the whole organism, particularly in hypoxic tissues, while no such effect occurs when breathing 100% oxygen in normobaric conditions as it only increases the percentage of oxygen bound to hemoglobin. University Hospital Rijeka acquired a Haux® chamber (STARMED-230) with 8 seating positions, and 2 additional seats in the pre-chamber. Chamber has a built-in mechanical ventilator (SIARETRON 1000 IPER Siare), vital functions monitoring and perfusion systems approved for use inside the chamber, which altogether enables treatment of severely ill patients. It also has a defibrillator/pacing unit with 12 channel ECG, SpO₂, IBP options.

Our HBOT team is highly specialized consisting of a surgeon specialized in wound treatment, pulmonologist versed in screening, evaluating and approving patients for HBOT and an experienced anesthesiologist on our team, with 2 anesthesia and intensive care medicine residents and 1 emergency medicine resident which allows us to monitor and treat critically ill patients.

Current application of intensive care in hyperbaric chamber is based on close cooperation with the department of anesthesiology and intensive care medicine. We have so far treated patients with traumatic brain injury, diffuse axonal injury, hypoxic-anoxic injury. Medical personnel has to be highly trained and well educated in managing such a patient. Equipment used has to be certified for use in conditions of elevated atmospheric pressure. Moreover, use of such equipment has to be in a manner of high safety and security standards due to an increased possibility of adverse events such as fire. As we can see, challenges of treating such patients in such an environment are many, as so are the potential rewards in seeing improvement in their health.

Key words: hyperbaric oxygen therapy, intensive care medicine, team, equipment
Hyperbaric oxygen therapy in the treatment of trauma related to sports injuries
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\textsuperscript{2}Affiliated Researcher Institute for Life Sciences - Scuola Superiore Sant’Anna, Pisa, Italy

In sports injuries, Hyperbaric Oxygen Therapy (HBOT) is appropriate in brain concussion and orthopedic damage. The cerebral vascular architecture is well structured: the distance between a neuron a capillary is never more than 10 microns. In brain concussion the destruction of the Blood-Brain Barrier (BBB) is an early event (Hay J., 2015). The consequences (which may persist for several years) are: Post traumatic epilepsy; Parkinson’s / Alzheimer’s and similar diseases; Chronic encephalopathy. The trauma causes extravasation of albumin in brain tissues (which is captured / absorbed by astrocytes). The damage is worsened by the reduced functionality of the potassium channels (aquaporin 4, glutamate) and the up-regulation of TGF\textbeta (pro-inflammatory). There is a positive synergy between HBOT and inhibitors drugs of albumin uptake (eg losartan potassium that inhibits TGF\textbeta receptors) in reducing the outcome of concussion, such as post-traumatic epilepsy.

Another consequence of brain injury in head trauma is the increase of intracellular calcium, which is neurotoxic. The HBOT acts on the calcium channels (in particular on the voltage sensor subunit) blocking the entry of calcium into the cell. In the concussion there is an increase in dopamine and serotonin; the GABA (in the brain) and the glycine (in the marrow), that have inhibitory activity, increase due to non-deactivation.

Post-traumatic brain edema is caused by mitochondrial dysfunction. Protecting the mitochondria is the new therapeutic strategy to reduce the consequences of a head trauma (Vlodavsky E et al, 2017). The HBOT avoids the opening of the mitochondrial Permeability Transition Pore (mPTP) and the 18-kDa translocator protein (TSPO) also known as the “peripheral simil-benzodiazepine receptor”. The HBOT inhibits apoptosis as a result of concussion. After a traumatic damage, the Hypoxia-Inducible Factor-1\alpha (HIF-1\alpha) is present in the brain tissue in high concentration and can bind to tumor suppression factor p53 by activating apoptosis. In a randomized study [Palzur, 2008], the HBOT (2.8 bar, 45 minutes) - unlike normobaric oxygen - has significantly reduced the percentage of apoptotic cells (also thanks to the increase in Bcl-2 anti apoptosis) and the area of the lesion.

In orthopedic trauma with an exposed fracture complicated by ischemia and / or neuropathy (grade 3B and 3C of Gustilo classification) or in crush syndrome, good practice consists in 1) stabilization of fluid balance and acid / base. 2) Debridment
/ fasciotomy (open the compartment to reduce the pressure). 3) Bone synthesis. 4) Revascularization / thrombolytics. 5) Antibiotic prophylaxis. 6) Hyperbaric oxygen therapy to increase tissue perfusion, reduce edema, tissue damage and risk of infection.

The ECHM Consensus Conference on Hyperbaric Oxygen Therapy (Lille, 2016) has recognized the following indications (in brackets the levels of evidence): Open fracture with crush injury (IB); Crush injury without fracture (IIC); Necrotizing soft tissue infection (IC); Compromised skin grafts and musculo-cutaneous flaps (IIC); Refractory chronic osteomyelitis (IIC); Post-vascular procedure reperfusion syndrome (IIIC); Limb replantation (IIIC).

Evaluation and Prognostic Values of Microcirculation Measurement with Transcutaneous Oxymetry (TCpO2) and Laser Doppler Flowmetry (LDF)

Mario Franolić
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We are witnessing to a real epidemic of various diseases connected to the development of human civilization. Most common among them are disorders of circulation of different origin: all stages of blood vessel diseases as a result of diabetes, Bürger’s disease, Raynaud’s disease and syndrome, atherosclerosis, systemic autoimmune diseases (lupus, scleroderma, vasculitis ...). Having in mind that health care, but also general culture and health culture, are not at the desired level, there is also an increasing number of patients with chronic wounds (venous ulcers, decubitus, diabetic foot ...). And we must have in mind posttraumatic ischemia also. One of the major common denominators of all these diseases is microangiopathy - a change in the structure of small blood vessel walls. By shrinking, little blood vessels bring less and less blood to the cells “in the periphery,” far from the major arteries, causing peripheral ischemia and hypoxia. The most notable diagnostic methods of circulatory examination - arteriography and ultrasound - are unable to record the condition and functionality of the microvascular network. They are usually clinically evaluated on the basis of external manifestations on soft tissues (changes in appearance and skin color, opening of wounds, gangrene etc.). Using a non-invasive and completely painless method without risk, through the skin electrode, we can accurately measure the state of microcirculation, the smallest blood vessels not seen by conventional radiological methods (perfusion). We are able to evaluate the condition of these small blood vessels, their maximum capacity as well as the amount of oxygen coming into the peripheral tissues (partial oxygen pressure, pO2). This can be used to estimate the degree of risk for survival of extremities or other
tissues with great certainty and to give a prognosis of the outcomes of the treatment. Thanks to the application of this diagnostic method in triage of patients, the choice of therapy type, prediction of outcome and the treatment of critical ischemia and wounds are much more precise and better and the cost of treatment is significantly reduced. We are talking about Laser Doppler Flowmetry (LDF) and Transcutaneous Oximetry (TCpO2). The basic application of these methods is in the treatment of chronic wounds, vascular surgery, traumatology and hyperbaric medicine, but a list of possible applications and indications is even larger. In fact, there are two devices: the LDF measures the local tissue perfusion and the maximal perfusion capacity of capillaries, arterioles, venules and shunts, indicating also the existence and degree of inflammation, while TCpO2 measures the partial oxygen pressure - pO2. For precise diagnosis of tissue status, it is necessary to know local perfusion and local tissue oxygenation. LDF is supplemented with TCpO2 when capillary circulation is impaired, and macrocirculation (large blood vessels) is normal. TCpO2 is supplemented with a Laser Doppler to determine whether low values are a result of micro or macrostructure damage. The combination of these two diagnostic methods provides precise information on the state of microcirculation and the prognosis of cureability. The measuring electrodes are placed on the characteristic ischemic spots on the extremities, around the wound and in them. After basic recording, the provocation is carried out by heating and by inhaling pure oxygen at normal pressure and, if necessary, in hyperbaric chamber.
41-year old Caucasian for two months suffers frequent attacks of pain in left hip worsening on weight bearing, and subsiding with unload and rest. Oral analgesics have minimal and transient effect. Radiographs are normal, but MRI demonstrates changes consistent with grade II of avascular necrosis (AVN) of femur head. The patient receives series of 40 sessions of hyperbaric oxygenotherapy (HBOT). Clinical improvement appears after first 20 sessions, and on discharge patient is symptom free, fully mobile with no crutches. Control MRI done two months after the treatment shows complete regression of pathological changes.

HBOT is procedure with inhaling high partial pressure of oxygen, aiming to increase its fraction dissolved in blood plasma. In such form oxygen can reach deprived tissues with impaired circulation, independently of normal transport with hemoglobin in RBC.

Exact cause of AVN is unknown and combined multifactorial etiology with inter- and intra-individual variations is hypothesized. Natural course of AVN suggests self-containment only in early stages, but later, after critical accumulation of primary and secondary changes, irreversible progression. The self-containment and frequent reversal of changes in early stages might be result of timely removal or change of intensity or quality of unknown cause(s), before the start of irreversible destruction. That is possible explanation of beneficial effect of HBOT in I and II grade of AVN, where correction of hypoxia and restitution of aerobic metabolism in affected bone retards pathological process and enhances reparation processes, as demonstrated in our presentation.

**Key words:** Avascular bone necrosis, hyperbaric oxygenotherapy, case presentation