KAUHM
Korean Academy of Undersea and Hyperbaric Medicine

2018년 제2회
대한고압의학회
추계학술대회

일시. 2018년 11월 23일(금) 10시~16시 30분

장소. 쉐라톤 호텔 강남(구 팔레스호텔)

INVITATION

2018년 제2회 **대한고압의학회 추계학술대회**

안녕하십니까

1960년대 고압산소치료가 우리나라에 도입된 이후 여러 선구자들의 노력으로 고압의학이 발전해 왔습니다. 그 동안 열악한 환경과 낮은 수가에도 고압의학 관계자들은 의료 최일선에서 국민의 건강과 생명을 지키기 위해 최선의 노력을 해왔습니다. 고압산소치료 효과가 확인된 질환은 일산화탄소중독, 잠수병, 가스색전증, 혐기성 세균 감염증, 시안화물중독, 화상, 버거씨병, 식피술 또는 피판술 후, 수지 접합수술 후, 방사선치료 후 발생한 조직괴사, 돌발성 난청 등이 있으며 그 적용은 점점 확대되고 있습니다. 이에 고압의학 관련 진료, 교육, 연구, 인증, 정책 수립 및 이와 관련된 국내외 협력을 통하여 고압의학 발전에 기여함을 목표로 대한고압의학회가 설립되었고 벌써 1년이 흘렀습니다.

이번에 두 번째로 개최되는 대한고압의학회 추계 학술대회는 고압의학 관계자들에게 더욱 의미 있는 자리가 될 것입니다. 학술대회에 많은 관심과 참여 및 지도편달을 진심으로 바라며 저희는 항상 열린 마음으로 여러분과 소통하는 학회가 되도록 노력하겠습니다.

감사합니다.

대한고압의학회 회장 **허 탁**

PROGRAM

2018년 제2회 **대한고압의학회 추계학술대회**

Main topic

좌장: 순천향의대 응급의학과 **임 훈** 교수

Oxygen transport & mitochondria as a target for HBOT

연세원주의대 응급의학과

김 현 교수

Definition

- Hyperbaric oxygen therapy:
  Breathing 100% oxygen within hyperbaric chambers compressed to greater than 1.4 atm of absolute pressure (hyperoxygenation)

O_2 transport

- No animal can live in an atmosphere where a flame does not burn - *Leonardo da Vinci, 1500*
- The first concern in any life-threatening to maintain an adequate supply of oxygen to sustain oxidation metabolism.
- The delivery of O_2 and its utilization: integration of the respiratory, cardiovascular, and **microvascular systems**.

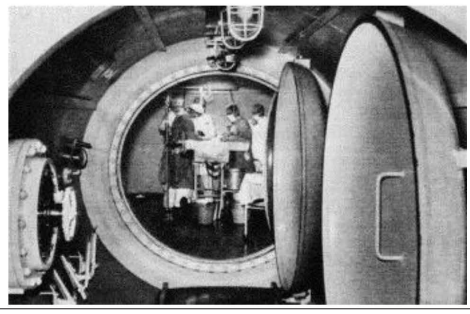
Major contributions (legacy)

Dr. Ite Boerema

- 1902-1978, professor of surgery at the university of Amsterdam
- Surgeon and engineer with a double-Dutch legacy to medical technology
- Red cell remove and plasma-dissolved oxygen to sustain life
- Gas gangrene: sudden decline in mortality rate (from 66% to 23%)
- Gas-forming soft tissue infections, carbon monoxide exposure, thermal burns, and radiotherapy-induced osteonecrosis

Life without blood

- A study of the influence of **high atmospheric pressure** and **hypothermia** on dilution of the blood (J Cardiovasc Surg 1959;13:133-146)
- 1948: hypothermia (27도)
- Hg: 0.4%, plasma solution, 3 ATA (45 mins)
- EEG: no pathologic changes
- Recovery: reinfusion of normal blood



Like all good experiments, Dr. Boerema has been replicated many times with like results.

Hyperoxygenation: Physiology (The Start)

Dr. Ite Boerema

Life without blood

(A study of the influence of high atmospheric pressure and hypothermia on dilution of the blood)

by

I. BOEREMA (*), N. G. MEYNE, W. K. BRUMMELKAMP
S. HOUMA, H. H. HENNEN, F. KAMERMANS, M. STERN HANF
and W. VAN AALDEREN
(from the Surgical Department of the University of Amsterdam)

When in 1948 we (first al research) started our experiment on hypothermia [1] our ultimate aim was to reduce the metabolism of a warm-blooded animal to such an extent that all the physiological processes would almost come to a standstill.

If successful, this would enable the heart to be clamped off for a period long enough to allow for a major intracardiac operation to take place. When, however, we presented our results to the Netherlands Society of Surgeons in 1950 this aim had not been achieved by any means. In a hypothermic animal at about 27° C., the circulation could be stopped with good chances of survival for about twice as long as in a normothermic animal. The gain in time, about 100 per cent, was relatively great, but absolutely it was very modest, amounting to about five minutes; the reason for this was that below 20° C the physiology was altered too much and the normal harmony of life processes disturbed too much to allow for continuation of life or normal recovery by warming up.

Efforts to achieve safe conditions at a lower level of hypothermia so as to gain a greater period of time for clamping off the heart failed until recently, at any rate for animals with the same weight as human patients. So in 1956 we presented a series of experiments which showed that it was possible to clamp off the circulation for a greater length of time without lowering the temperature further than 27° C. [1] [2] We operated on the animal in a pressure chamber at an absolute pressure of three atmospheres. The animal breathed pure oxygen, the investigators naturally breathed air.

Through the combination of inhaling pure oxygen and being under three atmospheres of pressure, the whole body was supersaturated with oxygen in physical solution.

(*) Professor of Surgery.

Again we are much indebted to T.N.O. (Institute for Applied Physical Research) for financial aid, to the chief of the Mijnendienst of the Royal Dutch Navy at Den Helder and to our advisor on technical matters, P. Bouma.

Mechanisms of HBOT
(Primary & Secondary)

Primary

- Hyperoxygenation (O_2 tensions)
- Direct effects of pressure

Dose dependent

Secondary

- Hyperoxygenation
  (enriched O_2 -> cellular functions)

Accumulative

Mechanisms of HBOT
(Primary & Secondary)

Primary

- Hyperoxygenation (O_2 tensions)
- Direct effects of pressure

Oxygen delivery/uptake
Bubble size reduction

Secondary

- Hyperoxygenation
  (enriched O_2 -> cellular functions)

Anti-bacterial
Anti-inflammation (cytokine)
Ischemia-reperfusion injury
Neovascularization
Stem cell migration

Mechanisms of HBOT
(Primary & Secondary)

Primary

- Hyperoxygenation (O_2 tensions)
- Direct effects of pressure

Oxygen delivery/uptake
Bubble size reduction

Secondary

- Hyperoxygenation
  (enriched O_2 -> cellular functions)

Anti-bacterial (PMN cells)
Anti-inflammation:cytokine (leukocyte)
Ischemia-reperfusion injury (endothelium, PMN activation
Neovascularization
Stem cell migration
Fibroblast GF

Mechanisms of HBOT
(Primary & Secondary)

Primary

- Hyperoxygenation (O_2 tensions)
- Direct effects of pressure

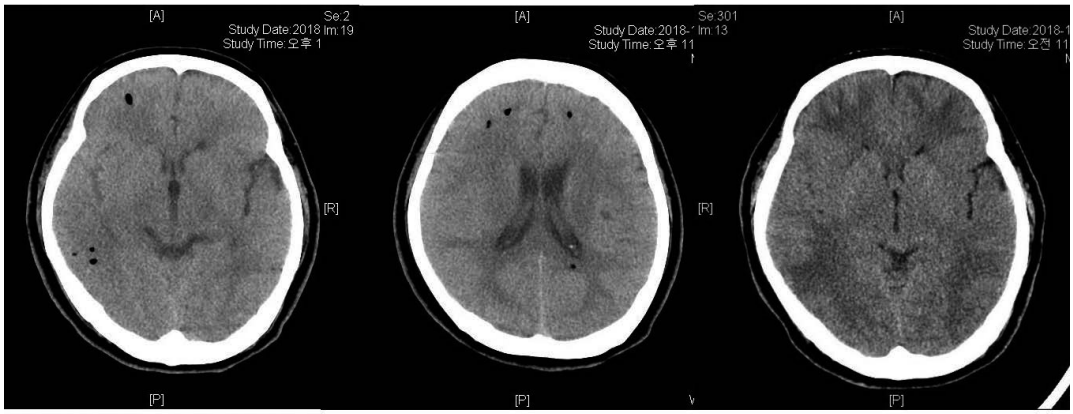
Oxygen delivery/uptake
Bubble size reduction

Secondary

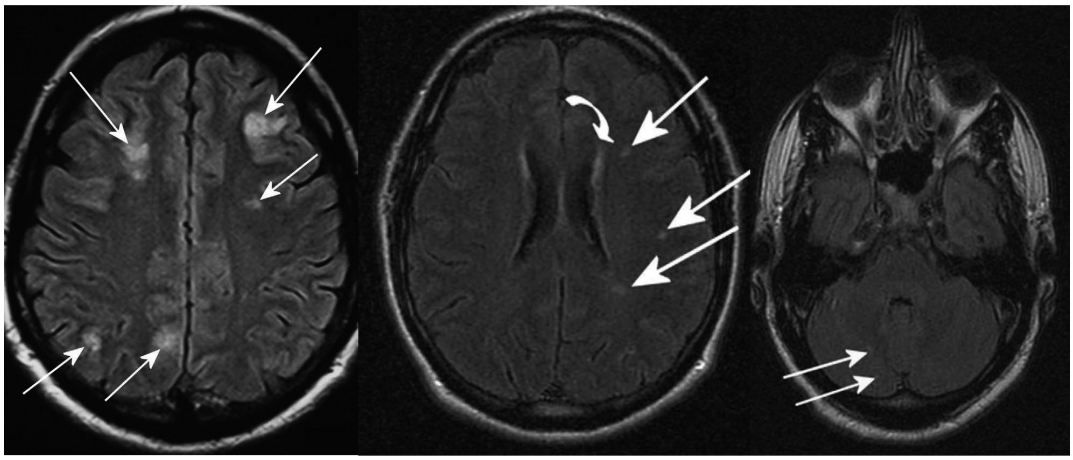
- Hyperoxygenation
  (enriched O_2 -> cellular functions)

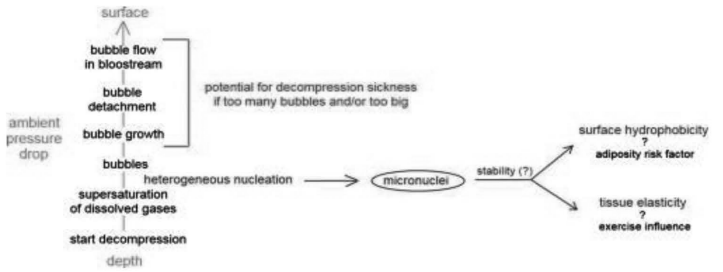
PMN cells
Leukocyte
Ischemia-reperfusion injury
Neovascularization
Stem cell migration

HBOT: primary mechanism (AGE)



HBOT: primary mechanism (DCS)

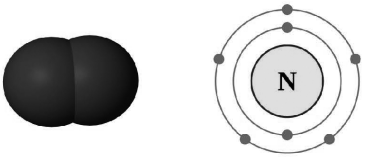




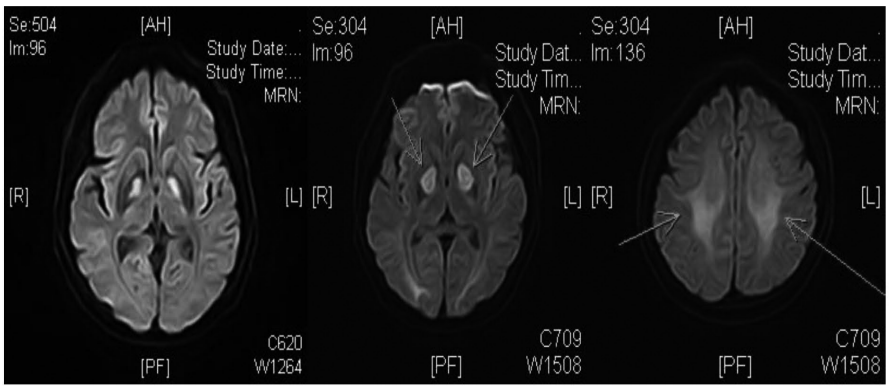
(*Papadopoulou et al (2013)A critical review of physiological bubble formation in hyperbaric decompression Advances in Colloid and Interface Science. Elsevier. 191–192 (191–192): 22–30*)

Nitrogen

- N_2 solubility in blood: 0.0138 to 0.0148, dissolved by whole blood under nitrogen pressures varying from 1 to 6 atmospheres (absolute) has been found directly proportional (1.3 -> 7.9)
- 단백질(아미노산), 알칼로이드



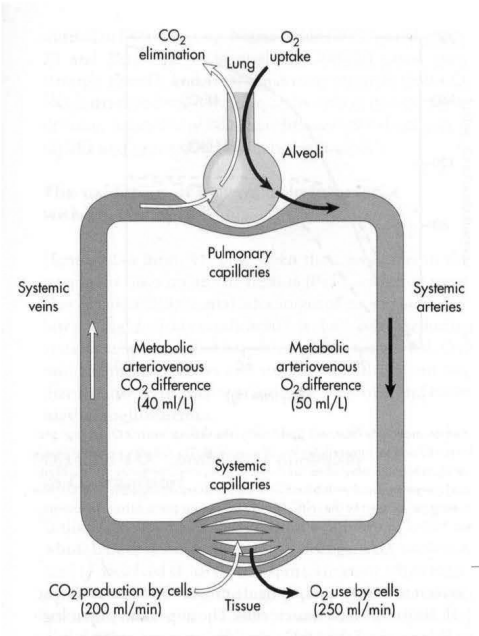
HBOT: primary mechanism (CO)



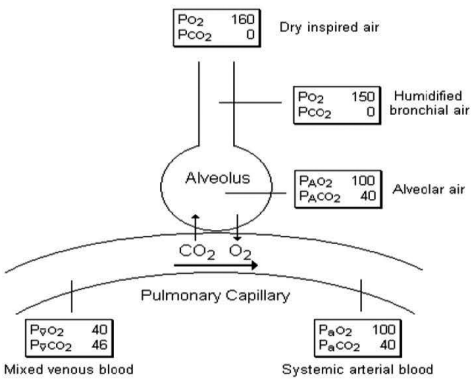
HBOT: primary mechanism

- Emergent condition (time dependent)
- Direct bubble size reduction: AGE, DCS
- Hyperoxygenation: CO, cyanide, toxic gases

O_2 transport (cellular level)

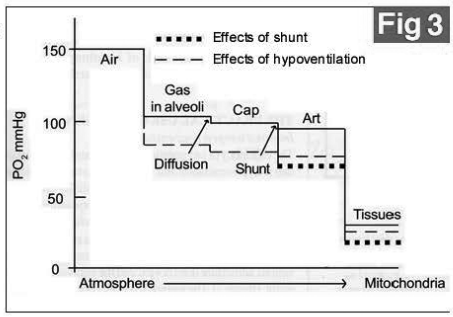


Movement of O_2 down



Movement of O_2 down

- Oxygen concentration: down (air -> cell)
- PO_2: the lowest level (4-20 mmHg) in the mitochondria



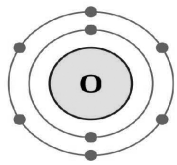
Oxygen transport

- 4 factors:
  - O_2 content of whole blood (CaO_2)
  - O_2 delivery (DO_2)
  - O_2 uptake
  - Fractional extraction of O_2 from capillary blood

O_2 content (CaO_2)

- CaO_2= 1.34 x Hb x SaO_2 + 0.003 x PaO_2
- NL CaO_2= 1.34 x 15 x 0.98 (19.7) + 0.003 x 100 (0.3)
  = 20.0 ml/100 ml (or 200 ml/L)
- Which is more influence in CaO_2? Hb vs PaO_2
- Solubility of O_2: 0.028 ml O_2/L/mm Hg

Solubility of O_2 & CO_2 in plasma



Temp	ml O_2/L/mm Hg	ml CO_2/L/mm Hg
25	0.033	0.892
30	0.031	0.802
35	0.028	0.713
37	0.028	0.686
40	0.027	0.624

Christoforites C et al. J Appl Physiol 1969;26:56
Severinghaus JW et al. J Appl Physiol 1956;9:189

Concentration of O_2 & CO_2 in whole blood

	Arterial	Venous	A-V difference
PO_2	90 mmHg	40 mmHg	50 mmHg
Dissolved O_2	3 ml/L	1 ml/L	2 ml/L
Total	200 ml/L	150 ml/L	50 ml/L
PCO_2	40 mmHg	45 mmHg	5 mmHg
Dissolved CO_2	26 ml/L	29 ml/L	3 ml/L
Total CO_2	490 ml/L	530 ml/L	40 ml/L

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	19.7	0.3	20.0
2 ATA HBOT	19.7	3.0	22.7
3 ATA HBOT	19.7	4.5	24.2

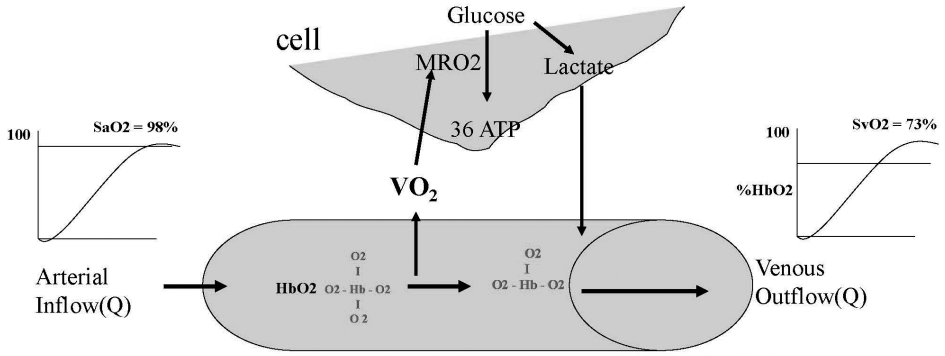
Oxygen delivery (DO_2)

- DO_2 = cardiac output(Q) x CaO_2
  = Q ($mL/min/m^2$) x 1.34 x Hb x SaO_2 x 10
  = NL (520 - 570 $mL/min/m^2$)

Oxygen uptake (VO_2)

- VO_2 = Q x (CaO_2 - CvO_2)
  = Q($mL/min/m^2$) x 1.34 x Hb x SaO_2 x 10
  - Q($mL/min/m^2$) x 1.34 x Hb x SvO_2 x 10
  = Q x 1.34 x Hb x (SaO_2 - SvO_2)
  = NL(110 - 160 $mL/min/m^2$)

Tissue Oxygen Balance



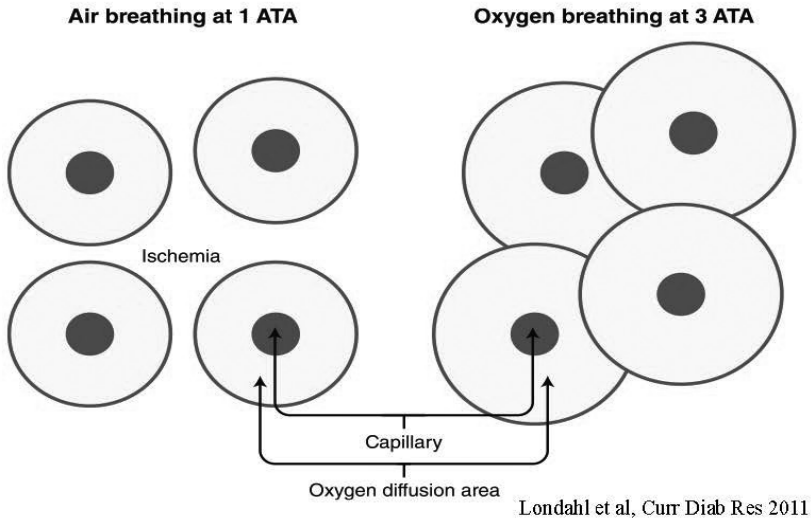
$$VO2 = Q \times 1.34 \times Hb \times (SaO_2 - SvO_2)$$

TABLE 5. EFFECTS OF O_2 INHALATION AT 3.5 ATM. UPON BLOOD O_2 AND CO_2 TRANSPORT (*Group III*, 12 SUBJECTS)

BLOOD MEASUREMENTS	1 Atm Air			3.5 Atm. Oxygen		
	Arterial	Internal Jugular	A-V Difference	Arterial	Internal Jugular	A-V Difference
O_2 Content (vols %)	18.7	12.6	6.1	26.0	17.8	8.2
Hb Sat (%)	96.1	65.2	30.9	100.0	89.3	10.7
pO_2 (mm. Hg)	91.0	38.0	53.0	2100.0	75.0	2025.0
CO_2 Content (vols %)	50.0	55.7	5.7	46.9	55.2	8.3
pH	7.40	7.34	0.06	7.43	7.31	0.12
pCO_2 (mm Hg)	39.0	50.0	11.0	34.0	53.0	19.0

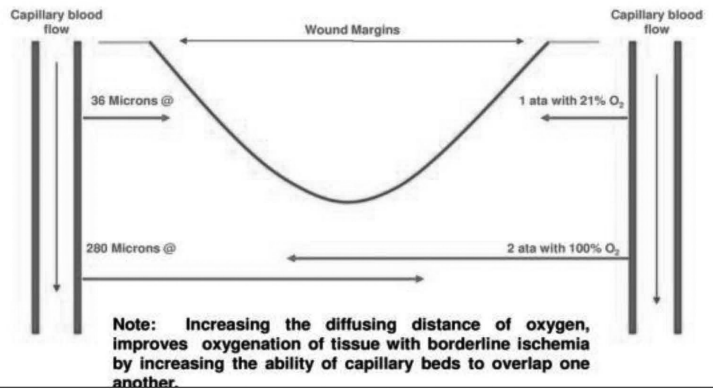
Lambetsen, DJ, et al. 1953. J. Appl. Physiol.
Venous Hb sat. at almost arterial levels at 3.5 ATM of oxygen

Oxygen diffusion area



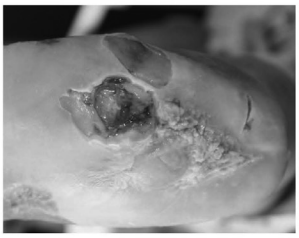
Londahl et al, Curr Diab Res 2011

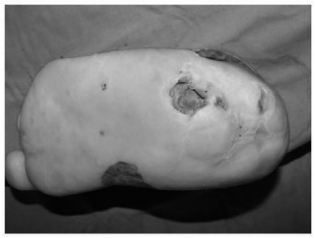
Diffusion distance of oxygen from functioning capillaries



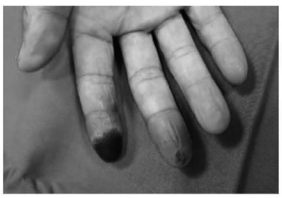
Note: Increasing the diffusing distance of oxygen, improves oxygenation of tissue with borderline ischemia by increasing the ability of capillary beds to overlap one another.

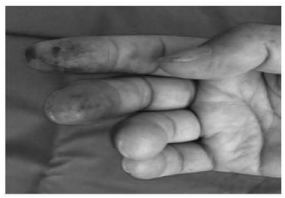
당뇨발

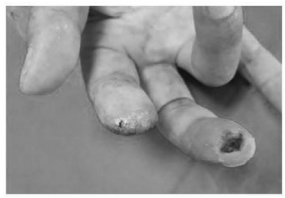




말초혈관 장애



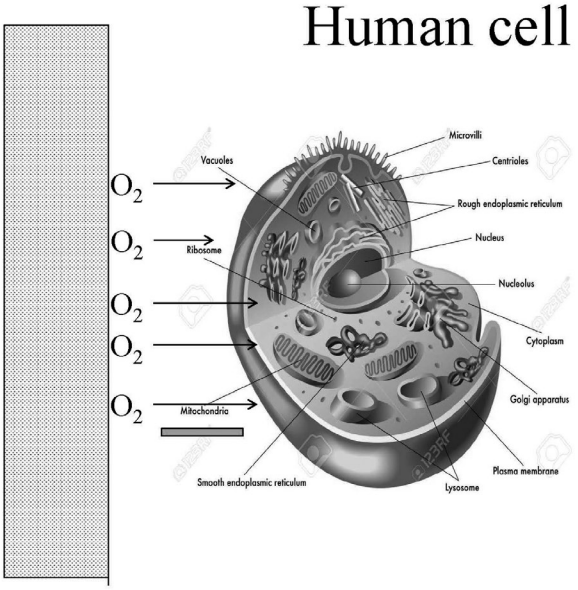




HBOT: secondary mechanism

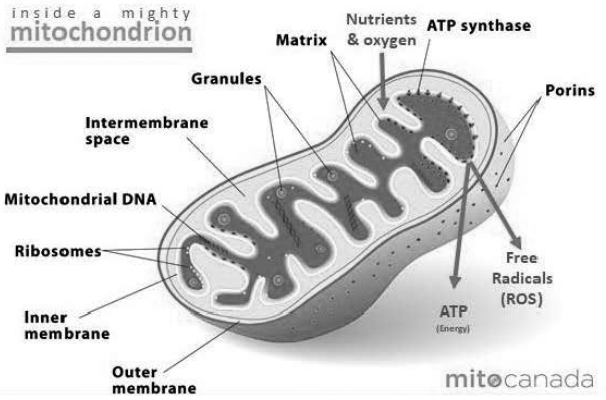
- Non-emergent condition (frequency dependent)
- Anti-bacterial
- Anti-inflammation
- Neovascularization
- Fibroblast (collagen)
- Stem cell migration

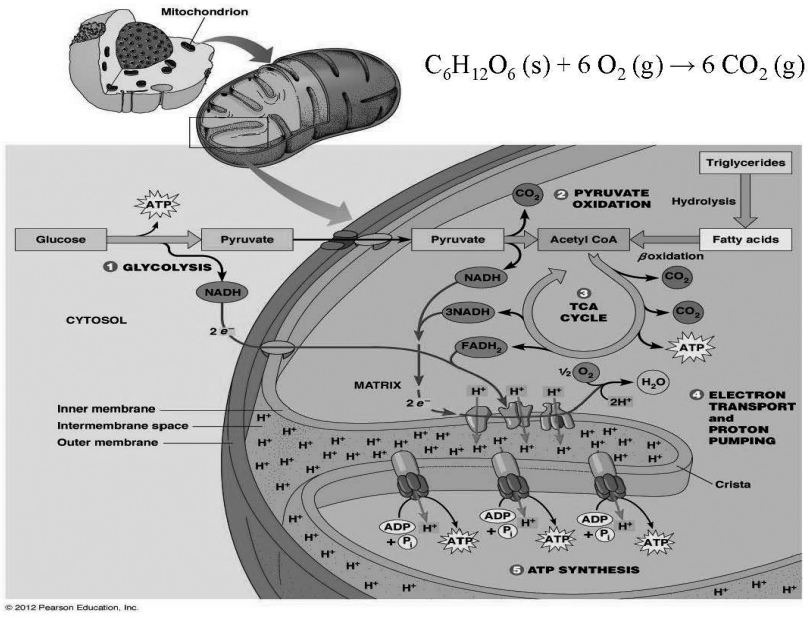
Human cell



- O2 consumption: 250 ml/min
- 90%: mitochondria

Mitochondrion





$$C_6H_{12}O_6\,(s) + 6\,O_2\,(g) \rightarrow 6\,CO_2\,(g) + 6\,H_2O\,(l) + E$$

Cellular respiration

1. Glycolysis: 2 ATPs + Glucose → 2 Pyruvic Acid + 4 H^+ + 4 ATPs

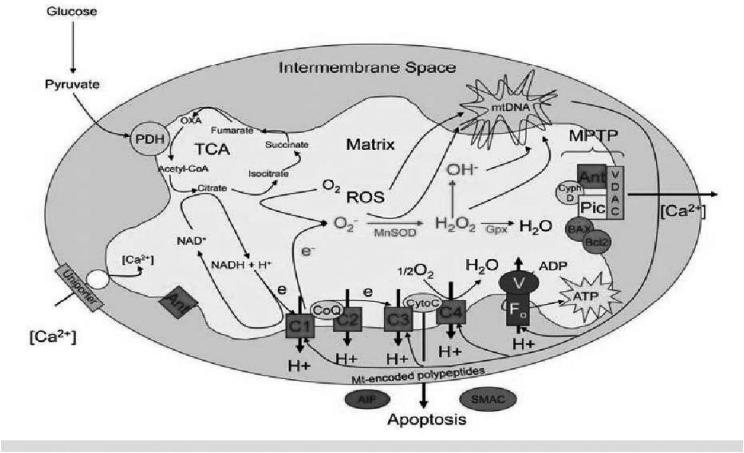
2. Formation of Acetyl CoA: 2 Pyruvic Acid + 2 CoA → 2 Acetyl CoA + 2 CO_2 + 2 H^+

3. Krebs Cycle: 2 Acetyl CoA + 3 O_2 → 6 H^+ + 4 CO_2 + 2 ATPs

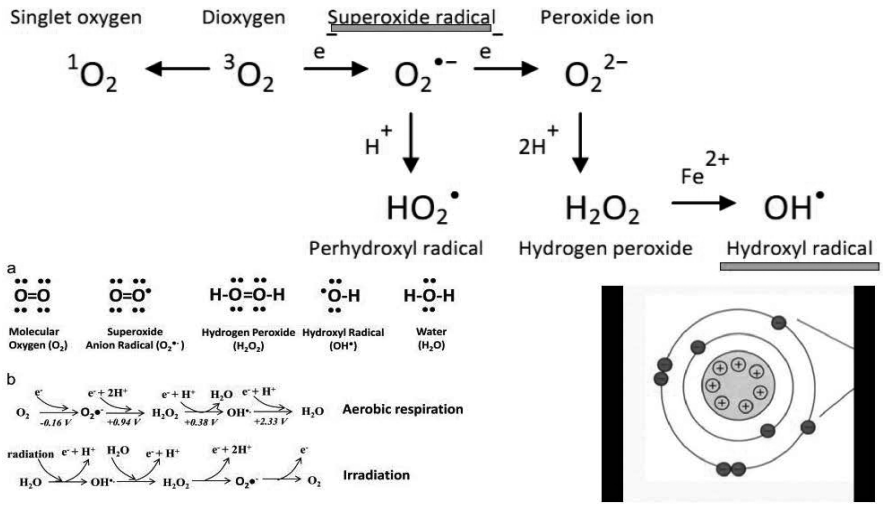
4. Electron Transport System: 12 H^+ + 3 O_2 → 6 H_2O + 32 ATPs

Overall Reaction: Glucose + 6 O_2 → 6 CO_2 + 6 H_2O + 36 ATPs

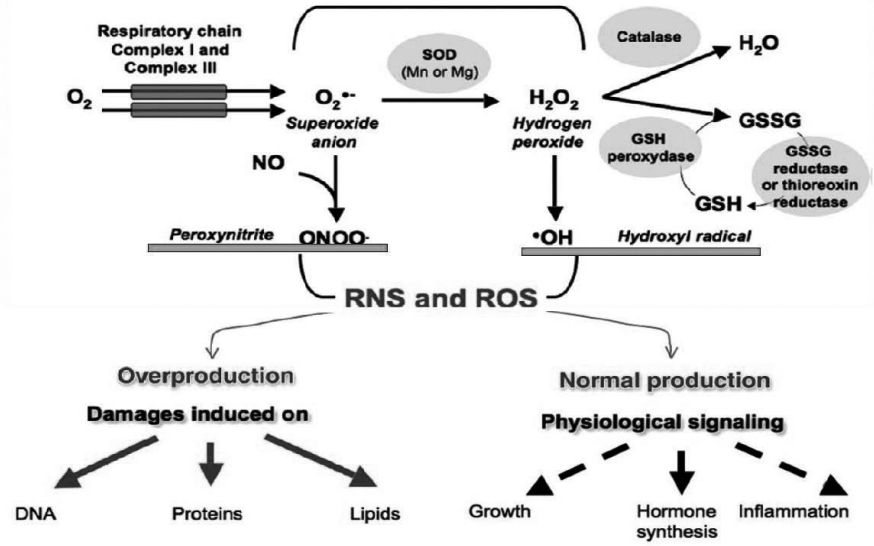
Cellular respiration



Oxygen free radical



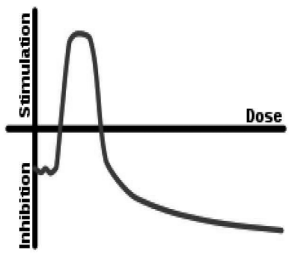
Reactive nitrogen & oxygen



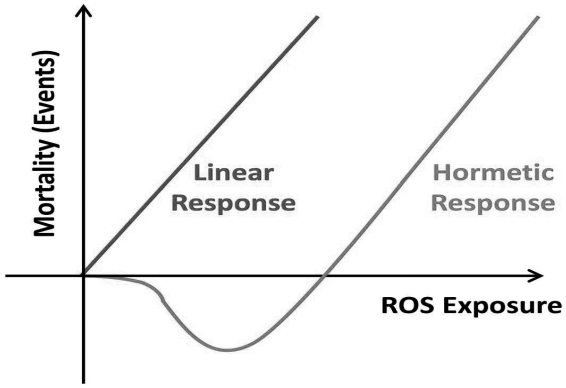
Comprehensive Physiology 2017

Hormesis

- Biphasic response: increasing amounts of a substance or condition
- Within the hormetic zone: favorable biological response to low exposures to toxins and other stressors
- Physical exercise, alcohol, mitochondria



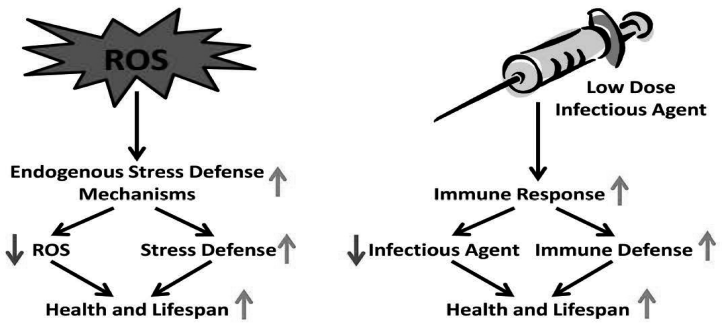
Mitochondrial hormesis (mitohormesis)



Free radical theory: linear dose-response relationship (ROS and oxidative stress and mortality)
Mitohormesis: non-linear dose-response relationship (low doses of ROS exposure decrease mortality, while higher doses promote mortality)

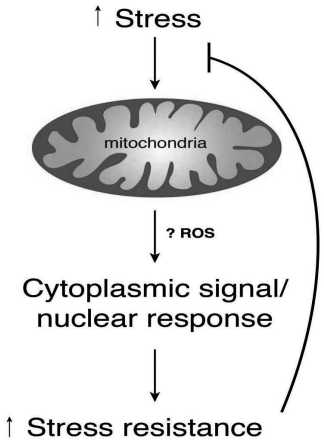
Dose Response. 2014 May; 12(2): 288–341

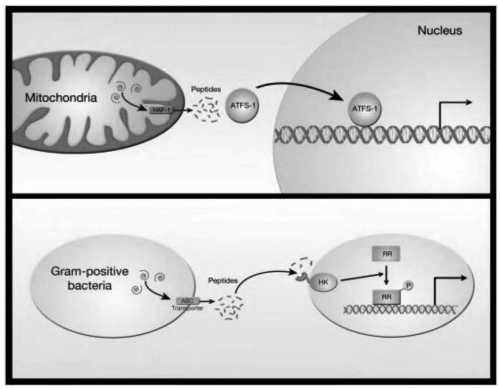
Mitochondrial hormesis (mitohormesis)



Dose Response. 2014 May; 12(2): 288–341

Mitochondrial hormesis (mitohormesis)

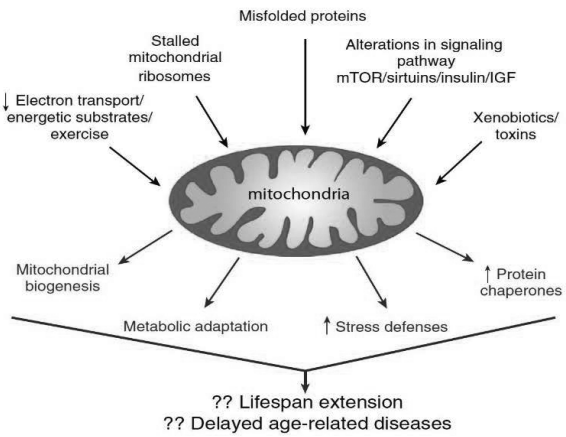




Potential Parallels (mitochondrial unfolded protein response and quorum sensing in G + bacteria)

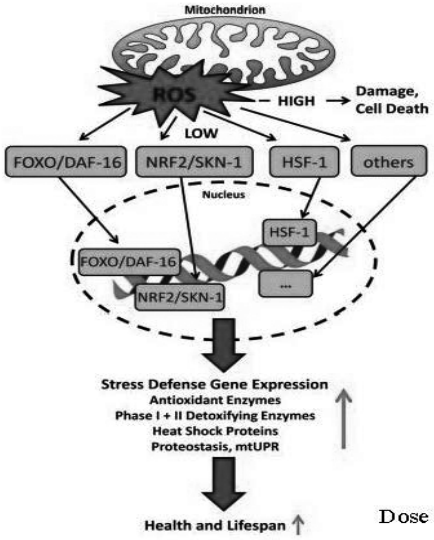
Cell metabolism 2014 May; 19(5): 757–766

Mitochondrial hormesis (mitohormesis)

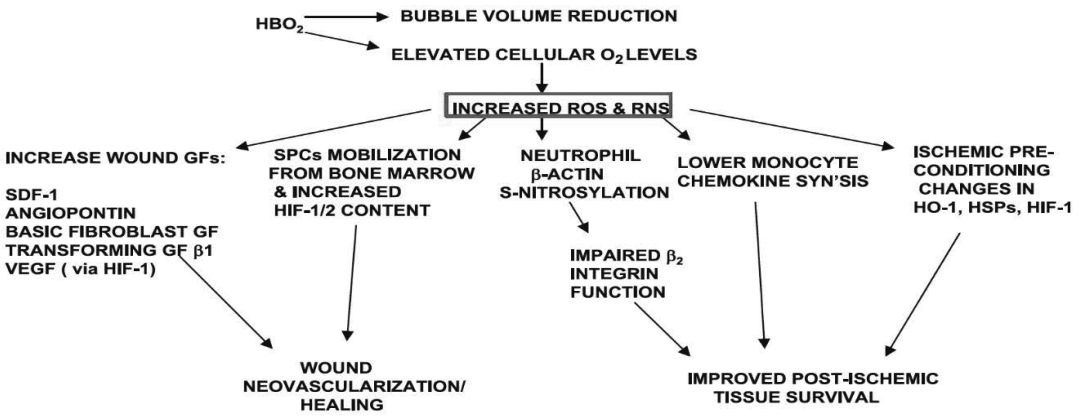


Cell metabolism 2014 May; 19(5): 757–766

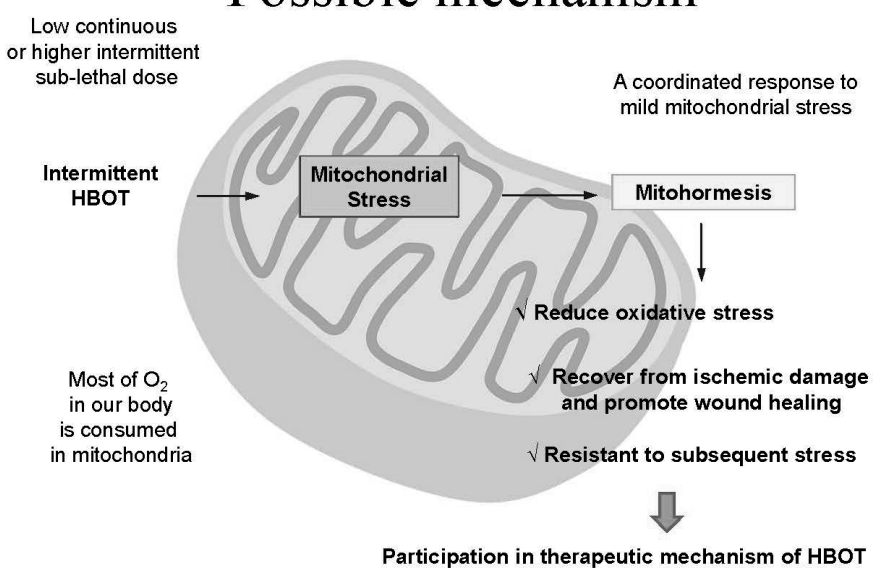
Mitochondrial hormesis (mitohormesis)



Dose Response. 2014 May; 12(2): 288–341



J Appl Physiol (2009)



Mechanisms of HBOT
(Primary, secondary & tertiary)

Primary	Secondary	Tertiary
• Hyperoxygenation (O_2 tensions)	• Enriched O_2 (cellular functions)	• Oxidative stress in mitochoondria
• Direct effects of Oxygen delivery/uptake Bubble reduction	Anti-bacterial Anti-inflammation (cytokine) Ischemia-reperfusion injury Neovascularization Stem cell migration	Mitohormesis ? ? ?

Future

- Research of mechanism
- Clinical application
- Spectrum of HBOT

Session 1

좌장: 서울의료원 응급의학과 **박상현** 주임과장

Decompression illness for military divers

해군해양의료원

허정필 원장

해군해양의료원 산하 해양의학지원소는 해군의학 관련 진료 및 연구, 잠수의무요원 육성 및 관리, 챔버 운용 및 교육 등 다양한 역할을 수행하고 있다. 해군은 해양의학지원소 챔버 뿐 아니라 육상 및 함정에서 다수의 다인용 및 일인용 챔버를 운용하고 있다. 군대에서 다이버들은 임무에 따라 크게 두 종류로 나누어진다. 해난구조대(SSU: Ship Salvage Unit)로 대표되는 구조 다이버들과 특수전전단(UDT: Underwater Demolition Team)으로 대표되는 전투 다이버들이다. 군대 다이버들은 군사작전 뿐 아니라 민간사고에도 투입되고 있으며 이때 항상 잠수의무요원들이 같이 활동을 한다. 특히, 세월호 의무지원은 역대 최다 · 최장기간 다이버 및 잠수의무요원들이 투입된 작전으로 작전 간 챔버치료 건수 만 125건에 이르렀다. 산업 및 레저 다이버들을 대상으로 한 여러 외국 연구에서는 장기간 다이빙 시 이압성 골괴사, 난청, 폐기능 저하 및 신경학적 이상 등이 생길 수 있다고 보고되고 있다. 해군은 잠수함 근무환경의 유해인자 분석 및 특수건강검진을 포함한 잠수함 승조원들에 대한 건강관리모델을 민간대학병원과 협력하여 개발 중이다. 향후 이러한 건강관리 체계를 다이버들에게도 확대 적용하여 평생 건강관리 방안을 마련할 필요가 있다.

CRAO

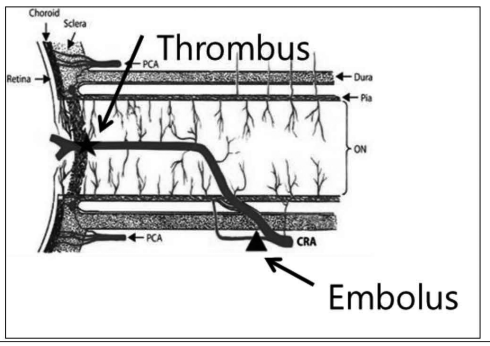
연세원주의대 응급의학과

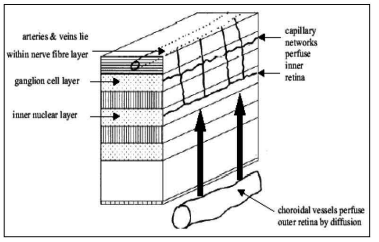
차용성 교수

Introduction (1)

- Central retinal artery occlusion (CRAO) is a devastating and common ophthalmologic condition.
- CRAO presents as a sudden, unilateral, and painless loss of vision.
- Even when treated promptly, an acute obstruction of the central retinal artery usually results in severe and permanent loss of vision.

Hayreh SS, et al. *Am J Ophthalmol* 2005;140:376. 1-18.





Varma, D., Cugati, et al. *Eye,* 2013 *27*(6), 688.

Introduction (2)

- Traditional CRAO treatment
  - Focuses on moving the embolus downstream by lowering intraocular pressure and producing vasodilatation.
    - ocular massage
    - anterior chamber paracentesis
    - intraocular pressure-lowering medications
    - vasodilators and oral diuretics

- However, there are currently no effective therapies available for CRAO.

Purnima S. Patel, SriniVas R. Sadda. *Retina 5th. Chap 51.*

Introduction (3)

- Another treatment for CRAO is hyperbaric oxygen therapy (HBOT).
  - This involves the inhalation of 100% oxygen at pressures exceeding 1 ATA.
  - During HBOT, the volume of oxygen dissolved in the plasma increases up to 20–30 times.

Fosen KM, et al. *Antioxid Redox Signal* 2014;21:1634-47.

Case

Case report (1)

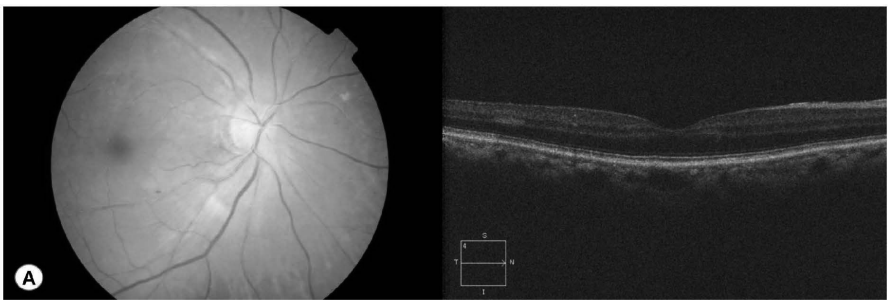
- 81/F
- Chief complaint: sudden, painless loss of vision in the right eye (OD)
- Duration: 10 hours prior to the ED admission
- Past history
  - Heart failure, atrial fibrillation, and a renal infarct
  - Cataract surgery on both eyes

Case report (2)

- Physical examination

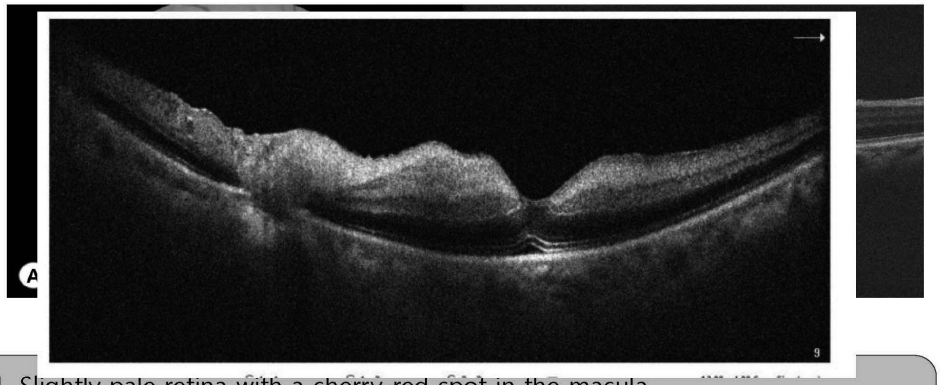
  - Non-corrected visual acuity at the ED
    - hand motion only in the OD (right eye) and 0.4 on a decimal scale in the OS (left eye).
  - Intraocular pressure: 14 mmHg in the OD and 13 mmHg in the OS.
  - Relative afferent pupillary defect at her right eye
  - The anterior segment of her eyes: no abnormal findings.
  - A dilated fundus examination: slightly pale retina with a cherry-red spot in the macula.

Case report (3)



1. Slightly pale retina with a cherry-red spot in the macula
2. A domain optical coherence tomography scan: mild increase in the reflectivity of the inner retinal layer

Case report (3)



1. Slightly pale retina with a cherry-red spot in the macula
2. A domain optical coherence tomography scan: mild increase in the reflectivity of the inner retinal layer

Case report (4)

- Digital massage on her right eye + topical brimonidine & dorzolamide/timolol → maximize the perfusion pressure.
- ED: oxygen therapy for 30 minutes via facial mask with a reservoir bag (15 L/min) → no significant improvement in the patient's vision.
  → treat the patient with HBOT.

Case report (5)

2 and 2.4 ATA

No significant improvement

2.8 ATA

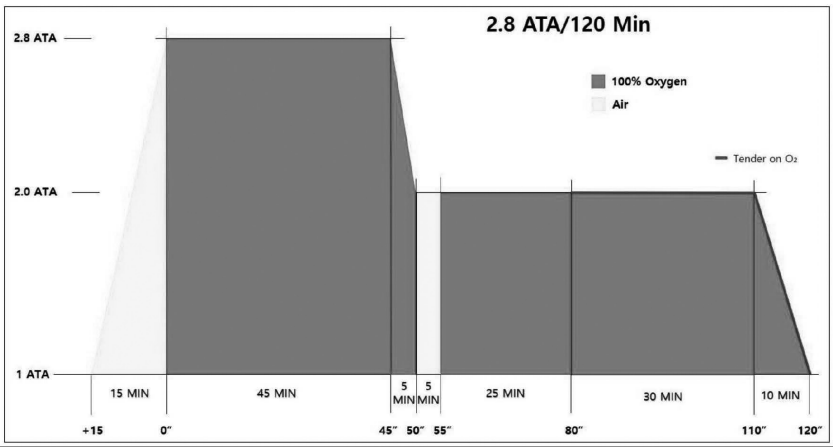
Almost the same vision

2.8 ATA

2.8 ATA for 140 minutes in the multiplace chamber

HBOT treatment

Case report (6)



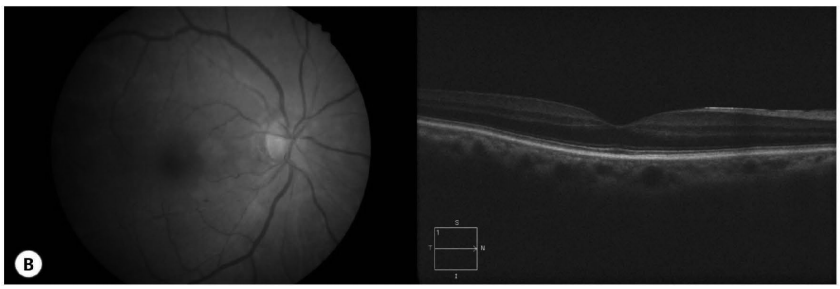
HBOT protocol

Case report (7)

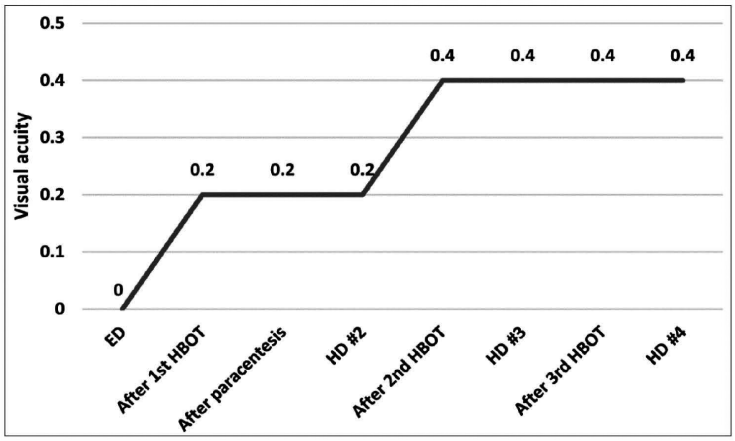
- After her admission (HD #1)
  - Anterior chamber paracentesis by an ophthalmologist
    - the visual acuity did not improve.
  - Start on intermittent oxygen therapy for 15 minutes every hour, alternating with 45 minutes of breathing room air.
  - The patient was also administered a 6 L/min supply of oxygen while sleeping.
  - If her visual acuity decreased, we planned to restart the HBOT.

Case report (8)

- HD #2
  - NO the cherry-red spot on the fundus



Case report (9)



Backgrounds (1)

- Arterial blood supply
  - Internal carotid artery ⇒ Ophthalmic artery ⇒ Orbital structures +Tissues of the globes (central artery of the retina, short and long posterior ciliaries, anterior ciliaries).
  - The central retinal artery enters the globe within the substance of the optic nerve and serves the inner layers of the retina through its many branches.
  - **The long posterior ciliary arteries provide blood to the choroid and the outer layers of the retina.**
- The visual signs and symptoms of vascular occlusive diseases of the retina are dependent on
  - The particular vessel occluded, the degree of occlusion, the location of the occlusion, and the presence or absence of a cilioretinal artery.
  - 15-30%에서 cilioretinal artery is present
    - Supplies the area of the retina around the macula
    - Central vision may be preserved in central retinal artery occlusion.

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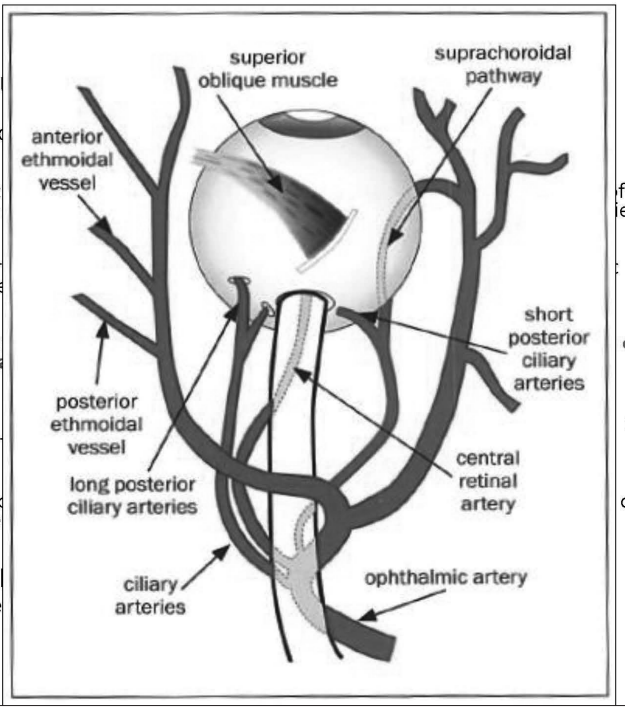

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    - Supplies the area of the retina around the macula
    - Central vision may be preserved in central retinal artery occlusion.

Rationale (1)

- In CRAO, the inner retinal layers
  - Normally served by the retinal circulation → Responsible for the vision loss
  - Normally, the choroidal circulation supplies the majority of the oxygen to the retina.
  - **Obtain enough oxygen via diffusion from the choroidal circulation → maintain viability**
    - Under normoxic conditions, approximately 60% of the retina's oxygen comes from the choroidal circulation.
    - Under hyperoxic conditions the choroid is capable of supplying 100% of the oxygen needed by the retina.

Rationale (2)

- In considering the effect of treating CRAO with supplemental oxygen
  - HBOT initiated ***before the retinal tissue damage is irreversible***.
  - Degree of occlusion of the blocked vessel matter.
    - If level of occlusion is at ophthalmic artery, there is no collateral circulation to provide oxygenation of the inner layer of the retina.
  - **Necessary oxygenation must be maintained to keep the retina viable** until natural recanalization, which usually occurs **within 72 hours.**

Patient selection criteria

- **Patients presenting within 24 hours of symptom onset should be considered for HBOT**
  - **Few case reports of patients presenting after this time interval having positive results.**

Ophthalmologic Tx

+

HBOT

Protocol of UHMS

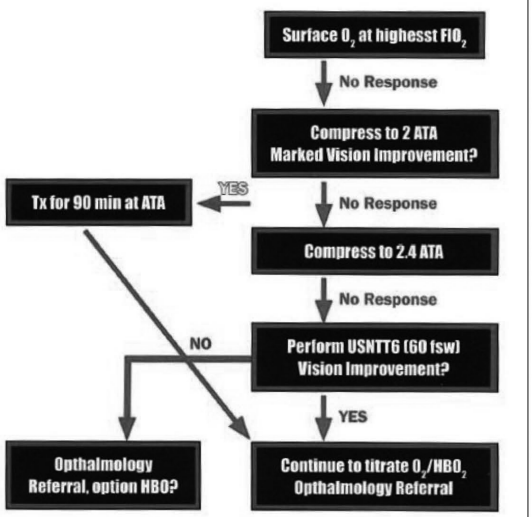
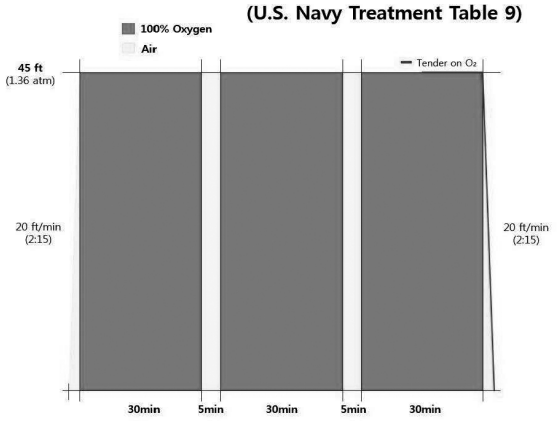


Figure 2. CRAO Treatment Algorithm (Murphy-Lavoie 2012).

WSCH HBOT treatment protocol #5

(U.S. Navy Treatment Table 9)



Total Elapsed Time : 102 min 15 sec
(not including descent time)

적응증

1. 감압병(DCS) 반복 치료시 사용가능
Air breaks는 생략할 수 있음

1. 가압속도 - 20 ft/min
(환자의 견디는 정도를 보고 조절한다.)

2. 감압속도 - 20 ft/min. 감압 속도는 환자의 medical 상태에 따라 1ft/min까지 조정 가능하다.

3. 경련이 나타나면 산소마스크를 벗기던지 산소중단을 멈추고 모든 증상이 사라진 이후 15분 뒤 산소호흡을 다시 시작하고 중단된 시점을 기준으로 치료 프로토콜을 이어 진행한다.

4. 동승자는 45ft 압력유지 기간의 마지막 15분 부터 100%산소를 시작하며, 감압 속도와 관계없이 모든 감압이 이루어질 때까지 산소호흡을 유지한다.

5. 환자는 감압하는 동안 100% 산소 또는 공기 호흡을 할 수 있다.

6. 만일 환자가 45 ft에서 산소에 잘 적응을 못할 경우 이 치료 유지 압력은 30 ft로 변경 가능하다. 산소 호흡 시간은 최대 3-4 시간으로 연장 가능하다.

WSCH HBOT treatment protocol #4

1. 가압속도 - 20 ft/min (환자의 견디는 정도를 보고 조절한다.)

2. 감압속도 - 1 ft/min를 초과하지 않는다. 감압속도를 더 느리게 하였다고 그것을 보상하기 위해서 빠르게 하지 말고 반대로 감압속도가 빨랐다면 감압을 잠깐 중단하여 빠른 감압속도를 보상하여라.

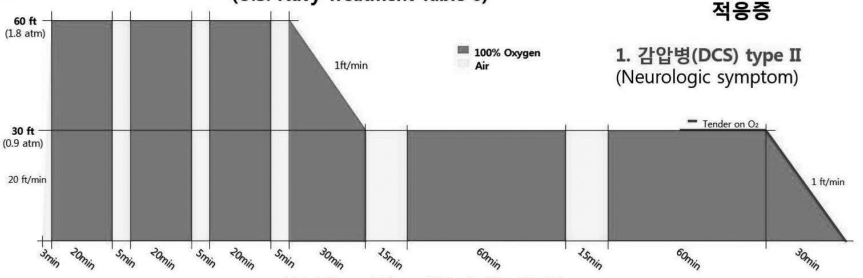
3. 환자는 60ft에 도달한 이후부터 100% 산소 호흡을 시작한다.

4. 경련이 나타나면 산소마스크를 벗기던지 산소중단을 멈추고 모든 증상이 사라진 이후 15분 뒤 산소호흡을 다시 시작하고 중단된 시점을 기준으로 치료 프로토콜을 이어 진행한다.

5. Table 6에서 60ft 압력에서 25분 Period(100% 산소 20분, Air break 5분)를 2회 더 추가하여 연장할 수 있고 또는 30ft 압력에서 75분 Period(Air break 15분, 100% 산소 60분)를 2회 더 추가하여 진행 가능하다. 또한 양쪽 모두 2번 추가 진행 가능하다.

6. 본 table을 변형하지 않았을 경우나 30 또는 60 feet에서 한 번의 추가 실시가 있었을 경우는 동승자는 30ft 압력유지 구간의 마지막 30분 부터 모든 감압이 이루어질 때까지 100%산소호흡을 유지한다. 30 또는 60 feet에서 한 번 이상의 추가 실시가 있었을 경우는 동승자는 30ft 압력유지 구간의 마지막 60분 부터 모든 감압이 이루어질 때까지 100%산소호흡을 유지한다. 만일 동승자가 18시간 이전에 고압 노출이 있었다면 30ft에서 추가 60분의 100% 산소 유지가 필요하다.

(U.S. Navy Treatment Table 6)



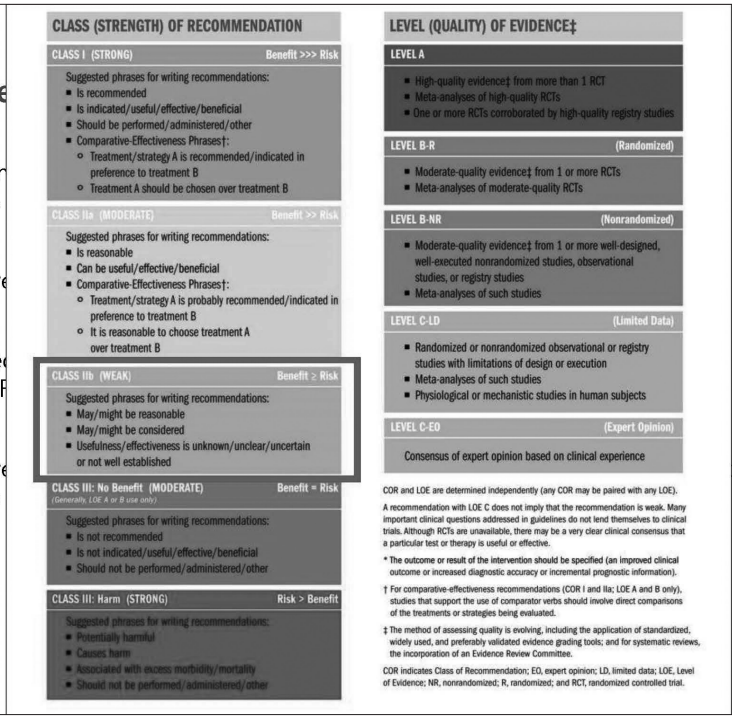
Total Elapsed Time : 285 min (4hr 45min)

적응증

1. 감압병(DCS) type II
(Neurologic symptom)

Evidence-Based Review

- Animal models of retinal injury have shown a reduction in apoptosis from 58% cell loss to 30% in animals treated with HBOT after experimental CRAO.
- There are many case reports.
- Based on the American Heart Association classification of evidence, treatment of CRAO with HBOT is class IIb.
- There is fair to good evidence to support its use with retrospective controlled case series but no prospective randomized controlled trials.



CLASS (STRENGTH) OF RECOMMENDATION

CLASS I (STRONG) — Benefit >>> Risk

Suggested phrases for writing recommendations:
- Is recommended
- Is indicated/useful/effective/beneficial
- Should be performed/administered/other
- Comparative-Effectiveness Phrases†:
  - Treatment/strategy A is recommended/indicated in preference to treatment B
  - Treatment A should be chosen over treatment B

CLASS IIa (MODERATE) — Benefit >> Risk

Suggested phrases for writing recommendations:
- Is reasonable
- Can be useful/effective/beneficial
- Comparative-Effectiveness Phrases†:
  - Treatment/strategy A is probably recommended/indicated in preference to treatment B
  - It is reasonable to choose treatment A over treatment B

CLASS IIb (WEAK) — Benefit ≥ Risk

Suggested phrases for writing recommendations:
- May/might be reasonable
- May/might be considered
- Usefulness/effectiveness is unknown/unclear/uncertain or not well established

CLASS III: No Benefit (MODERATE) — Benefit = Risk
(Generally, LOE A or B use only)

Suggested phrases for writing recommendations:
- Is not recommended
- Is not indicated/useful/effective/beneficial
- Should not be performed/administered/other

CLASS III: Harm (STRONG) — Risk > Benefit

Suggested phrases for writing recommendations:
- Potentially harmful
- Causes harm
- Associated with excess morbidity/mortality
- Should not be performed/administered/other

LEVEL (QUALITY) OF EVIDENCE‡

LEVEL A
- High-quality evidence‡ from more than 1 RCT
- Meta-analyses of high-quality RCTs
- One or more RCTs corroborated by high-quality registry studies

LEVEL B-R (Randomized)
- Moderate-quality evidence‡ from 1 or more RCTs
- Meta-analyses of moderate-quality RCTs

LEVEL B-NR (Nonrandomized)
- Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies
- Meta-analyses of such studies

LEVEL C-LD (Limited Data)
- Randomized or nonrandomized observational or registry studies with limitations of design or execution
- Meta-analyses of such studies
- Physiological or mechanistic studies in human subjects

LEVEL C-EO (Expert Opinion)

Consensus of expert opinion based on clinical experience

COR and LOE are determined independently (any COR may be paired with any LOE).

A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

* The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).

† For comparative-effectiveness recommendations (COR I and IIa; LOE A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

‡ The method of assessing quality is evolving, including the application of standardized, widely used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee.

COR indicates Class of Recommendation; EO, expert opinion; LD, limited data; LOE, Level of Evidence; NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.

Case series in WSCH

- **Purpose**
  - To analyze the effects of hyperbaric oxygen therapy in central retinal artery occlusion.

- **Method**
  - Retrospective chart review
  - Yonsei Wonju Severance Hospital
  - Mar 1st 2011 ~ Sep 30th 2018
  - Exclusion criteria
    - Lost to follow up within 1 month

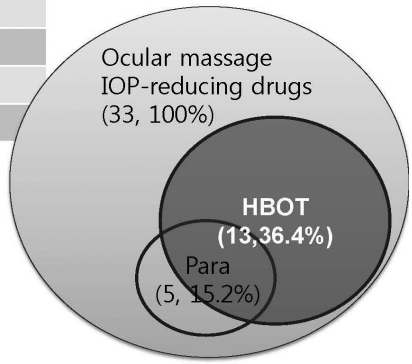
Results

- Totally 33 Eyes of 33 patients were reviewed.

Variables		
Age	73.55 ± 9.66	
Sex	M : 24 (72.7%)	F : 9 (27.3%)
HTN	24 (72.7%)	
DM	8 (24.2%)	
Cilioretinal artery	5 (15.2%)	
Symptom duration (in hours)	78.12 ± 135.91	
Initial Visual Acuity (LogMAR)	2.81 ± 1.03	

Treatment

Treatment	N (%)
Ocular massage IOP-reducing drugs	33 (100)
Hyperbaric oxygen therapy (HBOT)	13 (36.4)
Anterior chamber paracentesis	5 (15.2)
Single treatment	19 (57.6)
2 Combined treatment	10 (30.3)
3 Combined treatment	4 (12.1)



Outcome

Legal blindness (visual acuity < 0.1)	26 (78.8%)
Visual acuity over 0.5 (Decimal)	3 (9.1%)

Result (1)

	V/A over 0.5 (3)		V/A under 0.5 (30)		P-value
Age	55, 72, 82		75 [64 - 80]		0.657
Sex	M : 2	F : 1	M : 22	F : 8	1.000
Time	6 [10 – 24]		36 [11 - 96]		0.188
HTN	1 (33.3%)		23 (76.7%)		0.174
DM	0 (0.0%)		8 (26.7%)		0.560
Cilioretinal artery	1 (33.3%)		4 (13.3%)		0.400
HBOT	3 (100.0%)		10 (33.3%)		0.052
Paracentesis	2 (66.7%)		3 (10.0%)		0.053
Initial visual acuity	3.00 [3.00 - 3.00]		3.00 [2.00 – 3.00]		0.491
Final visual acuity	0.15 [0.22 - 0.30]		3.00 [1.30 – 5.00]		<0.001

Result (2)

	HBOT (13)	No HBOT (20)	P-value
Age	77 [67 - 81]	74 [64 - 79]	0.478
Sex	M : 9 F : 4	M : 15 F: 5	1.000
Time	24 [7 - 72]	36 [24 - 96]	0.478
HTN	9 (69.2%)	15 (75.0%)	1.000
DM	3 (23.1%)	5 (25.0%)	1.000
Cilioretinal artery	2 (15.4%)	3 (15.0%)	1.000
Paracentesis	4 (30.8%)	1 (5.0%)	0.066
Initial visual acuity	3.00 [2.50 – 3.00]	3.00 [2.00 – 3.00]	0.392
Change in V/A	0.70 [-1.50 – 2.74]	0.00 [0.00 – 0.63]	0.281
Final visual acuity	2.00 [0.80 – 4.00]	3.00 [1.38 – 5.00]	0.392

Conclusion

- **Hyperbaric oxygen therapy** may have additive beneficial effects on central retinal artery occlusion.
- Further research on larger samples should be performed.

Non-listed/Non-approved/ Controversial indications

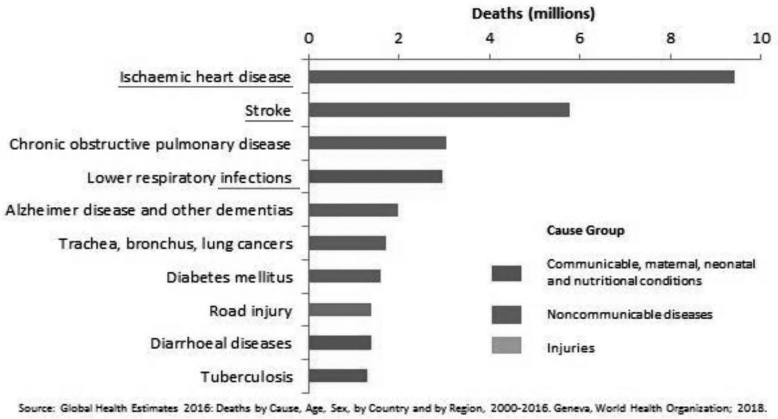
아주의대 응급의학과

최상천 교수

Now, first things first !

I have got no conflict of interest to declare.

Top 10 global causes of deaths, 2016

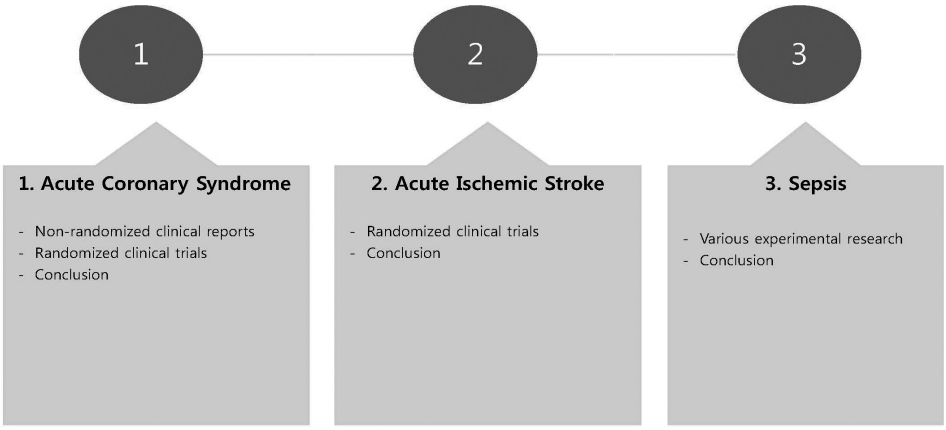


Source: Global Health Estimates 2016: Deaths by Cause, Age, Sex, by Country and by Region, 2000-2016. Geneva, World Health Organization; 2018.

Sepsis

- The global epidemiological burden of sepsis is difficult to ascertain.
- It is estimated to affect more than 30 million people worldwide every year, potentially leading to 6 million deaths.

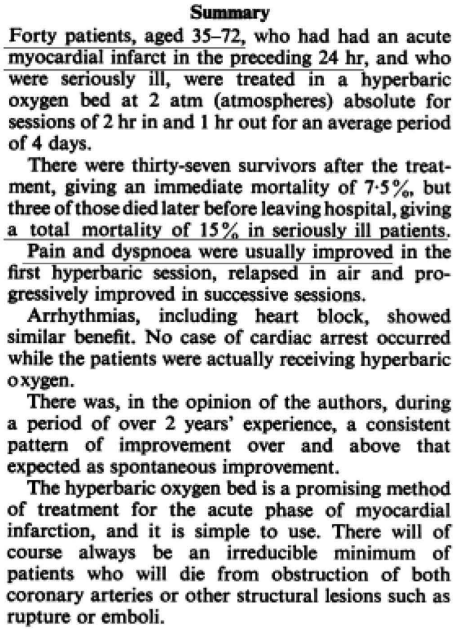
Contents



Acute Coronary Syndrome

Non-randomized clinical reports to treat ACS

Trial	Methodology	Therapy	Outcomes
Cameron and collegues (1965)	10 patients with AMI within 24 hours	2 ATA HBOT; 1 treatment only	Decreased cardiac output, Increased SVR and SBP
Ashfield and collegues (1969)	40 patients with AMI within 24 hours	2 ATA 2 hours followed by 1 ATA on air; repeat for up to 4 days	15% mortality rate, improved pain and dyspnea
Veselka and collegues (1999)	17 patients with history of MI	Dobutamine stress echo followed by HBOT at 2 ATA for 90 mins	HBO can detect viable myocardium with about the same performance as dobutamine
Moon and collegues (1964)	1 patient in cardiogenic shock	48 hours of HBOT	Successful outcome
Hood (1968)	1 patient with refractory VT 3wks after anterolateral MI	3 ATA HBOT for 15 mins then 2 ATA for 7 hours; 2 such sessions	Improvement in dysrhythmia; discharged day 25



Summary

Forty patients, aged 35–72, who had had an acute myocardial infarct in the preceding 24 hr, and who were seriously ill, were treated in a hyperbaric oxygen bed at 2 atm (atmospheres) absolute for sessions of 2 hr in and 1 hr out for an average period of 4 days.

There were thirty-seven survivors after the treatment, giving an immediate mortality of 7·5%, but three of those died later before leaving hospital, giving a total mortality of 15% in seriously ill patients.

Pain and dyspnoea were usually improved in the first hyperbaric session, relapsed in air and progressively improved in successive sessions.

Arrhythmias, including heart block, showed similar benefit. No case of cardiac arrest occurred while the patients were actually receiving hyperbaric oxygen.

There was, in the opinion of the authors, during a period of over 2 years' experience, a consistent pattern of improvement over and above that expected as spontaneous improvement.

The hyperbaric oxygen bed is a promising method of treatment for the acute phase of myocardial infarction, and it is simple to use. There will of course always be an irreducible minimum of patients who will die from obstruction of both coronary arteries or other structural lesions such as rupture or emboli.

Randomized clinical trials to treat ACS

Study	Methods	Participants	Interventions	Outcomes
Stavitsky (1998)	Multicenter RCT; non-blind	138 patients with AMI clinical dx and eligible for thrombolysis enrolled in ED	Control: thrombolysis, aspirin, heparin and NTG H: same + 2 ATA HBOT for 2 hrs	Death, time to pain relief, enzyme change, LVEF
Shandling (1997)	Same as above	82 patients (41:41)	Same as above	
Sharifi (2004)	RCT; non-blind; 5 patients crossed allocation	69 Patients (H33:C36) with AMI or unstable angina	Control: stent, ASA, heparin, clonpidogrel H: same + 2 ATA HBOT for 90 mins at 1 and 18 hours	MACE, adverse events

Am Heart J. 1997 Sep;134(3):544-50.

Hyperbaric oxygen and thrombolysis in myocardial infarction: the "HOT MI" pilot study.

Shandling AH[1], Ellestad MH, Hart GB, Crump R, Marlow D, Van Natta B, Messenger JC, Strauss M, Stavitsky Y.

⊕ Author information

Abstract

Hyperbaric oxygen treatment (HBO) in combination with thrombolysis has been demonstrated to salvage myocardium in acute myocardial infarction in the animal model. Therefore a randomized pilot trial was undertaken to assess the safety and feasibility of this treatment in human beings. Patients with an acute myocardial infarction (AMI) who received recombinant tissue plasminogen activator (rTPA) were randomized to treatment with HBO combined with rTPA or rTPA alone. Sixty-six patients were included for analysis. Forty-three patients had inferior AMIs (difference not significant) and the remainder had anterior AMIs. The mean creatine phosphokinase level at 12 and 24 hours was reduced in the patients given HBO by approximately 35% ($p = 0.03$). Time to pain relief and ST segment resolution was shorter in the group given HBO. There were two deaths in the control group and none in those treated with HBO. The ejection fraction on discharge was 52.4% in the group given HBO compared with 47.3% in the control group (difference not significant). Adjunctive treatment with HBO appears to be a feasible and safe treatment for AMI and may result in an attenuated rise in creatine phosphokinase levels and more rapid resolution of pain and ST segment changes.

Cardiology. 1998 Oct;90(2):131-6.

Hyperbaric oxygen and thrombolysis in myocardial infarction: the 'HOT MI' randomized multicenter study.

Stavitsky Y[1], Shandling AH, Ellestad MH, Hart GB, Van Natta B, Messenger JC, Strauss M, Dekleva MN, Alexander JM, Mattice M, Clarke D.

⊕ Author information

Abstract

In a previous pilot study, we demonstrated that adjunctive treatment with hyperbaric oxygen (HBO) appears to be feasible and safe in patients with acute myocardial infarction (AMI) and may result in an attenuated rise in creatine phosphokinase (CPK), more rapid resolution of pain and ST changes. This randomized multicenter trial was organized to further assess the safety and feasibility of this treatment in human subjects. Patients with an AMI treated with recombinant tissue plasminogen activator (rTPA) or streptokinase (STK), were randomized to treatment with HBO combined with either rTPA or STK, or rTPA or STK alone. An analysis included 112 patients, 66 of whom had inferior AMIs (p = NS). The remainder of the patients had anterior AMIs. The mean CPK at 12 and 24 h was reduced in the HBO patients by approximately 7.5% (p = NS). Time to pain relief was shorter in the HBO group. There were 2 deaths in the control and 1 in those treated with HBO. The left ventricle ejection fraction (LVEF) on discharge was 51.7% in the HBO group as compared to 48.4% in the controls (p = NS). The LVEF of the controls was 43.4 as compared to 47.6 for those treated, approximately 10% better (no significant difference). Treatment with HBO in combination with thrombolysis appears to be feasible and safe for patients with AMI and may result in an attenuated CPK rise, more rapid resolution of pain and improved ejection fractions. More studies are needed to assess the benefits of this treatment.

Am J Cardiol. 2004 Jun 15;93(12):1533-5.

Usefulness of hyperbaric oxygen therapy to inhibit restenosis after percutaneous coronary intervention for acute myocardial infarction or unstable angina pectoris.

Sharifi M[1], Fares W, Abdel-Karim I, Koch JM, Sopko J, Adler D; Hyperbaric Oxygen Therapy in Percutaneous Coronary Interventions Investigators.

⊕ Author information

Abstract

The purpose of this trial was to assess whether the addition of hyperbaric oxygen to percutaneous coronary intervention can reduce clinical restenosis. Major adverse cardiac events at 8 months were found in only 1 of 24 patients (4%) who received hyperbaric oxygen compared with 13 of 37 patients (35%) who did not.

Randomized clinical trials to treat ACS (2)

Study	Methods	Participants	Interventions	Outcomes
Swift (1992)	RCT	34 patients (H24:10C) with clinical dx of AMI within 1 wk +abnormal WMA	Control: echo, 2 ATA, echo HBOT: 2 ATA between echoes	Improved left ventricular function on echo
Thurston (1973)	RCT; no blinding after allocation to group	208 patients (H103:C105) with strong clinical probability of AMI at adx	Control: oxygen mask HBOt: 48 hrs of oxygen at 2 ATA for 2 hrs, followed by 1 hour on air at 1 ATA	Death, significant dysrhythmias, adverse effects
Dekleva (2004)	RCT	74 patients (37:37) with AMI within 24 hrs	Control: streptokinase 1.5 mU/L HBOT: + 2 ATA for 60 ms	Enzyme changes, LVEF

Am Heart J. 1992 Nov;124(5):1151-8.

Myocardial hibernation identified by hyperbaric oxygen treatment and echocardiography in postinfarction patients: comparison with exercise thallium scintigraphy.

Swift PC[1], Turner JH, Oxer HF, O'Shea JP, Lane GK, Woollard KV.

⊕ Author information

Abstract

To evaluate the potential for hyperbaric oxygen (HBO) to produce transient improvement in function in areas of myocardium ischemic at rest (hibernating myocardium), 24 patients were studied within 1 week of acute myocardial infarction. Results were compared with single-photon emission computed tomography (SPECT) thallium-201 exercise scintigraphy. Echocardiography demonstrated improved contraction following HBO in 20 of 62 damaged left ventricular segments in 12 of 24 patients. Thirteen of the 28 segments and 9 of the 14 patients showing reversible ischemia on SPECT imaging showed improvement with HBO. There were eight segments with apparently normal resting contraction that showed a reversible thallium defect. Of 42 segments with fixed contraction abnormalities following HBO, eight had reversible thallium defects, four had normal thallium kinetics, and 30 had fixed thallium defects. Thus hyperbaric oxygen can demonstrate improvement in function in some segments of left ventricle after infarction. There is some overlap with viability as determined by thallium studies, but the two techniques may be complementary in describing myocardial ischemia.

Abstract

A randomized, controlled clinical trial was carried out to study the effect of hyperbaric oxygen (HBO) on mortality following recent acute myocardial infarction in patients aged under 70. One hundred and three patients were treated with HBO and are compared with 105 controls treated conventionally in the same coronary care unit. Seventeen of the patients treated with HBO died (16.5 per cent) compared with 24 (22.9 per cent) of the controls. When allowance is made by means of the Peel Index for the distribution of severely ill patients in the two groups this difference may be shown to be statistically significant at the 5 per cent level. The over-all mortality among the 80 patients who were exposed to a minimum of four hours in HBO was 11.3 per cent.

Detailed analysis suggests that mortality in high-risk groups may be reduced by HBO to about a half. The only three patients with cardiogenic shock to survive were in the HBO group and the effect of the therapy in certain disorders of conduction is described. The advantages and disadvantages of HBO therapy are discussed.

It is concluded that the evidence in favour of HBO treatment is sufficiently strong to justify its use in selected patients where facilities already exist and to warrant a more extensive trial.

Am Heart J. 2004 Oct;148(4):E14.

Adjunctive effect of hyperbaric oxygen treatment after thrombolysis on left ventricular function in patients with acute myocardial infarction.

Dekleva M[1], Neskovic A, Vlahovic A, Putnikovic B, Beleslin B, Ostojic M.

⊕ **Author information**

Abstract

BACKGROUND: The role of hyperbaric oxygen in patients with acute myocardial infarction is controversial, ranging from not beneficial to having a favorable effect. This randomized study was conducted to further assess the benefit of hyperbaric oxygen treatment after thrombolysis on left ventricular function and remodeling in patients with acute myocardial infarction.

METHODS: Seventy-four consecutive patients with first acute myocardial infarction were randomly assigned to treatment with hyperbaric oxygen treatment combined with streptokinase (HBO+) or streptokinase alone (HBO-).

RESULTS: There was a significant decrease of end-systolic volume index from the first day to the third week in HBO+ patients compared with HBO- patients (from 30.40 to 28.18 vs from 30.89 to 36.68 mL/m2, P <.05) accompanied with no changes of end-diastolic volume index in HBO+ compared with increased values in HBO- (from 55.68 to 55.10 vs from 55.87 to 63.82 mL/m2, P <.05). Ejection fraction significantly improved in the HBO+ group and decreased in the HBO- group of patients after 3 weeks of acute myocardial infarction (from 46.27% to 50.81% vs from 45.54% to 44.05 %, P <.05).

CONCLUSIONS: Adjunctive hyperbaric oxygen therapy after thrombolysis in acute myocardial infarction has a favorable effect on left ventricular systolic function and the remodeling process.

Conclusions

- The rationale for the use of HBOT for ACS is clear
- Both the animal and uncontrolled human data suggest there may be a window of opportunity after both the primary event and revascularization
- No reliable data from the previous studies exist to confirm or refute any effect of HBOT on mortality, length of stay, or LV contractility.

Conclusions (2)

- Risk for death and adverse effects regarding HBOT on ACS would be of great clinical importance and deserves further investigation.
- Given the activity of HBOT in modifying IR injury, combinations of HBOT and thrombolysis in the early state of ACS and the prevention of restenosis after stent placement may deserve attention to potential clinical application.

Acute Ischemic Stroke

Table 21.9 Summary of Animal Studies of Focal Cerebral Ischemia Where HBOT Was Compared with Normobaric Air or Oxygen

RIAL, ANIMAL, AND VESSEL OCCLUDED	TIME TO HBOT	OXYGEN DOSE	OUTCOME
Weinstein (1986),[102] gerbil, 20-minute bilateral CCA	0	1.5 ATA for 15 minutes once	Improved survival
ang (2002),[192a] rat, 1-hour MCA	0	2.8 ATA for 15 minutes once	Neuroprotection implied by reduced extracellular dopamine
Mrkonjic-Lollic and colleagues (2003),[74] rat, 1-hour MCA	0	3 ATA for 1 hour once	Reduced infarct volume, leukocyte infiltrate, and myeloperoxidase
Tamami (2000),[192b] rat, permanent right MCA and right CCA	10 minutes	3 ATA for 2 hours once	Reduced infarct volume
Veloso (2002),[192c] rat, permanent MCA*	10 minutes	2 ATA for 3 hours 50 minutes once	No difference in ischemic volume or myeloperoxidase
Veltkamp and colleagues (2000),[81] rat, 1-hour 15-minute MCA	15 minutes	1.5 ATA for 1 hour or 2.0 ATA for 1 hour	Reduced infarct volume and better behaviorally with 2.5 ATA HBO
Burt (1987),[192d] gerbil, permanent CCA	<30 minutes	1.5 ATA for 36 or 18 hours with long air breaks† once	Reduced chance of infarct with intermittent, shorter HBO
Chaitan (1990),[78] gerbil, permanent CCA	40 minutes	2.5 ATA for 2 or 4 hours†	Improved survival
Veltkamp (2005),[192e] rat, 2-hour MCA	40 minutes	3.0 ATA for 1 hour once	Reduced BBB permeability, smaller infarcts
Veltkamp and colleagues (2006),[82] rat, 2-hour MCA	45 minutes	3.0 ATA for 1 hour once	Reduced evidence of ischemic biochemical degradation
Barkell (1985),[192f] gerbil, permanent CCA	1 hour	1 or 1.5 ATA for up to 1 hour† once	More HBO reduced color density differences between sides
Buenther (2005),[192g] rat, permanent MCA	15 minutes to *6 hours**	2.5 ATA for 90 minutes once *or 4 times on day 1**	Early HBO reduced infarct size, *late at 6 hours and repeated HBO did not**
Ross (1998),[192h] rat, 3-90-minute MCA*	Not stated. Probably immediately after occlusion	2 ATA for 30 minutes once or daily for 4 days	No benefit
Sunabitz (2004),[79] rat, permanent MCA	2 hours	2 ATA for 1 hour once	Reduced infarct volume and deficit
Calvert (2006),[192] rat, permanent CCA*	2 hours	2.5 ATA for 2 hours once vs. NBO control	No difference in the reduction of hypoxia inducible factor
Nawamura (1990),[193] rat, 4-hour MCA	3 hours	2 ATA for 30 minutes once	Reduced infarct volume and edema
Liao (2006),[192i] rat, 90-minute MCA	3 hours	3 ATA for 1 hour once	Reduced infarct area and improved deficit
Liao and colleagues (2004),[193] rat, 90-minute MCA and *permanent MCA**	3, 6, and 12 hours	3 ATA for 1 hour once	Transient: improved outcome early, *worse outcome late** *Permanent: worse outcome**
Weinstein and colleagues (1987),[192k] cat, *6- and 24-hour** MCA	Variable up to 6 hours	1.5 ATA 40 minutes once at 6 or 24 hours†	Function improved and reduced infarct size with HBO up to 3rd hour of 6-hour occlusion, *but not 4th of 12-hour occlusion**
Badr and colleagues (2001),[187] rat, 2-hour MCA	6 hours	3 ATA for 1 hour once	Reduced biochemical evidence of ischemia
Yin (2005),[192l] rat, 2-hour MCA	6 or 24 hours	2.5 ATA 2 hours daily for 6 days	Improved outcome at both times
Yin (2002),[192c] rat, 2-hour MCA	8 hours	3 ATA for 1 hour once	Reduced infarct area
Yin (2003),[192d] rat, 2-hour MCA occlusion	8 hours	2.5 ATA for 2 hours once	Reduced infarct area, neurologic scores and apoptosis

Randomized clinical trials to treat Acute ischemic stroke

Study	Methods	Participants	Interventions	Outcomes
Anderson (1991)	RCT stratified for disease severity and blinded	39 patients with ischemic stroke within 2 wks	Control: sham 1.5 ATA for 60 ms within 6 hrs and then every 8 hrs to a total of 15 over 5 days HBOT: 100% oxygen as above	Neurologic examination at day 5, week 6, year 1; infarct volume on CT at 4 mth
Nighoghos sian (1995)	RCT with sham tx	34 patients with stroke confirmed with CT within 24 hrs suggestive of MCA occlusion and scoring less than 80 on the Orogogozo scale	Low dose heparin and supportive care Control: sham at 1.2 ATA for 40 ms for 10 dys HBOT: 1.5 ATA on the same schedule	Neurologic examonination on 3 scales: Orgogozo, Trouillas, and Rankin disability scales; adverse of HBOT
Rusyniak (2003)	RCT stratified by time to 24 hrs with allocation concealment and blinding of patients and investigators	33 patients with ischemic stroke presenting within 24 hrs of onset of neurologic deficit; all patients < the NIHSS score 23	Control: sham at 1.14 ATA for 60 mins HBOT: 2.5 ATA on the same schedule	NIHSS score at 24 hrs and 90 days; Barthel index, Rankin Scale, and GCS at 90 days; mortality; adverse effects

A pilot study of hyperbaric oxygen in the treatment of human stroke.

Anderson DC[1], Bottini AG, Jagiella WM, Westphal B, Ford S, Rockswold GL, Loewenson RB.

⊕ **Author information**

Abstract

We administered hyperbaric oxygen or air in a double-blind prospective protocol to 39 patients with ischemic cerebral infarction. We interrupted the study when we noticed what appeared to be a trend favoring the air-treated patients, whose neurological deficits were less severe (mean +/- SEM score on graded neurological examination: air, 25.6 +/- 4.9; oxygen, 34.5 +/- 7.5) and whose infarcts were smaller (air, 29.0 +/- 12.2 cm3; oxygen, 49.2 +/- 11.7 cm3) at 4 months. The trend, we decided, was probably an artifact of the randomization process. Nevertheless, we chose not to resume the trial because the treatment was difficult to administer by schedule (for various reasons the treatment protocol was broken in 15 of the 39 patients), was poorly tolerated (eight of the 39 patients refused to continue treatments), and did not produce dramatic improvement.

Stroke. 1995 Aug;26(8):1369-72.

Hyperbaric oxygen in the treatment of acute ischemic stroke. A double-blind pilot study.

Nighoghossian N[1], Trouillas P, Adeleine P, Salord F.

⊕ Author information

Abstract

BACKGROUND AND PURPOSE: The effects of hyperbaric oxygen (HBO) therapy on humans are uncertain. Our study aims first to outline the practical aspects and the safety of HBO treatment and then to evaluate the effect of HBO on long-term disability.

METHODS: Patients who experienced middle cerebral artery occlusion and were seen within 24 hours of onset were randomized to receive either active (HBO) or sham (air) treatment. The HBO patients were exposed daily to 40 minutes at 1.5 atmospheres absolute for a total of 10 dives. We used the Orgogozo scale to establish a pretreatment functional level. Changes in the Orgogozo scale score at 6 months and 1 year after therapy were used to assess the therapeutic efficacy of HBO. In addition, we used the Rankin scale and our own 10-point scale to assess long term-disability at 6 months and 1 year. Two sample t tests and 95% confidence intervals were used to compare the mean differences between the two treatment groups. Student's two-tailed test was used to compare the differences between pretherapeutic and posttherapeutic scores at 6 months and 1 year in the two treatment groups.

RESULTS: Over the 3 years of study enrollment, 34 patients were randomized, 17 to hyperbaric treatment with air and 17 to hyperbaric treatment with 100% oxygen. There was no significant difference at inclusion between groups regarding age, time from stroke onset to randomization, and Orgogozo scale scores. Neurological deterioration occurred during the first week in 4 patients in the sham group, 3 of whom died; this worsening was clearly related to the ischemic damage. Treatment was also discontinued for 3 patients in the HBO group who experienced myocardial infarction, a worsening related to the ischemic process, and claustrophobia. Therefore, 27 patients (13 in the sham group and 14 in the HBO group) completed a full course of therapy. The mean score of the HBO group was significantly better on the Orgogozo scale at 1 year ($P < .02$). However, the difference at 1 year between pretherapeutic and posttherapeutic scores was not significantly different in the two groups ($P < .16$). Moreover, no statistically significant improvement was observed in the HBO group at 6 months and 1 year according to Rankin score ($P < .78$) and our own 10-point scale ($P < .50$).

CONCLUSIONS: Although the small number of patients in each group precludes any conclusion regarding the potential deleterious effect of HBO, we did not observe the major side effects usually related to HBO. Accordingly, it can be assumed that hyperbaric oxygen might be safe. We hypothesize that HBO might improve outcome after stroke, as we detected an outcome trend favoring HBO therapy. A large randomized trial might be required to address the efficacy of this therapy.

Stroke. 2003 Feb;34(2):571-4.

Hyperbaric oxygen therapy in acute ischemic stroke: results of the Hyperbaric Oxygen in Acute Ischemic Stroke Trial Pilot Study.

Rusyniak DE[1], Kirk MA, May JD, Kao LW, Brizendine EJ, Welch JL, Cordell WH, Alonso RJ; Hyperbaric Oxygen in Acute Ischemic Stroke Trial Pilot Study.

⊕ Author information

Abstract

BACKGROUND AND PURPOSE: Hyperbaric oxygen therapy (HBO) has promise as a treatment for acute stroke. This study was conducted to evaluate the efficacy, safety, and feasibility of using HBO in acute ischemic stroke.

METHODS: We conducted a randomized, prospective, double-blind, sham-controlled pilot study of 33 patients presenting with acute ischemic stroke who did not receive thrombolytics over a 24-month period. Patients were randomized to treatment for 60 minutes in a monoplace hyperbaric chamber pressurized with 100% O2 to 2.5-atm absolute (ATA) in the HBO group or 1.14 ATA in the sham group. Primary outcomes measured included percentage of patients with improvement at 24 hours (National Institutes of Health Stroke Scale [NIHSS]) and 90 days (NIHSS, Barthel Index, modified Rankin Scale, Glasgow Outcome Scale). Secondary measurements included complications of treatment and mortality at 90 days.

RESULTS: Baseline demographics were similar in both groups. There were no differences between the groups at 24 hours ($P=0.44$). At 3 months, however, a larger percentage of the sham patients had a good outcome defined by their stroke scores compared with the HBO group (NIHSS, 80% versus 31.3%; $P=0.04$; Barthel Index, 81.8% versus 50%; $P=0.12$; modified Rankin Scale, 81.8% versus 31.3%; $P=0.02$; Glasgow Outcome Scale, 90.9% versus 37.5%; $P=0.01$) with loss of statistical significance in a intent-to-treat analysis.

CONCLUSIONS: Although our HBO protocol appears feasible and safe, it does not appear to be beneficial and may be harmful in patients with acute ischemic stroke.

Conclusions

- The animal and uncontrolled human data suggest early treatment is more likely to produce benefit and that late treatment (around 24 hrs) may be harmful.
- There is, however, no convincing evidence from RCTs that HBOT improve outcome.
- Little clinical data exist on which to base treatment recommendations.
- Given the small numbers of subjects in the trials included, we can not be certain that a benefit from HBOT has been exclude.

Sepsis

Effects of hyperbaric oxygen treatment on liver functions, oxidative status and histology in septic rats

Intensive Care Med (2005) 31:1262–1268
DOI 10.1007/s00134-005-2701-6

Induction of sepsis

Rats in the SEP, SEP+HBO, SEP+CEF and SEP+CEF+HBO groups received an intraperitoneal inoculum of 1 ml saline containing viable *E. coli* cells (2.1×10^9 cfu). *E. coli* were isolated from (2.1×10^9 cfu). A total of six HBO sessions were performed at 2 atm absolute for 90 min at 6-h intervals. CEF was administered intraperitoneally at a dose of 50 mg/kg twice daily. Animals were killed 48 h after sepsis induction. Their liver and

Experimental procedure

HBO and/or CEF treatments were started 1 h after sepsis induction. A preliminary study was performed to determine 50% survival of sepsis-induced animals, and 48 h was found to be an appropriate time point; this provided a sufficient number of animals for bio-

Table 2 Serum transaminases, ALP, protein and bilirubin levels

	ALP (U/l)	ALT (U/l)	AST (U/l)	Total protein (g/dl)	Albumin (g/dl)	Total bilirubin (mg/dl)	Direct bilirubin (mg/dl)
Control	115.5±8.7	172.0±14.1	81.4±14.0	5.74±0.13	2.88±0.08	0.28±0.04	0.13±0.02
HBO	141.6±23.7	188.8±33.4	87.3±27.5	5.35±0.19	2.78±0.10	0.23±0.03	0.10±0.02
SEP	259.4±37.8*	248.3±20.4*	180.6±18.0*	5.40±0.16	2.81±0.10	0.34±0.05	0.14±0.03
SEP+HBO	178.6±35.8*	207.1±11.1	149.2±6.5*	5.64±0.18	2.81±0.14	0.21±0.03	0.08±0.02
SEP+CEF	157.6±11.1**	250.4±26.9*	117.8±19.4**	5.03±0.18	2.36±0.15	0.29±0.07	0.10±0.04
SEP+CEF+HBO	130.3±9.4**	203.1±23.6	101.1±15.7**	5.16±0.12	2.28±0.09	0.40±0.11	0.14±0.04

*$p<0.05$ vs. control, **$p<0.05$ vs. SEP

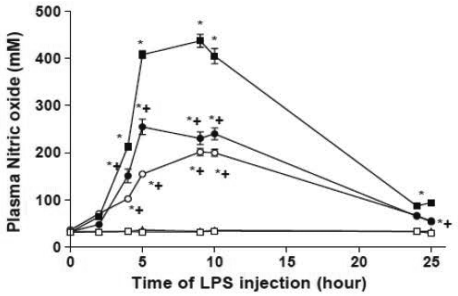
	TBARS (nmol/g protein)	SOD (μg/mg protein)	CAT (μg/g protein)
Control	4.54±0.28	2.65±0.03	25.12±2.88
HBO	4.94±0.23	4.48±0.03*	40.17±3.11*
SEP	7.21±0.71*	1.45±0.01*	12.68±1.96*
SEP+HBO	5.97±0.65*,**	2.27±0.01**	18.56±1.12*
SEP+CEF	5.35±0.51**	2.34±0.02**	30.45±2.57**
SEP+CEF+HBO	5.15±0.21**	4.57±0.03*·***	42.89±3.48*·***

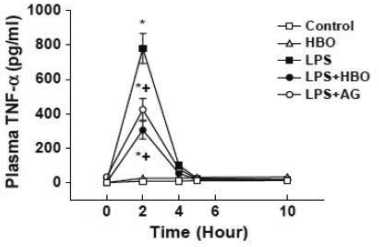
	No. before experiment	No. after experimental procedure	Survival rate (%)
Control	15	15	100
HBO	15	15	100
SEP	15	8	53.3
SEP+HBO	15	7	46.7
SEP+CEF	15	10	66.7
SEP+CEF+HBO	15	11	73.3

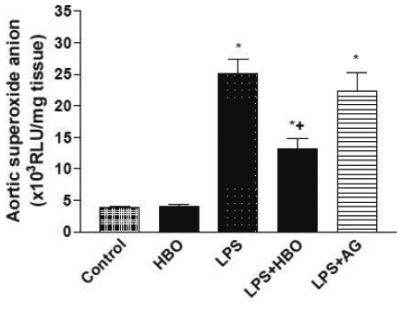
Hyperbaric oxygen protects against lipopolysaccharide-stimulated oxidative stress and mortality in rats

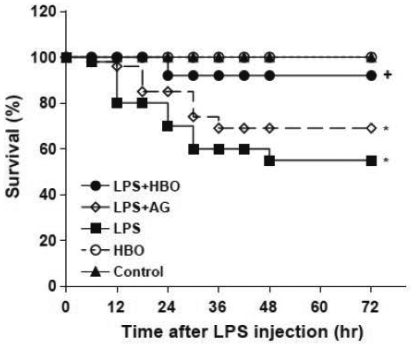
European Journal of Pharmacology 508 (2005) 249–254

Rats were exposed to hyperbaric oxygen (100% oxygen; 2 atm absolute pressure, ATA) for a 60 min compression period 1, 4, 9, and 24 h after lipopolysaccharide treatment.


Plasma Nitric oxide (mM)
Time of LPS injection (hour)


Plasma TNF-α (pg/ml)
Time (Hour)
Control
HBO
LPS
LPS+HBO
LPS+AG

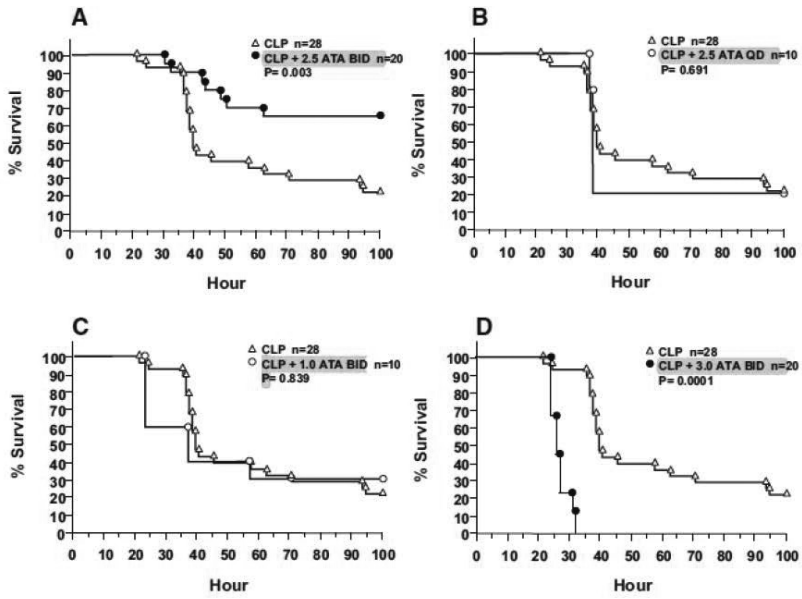

Aortic superoxide anion
(x10³RLU/mg tissue)
Control
HBO
LPS
LPS+HBO
LPS+AG


Survival (%)
Time after LPS injection (hr)
LPS+HBO
LPS+AG
LPS
HBO
Control

Hyperbaric oxygen protects from sepsis mortality via an interleukin-10–dependent mechanism*

Crit Care Med 2006 Vol. 34, No. 10

Interventions: Sepsis was induced by CLP. Mice were randomized to receive a 1.5-hr HBO_2 treatment at either 1, 2.5, or 3 atmospheres absolute every 12 hrs or HBO_2 at 2.5 atmospheres absolute every 24 hrs.



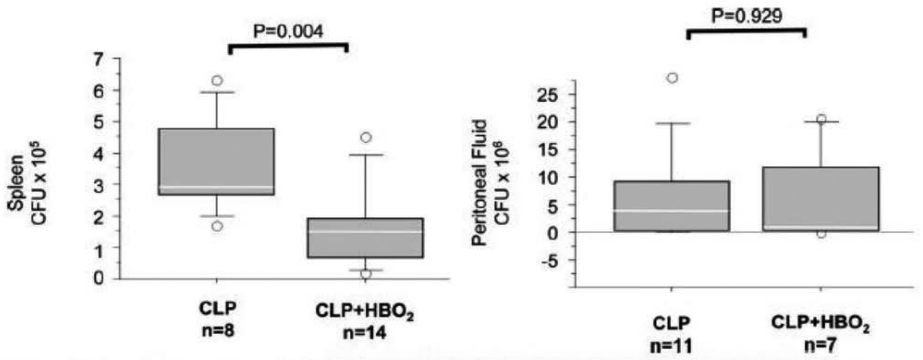


Figure 2. Hyperbaric oxygen (HBO_2) reduces splenic but not peritoneal fluid bacterial load. Mice were

Ozone Therapy and Hyperbaric Oxygen Treatment in Lung Injury in Septic Rats

International Journal of Medical Sciences
2011; 8(1):48-55

5-days of experimental period. The onset of sepsis was determined by clinical follow-up, heart rate count and rectal temperature measurements. The other 40 rats were randomly divided into four groups containing ten rats in each, sham, control, HBO, and OT groups.

All treatments were started 10 hours after *E.coli* inoculation; the sham animals had been injected physiological saline (10 ml/kg) while the control group received cefepime HCl (50 mg/kg) every 12 hours intraperitoneally (i.p.) for five consecutive days; HBO had been administered at 2.8 atm pressure with 100% O_2 inhalation for 90 minutes twice daily and OT was

Table 1. Schedule for sepsis induction and timing of treatments.

		Study groups			
Day of experiment	Treatment time	Sham	Control	HBO	Ozone
Day 0	8 a.m.	---	*E.coli*	*E.coli*	*E.coli*
	6 p.m.	Saline	Cefepime	Cefepime + HBO	Cefepime + OT
Day 1	6 a.m.	Saline	Cefepime	Cefepime + HBO	Cefepime
	6 p.m.	Saline	Cefepime	Cefepime + HBO	Cefepime + OT
Day 2	6 a.m.	Saline	Cefepime	Cefepime + HBO	Cefepime
	6 p.m.	Saline	Cefepime	Cefepime + HBO	Cefepime + OT
Day 3	6 a.m.	Saline	Cefepime	Cefepime + HBO	Cefepime
	6 p.m.	Saline	Cefepime	Cefepime + HBO	Cefepime + OT
Day 4	6 a.m.	Saline	Cefepime	Cefepime + HBO	Cefepime
	6 p.m.	Saline	Cefepime	Cefepime + HBO	Cefepime + OT
Day 5	6 a.m.	Saline	Cefepime	Cefepime + HBO	Cefepime
	4 p.m.	Sacrificing			

During the study period, all animals were survived, and no complication was seen related to in-

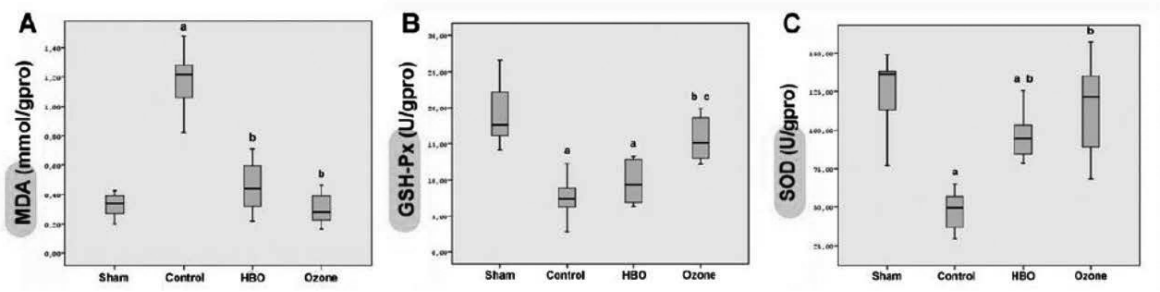
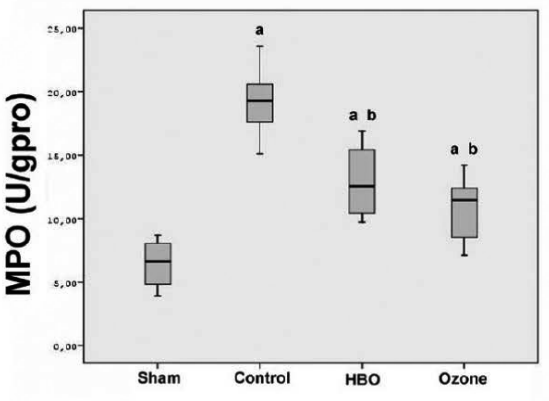


Figure 1. Oxidative stress indices in lung tissue. A: MDA levels were found to be significantly increased and antioxidant


MPO (U/gpro)
25,00
20,00
15,00
10,00
5,00
0,00
a
a b
a b
Sham
Control
HBO
Ozone

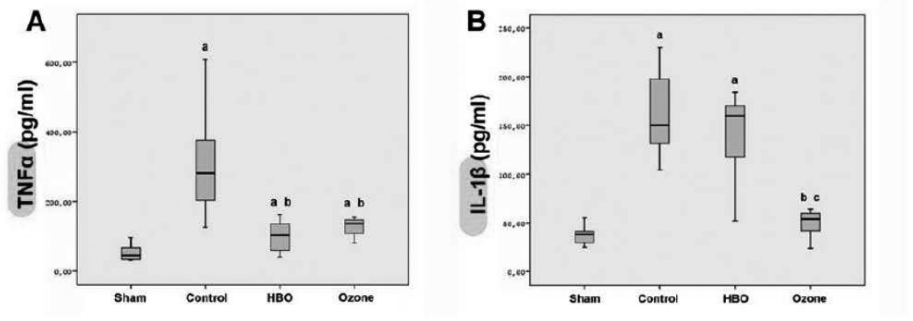

A
TNFα (pg/ml)
a
a b
a b
Sham
Control
HBO
Ozone
B
IL-1β (pg/ml)
a
a
b c
Sham
Control
HBO
Ozone

Table 2. Histologic scores of lung injury (median and range).

	Sham	Control	HBO	Ozone
Edema	0 (0-1)	2 (1-3)	1 (1-3)	1 (1-3)
Hemorrhage	0 (0-1)	3 (3-4)	3 (1-4)	1 (1-3)
Leukocyte infiltration	0 (0-1)	3 (2-4)	2 (1-3)	1 (1-3)
Septal thickening	0 (0-1)	4 (2-4)	2 (2-3)	2 (1-2)
Total injury score	0 (0-4)	12 (8-15) [a]	8 (5-13) [a,b]	5 (4-11) [a,b,c]

[a] $p<0.05$ vs sham, [b] $p<0.05$ vs control (cefepime), [c] $p<0.05$ vs HBO groups.

The effects of hyperbaric oxygen therapy on blood–brain barrier permeability in septic rats

BRAIN RESEARCH 1412 (2011) 63–72

decompression for 15 min each. A total of 4 HBOT sessions were applied at 1, 7, 13 and 19 h after CLP operation. Animals were subsequently transferred to their cages.

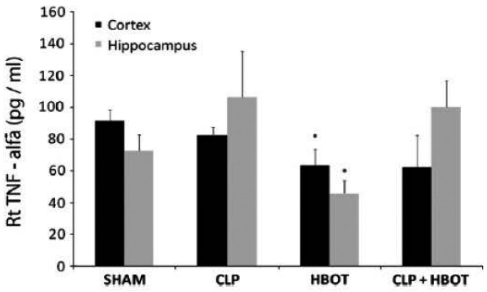
4.4. Measurement of arterial blood pressure and rectal temperature

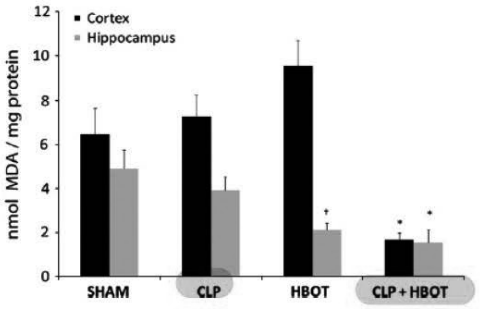
Arterial blood pressure and rectal temperature of the rats were recorded as criteria to confirm the development of sepsis findings (hypotension and fever) following CLP operation. 24 h after CLP operation, the rectal temperature of rats were

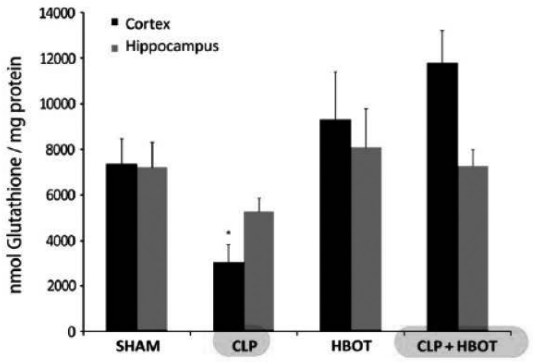
In cerebral cortex and hippocampus, TNF-α level was assessed as a marker of inflammation and MDA and GSH levels were assessed as markers of oxidative stress. Animals were sacrificed by decapitation 24 h after CLP surgery. Cerebral

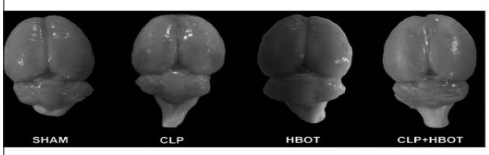
Table 1 – Physiological parameters of rats in experimental groups.

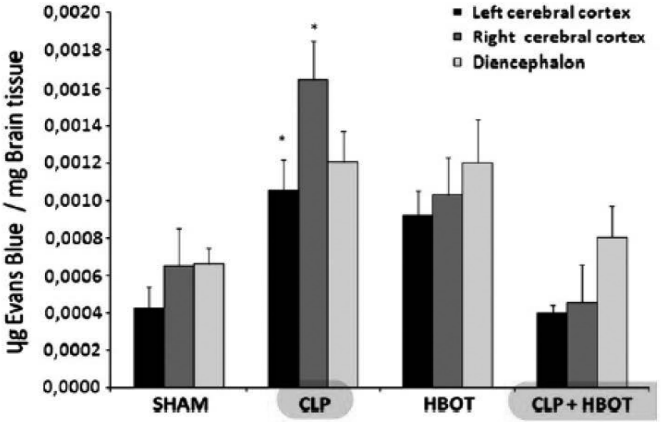
Groups	n	Mean arterial blood pressure (MABP) (mm Hg)	Rectal temperature (°C)
Sham	8	122,14± 2,40	37,93±0,31
CLP	8	95,13± 6,25 *	38,34±0,14 #
HBOT	8	128,50± 2,63 **	36,86±0,14
CLP + HBOT	8	102,75± 3,53	37,23±0,23









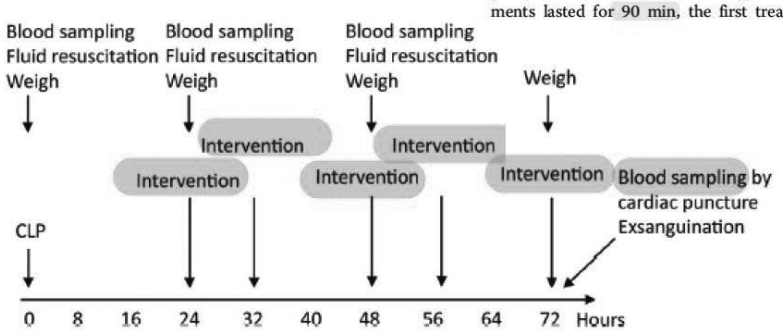


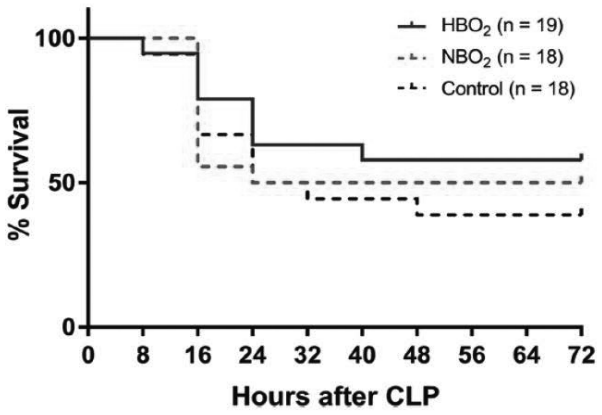
Treatment with 24 h–delayed normo- and hyperbaric oxygenation in severe sepsis induced by cecal ligation and puncture in rats

Journal of Inflammation (2017) 14:27

HBO_2 treatment has been demonstrated experimentally, but optimal timing remains uncertain. We investigated the effects of 24 h delayed normobaric oxygen (NBO_2) and HBO_2 treatment on the endogenous production of the

group allocation. All normo- or hyperbaric oxygen treatments lasted for 90 min, the first treatment being ad-





In total, there was a 42% mortality (8 died out of 19) during the observation period in rats treated with HBO_2, whereas we saw 50% mortality (9 died out of 18) in rats treated with NBO_2 group and 61% mortality in the control group (11 died out of 18). Treatment with 2.5 bar

Table 2 Differences in median TNF-α concentration

Hours	HBO_2	NBO_2	Control
0	25.7 (0.4–102.9)	11.9 (0.4–188.7)	57.2 (0.4–114.9)
24	59.4 (0.4–138.5)	33.5 (0.4–136.3)	64.0 (0.4–157.8)
48	9.5 (0.4–74.0)	0.4 (0.4–93.2)	47.0 (1.2–137.6)
72	55.8 (0.4–122.4)	62.1 (0.4–125.5)	5.1 (0.4–131.8)

Table 3 Differences in median IL-6 concentration

Hours	HBO_2	NBO_2	Control
0	2.8 (2.8–30.5)	2.8 (2.8–153.2)	33.4 (2.8–70.0)
24	161.5 (14.6–296.5)	197.9 (2.8–278.6)	144.3 (53.4–322.9)
48	2.8 (2.8–77.9)	2.8 (2.8–101.3)	2.8 (2.8–95.5)
72	5.5 (2.8–134.9)	2.8 (2.8–224.7)	2.8 (2.8–182.6)

Table 4 Differences in median IL-10 concentration

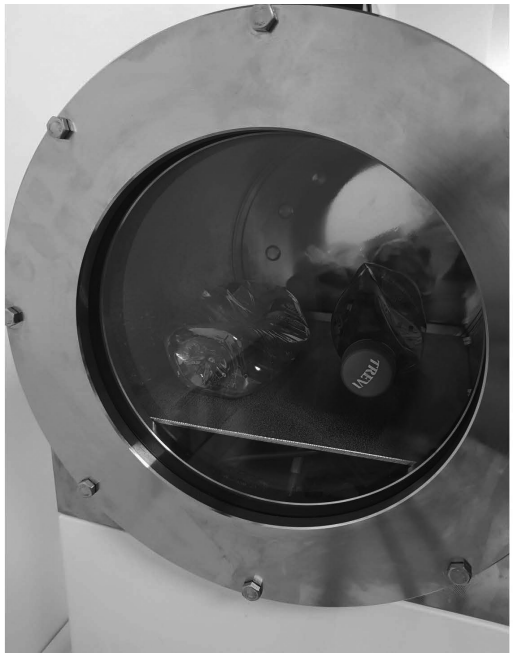
Hours	HBO_2	NBO_2	Control
0	64.7 (0.1–148.5)	0.1 (0.1–278.8)	79.4 (0.1–155.9)
24	591.0 (336.2–969.1)	405.8 (296.6–579.9)	517.2 (289.6–1138)
48	489.4 (332.7–650.1)*	337.8 (203.6–453.4)	228.6 (40.4–313.2)*
72	250.9 (99.4–330.5)	72.5 (0.1–538.5)	169.5 (585.8–0.1)

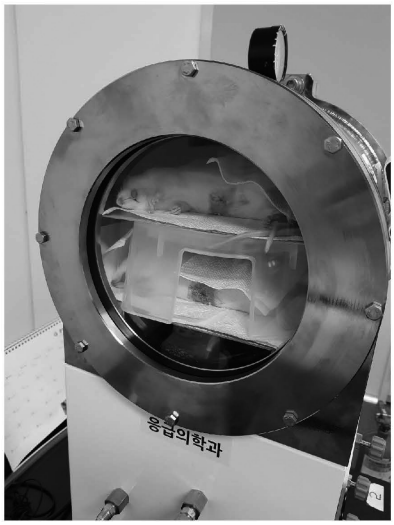
Table 5 Difference between survivors and non-survivors in median cytokine levels at hour 24

	Non-survivors	Survivors	*P*-value
TNF-α	59,37 (0.37–179.8)	37.52 (0.37–122.6)	0.31
IL-10	1050 (274.1–5318)	450.2 (342.2–627.4)	0.13
IL-6	302.3 (102.8–581.6)	107.9 (20.5–252.2)	0.04*

Conclusion

- The animal data suggest that early HBOT is more likely to show beneficial effect such as reduced mortality and that delayed treatment (after 24 hrs) not to show benefit effect.
- There is, however, no convincing evidence from human data that HBOT improve outcome.
- Also, optimal timing and frequency remain uncertain.

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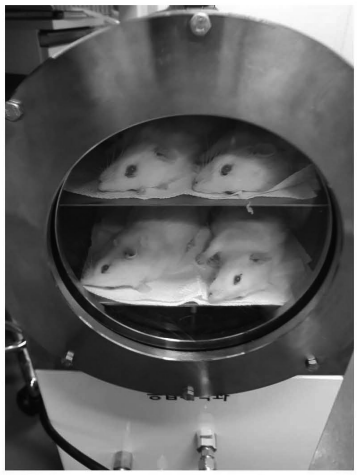


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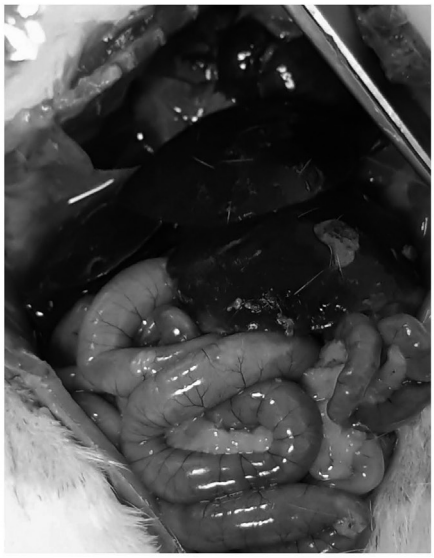
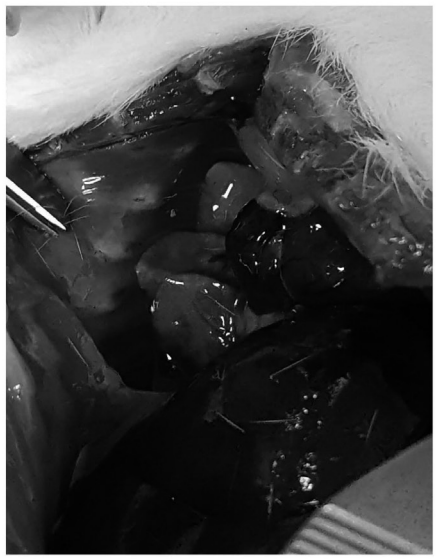




Take Home Message

- The rationale for the use of HBOT for ACS, acute ischemic strok e, and sepsis is clear.
- Little clinical data exist on which to base treatment recommendations.
- Given the small numbers of subjects in the trials included, we can not be certain that a benefit from HBOT has been exclude.

Q & A ?

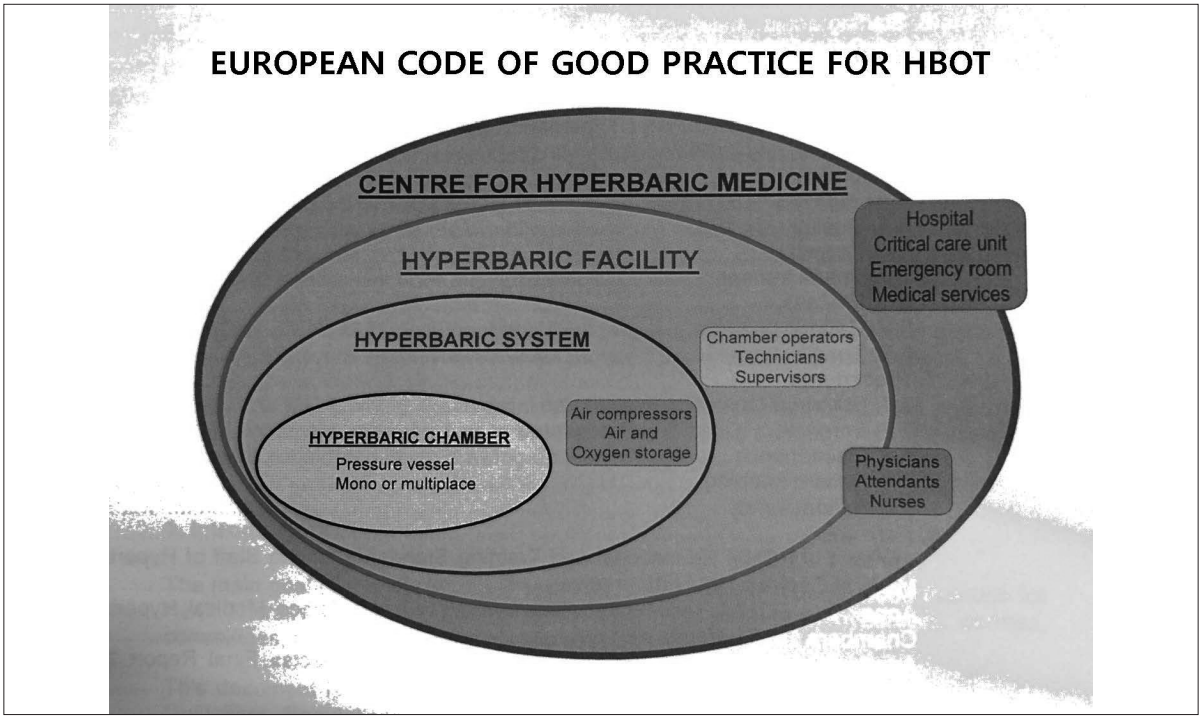
Session 2

좌장: 전남의대 응급의학과, 대한고압의학회 회장 **허 탁** 교수

고압산소치료센터 안전관리 및 인증

울산의대 응급의학과

오세현 교수


EUROPEAN CODE OF GOOD PRACTICE FOR HBOT
CENTRE FOR HYPERBARIC MEDICINE
HYPERBARIC FACILITY
HYPERBARIC SYSTEM
HYPERBARIC CHAMBER
Pressure vessel
Mono or multiplace
Air compressors
Air and
Oxygen storage
Chamber operators
Technicians
Supervisors
Hospital
Critical care unit
Emergency room
Medical services
Physicians
Attendants
Nurses


UHMS Position Statement:
Low-Pressure Fabric Hyperbaric Chambers
OXYNOVA 8
HYPERBARIC
Oxysys 4000

UHMS Position Statement: Low-Pressure Fabric Hyperbaric Chambers

• Hyperbaric oxygen therapy is defined as an intervention in which an individual breathes near 100% oxygen while [wholly enclosed] inside a hyperbaric chamber at a pressure equal to or greater than 1.4 ATA.

• Soft chamber treatments are FDA-approved for *acute mountain sickness only*.

• Exposure to treatment pressures less than 1.4 ATA while breathing air does NOT meet the definition of therapeutic hyperbaric oxygen therapy and does NOT achieve the minimum pressure and oxygen levels required for any UHMS-approved indication.

• ALL UHMS currently approved indications require that patients breath near 100% oxygen while enclosed in a chamber pressurized to a minimum of 2 ATA.

• Mild hyperbaric exposures with air deliver no more oxygen to the body than breathing oxygen by mask at sea-level pressure.

• The UHMS does not recommend the use of mild hyperbaric therapy for any medical purpose other than *acute mountain sickness*.

UHM 2018, 45(4)

UHMS Position Statement: Topical Oxygen for Chronic Wounds

• Devices, wound dressings, and topical medication designed to increase external wound exposure to oxygen should not be termed hyperbaric oxygen therapy. Doing so intentionally or unintentionally suggests that topical oxygen delivery methods are equivalent or identical to hyperbaric oxygen. Any report of methods and devices providing increased topical oxygen delivery to wounds should clearly state that topical oxygen and not hyperbaric oxygen is being delivered.

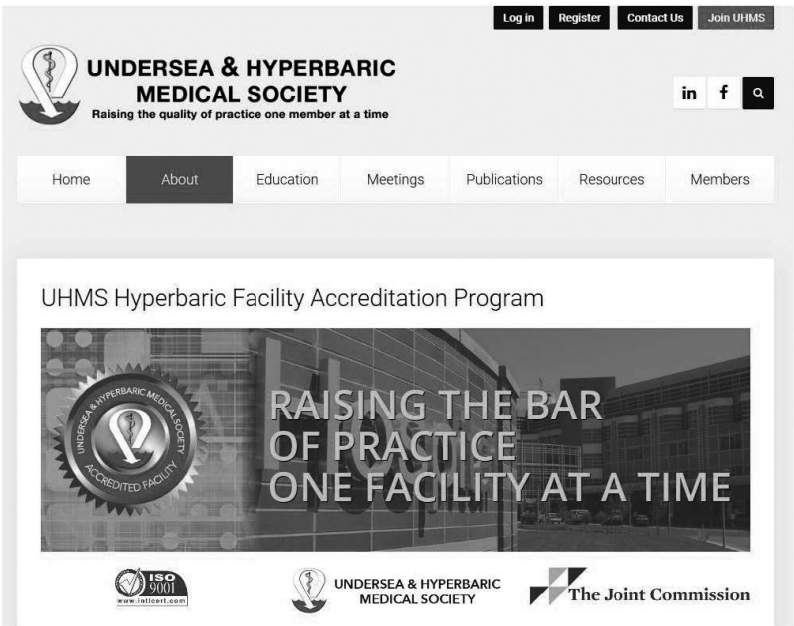
• Hyperbaric oxygen therapy provides mechanisms of action and physiological effects which are distinct from those of devices, wound dressings and topical medication that provide topical oxygen. Study results concerning hyperbaric oxygen should not be used to support topical oxygen therapy.

• Oxygen-containing and oxygen-generating wound dressings aim to address localized wound bed environments and are part of overall wound care strategies to optimize local conditions. They are a local treatment and lack the systemic effect of HBO2. These treatments are distinct from HBO2 as they lack the systemic effects of HBO2. Hyperbaric oxygen literature should not be used in support of their use and it should be clear that they are not HBO2.

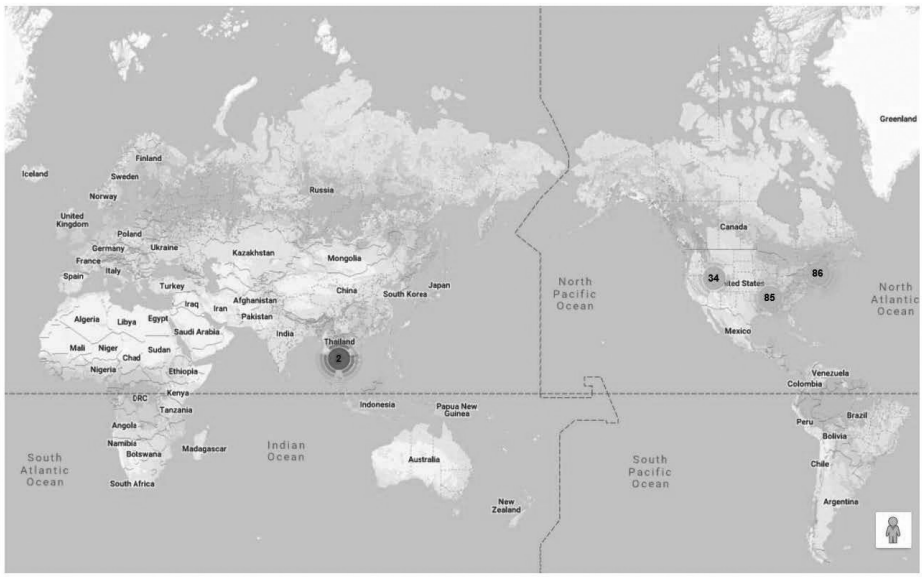
UHMS Position Statement: Topical Oxygen for Chronic Wounds

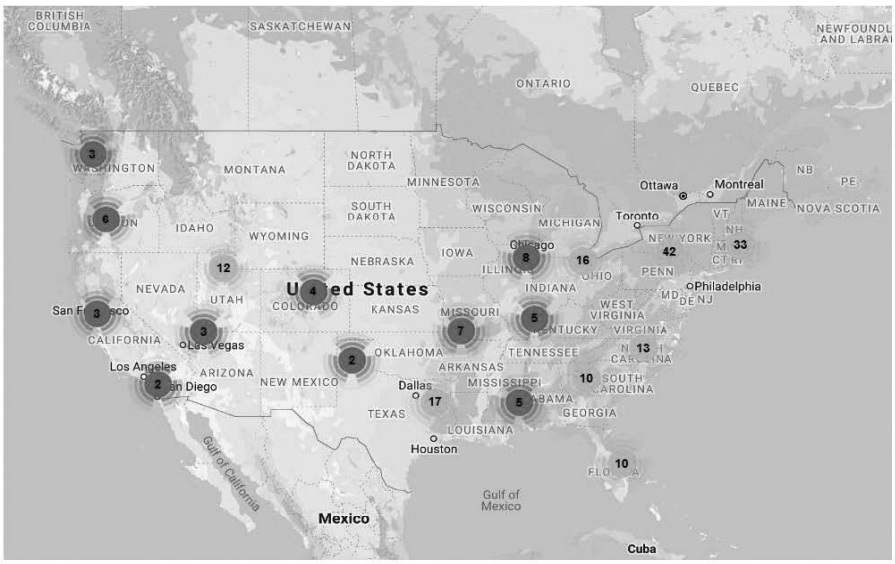
• Topical oxygen may be a promising treatment, based on some recent studies, but it cannot be recommended for routine clinical care at this time due to a restricted volume and quality of supporting scientific evidence. More investigation is necessary to determine if topical oxygen can be used in the clinical setting for wound care. In particular, we need better information on precise indications for use, optimal dosing regimens and standardized outcomes. Future clinical studies should address these issues.

• Before topical oxygen therapy can be recommended for non-healing wounds, its application should be subjected to additional scientific scrutiny to better establish indications for use, dosing and response to treatment.



UHMS Hyperbaric Facility Accreditation Program



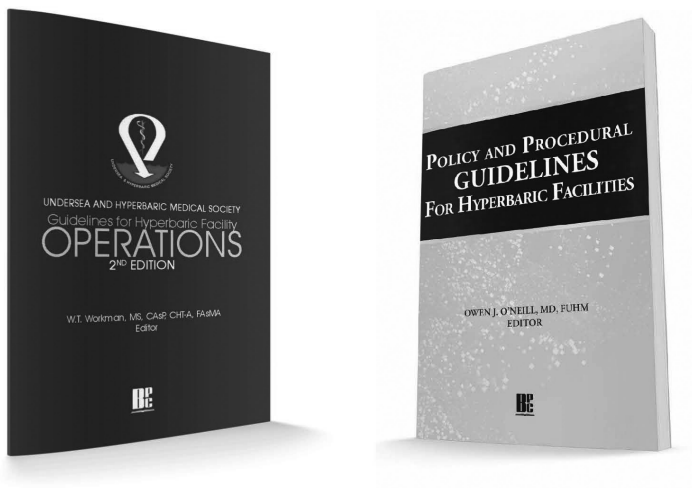


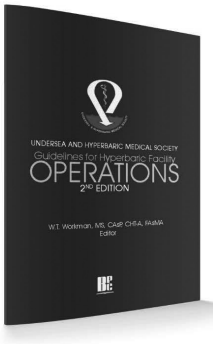
Clinical Hyperbaric Facility Accreditation Manual Fourth Edition

UHMS Accreditation Manual

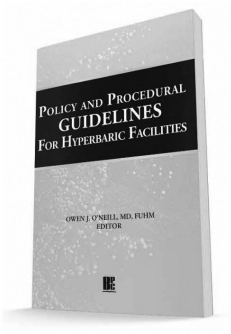
- Introduction

I. Accreditation policies and procedures
II. Standards
III. Survey rating process
IV. Document review guidance
V. Survey schedule
VI. Probe update log
VII. References





- Recommended training guidelines
  - Physician/nursing personnel
- Job descriptions and responsibilities
  - Clinical/nursing/technical/non-clinical job descriptions
- Staffing guidelines
- Safety program guidelines
- Quality assurance in clinical hyperbaric medicine



- Governance
- Administration
- Emergency procedures
- Patient care
- Hyperbaric chamber
- Quality improvement

인증(Certification, Accreditation)

- 정의: 어떠한 문서나 행위가 정당한 절차로 이루어졌다는 것을 공적 기관이 증명함
- 목표: 환자안전, 의료 질 향상을 위한 자발적이고 지속적인 노력 유도
- 절차, 과정: 시설, 인력, 장비, 운영, 질관리

필요한 과정

- Position statement
- 시설, 장비 기준 마련
- 인력 기준/교육 프로그램 양성
  - 의사
    - 세부전문의
    - 인정의
    - 인증의
  - 간호사, 응급구조사, 운영사 등

HYPERBARIC CHAMBER

HYPERBARIC SYSTEM

HYPERBARIC FACILITY

CENTER FOR HYPERBARIC MEDICINE

GangNeung Asan Hospital

고압산소치료센터

강릉아산병원
고압산소치료센터

의학적 근거에 따른 보험급여: Hard chamber vs Soft Chamber 치료 기간 및 적응증 확대

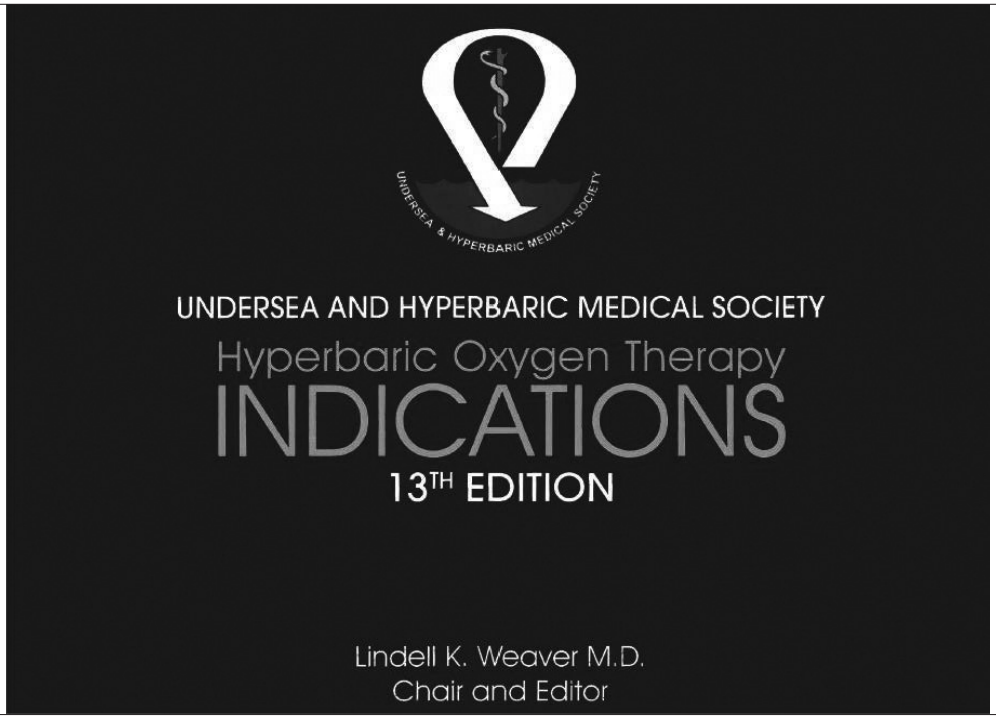
순천향의대 응급의학과

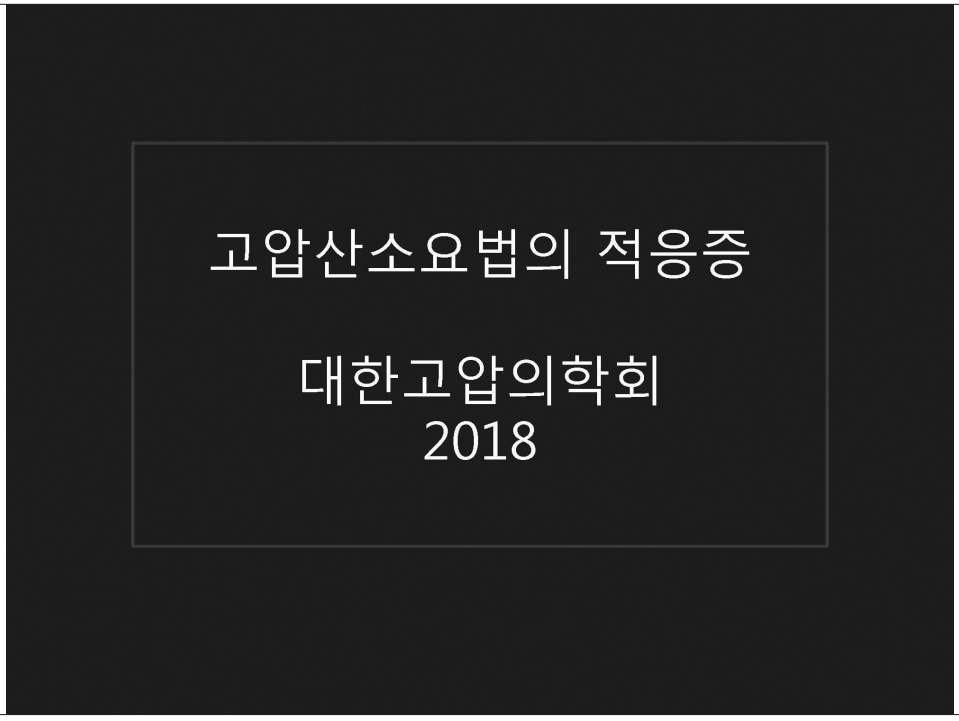
김기운 교수

Conflicts of Interest on this lecture

발표 내용은 발표자의 개인적인 의견이며,
의학적 관점에서 수정할 수 있음

None


UNDERSEA & HYPERBARIC MEDICAL SOCIETY
UNDERSEA AND HYPERBARIC MEDICAL SOCIETY
Hyperbaric Oxygen Therapy
INDICATIONS
13TH EDITION
Lindell K. Weaver M.D.
Chair and Editor


고압산소요법의 적응증
대한고압의학회
2018

현대 고압의학의 역사

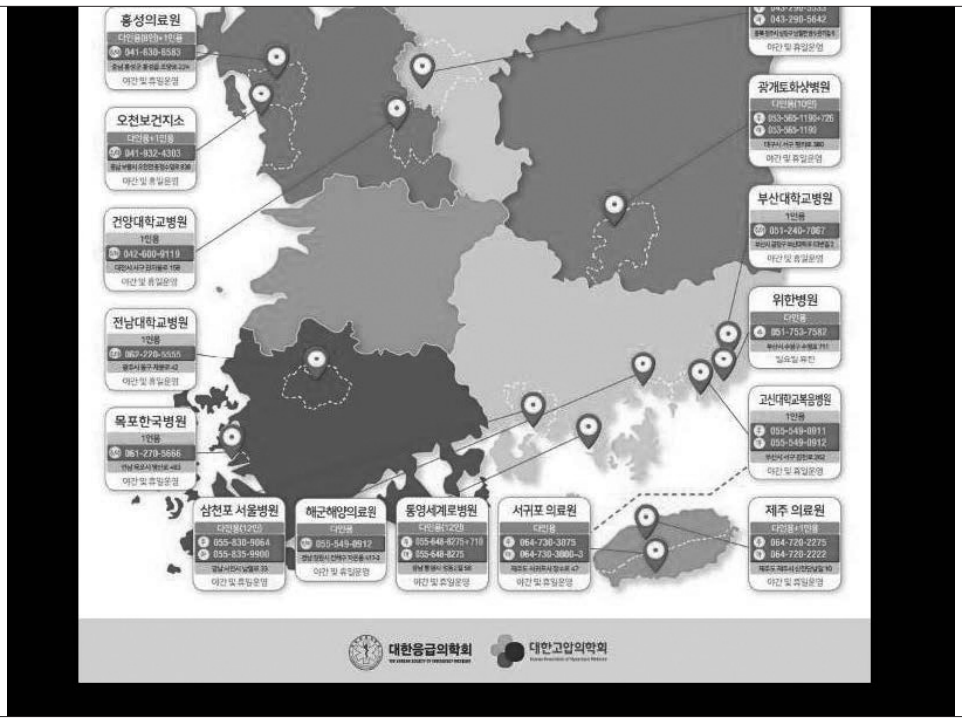
- Undersea Medical Society 설립(1967년)
  - 미해군 잠수의학 군의관 6명
  - 임상의학에서 관심을 갖고 모이기 시작함
- Undersea and Hyperbaric Medical Society 설립(1986년)
  - 의학 학술 단체

대한고압의학회

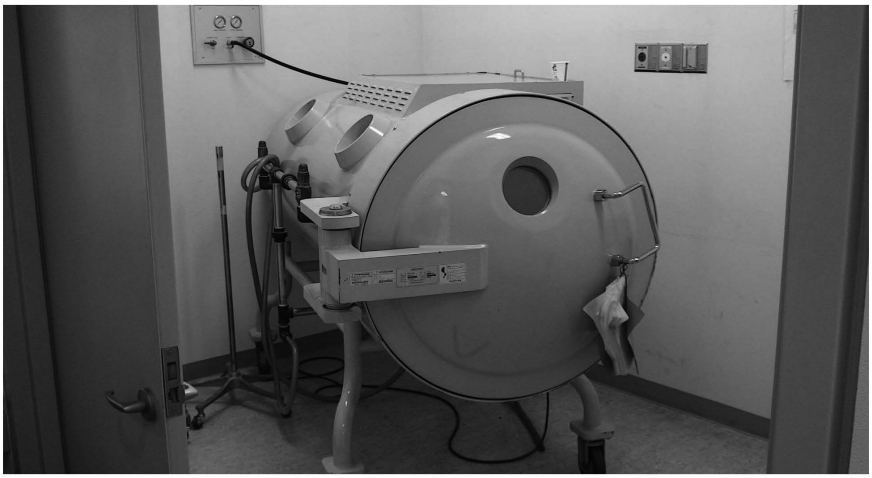
- 대한고압의학협회 창립(2013. 4. 18)
- 대한고압의학회 창립(2017. 11. 24)

첫돌을 축하합니다.

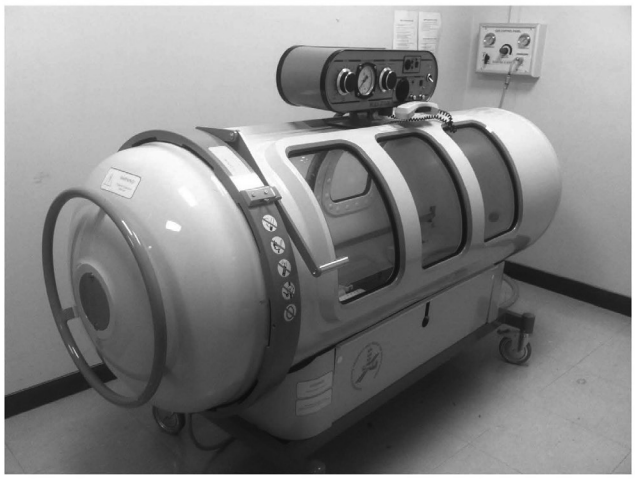

전국 고압산소챔버 운영 현황
2017년 6월 자료
다인용 / 다인용 +1인용
1인용
• 진료시 문의요망
• 공군항공우주의료원, 해군해양의료원 - 군인, 경찰만 가능
한양대학교병원
서울아산병원
한림대학교춘천성심병원
원주세브란스기독병원
순천향대학교 부천병원
인하대학교병원
홍성의료원
오천보건지소
건양대학교병원
강릉아산병원
공군항공우주의료원
광개토화상병원
부산대학교병원


홍성의료원
041-630-8583
오천보건지소
041-932-4303
건양대학교병원
042-600-9119
전남대학교병원
061-379-5555
목포한국병원
061-270-5666
삼천포 서울병원
055-830-9064
055-835-9900
해군해양의료원
055-549-8912
동영세계로병원
055-648-8275
055-648-8275
서귀포 의료원
064-730-3075
064-730-3880
제주 의료원
064-720-2275
064-720-2222
광개토화상병원
053-565-1190
053-565-1190
부산대학교병원
051-240-7067
위한병원
051-753-7582
고신대학교복음병원
055-549-0811
055-549-0912
대한응급의학회
대한고압의학회

1995'~2000'



부산대병원(2008')



1인용(Monoplace) chamber

Young baby CO intoxication

다인용 챔버(제주의료원)

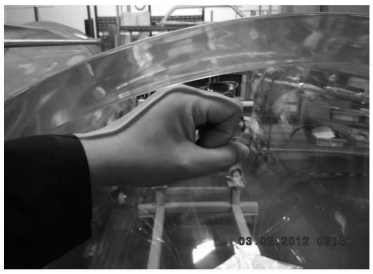
논점 1: 고압이란?

- 사전적(common sense) 정의 : 1.3 ATA 이상
- 의학적(therapeutic) 정의 : 2.0 ATA 이상

~6 ATA

~1.3 ATA

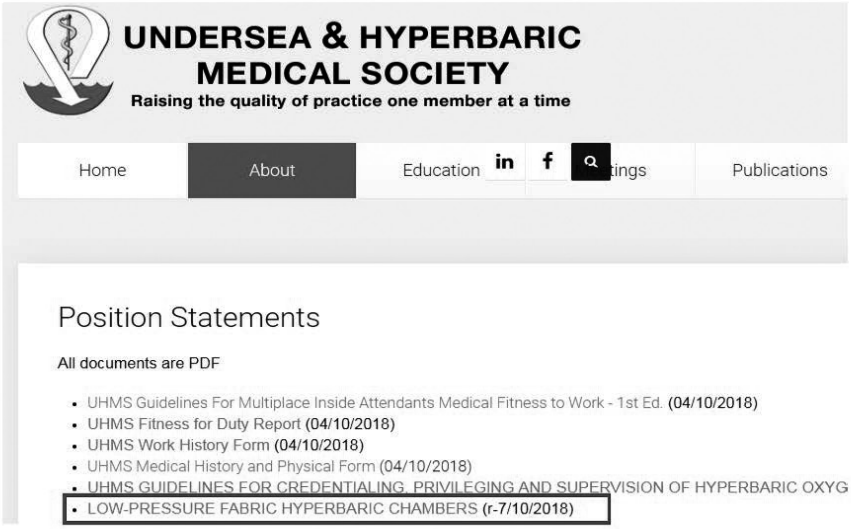
Soft vs. hard chamber



강화 PVC
철(다인용챔버)
고강도

천
플라스틱, 목재
저강도

Low pressure chamber(< 2.0ATA)



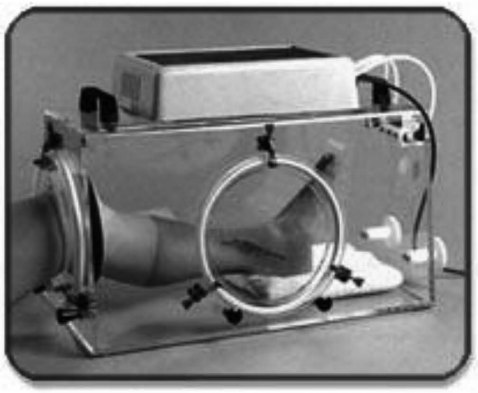
UNDERSEA & HYPERBARIC MEDICAL SOCIETY
Raising the quality of practice one member at a time

CONCLUSIONS AND RECOMMENDATIONS:

- Hyperbaric oxygen therapy is defined as an intervention in which an individual breathes near 100% oxygen while [wholly enclosed] inside a hyperbaric chamber at a pressure equal to or greater than 1.4 ATA.
- Soft chamber treatments are FDA-approved for *acute mountain sickness only*.
- Exposure to treatment pressures less than 1.4 ATA while breathing air does NOT meet the definition of therapeutic hyperbaric oxygen therapy and does NOT achieve the minimum pressure and oxygen levels required for any UHMS-approved indication.
- ALL UHMS currently approved indications require that patients breathe near 100% oxygen while enclosed in a chamber pressurized to a minimum of 2 ATA.
- Mild hyperbaric exposures with air deliver no more oxygen to the body than breathing oxygen by mask at sea-level pressure.
- The UHMS does not recommend the use of mild hyperbaric therapy for any medical purpose other than *acute mountain sickness.*

- Soft chamber 치료는 단지 고산병에만 허가(FDA)
- 1.4 이하의 압력은 치료적 고압산소요법의 압력에 도달하지 않으며, 학회 적응증에 맞지 않음
- 고압산소요법의 치료 적응증은 모두 2기압 이상임
- 경도의 고압에 노출은 해발 zero 압력에서 산소 마스크를 통해 산소를 투여하는 것과 차이가 없음

근거 미약 또는 퇴출



저압 챔버 또는 soft chamber

- 미용
- 건강 증진 또는 대체적 사용
- 만성피로, 피부 탄력 저하, 비특이적 통증…

→ 급여 외(外) 사용

저압 챔버의 급여

- 심평원 기준에 '치료적 고압'의 정의가 누락
- 기존 제품의 사용 연한을 고려하여 한시적 급여 후 '치료적 고압산소요법'을 할 수 있는 제품으로 교환 독려

- 먼저 저압챔버의 사용 빈도를 파악해야…

논점 2: 급여기준과 횟수

UHMS standard review

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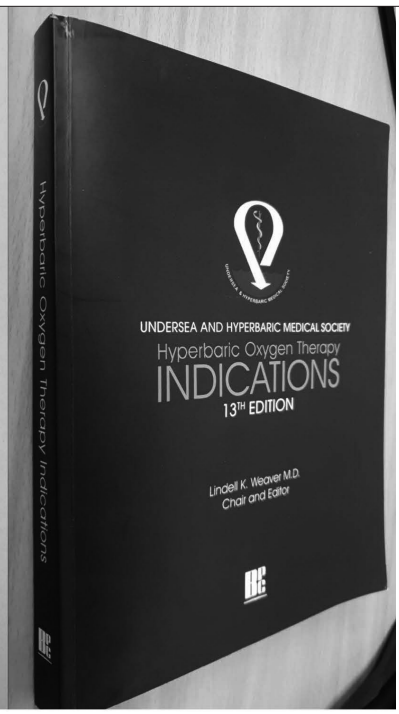

Treatment Details

적응증	Pressure	Treatment Times	Total treatment	consideration
1. Air or Gas Embolism	2.8 ATA for 90 min	2~3 times for first day and 1 / day next day		Pneumothorax treatment Hours to days gas bubbles are present→ hours to days delayed treatment is recommended
2. Carbon Monoxide Poisoning/cyanide poisoning	2.5 ATA for 90 min	2~3 times for first day and 1 / day next day	7	Over Age 36 yr, LOC(+), COHb>25%, CO exposure >24 hr, pregnant>10%
3. Clostridial Myositis and Myonecrosis (Gas Gangrene)	3.0 ATA for 90 min	3 times first day then 2 times / day	5	*Clostridium perfringens* type A 95%
4-1. Crush Injury Traumatic Ischemias	2.0 ATA for 90 min	Tid 2 days→ bid 2 days→ qd 2 days	8~12	Clinical improvement is the count of treatments
4-2. Compartment Syndrome	2.0 ATA for 90 min	Bid first day and once more day 2	3	HBOT is not a substitute for fasciotomy; use it for the impending stage of the compartment syndrome
4-3. residual problems Threatened flaps and grafts	2.0 ATA for 90 min	Bid for 7 days	14	
4-4. problem wounds/infectced wounds	2.0 ATA for 90 min	Bid for 7 days, daily for 7 days	21	
4-5. refractory osteomyelitis	2.0 ATA for 90 min	Daily for 21 days	21	Possible extension to 40 treatments
4-6. post-fasciotomy concerns, compartment syndrome	2.0 ATA for 90 min	Bid for 7 days	14	Concerns include massive swelling, threatened flaps, unclear demarcation, neuropathy
5. Decompression Sickness	2.8 ATA 90 min	Next	Next	Multichamber table
6-1. Arterial Insufficiencies: Central retinal artery occlusion	2.0 ATA 90 min→ 2.4~2.8 ATA 90 min	2~3/day	5	Time is vision, up to 2 weeks delay
6-2. Arterial Insufficiencies: diabetic foot ulcer	2.0 ATA 90 min	1 / day	20	Wagner grading system for DFU 3 or higher. Deeper with abscess, osteomyelitis, tendinitis, gangrene
6-3. Arterial Insufficiencies: hypoxic lower extremity wounds/venous stasis ulcer/pressure ulcer	2.5 ATA for 90 min	1 / day	30	
7. Severe Anemia	-	-	-	Multichamber option – 3~4 times at 2~3 ATA with air breaks for up to 3 ~ 4 hr
8. Intracranial Abscess	2.5 ATA for 90 min	2 / day	40	
9-1. Necrotizing Soft Tissue Infections	2.5 ATA for 90 min	Bid for a 4 days		
9-2. Necrotizing Soft Tissue Infections: clostridial myositis	2.5~3.0 ATA for 90 min	tid for first day, and bid until stable		More higher pressure and more times of treatments than other bacterial infection
10. Osteomyelitis (Refractory)	2~3 ATA for 90 min	Bid for first two days, and qd	20~40	Patient status and involved area- long bone, spine etc. are complicated factors

Treatment details-Cont'

11. Delayed Radiation Injury (Soft Tissue and Bony Necrosis)- mandible, larynx, chest wall, bladder, prostate, small and large intestine, extremity, CNS, optic neuritis	2.5~3.0 ATA for 90 min	Qd for 30 days	About 30	Each location has different treatment protocol by previous researches Read the manual
12. Compromised Grafts and Flaps	2~2.5 ATA for 90~120 min	Consider more times by flap status	20	As soon as signs of flap or graft compromise appear
13. Acute Thermal Burn Injury	2.4 ATA for 90 min	Tid for first 3 days and qd	30	
14. Idiopathic Sudden Sensorineural Hearing Loss	2~2.5 ATA for 90 min	qD	10~20	Moderate or worse range > 40 dB within 14 days

1. 수기료 및 재료

주 : 산소는 별도 산정한다.

	항목코드	처방명칭	수가코드	수가명칭	단가(원)*
	OXY050	O2 고압[50L/min]	GO250	고압산소[50L/분,10분단위]	200
자586-가	M0586	고압산소요법 [1시간까지]	M0586	고압산소요법 [1시간까지]	12,570
자586-나	M0587	고압산소요법 [1시간을 초과하여 2시간까지]	M0587	고압산소요법 [1시간을 초과하여 2시간까지]	30,570
자586-다	M0588	고압산소요법 [2시간 초과](1일당)	M0588	고압산소요법 [2시간 초과](1일당)	79,340

* 중별 가산 제외 금액

2. 수가산정 방법

- 고시 제2017-118호(행위), 2017. 6. 30시행 -

고압산소요법을 동일날 오전·오후로 나누어 시행할 경우에는 실 처치시간을 합산하여 자586 고압산소요법 해당 항목 소정점수를 1회만 산정하며, 각 적용증별 수가 산정방법은 다음과 같이 함.

- 다 음 -

가. 일산화탄소중독, 감압병(잠수병), 가스색전증, 혐기성세균감염증(가스괴저증), 시안화물중독증에 고압산소요법시는 자586 해당항목 소정점수 산정

나. 화상, 버거씨병, 식피술 또는 피판술 후, 수지접합수술 후, 방사선치료 후 발생한 조직괴사 등에 고압산소요법시는 처치시간 1시간 이내는 자586가 소정점수를 산정하고, 1시간 초과시는 자586가 소정점수의 200%를 산정함. 단, 통상 2주 이내로 실시함을 원칙으로 하되, 연장실시가 반드시 필요한 경우에는 사례별로 인정함.

다. 초기 청력역치 80dB 이상의 돌발성 난청환자에서 고압산소요법을 1회 60~120분이내로 실시한 경우 인정하며, 처치시간에 따라 자586 해당항목 소정점수를 산정함.

우리나라 급여 기준

- 일산화탄소중독
- 감압병(잠수병)
- 가스색전증
- 혐기성세균감염증(가스괴저증)
- 시안화물중독증
- 돌발성 난청환자(80dB 이상)
- 화상
- 버거씨병
- 식피술/피판술 후, 수지접합수술 후
- 방사선치료 후 발생한 조직괴사

한국 기준(old standard, 근거 기반 **반대**로 되어 있음)

일산화탄소중독
감압병(잠수병)
가스색전증
혐기성세균감염증(가스괴저증)
시안화물중독증
돌발성 난청환자(80dB 이상)

] 시간에 따라 횟수 제한 없음

화상
버거씨병
식피술/피판술 후, 수지접합수술 후
방사선치료 후 발생한 조직괴사

] M0586
1시간 초과- M0586 X 2
횟수 14회까지

UHMS criteria

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횟수와 시간은 근거기반으로 지침서에 정함

유럽

The Society | DHM Journal | Activities | Membership | Members area

You are here: Home › News & informations › ** ECHM 2016 Consensus Recommendations on Indic
of HBOT – OPEN ACCESS

← MEMBERS AREA SECTION ** Open Letter to A

**** ECHM 2016 Consensus Recommend
on Indications and Practice of HBOT –
ACCESS**

24/03/2017 | Filed under: News & informations and tagged with: Guidelines

Table 2
Recommendations on the indications accepted for HBOT (there was no Level A evidence)

Condition	Level of evidence B	Level of evidence C	Agreement level
Type 1			
CO poisoning	X		Strong agreement
Open fractures with crush injury	X		Strong agreement
Prevention of osteoradionecrosis after dental extraction	X		Strong agreement
Osteoradionecrosis (mandible)	X		Strong agreement
Soft tissue radionecrosis (cystitis, proctitis)	X		Strong agreement
Decompression illness		X	Strong agreement
Gas embolism		X	Strong agreement
Anaerobic or mixed bacterial infections		X	Strong agreement
Sudden deafness	X		Strong agreement
Type 2			
Diabetic foot lesions	X		Strong agreement
Femoral head necrosis	X		Strong agreement
Compromised skin grafts and musculo-cutaneous flaps		X	Strong agreement
Central retinal artery occlusion (CRAO)		X	Strong agreement
Crush Injury without fracture		X	Agreement
Osteoradionecrosis (bones other than mandible)		X	Agreement
Radio-induced lesions of soft tissues (other than cystitis and proctitis)		X	Agreement
Surgery and implant in irradiated tissue (preventive treatment)		X	Agreement
Ischaemic ulcers		X	Agreement
Refractory chronic osteomyelitis		X	Agreement
Burns, 2nd degree more than 20% BSA		X	Agreement
Pneumatosis cystoides intestinalis		X	Agreement
Neuroblastoma, stage IV		X	Agreement
Type 3			
Brain injury (acute and chronic TBI, chronic stroke, post anoxic encephalopathy) in highly selected patients		X	Agreement
Radio-induced lesions of larynx		X	Agreement
Radio-induced lesions of the CNS		X	Agreement
Post-vascular procedure reperfusion syndrome		X	Agreement
Limb replantation		X	Agreement
Selected non-healing wounds secondary to systemic processes		X	Agreement
Sickle cell disease		X	Agreement
Interstitial cystitis		X	Agreement

일본

위원회보고

고기압 산소 치료 증거 보고서 발간에 즈음

- 난치성 궤양 (당뇨, 동맥 또는 정맥 혈류 장애)
- 방사선 장해 (Delayed radiation injury)
- 방사선 또는 항암제와 병용되는 악성 종양
- 저산소 뇌증 (hypoxic encephalopathy)
- 감압 증
- 가스 색전증
- 일산화탄소 중독
- 돌발성 난청
- 급성 두부 외상
- 망막 동맥 폐색
- 장폐색
- 척수・신경 질환
- 뇌경색
- 급성 관상 동맥 증후군

진단에 따른 개선안

- 일산화탄소/시안화물 중독Carbon monoxide cyanide poisoning
- 감압병Decompression sickness
- 공기색전증Air or gas embolism
- 가스괴저증Gas gangrene
- 식피술/피판술/수지접합 후 탈락 위험Compromised skin grafts and flaps
- 화상burn
- 말초허혈증peripheral ischemias ←버거씨 병
- 지연성 방사선 손상Delayed radiation injury (soft tissue and bony necrosis)
- 돌발성난청(00 Db 이상)Idiopathic Sensorineural hearing loss
- 압착손상Crush injury 과 구획증후근Compartment syndrome
- 중증 빈혈severe anemia
- 괴사성 연부조직염Necrotizing soft tissue infections
- 당뇨족 궤양 diabetic foot ulcer 골수염Osteomyelitis
- 뇌내 농양 intracranial abscess

진단에 따른 개선안

1. 일산화탄소/시안화물 중독Carbon monoxide cyanide poisoning
2. 감압병Decompression sickness
3. 공기색전증Air or gas embolism
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14. 뇌내 농양 intracranial abscess

시행 횟수에 따른 개선안

적응증	기존 횟수	개선안	비고
일산화탄소/시안화물 중독	제한없음	7	DNS 발생시 +20
감압병	제한없음	14	신경증상 +20
공기색전증	제한없음	7	
가스괴저증	제한없음	7	
식피술/피판술/수지접합 후 탈락 위험	14	20	
화상	14	30	
말초허혈증(버거씨 병)	14	30	
지연성 방사선 손상	14	30	
돌발성난청(00 Db 이상)	제한없음	14	
압착손상과 구획증후근	신규	14	
중증 빈혈	신규	30	
괴사성 연부조직염	신규	7	
골수염, 당뇨족	신규	21	
뇌내 농양	신규	40	

DNS- delayed neurologic sequelae

요약

- 고압산소치료의 급여의 근거기반화
  - 의학적 효과에 근거한 기준 마련
  - 안전한 관리: 인력의 인증, 시설(장비)의 인증
- 국가 주도 정책 수립 필요

주관 기관(심평원 또는 보건복지부)의 상황의 인식 및 주도적 진행 필요!

고압의학 정착을 위한 정책

보건복지부 예비급여과

황호평 사무관

Oral Presentation

좌장: 인하의대 응급의학과 **백진휘** 교수

The experience for the operation of hyperbaric oxygen therapy center in a single tertiary academic hospital in Korea where hyperbaric medicine is at the beginning stage

Division of hyperbaric medicine, Department of Emergency Medicine,
Yonsei University Wonju College of Medicine, Wonju, Republic of Korea

Yoon Seop Kim, MD, Yoonsuk Lee, MD, Sung Oh Hwang, MD, PhD, Yong Sung Cha, MD*, Hyun Kim, MD, PhD*

Purpose

Hyperbaric medicine is at the beginner level in Korea when compared to other developed countries, such as the United States and Japan. Our facility has been managed by physicians with certifications for diving and clinical diseases from the Undersea and Hyperbaric Medical Society (UHMS) and National Oceanic and Atmospheric Administration form October 2016. This study was conducted to share similar issues can be encountered whenever a program is established in a new area through our experiences on the operation of the hyperbaric oxygen (HBO2) therapy center

Method

In this retrospective observational study, we collected the data of HBO2 patients treated in our center between October 2016 and June 2018 after HBO2 was operated by certified physicians. We then compared demographic data of patients with those treated during the period from January 2011 to September 2015, before HBO2 was operated by certified physicians.

Result

A total of 692 patients received 5,130 treatments. Twelve indicated diseases were treated with HBO2. Fifty-six critically ill patients with intubation received HBO2. About 16.6% of the treated patients withdrew during the treatment course without completing the recommended number of HBO2 sessions because they were discharged without completing the treatment.

Conclusion

After the establishment of the HBO2 center that is operated by physicians with certification made by a robust institute such as UHMS, more patients including critically ill patients received HBO2, which is indicated in increasing numbers of diseases. In order to improve the use of hyperbaric medicine in Korea, KAUHM and advanced and well-organized academic society should constantly communicate and aim to set Korean education programs as various course levels of UHMS and increase reimbersement of HBO2.

Severe soft tissue infection treated with antibiotics and hyperbaric oxygen therapy

인하대학교병원 응급의학과
강 수*, 김요한, 백진휘

Introduction

중증연부조직감염은 사망률과 이환율이 높으며 항생제 치료가 근간이지만 수술적 치료가 반드시 필요한 경우도 있다. 또한 효과에 대하여 논란이 있지만 고압산소치료를 병행하는 경우도 있다. 우리는 항생제와 고압산소치료로 성공적인 치료를 한 중증연부조직감염 1례를 보고하는 바이다.

Case presentation

특이 병력 없는 20세 여자 환자로 응급실 내원 전일 스킨 스쿠버 중 왼쪽 발등을 산호더미로 추정되는 부분에 부딪힌 후 발생한 발등의 검붉은 색 발진 및 부종을 주소로 내원하여 봉와직염 의증하에 cefazoline 정맥주사 후 귀가하였다. 환자는 익일 괴사성 근막염 의증으로 감염내과 입원하여 MRI 및 항생제 정맥 주사 투여하였다. tibia & foot MRI에서 비과사성근막염을 동반한 봉와직염 및 화농성근육염의 소견을 보이고 있었으며 3일간의 항생제 치료 시행 후 부종은 호전을 보이나 순환감소에 의한 피부변색 및 통증 소견보여 입원 3병일부터 3일간 총 5회의 고압산소치료 진행하였다. 고압산소치료 시행 후 통증 및 피부변색 소견 호전보여 항생제 치료 유지 후 입원 13병일에 퇴원하였다.

Conclusion

고압산소치료는 중증연부조직감염 환자의 생존율 증가 및 병변의 회복에 연관이 있을 것으로 사료된다. 따라서 중증연부조직감염의 병행치료로 고압산소치료를 고려해야 한다.

The experience of hyperbaric oxygen therapy in delayed radiation cystitis in a single tertiary academic hospital

Division of hyperbaric medicine, Department of Emergency Medicine,
Yonsei University Wonju College of Medicine, Wonju, Republic of Korea
Yoonsuk Lee, Tae Hyeon Kwon, Hyun Kim, Yong Sung Cha

PURPOSE

A delayed radiation injury is typically seen after a latent period of six months or more from a radiation exposure. It may occasionally develop many years after the exposure. Delayed radiation induced cystitis (DRC) is not a common complication but can be very difficult to manage when it occurs. Hyperbaric oxygen therapy (HBOT) has been applied as an adjunct therapy for delayed radiation injury for more than 30 years, clinically. We reported our experience of HBOT in the DRC.

METHODS

From November 2016 to December 2017, 10 patients with the DRC were treated with HBOT at WSCH. Those patients were primary managed by the urologist, with drugs or an endoscopy, but failed. The protocol of HBOT is one treatment per day at a pressure of 2.0 atmospheres absolute for 100 minutes for at least total 30 sessions. Data were collected from a retrospective review of medical records.

RESULTS

Among ten patients with a hematuria due to DRC, a hematuria was stopped in four cases, and three reported fewer bleeding episodes. Four patients reported a frequency, and all of them were relieved from a frequency. Seven patients completed the planned course of therapy and other 3 patients withdrew the treatment by personal reasons, and showed no responses. Overall 70% of patients had a partial to good response.

CONCLUSIONS

Series of the radiation induced cystitis cases, that the primary managements had failed, showed a good overall response rate to hyperbaric oxygen therapy. Hyperbaric oxygen therapy is generally well tolerated to patients. This review emphasizes the importance of selecting HBOT as a treatment option for this common and difficult disease.

Poster Presentation

좌장: 전남의대 응급의학과 **이성민** 교수

고압산소치료 후 발생한 폐부종 2례

인하대학교병원 응급의학과
박진수*, 신승렬, 백진휘

Introduction

일산화탄소 중독 환자에 고압산소치료의 이득에 대해서 여러 의견들이 있으나, 급성기 및 만성기 심장, 뇌, 폐, 신장, 근육 등 주요 장기 손상의 진행 억제를 위해 전세계 많은 의료기관에서 일산화탄소 중독 환자에게 고압산소치료가 추천되어 시행되고 있다. 25% 이상(임산부의 경우 20% 이상)의 일산화탄소 수치, 의식소실, 심한 대사성 산증(pH〈7.1), 종말기관 허혈의 증거(심전도 변화, 흉통, 의식변화)가 있는 경우 고압산소치료의 적응증이 된다. 고압산소치료 시 발현 가능한 합병증으로는 피로, 폐손상 중이 손상, 비강손상, 시력손상, 산소중독에 의한 폐부전, 폐부종, 경련 등이 있다. 저자들은 고압산소치료 후 발생한 폐부종 2례를 경험하였기에 보고하는 바이다.

Case presentation

Case #1.

기저질환으로 고혈압, 고지혈증, 우울증을 앓고 있는 74세 여자가 연탄 때우는 집의 밀폐된 공간에서 의식없는 모습으로 보호자들에게 발견되어 119통해 본원 응급실 내원하였다. 의식 상태는 혼수로 Glasgow comma scale 3점이었고, 내원시 혈압 133/72mmHg, 심박수 98회/분, 체온 37.8℃, 호흡수 22회/분으로 기도 유지 위해 기관 삽관 시행하였다. COHb 26.9%, pH 7.43, pCO2 22.0mmHg, pO2 82.3mmHg, HCO3 14mmol/L 확인되었고, lactic acid 8.30 mmol/L, CK 194IU/L, Troponin-I 0.480ng/ml로 상승되어 있었다. 심전도는 심박수 138회/분의 동성빈맥이었으며, 뇌 CT에서는 대뇌 피질에 허혈성 변화 외 급성 손상 소견은 보이지 않았다. 기관삽관 후 시행한 흉부 X-ray에서는 우상엽의 부분 무기폐 외 급성 폐병변은 보이지 않았다.

자발호흡 유지 확인된 3일 후 T-piece로 변경하여 고압산소치료를 시도하였다. 고압산소치료 시도 중 산소포화도 감소 확인되어 치료 중단하였고, 이후 시행한 흉부 X-ray에서 폐부종 소견이 확인되었다. 이후 6일간 고압산소치료 재시도 하였으나, 산소포화도 감소 및 폐부종 호전 악화 반복되어 7일 만에 중단하였다. 이후 저산소성 뇌손상 진행되어 기관절개 시행하였고, 재원 20일 만에 요양병원으로 전원되었다.

Case #2

기저질환으로 고혈압 외 특이질환 없는 80세 남자가 집 창문 너머로 움직임 없는 모습 이웃에 발견되어 119 통해 본원 응급실 내원하였다. 초기 의식은 기면상태였고, 묻는 말에 고개 끄덕이나 대답하지 못하는 운동 실어증 보였으며, 운동단계는 3/3/4/4확인되었다. 내원시 혈압 134/57mmHg, 심박수 94회/분, 체온 36.2℃, 호흡수 18회/분이었으며, 의식저하 원인 확인 위해 시행한 뇌 CT, 뇌 MRI에서는 양측 대뇌피질의 허혈성 변화 외 급성 병변 보이지 않았다. 의식저하의 다른 원인 확인 위해 시행한 응급약독물 검사에서 진통제 성분 외 검출되지 않았고, ABGA+CO-Oximeter에서 CO Hb 33.3%, pH 7.42, pCO2 34.8mmHg, pO2 92.1mmHg, HCO3 22mmol/L 확인되었다. 초기 흉부 X-ray에서는 급성 폐병변은 보이지 않았으며(Fig.3), 일산화탄소 중독 치료 위해 응급의학과 입원하였다. 1차 고압산소치료는 무리없이 진행하였으나, 2차 진행 중 가슴 답답함 호소하여 1시간 만에 중단, 3차 진행 중 심각한 호흡곤란 및 빈호흡 지속되어 기관 삽관 시행하였다. 이후 시행한 흉부 x-ray(Fig.4), 흉부 CT에서 급성 폐부종 소견 보여 6일 간 인공호흡기 치료 후 폐상태 호전, 의식 명료하여 기관 삽관 제거 후 다시 고압산소치료 시도하였다. 이후 4일 간 고압산소치료 재시도하였으나, 시도 할 때마다 가슴 답답함 호소하여 중단하였다.
이후 호흡기 증상 호전되어 재원 15일 만에 퇴원하였다.

Conclusion

두 증례를 통해 고압산소치료의 이득과 해에 대해 환자들마다 개별적인 평가가 이루어져야 함을 알게 되었고, 특히 고령 환자들의 경우 폐부종 발생의 위험성을 고려 및 주의해야한다.

Case report: successful healing after hyperbaric oxygen therapy in the osteoradionecrosis of jaw

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Yonsei University Wonju College of Medicine, Wonju, Republic of Korea
Joo Hong Han, Yoonsuk Lee, Yong sung Cha, Hyun Kim

Introduction

A delayed radiation injury is typically seen after a latent period of six months or more and may occasionally develop many years after the radiation exposure. Osteoradionecrosis (ORN) of the jaw is an infrequent yet potentially devastating complication of the head and neck radiation therapy. Hyperbaric oxygen therapy (HBOT) has been applied as a therapy for delayed radiation injury for more than 30 years, and the most widely applied and most extensively documented indication for hyperbaric oxygen in chronic radiation injury is its application on a radiation necrosis of the mandible. We reported one successfully treated case of ORN by early administration of HBO for the first time in Korea.

Case report

Sixty-four years old female had been treated with radiation therapy for parotid cancer 30 years ago. She was referred to HBOT center because she had not recovered her gums and bones after tooth extraction which was done 6 months ago. She was treated with HBOT (2 atmospheres absolute for 100 minutes every day) at HBOT center of Wonju Severance Christian Hospital from April 2017 to May 2017 (total 30 sessions). After HBOT, the ORN was treated successfully.

Discussion

Robert Marx, DDS elucidated many basic principles in the etiology and management of mandibular

ORN that have led to a rational approach to its management. He has demonstrated that infection is not the primary etiology of mandibular necrosis. Osteoradionecrosis is the result of an avascular, aseptic necrosis. Marx has also shown that for hyperbaric oxygen to be consistently successful. HBO2 induces neovascularization in hypoxic tissues. It has been demonstrated the enhanced vascularity and cellularity in heavily irradiated tissues after hyperbaric oxygen therapy by comparing histologic specimens from patient, pre- and post-hyperbaric oxygen. Feldmeier and his colleagues have shown that hyperbaric oxygen given seven weeks after radiation can reduce the degree and mechanical effects of fibrosis by being applied prior to the manifestation of radiation injury. The hyperbaric study group headed up by Dr. Thom had published studies demonstrating the mobilization of stem cells mediated through nitric oxide with HBO2.

Conclusion

HBOT is a successful treatment in the osteoradionecrosis of jaw. The impact of hyperbaric oxygen in terms of its beneficial effects is likely to involve all three of these mechanisms in irradiated tissues. HBO2 stimulates angiogenesis and secondarily improves tissue oxygenation. It reduces fibrosis and it mobilizes and induces an increase of stem cells within irradiated tissues.

The experience of treating iatrogenic arterial gas embolism with the hyperbaric oxygen therapy at a tertiary academic hospital

Division of hyperbaric medicine, Department of Emergency Medicine,
Yonsei University Wonju College of Medicine, Wonju, Republic of Korea
Joo Hong Han, Yoonsuk Lee, Yong Sung Cha, Hyun Kim

Introduction

Arterial gas embolism (AGE) is an arterial vessel blockage caused by one or more bubbles of air or other gas in the circulatory system. The symptoms and signs of AGE include loss of consciousness, seizure, focal neurologic deficits, cardiac arrhythmias, or ischemia. The primary treatment of AGE is hyperbaric oxygen therapy (HBOT).

Material & Method

From October 2016 to June 2018, the center treatment log was reviewed retrospectively. Three AGE cases were developed during central venous catheter removal or disconnection that was successfully treated at a hyperbaric oxygen therapy center of Wonju Severance Christian Hospital. Two patients suffered from mental change and shock (impending arrest), and one patient experienced mental change, shock, and status epilepticus, respectively.

Result

All patients were treated with HBOT and successfully recovered. HBOT is not routinely administered with the air embolism patient, including venous gas embolism, however HBOT is well known as a useful treatment in severe AGE cases. Although it's better to apply HBOT as soon as possible, but the benefit has been reported even with the delayed HBOT for up to 30 hours.

HBOT provides oxygen at pressures higher than the atmospheric pressure and at 100 percent concentration such that a “supra” physiologic level of systemic hyperoxia can be achieved. This degree of hyperoxia allows enormous gradients for mostly nitrogen to be displaced from inside the air bubble, which in turn, reduces air bubble size and the degree of mechanical obstruction to end arterial blood flow.

Conclusion

Currently, the HBOT center is limitary available in Korea and only few are specialized in treating critical patients with HBOT. Thus, the benefits of HBOT should be considered with the risk of transporting an unstable patient to the HBOT center.

국내 일개 고압산소치료센터에서의 치료 도중 발생한 산소독성에 의한 경련 환자

연세대학교 원주세브란스기독병원 응급의학과
이윤석, 차용성, 김 현

Background

고압산소치료는 국내에서는 환자와 의료진 모두에게 아직은 익숙하지 않은 치료이다. 국내에서는 급성 일산화탄소 중독과 잠수병이 대표적인 적응 질환이지만 점차 많은 질환에 확장적용되고 있다. 고압산소치료가 활성화 되어있는 미국, 일본, 유럽에서는 국내보다 많은 질환에 활발히 사용하고 있으며 치료 시 발생할 수 있는 합병증에 대비하여 고압의학에 전문 교육을 받은 인력을 치료시 반드시 상주하도록 한다. 이에 우리는 고압산소치료센터에서 발생한 산소독성에 의한 경련 환자 사례를 고찰하고 고압산소치료시 환자 감시와 전문 의료진의 중요성을 제시하려 한다.

Method

2016년 10월부터 2018년 6월까지 원주세브란스기독병원 고압산소치료센터의 치료일지와 운영일지를 후향적으로 검토하였다. 이 기간 중 발생한 위험한 합병증인 경련 증례를 분석한다.

Result

연구 기간 중 총 5182회의 환자 치료(월 평균 34명의 신환)가 있었고 치료과정에서 2(0.3%)사례의 경련이 확인되었다. 첫 번째 환자는 만성 알코올 복용 과거력의 49세 남환으로 자살을 목적으로 번개탄을 연소하여 발생한 일산화탄소중독을 주소로 내원하여 고압산소치료를 시행하였고 4번째 치료에서 압력이 2.8 atmosphere absolute (ATA)로 올라가자 마자 아무런 전구증상 없이 경련이 발생하였고 치료받는 환자들을 감시하던 전문 응급구조사와 가스전문가에 의해 마스크로 공급되던 산소가 중단된 후 경련은 멈추었고 다른 후유증은 확인 되지 않았다. 두 번째 환자는 매일 소주 1–2병을 마시는 과거력의 50세 남환이며 화재에 의한 일산화탄소중독환자로 고압산소치료를

시행하였고 5번째 치료에서 압력이 2.8 ATA로 올라가고 30분후 경련 발생하였다. 환자를 감시하던 의사에 의하여 산소가 중단되었고 그 후에도 경련 지속되어 챔버 안에 있던 전문 응급구조사가 기도확보를 실시하였고 그 후 경련 멈추었고, 특별한 후유증 없이 의식 회복되어 퇴원하였다.

Conclusion

이전 연구들에서 보고된 바에 의하면 고압산소치료 도중 경련의 발생은 5000건의 치료중 1명의 확률로 발생한다고 보고하였다. 국내 일개 3차대학병원 고압산소치료센터에서는 개소식 이후 21개월의 기간 중 총 5182회의 치료 중 2건이 확인되었다. 고압산소치료가 점점 늘어나는 국내 상황에서 고압의학에 대한 전문적인 지식을 가지고 응급상황에 대처 할 수 있는 전문의료인력은 필수적이다.

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