

above as required to qualify. Repeat at intervals (2nd treatment and again after 2 weeks and 4 weeks of treatments) to reinforce technique and monitor drift.

RESULTS: Primary outcome: reduction in tcpO₂ during air break Secondary outcomes: Mask fit training may be included in accreditation and demonstration of reproducible results. As well demonstrate to what extent an equilibrium (i.e. zero slope) is reached during an extended (15 min.) airbreak.

**-E5-
TRANSCUTANEOUS OXIMETRY REVEALS
BIBS MASK DYSFUNCTION IN THE
HYPERBARIC ENVIRONMENT**

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Combined oral presentation with #E4

BACKGROUND: Seizures are a time & pressure related result of HBOT. This is prevented partly by including air breaks during treatment at pressure, as well by keeping duration and depth within safe parameters. In our O₂ environment monoplace chambers, BIBS delivers compressed air during periods of compression, air breaks and decompression. We studied mask fit, using tissue oxygenation measurement as an assessment tool.

MATERIALS AND METHODS: Initial assessment of mask fit included visual inspection and feedback from patients. We later adopted a method of briefly occluding the flow to the mask. Our routine assessment of air delivery was to monitor deflection on an air line pressure gauge. To confirm mask effectiveness, we used transcutaneous monitoring, with a chest lead hooked up to a Radiometer TCM 400. Data was collected from patients, with air breaks varying from 5 to 15 minutes. To avoid operational delays, pre-pressurization (i.e. normobaric air) stable baseline of precordial tcpO₂ was not measured.

RESULTS: We assessed 15 patients, treating at 2.4 ATA for 90 minutes of O₂, while assessing the variability of different mask fittings. Data ranged from median tcpPO₂ of 1000 to 1350 mmHg at 2.4 ATA. Lowest levels reached during air break ranged from less than 200 to 900. The drop in tcpO₂ during air breaks clearly varied depending on mask seal, with visual inspection of mask fit and gauge deflection not proving to be a reliable indicator of mask seal.

CONCLUSIONS: TCOM proved to be a useful tool to ensure adherence to treatment protocols. It became obvious that masks do not necessarily have a good seal and thus do not always give adequate air breaks. Pretesting using occlusion method may be a good indicator of fit during use, as it is a method that the patient can understand and directly experience.

**-E6-
HYPERBARIC OXYGEN IN PATIENTS WITH
IMPLANTED CARDIAC DEFIBRILLATORS
AND PACEMAKERS**

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Oral Presentation

BACKGROUND: Patients with implantable cardioverter defibrillators (ICD) or pacemakers may need hyperbaric oxygen (HBO₂) therapy. These devices may not be suitable for pressurization or may expose the patient to increased risk during HBO₂.

METHODS: Survey of ICD and cardiac pacemaker manufacturers to determine which devices were approved for HBO₂.

RESULTS: Some ICD and pacer devices have been tested/approved for HBO₂ exposures. We could find no in vivo ICD testing in hyperbaric conditions. Titanium pacemakers/ICDs will deform at 5 atm abs. Some pacing rates can be affected by sudden changes in pressure. Faulty ICDs and pacemakers, identified by recalls, may increase patient risk during HBO₂. If ICD leads are damaged, ignition could occur if the ICD discharges (personal communication with Medtronic, 2005). It is unknown if ignition could cause patient combustion.

Manufacturer	Pacers Approved	Max Pressure (atm abs)	ICDs Approved	Max Pressure (atm abs)
Medtronic	Thera-I, Elite, Prodigy	2.5	Marquis Gem	Not disclosed
Guidant	Insignia+, Pulsar, Pulsar-Max I,II; Discovery I,II; Meridian; Virtus II; Intellis II	4.9	Contact Renewal 3,4 Ventak Prizm III	4.9
St. Jude Medical	All "current" models	8.0	Photon Micro (V194,V232,V2301IV) Epic (V197,V233,V235) Epic+ (V196,V236,V239) Atlas (V199,V240); EpicIII (V337,V338) Atlas+HF (V340,V343)	3.0
Biotronik, Inc.	Philos I,II, Protos, Cylus, Aetros	3.0	Lumos, Lexos, Xelos, Belos	3.0
E.L.A Medical	All models	4.0	9201, 614, 615, 624, 625	1.5

CONCLUSIONS: 1) Approved pressures are up to 8.0

atm abs for pacemakers and 4.9 atm abs for ICDs, but vary by manufacturer and model. 2) ICD defibrillation during HBO₂ has not been reported, nor tested. 3) If there is an ICD discharge (defibrillation) in the presence of lead damage, ignition could occur. We advise that present-day ICDs should be deactivated before HBO₂ therapy (monitoring the patient and the capability for defibrillation would therefore be important).

-E7-

HBO₂ IN PATIENTS WITH DELAYED VENTILATION

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Oral Presentation

BACKGROUND: Delayed ventilation from emphysema or lung bullae may increase risk for pulmonary barotrauma during decompression from hyperbaric oxygen (HBO₂) therapy,¹ which can be lethal.² However, recommendations about quantifying risk of HBO₂ in patients with delayed ventilation are lacking.

METHODS: Patients with lung disorders (emphysema, asthma, radiation therapy, pneumonia, trauma, surgery, smoking history) and referred for HBO₂ had chest radiography and if abnormal, or clinical suspicion for delayed ventilation, also had xenon ventilation scanning. Pulmonary function tests (PFT) and high resolution CT were done if clinically indicated.

RESULTS: From 1/2005 to 2/2006 we identified 19 patients at risk for delayed ventilation. Fifteen patients had abnormal chest radiographs (radiation scarring, wedge-resection, traumatic pneumatocele, pneumonia, nodules, or pleural effusion). Nineteen patients had ventilation scans. Eight patients had PFTs: obstruction-6, air-trapping-2, hyperinflation-1, normal-1. Seven patients had chest CT scans showing air trapping, nodules, granulomas, COPD, emphysema, or fibrotic changes. One patient with bullous emphysema (xenon washout >40 minutes) was excluded from HBO₂ treatment because of lung and cardiac risk factors. A second patient with delayed ventilation healed her wound without HBO₂, and a third had a normal ventilation scan. Sixteen patients with delayed ventilation (mean ± 1SD = 7.8±4.4, range 2 to 18 minutes) were treated with HBO₂ (425 hyperbaric sessions) without adverse events. HBO₂ therapy was discontinued early (session #15) secondary to breathlessness in one, and in another due to reevaluation of risk/benefit after 4 HBO₂ sessions. Twelve of 16 patients with delayed ventilation >5 minutes were decompressed slowly. Six of these 16 patients with reversible airflow obstruction were given bronchodilators immediately prior to each HBO₂ treatment.

CONCLUSIONS: Patients with delayed ventilation

may be treated with HBO₂. Bronchodilators may improve ventilation in some. HBO₂ was not offered to the patient with lung bullae, so we cannot extrapolate these results to bullous disease.

1Unsworth IP. Pulmonary barotraumas in a hyperbaric chamber. *Anaesthesia*. 1973 Nov; 28(6):675-8.

2Wolf HK, Moon RE, Mitchell PR, Burger PC. Barotrauma and air embolism in hyperbaric oxygen therapy. *Am J Forensic Med Pathol*. 1990 Jun; 11(2):149-53.

-E8-

CRITICAL CARE OF PATIENTS TREATED IN MONOPLACE HYPERBARIC CHAMBERS, PAST 20 YEARS

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BACKGROUND: There has not been a systematic review of treating intubated, critically ill patients in monoplace hyperbaric chambers, yet some critically ill patients may need hyperbaric oxygen (HBO₂) therapy delivered within monoplace chambers.

METHODS: From 1986 to 2006 we reviewed records of all critically ill, intubated patients treated with HBO₂ in Sechrist 2500B and 3200B monoplace chambers. Patients were ventilated with the Sechrist 500A or the Omni-vent ventilators. They were sedated and occasionally paralyzed. Data is reported as mean±SD (range).

RESULTS: Baseline characteristics: 182 patients (1,281 HBO₂ sessions in intubated patients) in 61 females, 121 males; age=44±19 (2-83) years; 90 with necrotizing fasciitis, 44-CO poisoning, 14-crush injury, 10-gangrene, 7-AGE, 4-mucormycosis, 4-arterial insufficiency, 4-failing flaps, 4-osteomyelitis, 1-radiation necrosis. Until 1995 we did myringotomies in 66 patients and from 1995 did not do them in 116 patients. Of 108 patients, the APACHE II=17.6±7.5(6-44). The average number of IV lines per session = 3.8±1.8 (1-11). Of 154 patients, 27 died from their disease or withdrawal of support. Complications necessitating decompression included: Cardiac arrest (post-HBO₂ hypoxia-1, VT/VF-1); hypoxemia with air breathing-2; arterial line/connectors-5; ventilator circuit-8; ventilator malfunction-2; seizures-3; air-trapping-4; inadequate sedation-5; arrhythmias-4 (35/1,281=2.7%).

Pressure (ATA)	Arterial Blood Gases (N=155)			Ventilator Parameters		
	PaO ₂	PaCO ₂	pH	Parameter	Baseline	Chamber
0.85	263±121 (66-545)	36.3±7.2 (21-55)	7.38±0.02 (7.01-7.55)	V _E (/min)	16±4 (7-37)	16±4 (8-15)
1.9-2.2	1023±520 (210-1255)	35.7±10.6 (26-68)	7.41±0.08 (7.24-7.50)	V _T (ml)	654±133 (200-950)	622±128 (200-930)
2.4-2.6	1117±205 (535-1504)	35.9±7.6 (21-59)	7.39±0.08 (7.21-7.56)	PEEP (cm H ₂ O)	3.2±3.4 (2-25)	6.9±3.6 (2-28)
2.8-3.0	1281±264 (711-1809)	40±8.3 (28-68)	7.36±0.09 (7.13-7.55)	P _i (cm H ₂ O)	24±8 (10-60)	31±9 (12-72)
				EtCO ₂ (side-stream)	34±15 (11-99)	17±8 (7-60)