2021.10.21

-제2회 고압의학 전공의 기초교육과정 연수강좌-

Physics and Mechanism of Hyperbaric Medicine

연세원주의대 응급의학과 차용성

대한고압의학회 총무이사

HBOT를 하는 이유?

UNDERSEA AND HYPERBARIC MEDICAL SOCIETY
Hyperbaric Oxygen Therapy
INDICATIONS
14TH EDITION
Richard E. Moon, MD
Chair and Editor

2017 UHMS

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The NEW ENGLAND JOURNAL of MEDICINE

CLINICAL PRACTICE

Carbon Monoxide Poisoning

Lindell K. Weaver, M.D.

This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.

A 39-year-old female executive has a several-month history of fatigue, headache, and memory lapse. Multiple specialists have performed evaluations, but no diagnosis has been established. During a period of feeling worse than usual, she called a friend, who arrived at the residence to find the woman semicomatose and called 911. The patient was given supplemental oxygen and transported to the emergency department, where she is alert and has nonfocal findings on examination. Her carboxyhemoglobin level is 18%. How should she be treated? What is the expected outcome?

The New England
Journal of Medicine

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HYPERBARIC OXYGEN FOR ACUTE CARBON MONOXIDE POISONING

LINDELL K. WEAVER, M.D., RAMONA O. HOPKINS, PH.D., KAREN J. CHAN, B.S., SUSAN CHURCHILL, N.P., C. GREGORY ELLIOTT, M.D., TERRY P. CLEMMER, M.D., JAMES F. ORME, JR., M.D., FRANK O. THOMAS, M.D., AND ALAN H. MORRIS, M.D.

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מרכז רפואי אסף הרופא
Assaf-Harofeh Medical Center
TEL AVIV UNIVERSITY
אוניברסיטת תל אביב
Non Healing Brain Wounds
Basic Characteristics and The Use of
Hyperbaric Oxygen Therapy
The Sagol Center for Hyperbaric Medicine and Research,
Assaf-Harofeh Medical Center, Israel.
Sackler School of Medicine and Sagol School of neuroscience,
Tel Aviv University, Israel
1

See you in Israel 09.09.19…

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Backgrounds (1)

Mesocardial

EXIT

SECHRIST
THE WORLD'S LEADER IN
HYPERBARIC TECHNOLOGY
& MEDICINE

2019 EUBS

EUBS 2019
HBOT & The Brain | Tel-Aviv
Hyperbaric Medicine & The Brain
09-12 September 2019, Tel-Aviv
WELCOME

<Mechanisms of hyperbaric oxygen>

High-pressure oxygen

The beneficial
and
toxic manifestation

Dr. Kindwall

Not supported by cellular "respiratory effects" alone

Progressive improvement of non-healing wound

"Pharmacological effect" of oxygen

LEGEND: In many respects the hyperoxygenation effect of HBO_2 acts like a drug with its dose-duration effects. Oxygen tensions remain elevated in subcutaneous tissues for four hours after a hyperbaric O_2 exposure. It takes about an hour exposure to HBO_2 before the O_2 tensions in muscles (MM) and subcutaneous tissues (SC) plateau.

Figure 10. Dose-duration effects of hyperoxygenation.

In the past decades

- Oxygen and nitrogen radicals ⇒ Highly toxic within biological cells and in the environment
- Their action is also vital in eliciting protective responses, mediating intracellular signaling, and affecting immune defenses, inflammatory reactions, and gene expression.

Review

J Appl Physiol 106: 988–995, 2009.
First published October 9, 2008; doi:10.1152/japplphysiol.91004.2008.

HIG
Div
Oxi

The majority of patients who undergo HBO2 are not treated for bubble-induced injuries; hence, therapeutic mechanisms are related to an elevated oxygen partial pressure!!!

Stephen R. Thom, M.D., Ph.D.
Philadelphia, Pa.

Background: This article outlines therapeutic mechanisms of hyperbaric oxygen therapy and reviews data on its efficacy for clinical problems seen by plastic and reconstructive surgeons.
Methods: The information in this review was obtained from the peer-reviewed medical literature.
Results: Principal mechanisms of hyperbaric oxygen are based on intracellular generation of reactive species of oxygen and nitrogen. Reactive species are recognized to play a central role in cell signal transduction cascades, and the discussion will focus on these pathways. Systematic reviews and randomized clinical trials support clinical use of hyperbaric oxygen for refractory diabetic wound-healing and radiation injuries; treatment of compromised flaps and grafts and ischemia-reperfusion disorders is supported by animal studies and a small number of clinical trials, but further studies are warranted.
Conclusions: Clinical and mechanistic data support use of hyperbaric oxygen for a variety of disorders. Further work is needed to clarify clinical utility for some disorders and to hone patient selection criteria to improve cost efficacy. (*Plast. Reconstr. Surg.* 127 (Suppl.): 131S, 2011.)

Mechanisms (Primary vs Secondary)

Primary

- ***Direct effect of increased oxygen partial pressure***
- Direct effects of pressure

Dose, frequency, duration, effects

Secondary

- Effects of enriched oxygen environment have on cellular functions.

Accumulative effects

TABLE 7. MECHANISMS OF HYPERBARIC OXYGEN

*Theoretical and/or limited clinical data to support the effectiveness of these secondary mechanisms.

Primary mechanism

Hyperoxygenation!!!!

Hyperoxygenation

- **Most important… mostly what HBO is all about**
- **Strongly physics (how) and physiologic (why) basis**
- **Immediate & directly observable effects**

O_2 is physically **forced into the plasma & tissue fluids** by pressure!!!

Blood oxygen content
(calculation)

$$
\begin{aligned}
\text{Total oxygen content} &= \text{oxygen bound to Hb} \\
&\quad + \text{oxygen dissolved in plasma} \\
&= \boxed{(1.36(\text{mL } O_2/\text{g Hb}) \times SO_2 \times \text{Hb}(\text{g}/100\,\text{mL}))} \\
&\quad + (0.0031\,\text{mL } O_2/100\,\text{mL/mmHg} \\
&\quad \times PO_2(\text{mmHg})).
\end{aligned}
$$

Normal Blood O_2 Content

Hemoglobin carried	19.5 Vol%	97.5%
Physically dissolved	0.5 Vol%	2.5%
Total	20.0 Vol%	100%

Vol% = blood 100ml에 녹아있는 oxygen의 양(ml)

Henry's law

Fundamental to HBOT & Decompression science

Blood oxygen content
(calculation)

$$\text{Total oxygen content} = \text{oxygen bound to Hb} + \text{oxygen dissolved in plasma}$$

$$= \boxed{(1.36(\text{mL } O_2/\text{g Hb}) \times SO_2 \times \text{Hb}(\text{g}/100\,\text{mL}))}$$

$$+ (0.0031\,\text{mL } O_2/100\,\text{mL/mmHg} \times PO_2(\text{mmHg})).$$

Physically dissolved

Effects of hyperoxygenation
(O_2 content of blood)

	Hemoglobin carried O_2 (Vol%)	Plasma dissolved O_2 (Vol%)	Total O_2 content (Vol%)
1 ATA air	19.5	0.5	20.0
2 ATA HBO	19.5	5.0%	24.5%
3 ATA HBO	19.5	7.5%	27.0%

Hyperoxygenation physiology

LEGEND: Physically dissolved oxygen in the plasma adds to the blood O_2 content. In room air 97.% of the O_2 is carried in the RBC. HBO_2 adds O_2 to the plasma. Eventually with HBO, enough O_2 becomes physically dissolved in the plasma to meet the normal A-V O_2 extraction and amounts to 5 vol%. When this occurred in Boerema's study, he was able to sustain life in

Boerema confirmed in pig study.

I. Boerema 1960: "Life Without Blood"

Clinical applications of hyperoxygenation

LEGEND: Hyperoxygenation has 4 clinical applications. Improving O_2 availability in low blood flow states is the most frequent clinical application of HBO_2 and is most frequently used as adjunct to managing problem wounds.

KEY: AGE = Arterial gas embolism, ATA = Atmospheres absolute, CN = Cyanide, CO = Carbon monoxide, DCS = Decompression sickness, HBO = Hyperbaric oxygen, O_2 = Oxygen

Figure 14. Clinical applications of hyperoxygenation.

Mass effect of hyperoxygenation

Mass effect (~ water running down hill)

3-Fold increases with HBO at 2 ATA

Helps O_2 to cross and/or penetrate relative barriers

LEGEND: The increased concentration of O_2 molecules in the tissue fluids achieved with HBO_2 provides a "driving force" for oxygen to move through and/or penetrate relative barriers such as edema fluid, cicatrix, thickened capillary basement membranes, suppuration, avascular bone, etc.

KEY: ATA = Atmospheres absolute, HBO = Hyperbaric oxygen, O_2 = Oxygen, ~ = Similar to/ as for example

Figure 15. The "driving force" for moving oxygen across relative barriers.

Mitigation of sluggish perfusion in the microcirculation with hyperbaric oxygen

LEGEND: Hemoglobin O_2 transport is the most flow limited of the substances carried in the blood due to the mass of the RBC. HBO_2 results in the physically dissolved O_2 in the plasma being no more flow limited than any of the other substances dissolved in the plasma

If there is no blood flow, HBO will prevent tissue death. However, in situations where demarcation between viable and nonviable tissue is not clear, it can assist in the demarcation process.

In acute low-flow states, HBO can be important “bridge therapy” that maintain tissue viability until the circulation is improved.

-CRAO, PAOD-

Properties of hyperoxygenation

- Immediate onset

- Transient
  - However, the transient effects appear to be sufficient to activate factors instrumented for wound healing and infection control.
  - The ”pulses” of HBO given once or twice a day for these Ix appear to be sufficient to maintain the healing process.

- Propotional to inhaled O_2 pressure

- Depend on plasma transport
  - Blood flow

Topical oxygen (Topox)

LEGEND: Topical oxygen should not be considered a variant of HBO therapy. Although it appears to enrich the oxygen environment of the superficial portions of a wound, it does not penetrate more than a millimeter or two into deeper tissues.

Note the constricting bands (elastic type on the left and rubber gasket on the right) used to maintain a positive pressure in the devices. These appear to interfere with venous return and lower the juxta-wound transcutaneous oxygen tensions.

Figure 20. Methods of implementing topical oxygen.

TABLE 9. COMPARISONS OF OXYGEN PRESSURES; HBO_2 versus TOPOX

Situation In usual terminology	fsw* Conversions For comparison purposes
HBO_2 (2 ATA)	33 Feet of seawater*
Systolic (120 mmHg)	5 fsw
Topox (1.03 ATA)	0.99 fsw
Venous (10 cm H_2O)	0.33 fsw

COMMENT: In terms of oxygen tension increments to the wound, topical oxygen only provides 1/33rd as much as hyperbaric oxygen.

Because of the constricting band or seal to maintain pressure in the topical oxygen system, the inflation pressure exceeds that of the venous return pressure. This may explain why the lowered transcutaneous oxygen tension measurements were observed with topical oxygen (Cotto-Cumba[29]).

- **Not HBO!!!!**
- Limited diffusion (~1mm) through surface of an open wound
- Elastic “tie” to maintain seal
    - Restricts venous return
    - Lowers transcutaneous oxygen tensions
- HBO provides **33 1/3 times** more O_2 than Topox

Pressure effect!!!

- Bubble reduction
  - Arterial gas embolism & decompression sickness
  - Double the pressure, reduce the bubble volume by one half, etc.
  - A direct effect of pressure (Boyle's law)

- Forces O_2 in plasma
  - The pressure effect "drives" O_2 into tissues (the gradient effect of Henrys law)

Secondary mechanism

Expanding biological observations

- Both the beneficial and toxic manifestations of high-pressure oxygen were not readily by cellular “respiratory effects” alone.
  - Progressive improvement of a non-healing wound over several weeks
  - “pharmacological effect” of oxygen, concept heralded by Kindwall

Kindwall EP. *Hyperbaric medicine practice.* 1994. p.1-16

TABLE 11. SECONDARY MECHANISMS OF HYPERBARIC OXYGEN

- **Vasoconstriction**
- **Host Responses**
- **Microbiological Effects**
- **Reperfusion injury**

- **Inert gas washout**
  (& isobaric counterdiffusion)
- RBC Deformability
- Blood Brain Barrier Permeability

Viability	**Primary mechanisms keep tissues alive**
Repair & Function	**Secondary mechanisms resolve the problem**—the long term, durable effects

NOTE: Several of the 2° mechanisms (in smaller font) have theoretical justification at this time. As more information becomes available, we anticipate these will receive further attention & help to justify HBO_2 for "off-label" uses.

Vasoconstriction

- Tissue oxygenation maintained (through hyperoxygenation) while blood flow is decreased
- Probably not important in chronic low blood flow states
  - -Atherosclerotic blood vessel walls
  - -Auto-sympathectomies (diabetics)

LEGEND: HBO_2 reduces blood flow in healthy blood vessels regulated by the sympathetic nervous system. With hyperoxygenation, oxygen tensions are maintained due to increased physically dissolved oxygen in the plasma.

KEY: HBO = Hyperbaric oxygen, O_2 = Oxygen

- Reduction of blood flow reduces vasogenic edema

(crushing injury, compartment syndrome, burns, and reimplantations)

- It also reduces cytotoxic edema

(brain, spinal cord, and radiation-induced ischemia)

Leg swelling after inducing a model compartment syndrome in a canine right leg[67,71,72]

Edema reduction after 3 HBO treatments in 12 hours

LEGEND: Swelling reduction is clinically obvious after the HBO_2 (hyperbaric oxygen) treatments. This was further substantiated with microscopic examinations and volume-weight measurements.

Figure 22. Edema reduction in a canine compartment syndrome model with HBO_2.

Harmful effects of edema!!

LEGEND: Normally fluid is filtered from the capillary to the tissue fluid spaces on the arterial side of the capillary due to perfusion pressure and reabsorbed on the venous side due to oncotic pressure. Trauma/inflammation increase inflow; HBO_2 decreases inflow 2° to vasoconstriction.

Figure 24. Mechanism of edema reduction with HBO_2.

Host cellular functions

For cells to function, tissue fluid O_2 tensions must be **30-40 mmHg** Hunt [11]

-There are three oxygen-dependent host cells associated with wound healing and infection control on which HBO can direct effects.

1) Fibroblast with associated with angiogenesis
2) Neutrophils
3) Osteoclasts

tensions are lowered at any level of this gradien chondrial function and will inhibit their ability to carry ou ypoxia is compensated by breathing pure O_2 and even more so with HBO_2.

Figure 25. The oxygen gradient from inspired air to the mitochondria.

-If oxygen tensions are low in the tissue fluids, HBO can improve them.

-Mitochondria will continue to generate energy for all the cell's metabolic process.

Fibroblast

LEGEND: Oxygen has multiple roles in fibroblast function that include growth factor inductions of its three primary functions (migration, secretion and proliferation) to biochemical reactions needed for collagen formation.

KEY: FGF = Fibroblast growth factor, HBO_2 = Hyperbaric oxygen, O_2 = Oxygen, PDGF = Platelet derived growth factor, TGF-β = Transforming growth factor beta, VEGF = Vascular endothelial growth factor

Figure 26. Oxygen roles in fibroblast functions.

LEGEND: In hypoxic environments , TGF-β1 is induced to form cicatrix, a dense, hypovascular type of scar tissue. The ✕'s indicate where hypoxia could interfere with the normal fibroblast oxygen dependent mechanisms. TGF-β1 appears to bypass these mechanisms (thick, dotted black arrow) to form cicatrix and is induced by a hypoxic environment.

Figure 27. The effects of hypoxia on fibroblast function and as an induction of transforming growth factor-beta1 (TGF-β1).

-Angiogenesis is delayed effect of HBO (granulation tissue: 10 days ~ 2 weeks)

-" Pulses" of HBO rather than sustained hyperoxygenated tissue oxygen tensions.

: Rationale for only once or twice a day for wound healing and infection control

-To achieve an angiogenesis effect, 14 to 21 HBO Tx are recommended.

-Once granulation tissue is well established, it indicate adequate oxygenation of the wound base, and HBO Tx are no longer needed for angiogenesis to continue.

-The termination point for HBO is justified by the clinical appearance of the wound.

Thom SR. *J Appl Physiol* 106:988-995, 2009

Neutrophil

For bacterial killing, the WBC must have O_2 to generate reactive oxygen species

--Superoxide

--Peroxide

Hohn, ***Surg Forum***[7]

LEGEND: The oxidative burst in the phagocytic vesicle is oxygen dependent.

Figure 30. White blood cell (neutrophil) killing.

Osteoclast

Appearance of Metatarsal Head after 4 weeks of antibiotics, debridements and wound care

Marked Resorption of Metatarsal Head Bone after 2 Weeks of HBO Treatments

LEGEND: A malperforans ulcer in a dysvascular diabetic foot was limb threatening after failing to improve over a month's time. HBO_2 treatments were then initiated in an attempt to avoid a lower limb amputation.

Marked resorption of the infected metatarsal head (encircled by black arrows) became apparent on x-rays. With this information, the metatarsal head was removed and the wound healed. The resorption of dead, infected bone was attributed to HBO_2's stimulation of the osteoclast.

Figure 32. Bone Resorption after initiating HBO_2 treatments in an infected metatarsal head.

Microbiological applications of HBO

- Bacteriostatic and bacteriocidal effects
  - Clostridial organisms (no killed but stop multiplying)
  - Anaerobic Streptococcus and Peptostreptococcus are killed by hyperoxic condition.
  - Bacteroids species killed by hyperoxia.
  - Fungal infection by Actinomyces species

- Cessation of the endotoxin formation produced by clostridial organisms

- Toxin inactivation has been best appreciated with the theta toxin produced by clostridial organisms.

- Additive effect with certain antibiotics
  - Aminoglycosides
  - Amphotericin
  - Possibly vancomycin

LEGEND: Hyperbaric oxygen has many roles, both well documented as well as potentially beneficial in mitigating the harmful effects of microorganisms.

Figure 34. Hyperbaric oxygen as an antibiotic with multiple mechanisms.

Mitigation of the reperfusion injury

- All tissues can survive and resume function after transient periods of ischemia
  - 3~4 minutes in the brain
  - 4~6 hours in muscles
  - 12~24 hours in bone
  - More longer in connective tissue and skin

- However, during the period of ischemia, the endothelium appears to become “sensitized” due to the hypoxic insult.
  - Leads to attachment of neutrophils to the sensitized endothelium.
  - Susceptible to critical tissue such as brain, heart, and skeletal muscle .

-Once attached, the neutrophil becomes activated.

-Release of superoxides and peroxides that this cell ordinarily uses for killing bacteria.

-This ROS then attack the surrounding tissues (similar to

-HBO interferes with the cascades of events.

-Thom reported that HBO exposures interrupt the neutrophil adherence to the endothelium through inactivating the beta-integrin attachment mechanism.

-Another potential benefit of HBO is that it helps generate oxygen free radical scavengers such as superoxide dismutase and peroxidase.

-Scavnger generation is an oxygen-dependent process.

-The consequences are an intense vasoconstriction, the so called “no-reflow phenomenon” of the reperfusion injury

-Irreversible tissue damage due to hypoxia from the vasoconstriction coupled with the destructive effects of the oxygen radicals.

LEGEND: Mitigation of neutrophil adherence to the capillary endothelium sensitized by hypoxia reduces/eliminates the release of toxic O_2 radicals (peroxides and superoxides) that cause the harmful effects of the reperfusion injury.

In addition, this prevents the cascade of reactive oxygen species with nitric oxide (NO)--in hypoxic environments--from forming highly toxic degradation products such as peroxynitrite (-OON).

NOTES & KEY: *Neutrophils adhering to the sensitized capillary endothelium are so indicated by white circles around them (Zamboni[10]); CO Px = Carbon monoxide poisoning, HBO_2 = Hyperbaric oxygen, WBC = White blood cell

Figure 35. Morphology of the reperfusion injury and roles of HBO_2 in mitigating its effects.

- Recommend starting HBO as soon as possible after an extended period of ischemia in critical tissue such as the brain and heart as well as skeletal muscles and perhaps in other organs such as the liver, kidney, pancreas, etc.

- HBO should be considered after revascularization surgeries associated with long ischemia times
  - Re-implantations
  - Threatened flaps
  - Other situations where there is a possibility that reperfusion injury will contribute to more morbidity.

Reason for HBOT in acute CO poisoning (25 days after discharge)

Gas washout

LEGEND: Gases in tissues equilibrate with gases in the plasma.

With high plasma O_2 partial pressures, inert gases & carbon monoxide move from tissues → plasma → lungs → expired air.

KEY: CO = Carbon monoxide, HBO = Hyperbaric Oxygen, O_2 = Oxygen, Via = By way of

Figure 36. Carbon monoxide (and inert gas) "washout" in response to gradients from HBO_2.

Toxic gas
Decompression sickness

Mechanism 강의는…

Review

J Appl Physiol 106: 988–995, 2009.
First published October 9, 2008; doi:10.1152/japplphysiol.91004.2008.

HIGHLIGHTED TOPIC | *The Physiology and Pathophysiology of the Hyperbaric and Diving Environments*

Oxidative stress is fundamental to hyperbaric oxygen therapy

Stephen R. Thom
Institute for Environmental Medicine and Department of Emergency Medicine, University of Pennsylvania Medical Center, Philadelphia, Pennsylvania

Submitted 1 August 2008; accepted in final form 1 October 2008

Oxidative stress is not synonymous with oxygen toxicity!!!

2 ATA 100% oxygen

Thom 2.5 – 2.8 ATA 100% oxygen

Dr. Thom

HBO2 → Oxidative → Many
stress responses
HBO2
Reactive O2 species (ROS)
Reactive Nitrogen species (RNS)
PMN Fxn
I-R events
STEM
CELLS
INFLAMMATION
Growth
Factors
IMMEDIATE 바로
LATE 늦은
HBO2 Therapy: Oxidative stress in moderation.
HBO2
Reactive O2 species (ROS)
Reactive Nitrogen species (RNS)
PMN Fxn
I-R events
STEM
CELLS
줄기세포
INFLAMMATION
염증
Growth
Factors
Overlaps between these two

2018 WSCH Hyperbaric Medicine International Symposium

Topic : Basic and Clinical Research in Hyperbaric Medicine

Stephen R. Thom, MD, PhD
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일시: 2018년 10월 16일 화요일 오후 1시 30분 ~ 오후 6시
장소: 연세대학교 원주세브란스기독병원 외래센터 7층 대회의실
주최: 연세대학교 원주의과대학 응급의학교실
주관: 연세대학교 원주세브란스기독병원

The 2nd WISH

Wonju International Symposium of Hyperbaric Medicine

제2회 고압의학 국제 심포지엄

연세대학교 원주세브란스기독병원 개원 60주년 기념 심포지엄

Topic : Up-to-date Hyperbaric Medicine in Korea

- **일 시** : 2019년 6월17일(월) 12:45~18:00
- **장 소** : 연세대학교 원주세브란스기독병원 외래센터 7층 대회의실
- **주 관** : 연세대학교 원주의과대학 응급의학교실 대한고압의학회
- **주 최** : 연세대학교 원주세브란스기독병원 대한응급의학회

※ 문의사항 및 연수 교육은 뒷면을 참고하세요.

원주세브란스기독병원
Wonju Severance Christian Hospital

INVITED SPEAKERS

Stephen R. Thom, M.D, PhD

Professor
Dept. of Emergency Medicine
University of Maryland

David S. Lambert, M.D

Assistant Professor
Dept. of Emergency Medicine
University of Pennsylvania

Enoch Huang, M.D

Co-chair of Annual Scientific Meeting
Program Committee of UHMS
Past President of UHMS
Dept. of Hyperbaric Medicine/Wound Healing
Legacy Emanuel Medical Center

고압산소치료센터

고압산소치료센터 확장 개소식
교수연구실

2018 WSCH
Hyperbaric Medicine International Symposium
English Session
Mechanism and Research in Hyperbaric Medicine
Professor Stephen R. Thom, MD
Thank you .... Questions??
Baltimore Inner Harbor

2018 WSCH Hyperbaric Medicine International Symposium
Topic : Basic and Clinical Research in Hyperbaric Medicine

STARMED 2700
HAUX

제3회 WISH

The 3rd WISH

Wonju International Symposium of Hyperbaric Medicine

제 3회 고압의학 국제심포지엄

- Topic : Hyperbaric Medicine in Neuroscience and Anti-aging-

COVID-19로 인해 진료 업무에 노고가 많으십니다

지난 2018년부터 진행해오던 국제고압의학심포지엄(WISH)이 올해 세 번째로 개최됩니다. 2020년은 코로나로 인해 실시하지 못했기에 올해 개최는 더욱더 의미가 있는 것 같습니다.

그동안 국제고압의학심포지엄(WISH)을 성공적으로 개최하는 데 도움과 관심을 주신 많은 분께 감사드립니다. 이번 제 3회 국제고압의학심포지엄(WISH)은 COVID-19 상황을 고려하여 대면·비대면 방식으로 동시 진행하게 되었습니다.

저희 연세대학교 원주세브란스기독병원 고압산소치료센터 및 고압의과학연구소는 항시 응급·중환자의 치료에 대비하여 10인용 기기(Multiplace chamber) 1대와 1인용 기기(Monoplace chamber) 3대를 운용하고 있으며, 2021년 8월 치료 건수 15,000례를 달성하였습니다. COVID-19로 제한된 진료 환경에서도 매월 20회 이상의 응급치료를 시행하고 있고 또한 다양한 임상 및 실험 연구를 통해 환자의 치료에 도움이 되고자 노력하고 있습니다.

현재 국내 고압산소치료의 적용 질환은 점차 확대되어 이전보다 다양한 질환에 적용되고 있습니다. 하지만 고압의학 선진국에 비교하여 적용 질환이 매우 제한적입니다. 고압의학 선진국에서는 다양한 뇌질환에 고압산소치료를 사용하고 있고, 많은 연구가 이루어지고 있습니다. 따라서 이번 심포지엄에서는 국내 고압 산소 치료 적응증을 더욱 확대하고자 뇌과학분야의 전문가이신 이스라엘 텔아비브 대학의 Dr. Shai Efrati와 Dr. Keren Doenyas-Barak을 초청하여 선진국에서 이루어지는 최신 연구 결과와 고압산소치료의 경험을 국내 의료진들과 공유하고자 합니다.

이번 심포지엄에서는 온·오프라인 동시통역이 진행됩니다. 많은 관심과 참석 부탁드립니다.

감사합니다.

연세대학교 고압의과학연구소장
원주세브란스기독병원 고압산소치료센터장
대한고압의학회 이사장
김 현

The 3rd WISH

Wonju International Symposium of Hyperbaric Medicine

2021 고압의학 국제 심포지엄

- Topic : Hyperbaric Medicine in Neuroscience and Anti-aging-

- 일시: 2021년 12월 3일(금) 오전 8시 45분 ~ 오후 6시
- 장소: 원주 오키드 호텔 / 온라인 주소는 사전등록자에 한하여 개별 발송
- 제한 인원: 49명(온라인 실시간 송출 동시 진행)
- 주관: 연세대학교 원주의과대학 고압의과학연구소 / 대한고압의학회
- 주최: 대한응급의학회
- 대한의사협회 연수교육 평점 6점 예정

※ 이번 심포지엄에서는 온·오프라인 동시통역이 진행됩니다.

Invited Speakers:

Shai Efrati, MD
Director of Sagol Center for Hyperbaric Medicine and Research, Shamir Medical Center
Director of Research & Development, Shamir Medical Center
Professor at Sackler School of Medicine and the Sagol School of Neuroscience at Tel Aviv University

Keren Doenyas-Barak, MD
Head of PTSD program at Sagol Center for Hyperbaric Medicine and Research
Deputy Director of Department of Nephrology at Shamir Medical Center

참고자료들…

UNDERSEA AND HYPERBARIC MEDICAL SOCIETY
Hyperbaric Oxygen Therapy
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Richard E. Moon, MD
Chair and Editor

Hyperbaric Medicine Practice
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Harry T. Whelan, MD
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Textbook of
Chronic Wound Care
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Review

J Appl Physiol 106: 988–995, 2009.
First published October 9, 2008; doi:10.1152/japplphysiol.91004.2008.

HIGHLIGHTED TOPIC | *The Physiology and Pathophysiology of the Hyperbaric and Diving Environments*

Oxidative stress is fundamental to hyperbaric oxygen therapy

Stephen R. Thom

Institute for Environmental Medicine and Department of Emergency Medicine, University of Pennsylvania Medical Center, Philadelphia, Pennsylvania

Submitted 1 August 2008; accepted in final form 1 October 2008

WOUND HEALING

Hyperbaric Oxygen: Its Mechanisms and Efficacy

Stephen R. Thom, M.D., Ph.D.

Philadelphia, Pa.

Background: This article outlines therapeutic mechanisms of hyperbaric oxygen therapy and reviews data on its efficacy for clinical problems seen by plastic and reconstructive surgeons.
Methods: The information in this review was obtained from the peer-reviewed medical literature.
Results: Principal mechanisms of hyperbaric oxygen are based on intracellular generation of reactive species of oxygen and nitrogen. Reactive species are recognized to play a central role in cell signal transduction cascades, and the discussion will focus on these pathways. Systematic reviews and randomized clinical trials support clinical use of hyperbaric oxygen for refractory diabetic wound-healing and radiation injuries; treatment of compromised flaps and grafts and ischemia-reperfusion disorders is supported by animal studies and a small number of clinical trials, but further studies are warranted.
Conclusions: Clinical and mechanistic data support use of hyperbaric oxygen for a variety of disorders. Further work is needed to clarify clinical utility for some disorders and to hone patient selection criteria to improve cost efficacy. (*Plast. Reconstr. Surg.* 127 (Suppl.): 131S, 2011.)

Thank you for your attention!!!