HBOT Indications

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Now, first things first !

I have got no conflict of interest to declare.

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Low Pressure Chamber

Low pressure chamber(< 2.0ATA)

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Position Statements

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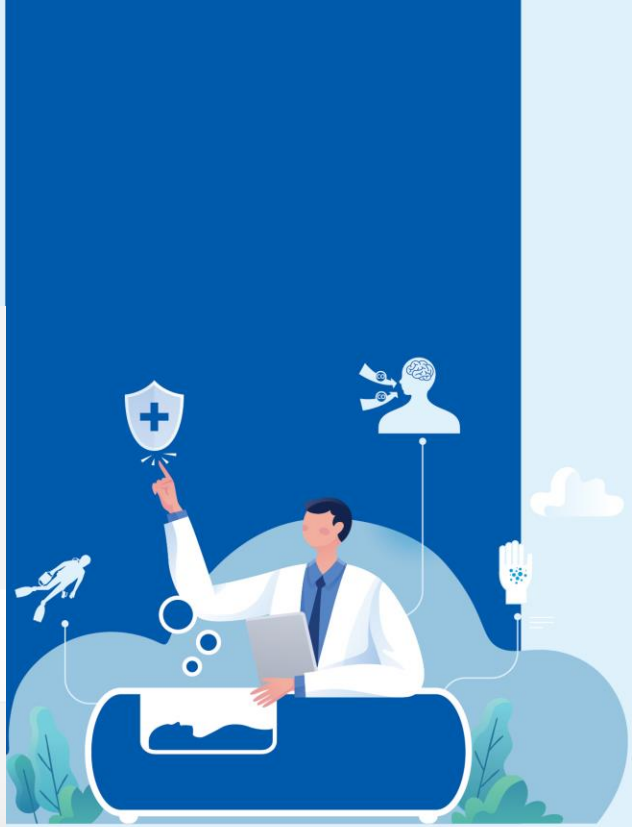
- UHMS Guidelines For Multiplace Inside Attendants Medical Fitness to Work - 1st Ed. (04/10/2018)
- UHMS Fitness for Duty Report (04/10/2018)
- UHMS Work History Form (04/10/2018)
- UHMS Medical History and Physical Form (04/10/2018)
- UHMS GUIDELINES FOR CREDENTIALING, PRIVILEGING AND SUPERVISION OF HYPERBARIC OXYG
- LOW-PRESSURE FABRIC HYPERBARIC CHAMBERS (r-7/10/2018)



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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

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저압 챔버 또는 soft chamber

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

→ 급여 외(外) 사용

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근거 미약 또는 퇴출

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Standard Indications

Indications for HBOT (US, UHMS)

Diving	Medical	Surgical	Infection
• Arterial gas embolism • Decompression sickness	• Acute blood loss anemia • Diabetic foot infection • CO/CN poisoning • Acute sensorineural hearing loss • Delayed radiation injury	• Acute peripheral ischemia • Burns • Crush injury & compartment syndrome • Gas gangrene • Necrotizing soft tissue infections • Central retinal artery occlusion • Preservation of flaps & grafts	• Actinomycosis bone infection • Cerebral abscess • Refractory osteomyelitis • Selected problem wound

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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	

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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

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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

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국내 적응증 (급여 기준)

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

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치료 회수 및 기간 (보험 기준)

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

시간에 따라
회수 제한 없음

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

M0586
1시간 초과- M0586 X 2
회수 14회까지

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고압산소치료법의 적응증 확대

잠수병 등에 필수적인 고압산소요법의
적응증도 당뇨성 족부궤양 등으로 대폭 확대하고 고압산소챔버 등 시설·
장비의 활용도도 높인다.

고압산소요법을 쓸 수 있는 질환은 당뇨성 족부궤양, 난치성
골수염, 머리 농양 등으로 확대된다.

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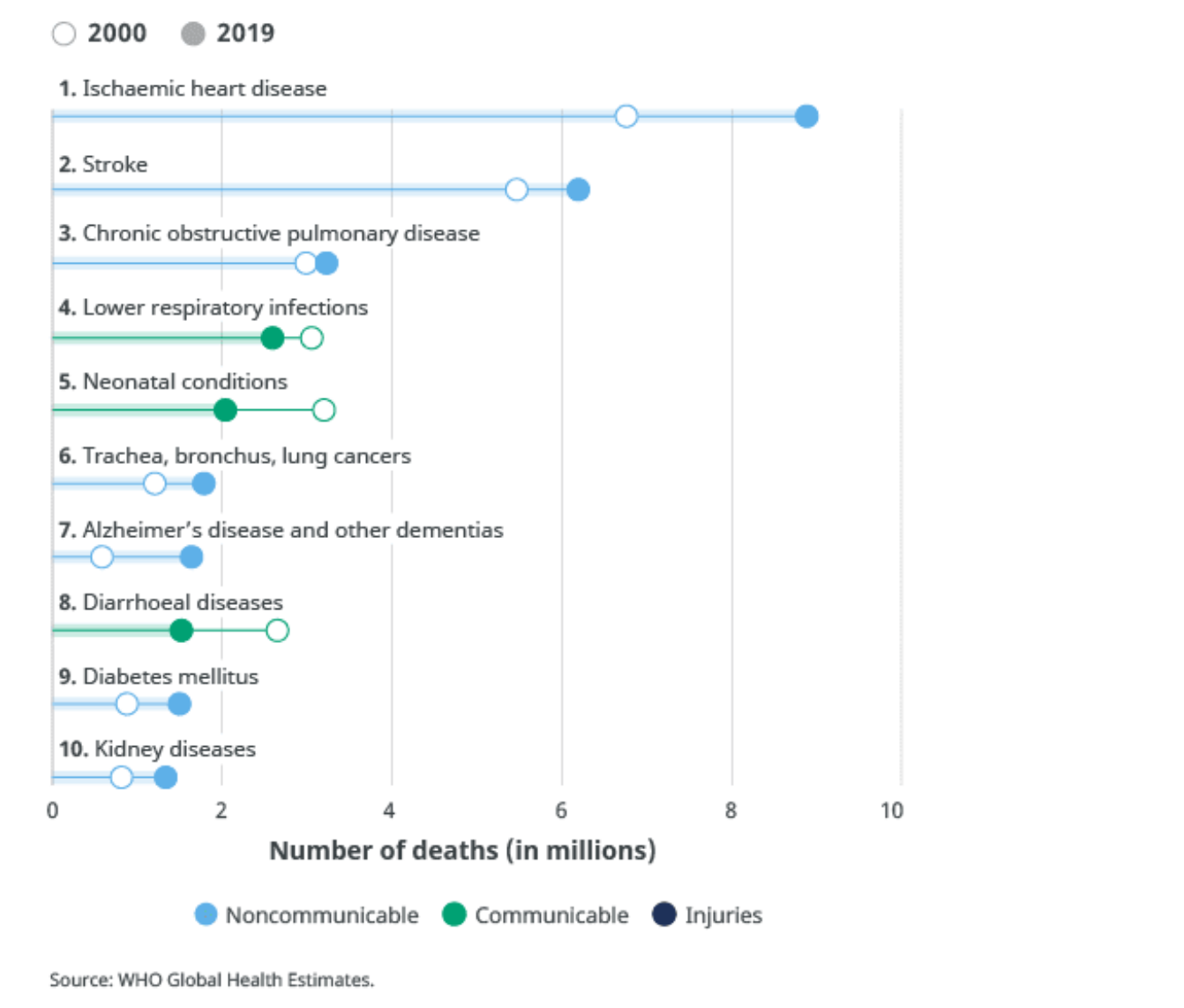
Non-approved Indications

Top 10 global causes of deaths, 2020



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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.

Acute Coronary Syndrome

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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 (DSE) followed by HBOT at 2 ATA for 90 mins	HBO can detect viable myocardium with about the same performance as DSE
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

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

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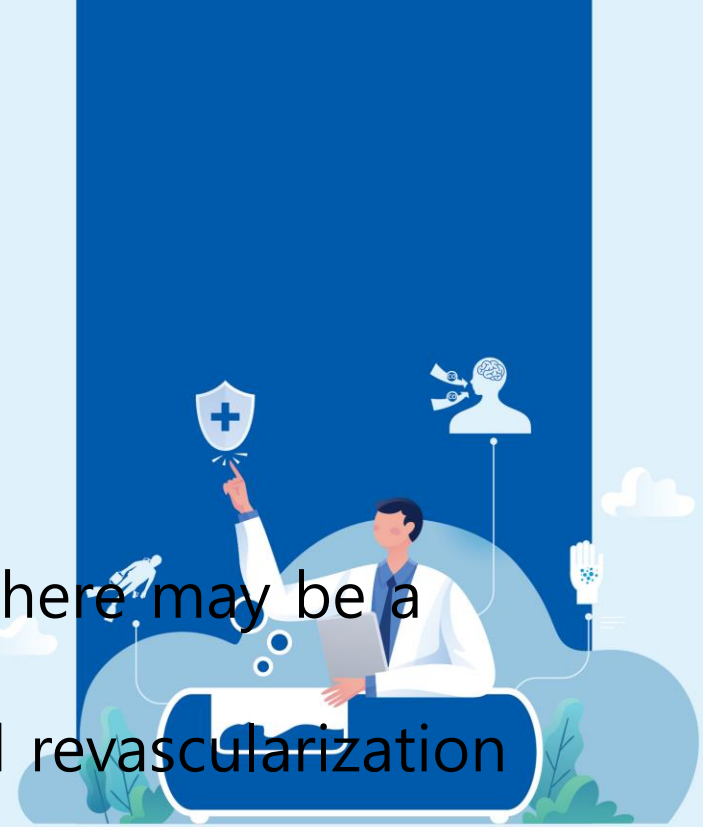
Randomized clinical trials to treat ACS (2)

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

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.

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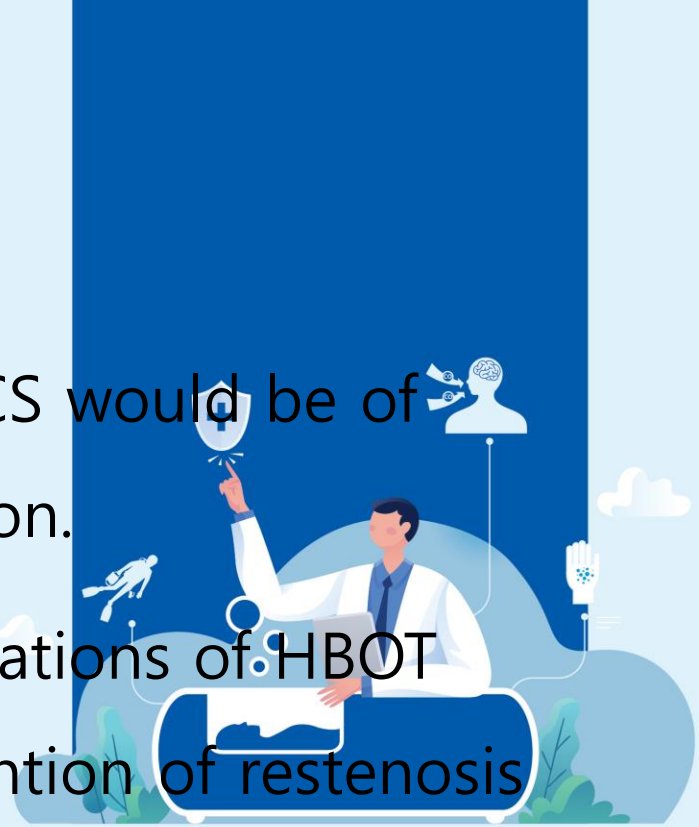
Conclusions (2)



- 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

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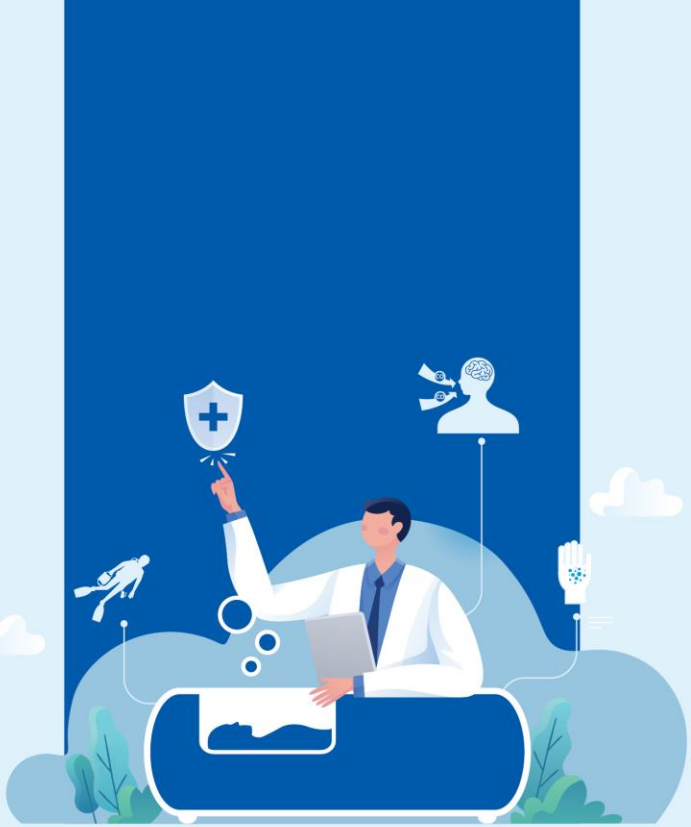
Table 21.9 Summary of Animal Studies of Focal Cerebral Ischemia Where HBOT Was Compared with Normobaric Air or Oxygen

TRIAL, ANIMAL, AND VESSEL OCCLUDED	TIME TO HBOT	OXYGEN DOSE	OUTCOME
Weinstein (1986),[192] gerbil, 20-minute bilateral CCA	0	1.5 ATA for 15 minutes once	Improved survival
Yang (2002),[192a] rat, 1-hour MCA	0	2.8 ATA for 15 minutes once	Neuroprotection implied by reduced extracellular dopamine
Milkovic-Lolic and colleagues (2003),[74] rat, 1-hour MCA	0	3 ATA for 1 hour once	Reduced infarct volume, leukocyte infiltrate, and myeloperoxidase
Sunami (2000),[192b] rat, permanent right MCA and right CCA	10 minutes	3 ATA for 2 hours once	Reduced infarct volume
Heide (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
Beitan (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
Darkill (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
Gunther (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**
Roos (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
Schäbitz (2004),[79] rat, permanent MCA	2 hours	2 ATA for 1 hour once	Reduced infarct volume and deficit
Calvert (2006),[192i] rat, permanent CCA*	2 hours	2.5 ATA for 2 hours once vs. NBO control	No difference in the reduction of hypoxia inducible factor
Kawamura (1990),[192j] rat, 4-hour MCA	3 hours	2 ATA for 30 minutes once	Reduced infarct volume and edema
Lou (2006),[192k] rat, 90-minute MCA	3 hours	3 ATA for 1 hour once	Reduced infarct area and improved deficit
Lou 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),[193a] 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),[193b] rat, 2-hour MCA	6 or 24 hours	2.5 ATA 2 hours daily for 6 days	Improved outcome at both times
Yin (2002),[193c] rat, 2-hour MCA	8 hours	3 ATA for 1 hour once	Reduced infarct area
Yin (2003),[193d] rat, 2-hour MCA occlusion	8 hours	2.5 ATA for 2 hours once	Reduced infarct area, neurologic scores and apoptosis



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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
Nighoghossian (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

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-

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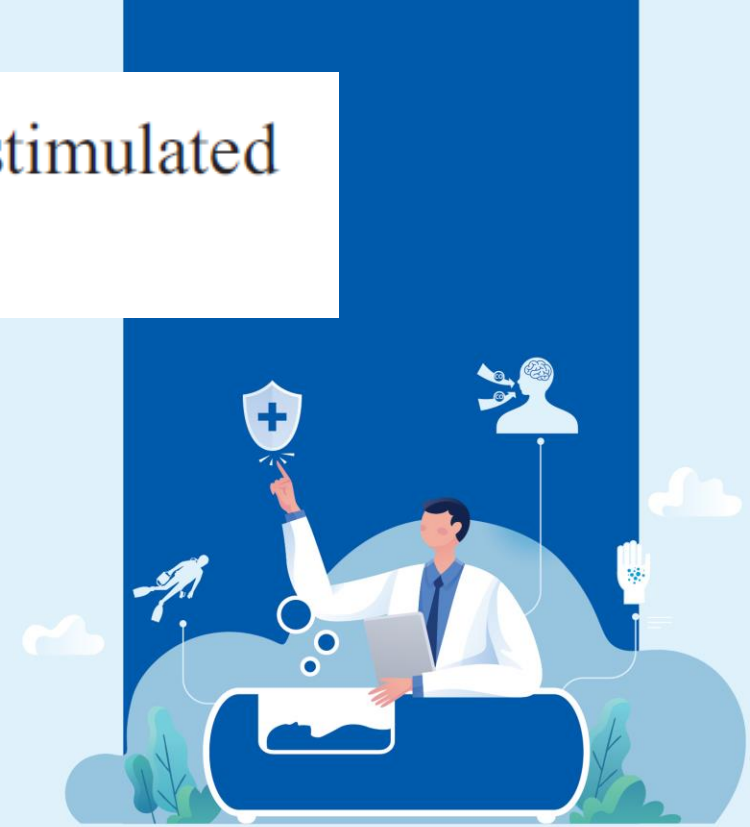
Hyperbaric oxygen protects against lipopolysaccharide-stimulated oxidative stress and mortality in rats

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.

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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.

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

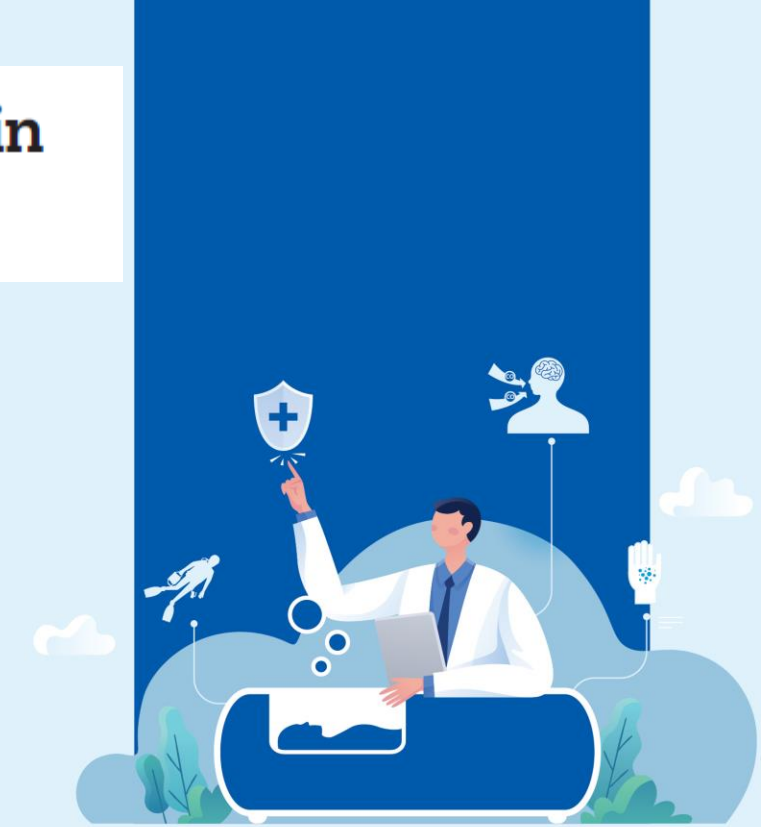
BRAIN RESEARCH 1412 (2011) 63–72

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



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life

MDPI

Article

Effect of Hyperbaric Oxygen Therapy on Acute Liver Injury and Survival in a Rat Cecal Slurry Peritonitis Model

Hee Won Yang [1], Sangchun Choi [1,*], Hakyoon Song [1], Min Ji Lee [2], Ji Eun Kwon [3], Han A. Reum Lee [4] and Kyuseok Kim [4,*]

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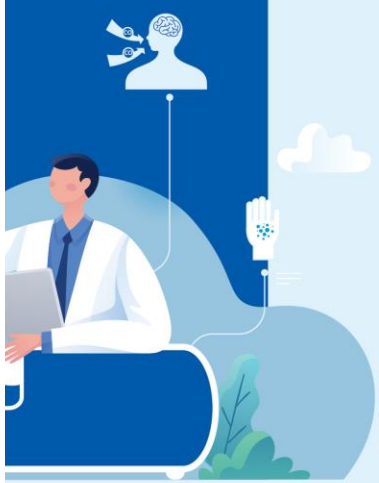

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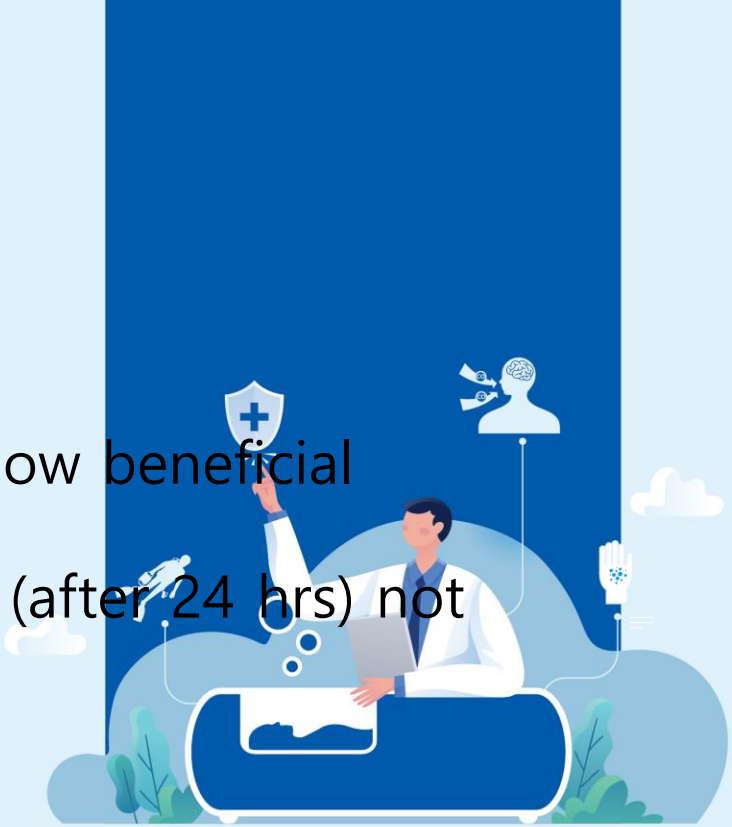
Conclusion



- 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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Take Home Message

- The rationale for the use of HBOT for ACS, acute ischemic stroke, 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.

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Q & A ?

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