The cardiac effects of hyperbaric oxygen at 243 kPa using in-chamber echocardiography

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Abstract

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Introduction: The adverse effects of hyperbaric oxygen (HBO) on cardiac physiology are considered a potential hazard during the treatment of some patients. The haemodynamic effects of HBO are poorly understood and the incompatibility of electrical equipment inside the chamber has made assessment difficult. At Fremantle Hyperbaric Unit, we have modified an ultrasound machine (Logiq™ e) for safe use within the hyperbaric environment. The aim of this study was to evaluate the cardiac changes that occur during HBO using in-chamber transthoracic echocardiography (TTE) in subjects without evidence of active cardiac disease.

Methods: Eleven patients and nine members of staff underwent comprehensive TTE examinations before and during HBO administered at a pressure of 243 kPa. The TTE examinations were reported by two independent cardiologists and statistically evaluated using paired Student's *t*-tests.

Results: There was a significant decrease in heart rate during HBO (65 vs. 70 bpm on air at atmospheric pressure, $P = 0.002$) resulting in a decrease in cardiac output (5.3 vs. 5.9 $L \cdot min^{-1}$, $P = 0.003$). Left ventricular outflow tract (LVOT) dimension was larger during HBO than baseline imaging (2.30 vs. 2.23 cm, $P = 0.0003$). LVOT velocity time integrals (VTI) decreased (19.9 vs. 21.5 cm, $P = 0.009$) and therefore a similar stroke volume was maintained (61 vs. 65 ml, $P = 0.5$). Ventricular and atrial volumes, intracardiac flows and minor valvular abnormalities were not significantly affected by HBO. No adverse cardiac effects were observed.

Conclusions: TTE can be safely performed within a hyperbaric chamber. Cardiac physiology is not adversely affected by HBO in individuals without active cardiac disease.

Key words

Physiology, cardiovascular, hyperbaric oxygen, echocardiography, hyperbaric research

Introduction

A number of potentially adverse changes occur in the cardiovascular system in response to hyperbaric oxygen (HBO), and these remain relatively little studied, in part owing to the incompatibility of electronic equipment inside the chamber.[1–5] At the Fremantle Hyperbaric Unit we have become the first, to our knowledge, to develop an ultrasound machine capable for use inside the chamber.[6]

HBO treatment is used in a wide range of patients for a variety of conditions including wound healing, delayed radiation tissue damage, necrotising infections and diving-related indications. Many of these patients are elderly with significant co-morbidities and the risk factors for the development of their primary complaints are similar to the potential risks for underlying cardiac disease. Chamber attendants are also subject to the physiological effects of breathing HBO.

Echocardiography continues to develop as an important tool in the recognition of cardiac disease and assessment of cardiac function. Previous literature has documented transthoracic echocardiography (TTE) findings before and after HBO.[7] Limited TTE studies have also been performed in hyperbaric conditions with the machine external to the chamber using the subject or an individual separate to the machine to acquire the images.[8,9] Both these studies highlighted some difficulties of imaging with the machine external to the chamber. Actual in-chamber 2-D TTE of subjects has never been performed.

The aim of this study was to evaluate the cardiac changes that occur during HBO using in-chamber TTE in subjects without evidence of active cardiac disease.

Methods

The study was approved by the Western Australian South Metropolitan Area Health Service Human Research Ethics Committee (approval no: 10/478), and conducted according to the principles of the Helsinki Declaration (revised 2008). Informed written consent was obtained from all subjects.

As previously described, with the assistance of Fremantle Hospital Biomedical Services and using available guidelines and recommendations, an ultrasound machine (Logiq™ e, GE Healthcare) was modified for safe use within the chamber.[6] The ultrasound machine had a cardiac software package and images were acquired with a 3 MHz cardiac probe. The cardiac software available did not have tissue Doppler capability.

Table 1
Acronyms used for physiological terms in this paper

LV – left vetricle
RV – right ventricle
LA – left atrium
RA – right atrium
LVOT –left ventricular outflow tract
RVOT – right ventricular outflow tract
VTI – velocity time integral
TR – tricuspid regurgitation
EDV –end diastolic volume
ESV – end systolic volume
EF – ejection fraction

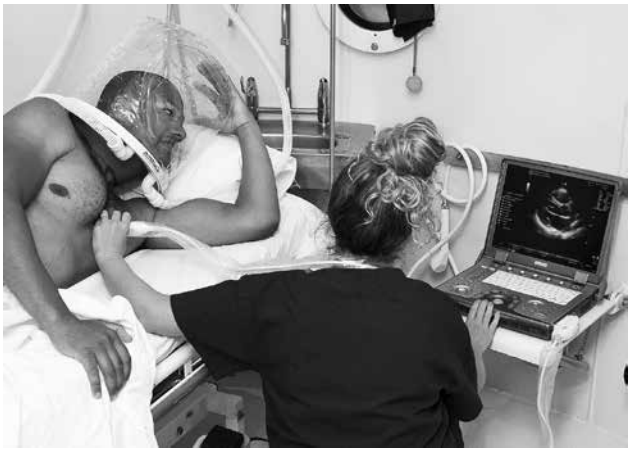
The subjects were a convenience sample of volunteers either being treated or working at Fremantle Hyperbaric Unit during the years 2011 and 2012. Patients and staff were recruited when there was both available space in the chamber and an available sonographer to conduct the examination. Examinations took place singularly within the chamber so privacy was not an issue. Patients undergoing HBO treatment for a range of conditions and available members of staff underwent TTE immediately before and during HBO. The patients were examined during their routine treatment and staff examined under the exact same conditions following 30 minutes of HBO. The chamber was pressurised to 243 kPa and 100% oxygen given through an Amron™ head hood at 30 $L \cdot min^{-1}$. The subjects were imaged on a trolley within the chamber in the left lateral position (parasternal long and short axis and apical views) and supine (subcostal views) as per a routine TTE examination (Figure 1).

A certified cardiac sonographer performed a comprehensive TTE examination. Assessments were made at room pressure breathing air before pressurisation and at pressure breathing 100% oxygen of cardiac chamber volumes and function; valve function; inflow velocities and outflow velocity time integrals and heart rate (see Table 1 for list of acronyms used). LV stroke volumes were calculated from LVOT VTI and LVOT diameter data. Cardiac outputs were derived from stroke volume and heart rate.

The TTE examinations were reported by two independent cardiologists blinded to the pressure, and subsequently reviewed if there was a discrepancy between the reported results. A consensus decision was then made on the findings.

The data were statistically evaluated using SPSS version 20. Paired Student's *t*-tests compared surface air and HBO measurements. Parameters were tested for normality of distribution before comparisons were made. Agreements between tests were measured using Kappa tests and correlations using Pearson tests. Significance was accepted as a *P*-value of 0.05 or less.

Figure 1
Subject undergoing echocardiography at 243 kPa pressure whilst breathing 100% oxygen from a head hood



Results

Eleven patients and nine members of staff were recruited. The 20 subjects (13 male, 7 female) were aged 48.8 (SD 15.7) years, their mean weight was 77.8 (SD 15.7) kg and body mass index 25.7 (SD 4.8) $kg \cdot m^{-2}$.

There were no significant differences between the patient and staff groups when compared using non-parametric testing. Therefore, the data were pooled for analysis. The TTE recorded measurements are shown in Table 2. Where possible, data were obtained under both surface air and HBO conditions, excepting that there was insufficient TR to estimate the right ventricular systolic pressure as paired data in 15 subjects (i.e., insufficient TR at atmospheric pressure, during HBO or both).

VALVULAR REGURGITATION AND STENOSIS

On surface imaging, there were 12 subjects with no TR, and five subjects with trace TR. There was one subject each with mild and moderate TR. During HBO imaging, TR was absent in 13 subjects and mild in four subjects. The apparent difference in severity of TR between surface air and HBO could not be compared statistically because of the small sample size. There was a sufficient envelope for estimation of right ventricular systolic pressure in four subjects during atmospheric imaging and two subjects during HBO conditions. HBO did not appear to have an important effect on the degree of TR. At atmospheric pressure on air, one subject had moderate aortic regurgitation, five subjects had mild pulmonary regurgitation, four had mild mitral regurgitation and another had moderate mitral regurgitation. No subjects had significant valvular stenosis. HBO conditions did not change the severity of valvular regurgitation identified at atmospheric pressure in any of the subjects.

Table 2

Haemodynamic data from surface air and hyperbaric oxygen (HBO) at 243 kPa derived from trans-thoracic echocardiographic imaging; * sample size too small for statistical comparison; † no correlation between measurements during atmospheric and HBO imaging (data presented for completeness)

Variable	*n*	Surface air Mean (SD)	HBO at 243 kPa Mean (SD)	*P*-value
LV diastolic diameter (cm)	16	4.8 (0.5)	4.7 (0.4)	0.3
LV diastolic volume (mL)	19	107.2 (22.9)	102.7 (25.8)	0.2
LV systolic volume (mL)	19	41.4 (17.5)	42.5 (18.8)	0.8
LV stroke volume (mL)	18	64.5 (14.6)	61.4 (15.2)	0.5
LV ejection fraction (%)	19	62.3 (9.6)	60.6 (10.5)	0.5
RA area (cm^2)	14	14.6 (3.4)	15.6 (2.6)	0.2
LA volume index ($mL \cdot m^{-2}$)	19	39.5 (11.4)	39.3 (14.0)	0.8
Aortic valve mean gradient (mmHg)	14	4.2 (1.6)	3.7 (1.2)	0.09
Aortic valve VTI (cm)	14	29.5 (5.8)	29.9 (5.7)	0.99
LV outflow tract (OT) diameter (cm)	20	2.23 (0.25)	2.30 (0.25)	<0.001
LVOT VTI (cm)	19	21.5 (6.0)	19.9 (6.3)	0.009
Heart rate (bpm)	20	69.7 (11.8)	64.9 (11.3)	0.002
Cardiac output ($L \cdot min^{-1}$)	19	5.9 (2.4)	5.3 (2.2)	0.003
Mitral inflow E wave ($cm \cdot s^{-1}$)	20	71.5 (22.5)	74.7 (25.7)	0.2
Mitral inflow A wave ($cm \cdot s^{-1}$)	18	59.1 (16.9)	55.3 (15.0)	0.2
Mitral inflow E:A ratio	18	1.4 (0.5)	1.5 (0.6)	0.06
E wave deceleration time (ms)†	19	219.0 (48.0)	227.0 (54.6)	0.6
Pulmonary vein S wave ($cm \cdot s^{-1}$)	12	43.9 (13.0)	44.1 (16.7)	0.9
Pulmonary vein D wave ($cm \cdot s^{-1}$)	12	44.0 (15.6)	45 6 (14.9)	0.9
TR peak velocity ($m \cdot s^{-1}$)*	4	2.6 (0.2)	2.2 (0.0)	n/a
RVOT VTI (cm)	18	16.0 (4.7)	14.4 (4.0)	0.07
Estimated pulmonary artery systolic pressure (mmHg)	5	32.5 (9.1)	30.7 (8.5)	0.3

CHAMBER DIMENSIONS AND VOLUMES

The LV dimensions using the standard, parasternal long-axis basal dimension, or apical LV volumes using Simpson's method,[10] were not different during atmospheric or HBO imaging (Table 2). However, the LVOT dimension was larger during HBO imaging than at atmospheric pressure (2.30 cm vs. 2.23 cm, $P = 0.0003$). Both the LA and RA sizes (volume and area, respectively) were no different between atmospheric and HBO imaging.

CARDIAC OUTPUT

LV stroke volume was measured using both LVOT (LVOT VTI and LVOT dimension) and Simpson's method (LVEDV – LVESV). The LV stroke volume and LV ejection fraction were no different between surface and HBO imaging by either of the two methods for their measurement. Along with the increase in the LVOT dimension under HBO conditions, the LVOT VTI decreased (21.5 cm vs. 19.9 cm, $P = 0.009$), thus maintaining a similar stroke volume.

There was a significant decrease in heart rate during HBO conditions (65 bpm during HBO vs 70 bpm at atmospheric pressure, $P = 0.002$). As a result, there was a significant decrease in cardiac output during HBO conditions (mean 5.3 $L \cdot min^{-1}$ vs. 5.9 $L \cdot min^{-1}$ at on surface air, $P = 0.003$; Table 2).

INTRA-CARDIAC FLOWS

There was no statistical difference between surface air and HBO conditions for mitral inflow E wave, A wave, mitral deceleration time, or pulmonary vein flows (Table 2). There was a trend toward a lower mitral inflow E:A ratio at ambient pressure vs. HBO (1.4 vs. 1.5 $m \cdot s^{-1}$, $P = 0.06$), consistent with the higher (but non-significant) early trans-mitral flows. Right-sided flows, reflected in the RVOT VTI measurements, trended toward lower values during HBO (14.4 vs. 15.8 cm, $P = 0.07$). The RVOT dimension was not measured during the study because of variable image quality of the region of interest.

INTERNAL CONSISTENCY OF DATA

To confirm the internal consistency of the data, correlational analysis was performed between the two conditions for each measurement described. Strong correlations ($r > 0.9$, $P < 0.001$) were found between surface air and HBO measurements for most variables, and similarly strong agreements were found using Kappa tests. Lesser degrees of agreement were found between Simpson's-derived cardiac output and ejection fraction ($r = 0.5$, $P = 0.02$ and $r = 0.6$, $P = 0.005$, respectively) hence LVOT-derived cardiac output and PLAX-derived LV EF were presented in Table 2 ($r = 0.96$, $P < 0.001$; $r = 0.9$, $P < 0.001$, respectively). No correlation

was observed between mitral deceleration time data pairs.

Discussion

Our study describes the cardiac physiology in response to HBO administered at 243 kPa. We describe that TTE is feasible and safe to perform inside a hyperbaric chamber. No adverse cardiac responses were observed in our group of individuals without evidence of active cardiac disease. Our findings provide a basis by which future studies on the cardiovascular effects of HBO could be considered in patients with cardiac disease. The observed fall in cardiac output during HBO in our study is a result of a decrease in heart rate. There was no significant change in stroke volume despite an increase in LVOT dimension and a decrease in LVOT flow. Ventricular and atrial volumes, intracardiac flows and minor valvular abnormalities were not affected importantly by HBO conditions.

It has been well documented that during HBO there is a decrease in cardiac output, primarily owing to bradycardia and increased afterload.[1–4] This decrease has previously been attributed to hyperoxia alone since, in animal models, cardiac output and heart rate do not significantly change under normoxic hyperbaric conditions.[3] However, other mechanisms may also play a role: animal models have demonstrated discrepancies between myocardial oxygen supply and demand, and the direct effect of hydrostatic pressure on cardiac pacemaker function may cause bradycardia.[2,11] There is no clear effect of HBO on myocardial contractility in either animal or human studies.[12–14] Our data show a reduction in heart rate, which appears to be the primary driver for the decrease in cardiac output, LVEF and stroke volume, both indirect measures of LV contractility, did not change.

Acute pulmonary oedema is considered a potential hazard during the treatment of patients with HBO. Case reports estimate the incidence of pulmonary oedema to be approximately 1 in 1,000 patients treated.[5,15] A postulated explanation for this was a disturbance in ventricular balance in patients with congestive cardiac failure.[5] Congestive cardiac failure remains a relative contra-indication to HBO treatment. In our study, we were unable to demonstrate any change in intracardiac flows or measures of left ventricular function during HBO. From our data, in a small patient cohort with no evidence of active heart disease, it appears that HBO does not predispose an individual to pulmonary oedema due to abnormal left ventricular systolic and/or diastolic function.

STUDY LIMITATIONS

Because of the relatively small number of subjects (20), we may have been unable to identify minor cardiac physiological effects of HBO. This includes trends observed in decreases in mitral inflow E:A ratio or RVOT VTI. We did not measure the effect of normoxic hyperbaric conditions, so are unable to exclude an effect of hyperbaric conditions specifically in the absence of hyperoxia.

We considered the possibility of variability in the echo imaging between HBO and surface conditions as an explanation for the results obtained. However point-to-point variation (test–retest variability) was extremely small between sonographer and independent observer. The overall agreement between atmospheric and HBO parameters was also good. Measurements were performed only on images felt to be of good quality.

Imaging of subjects at atmospheric and HBO conditions was slightly different in that a small positive pressure must be attained within the oxygen hood (maximum pressure < 1 cm H_2O) in order to prevent its collapse. Although we cannot exclude a minor effect from the positive pressure on cardiac physiology, we did not feel this to be an important factor.

An increase in the LVOT dimension under HBO conditions was not expected. There are no published data on the behaviour of the LVOT under HBO-loading conditions, so we are unable to verify our results from other studies. At atmospheric pressure, the LVOT does not vary significantly on repeated studies. However in our study, the increase in LVOT dimension was found by the sonographer performing the study, and by two independent cardiologists reviewing the study and blinded to the other analyses. Further, the increase in LVOT dimension offset the observed decrease in the LVOT VTI, preserving the stroke volume. The decrease in cardiac output we observed was driven by the decrease in heart rate under HBO conditions, rather than by a change in the LVOT dimension. Based on these observations, we consider this small increase to be a real phenomenon.

Conclusions

We have demonstrated that TTE is feasible within a hyperbaric chamber, and that cardiac physiology is not adversely affected by HBO conditions in patients and volunteers without evidence of active cardiac disease. The decrease in heart rate observed with HBO appears to drive the decrease in cardiac output, with no evidence for adverse effects of HBO on intracardiac flows or chamber volumes. Further study of the effects of HBO is required in individuals with significant cardiac disease.

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Conflicts of interest: nil

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