Heart Failure and HBO

CHF Strategies

Prediction

Can we reliably predict who will develop symptomatic heart failure (shortness of breath)?

Heart Failure and HBO

RESEARCH ARTICLE

Safety of hyperbaric oxygen therapy in patients with heart failure: A retrospective cohort study

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PLoS One. 2024 Feb 8;19(2):e0293484.

Heart Failure and HBO

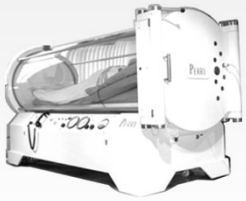
- **Acute pulmonary edema associated with HBO therapy is rare.**
- **Ejection Fractions alone are unreliable.**
- **No HBO specific predictors are available to identify who will go into heart failure.**
- **BNP levels offer promise but have not been studied within the heart failure pop.**

Yildiz S, BNP levels increase after HBO in diabetics. Clin Invest Med 2008;10: E231-234

Grassi P, BNP in Healthy Subjects After HBO Aviat Space Environ Med 2007; 78:52–3.

Heart Failure and HBO

- **BNP levels increase with wall tension and volume overload**



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Grassi P, BNP in Healthy Subjects After HBO. Aviat Space Environ Med 2007; 78:52–3.

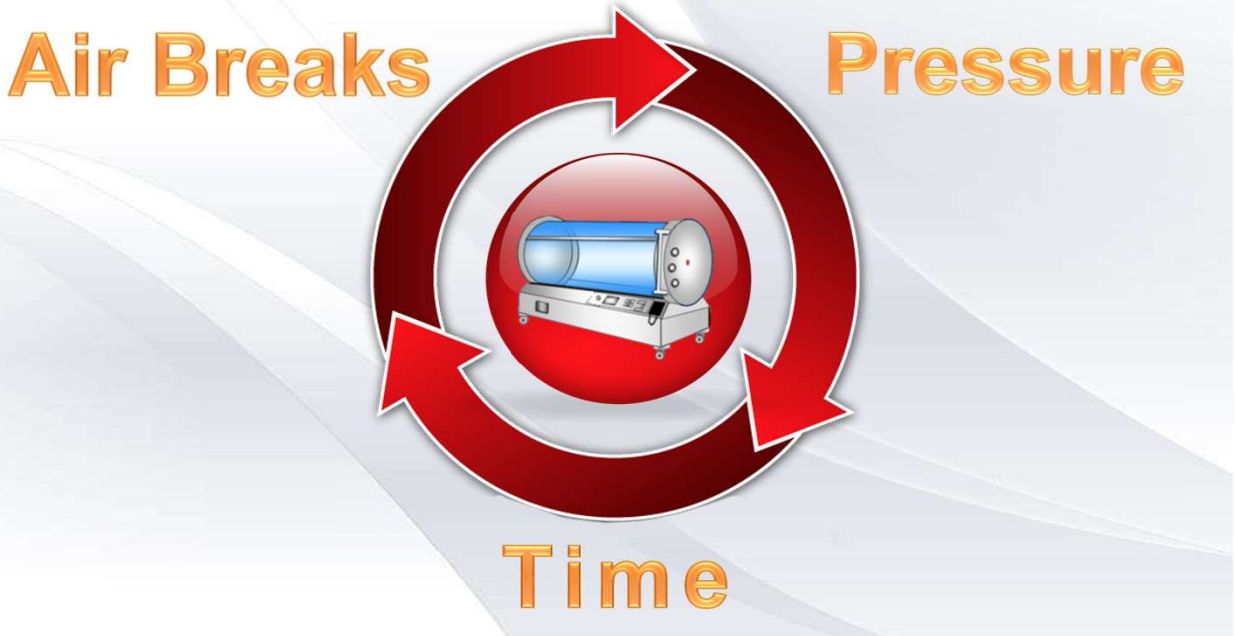
Known Physiology

1. HBO increases systemic vascular resistance and correspondingly a reduction in cardiac output.
2. Increase in pulmonary capillary wedge pressure occurs with high concentrations of normobaric oxygen in NY Heart Association class III or class IV heart failure patients (EF= 15 to 20%).
3. Oxygen radicals consume endothelial-derived nitric oxide, which decrease diastolic left ventricular distensibility.

Known Physiology

1. Systemic vascular resistance decreases rapidly when placed on air.
2. Systemic vascular resistance decreases with a reduction in pressure
3. "Flash" pulmonary edema does not mean there is an instantaneous transition from normal breathing to frothy, pink, sputum. It is a process which begins with decreased pulmonary compliance.

THREE KEY CONSIDERATIONS



CHF Strategies

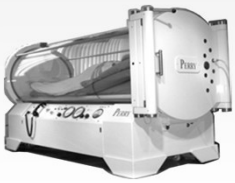
Step-1 MEDICAL STATUS

Is the patient medically optimized?

- Taking medications regularly?
- Recent change in exercise tolerance or PND?
- Consider weekly weights. Weight gain ≥ 5kg?
- Refer to PMD for weight gain | Medications

✔ **Yes** **Patient is Optimized**

→ **Begin HBO**



CHF Strategies

Listen GENERAL APPROACH

Communication

The most important element of case management is the early warning of Shortness of Breath!

Reminder before each treatment to notify if SOB.

Pulmonary edema takes time to evolve.

CHF Strategies

General Approach

If the patient becomes short of breath, place them on air, decompress, and evaluate.

- Placing the pt on air provides an immediate treatment of the problem, which is an elevated after load (inc SVR).
- In the typical pt there will be bibasilar rales which usually clear in 30 min as fluid redistributes.

CHF Strategies

Step-2 Pressure & Air-breaks

Is Treatment Pressure ≥ 2.4 ATA?

- Reduce pressure to 2.0 ATA

Is Treatment Pressure = 2.0 ATA?

- Add air-breaks using "30-10" schedule

Comp	30 min-O2	10 min-air	30 min-O2	10 min-air	30 min-O2	Decomp

Patient still symptomatic during HBO?

✔ **Yes** **Advance to step 3** (adjust time)

CHF Strategies

Step-3 ⇨ TIME

Reduce oxygen time by 15 min

Comp | 25 min-O2 | 10 min-air | 25 min-O2 | 10 min-air | 25 min-O2 | Decomp

Patient still symptomatic during HBO?

✔ Yes

Reduce oxygen time by additional 15 min

Comp | 20 min-O2 | 5 min-air | 20 min-O2 | 5 min-air | 20 min-O2 | Decomp

Patient still symptomatic during HBO?

✔ Yes **Advance to step 4 (adjust pressure)** ⇨

CHF Strategies

Step-4 ⇨ PRESSURE

Reduce pressure to 1.8 ATA | 60 min O_2 time

Comp | 20 min-O2 | 5 min-air | 20 min-O2 | 5 min-air | 20 min-O2 | Decomp

Patient still symptomatic during HBO?

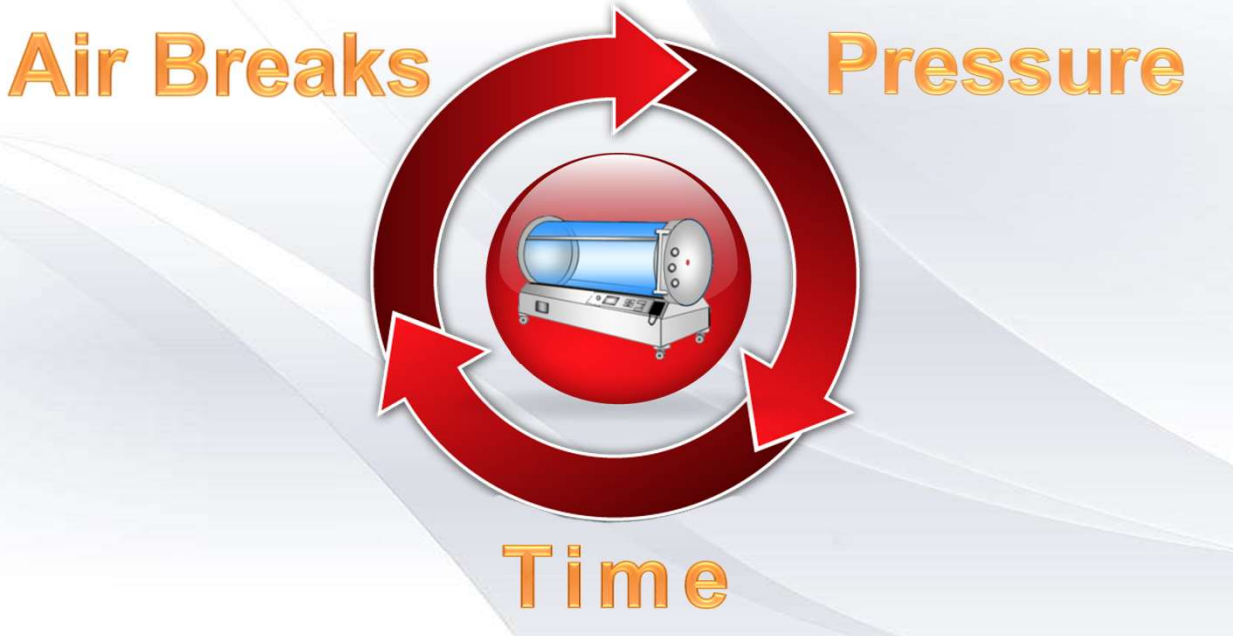
✔ Yes

STOP **Oxygen times less than 60 min with pressures less than 1.8 ATA may be sub-therapeutic.**

SUMMARY

- Acute heart failure with HBO therapy is rare.
- Pulmonary edema evolves over many minutes (not seconds)
- Ejection Fractions are unreliable predictors of HBO induced HF.
- No specific predictors are available to identify who will go into heart failure during HBO therapy.
- Check medical optimization
- Follow daily/weekly weights measured in the center
- Monitor – by checking for SOB several times during treatment
- Modify future treatment profiles if SOB occurs

THREE MODIFIERS



Linked Refs with Abstracts

- Yildiz S, Uzun G, Uz O, Ipcioglu OM, Kardesoglu E, Ozcan O. N-terminal pro-B-type natriuretic peptide levels increases after hyperbaric oxygen therapy in diabetic patients. Clin Invest Med. 2008 Oct 1;31(5):E231-5.
- Grassi P, Buscema G, Rinaldi A, Gobbato PE, Berlot G. B-type natriuretic peptide in healthy subjects after exposure to hyperbaric oxygen at 2.5 ATA. Aviat Space Environ Med. 2007 Jan;78(1):52-3.
- Vincent J, Ross MK, Pollock NW. Effect of hyperbaric oxygen treatment on patients with reduced left ventricular ejection fraction. Diving Hyperb Med. 2021 Sep 30;51(3):256-263.
- Leitman M, Efrati S, Fuchs S, Hadanny A, Vered Z. The effect of hyperbaric oxygenation therapy on myocardial function. Int J Cardiovasc Imaging. 2020 May;36(5):833-840.
- Schiavo S, Brenna CTA, Albertini L, Djaiani G, Marinov A, Katznelson R. Safety of hyperbaric oxygen therapy in patients with heart failure: A retrospective cohort study. PLoS One. 2024 Feb 8;19(2):e0293484.

Heart Failure and HBO

References - Heart Failure and Hyperbaric Oxygen:

- ## N-terminal pro-B-type natriuretic peptide levels increases after hyperbaric oxygen therapy in diabetic patients.

Yildiz S, Uzun G, Uz O, Ipcioglu OM, Kardesoglu E, Ozcan O. Clin Invest Med. 2008 Oct 1;31(5):E231-5.

Purpose: Diabetic patients receive hyperbaric oxygen therapy for non-healing lower extremity ulcers. Exposure to hyperbaric hyperoxia during hyperbaric oxygen therapy may affect cardiovascular functions by different mechanisms. Patients may experience serious problems such as pulmonary edema and death during hyperbaric oxygen therapy. The effect of hyperbaric oxygen therapy on cardiovascular functions in diabetic patients is not well documented. N-terminal pro-B-type natriuretic peptide (NT-proBNP) has been suggested as powerful biochemical marker of cardiac function. The aim of this study was to investigate the effect of hyperbaric oxygen therapy on NT-proBNP levels in diabetic patients.

Methods: Twenty-five diabetic patients (19 male and 6 female, 64.7 +/- 10.2 yr), who were planning to undergo hyperbaric oxygen therapy for non-healing lower extremity ulcers, were prospectively enrolled into the study. All patients were evaluated with echocardiography before the study. Heart rate and arterial blood pressure of patients were measured, and venous blood samples were drawn from each patient for NT-proBNP analysis before and immediately after the hyperbaric oxygen therapy.

Results: NT-proBNP levels increased from 815 +/- 1096 pg/ml to 915 +/- 1191 pg/ml after HBO2 therapy ($P < 0.05$). Heart rate and arterial blood pressure did not change with HBO2 therapy ($P > 0.05$).

Conclusion: Hyperbaric oxygen therapy induces considerable ventricular wall stress in diabetic patients. Care should be taken when a diabetic patient with cardiovascular disease is treated with hyperbaric oxygen therapy.

- ## B-type natriuretic peptide in healthy subjects after exposure to hyperbaric oxygen at 2.5 ATA

Grassi P, Buscema G, Rinaldi A, Gobbato PE, Berlot G. Aviat Space Environ Med. 2007 Jan;78(1):52-3.

Introduction: B-type natriuretic peptide (BNP) is a cardiac hormone used as a marker of cardiac dysfunction with diuretic and vasodilating properties secreted by the ventricles in response to wall stress. Hyperbaric oxygen (HBO) exposure is known to induce hemodynamic effects in humans which can be complicated by acute pulmonary edema. The aim of this study was to investigate if HBO has any effects on the secretion of BNP in healthy human subjects.

Methods: Eight healthy volunteers underwent the following HBO protocol in a hyperbaric chamber: compression to 2.5 atmospheres absolute (ATA); 45 min breathing 100% oxygen; 5 min breathing air; another 45 min in 100% oxygen; then decompression to atmospheric pressure. A venous blood sample was drawn before entering the chamber (To), immediately at the end of the treatment (T1), and at 5 h from To (T2). BNP concentration was determined using a rapid point-of-care immunoassay. Non-parametric statistics were used to analyze data.

Results: No difference in BNP levels was found between T0 and T1 or T2.

Discussion: The findings of this preliminary study show that in healthy subjects a single HBO exposure does not significantly modify BNP plasma levels. We hypothesize that this can be the net result between the stimulating effect of the HBO-induced vasoconstriction and the direct inhibitory effect on BNP secretion of myocyte hyperoxia. We conclude that HBO does not modify BNP secretion in healthy volunteers and that the direct effect of extreme hyperoxia on BNP secretion deserves further investigation.

- ## Effect of hyperbaric oxygen treatment on patients with reduced left ventricular ejection fraction

Vincent J, Ross MK, Pollock NW. Diving Hyperb Med. 2021 Sep 30;51(3):256-263.

Introduction: Hyperbaric oxygen treatment (HBOT) is available to a wide spectrum of patients, many with significant co-morbidities. Considering its effects on cardiac physiology and reports of pulmonary oedema following exposure, concerns exist about the safety of patients with compromised cardiac function. Few studies have described adverse events occurring during HBOT and even fewer reports address events arising in the hours following HBOT. A relation between adverse events and cardiac function has not been established. As medical guidance is limited, we aimed to evaluate the risk for patients with reduced left ventricular ejection fraction (LVEF) receiving HBOT.

Methods: This retrospective chart review of patients receiving HBOT from April 2003 through December 2019 at our hospital was designed to describe clinical characteristics of patients and to identify adverse events during HBOT and within 24 hours after HBOT. Patients ≥ 40 years of age with a documented LVEF of ≤ 40% were included. Data are presented as mean (SD) [range] or counts, as appropriate.

Results: A total of 23 patients were included in the final analysis, 2 (1) [0-4] patients per year. Patients received 25 (19) [1-60] treatments. Two patients had an episode of acute decompensated heart failure possibly linked to HBOT.

Conclusions: This study described the clinical characteristics of patients with reduced LVEF receiving HBOT and showed reassuring results, with a majority of patients with reduced LVEF tolerating HBOT well. Prospective research is required to more fully assess the risk

- ## The effect of hyperbaric oxygenation therapy on myocardial function

Leitman M, Efrati S, Fuchs S, Hadanny A, Vered Z. Int J Cardiovasc Imaging. 2020 May;36(5):833-840.

Hyperbaric oxygenation therapy is successfully implemented for the treatment of several disorders. Data on the effect of hyperbaric oxygenation on echocardiographic parameters in asymptomatic patients is limited. The current study sought to evaluate the effect of hyperbaric oxygenation therapy on echocardiographic parameters in asymptomatic patients. Thirty-one consecutive patients underwent a 60-sessions course of hyperbaric oxygenation therapy in an attempt to improve cognitive impairment. In all subjects, echocardiography examination was performed before and after a course of hyperbaric oxygenation therapy. Conventional and speckle tracking imaging parameters were calculated and analyzed. The mean age was 70 ± 9.5 years, 28 [90%] were males. History of coronary artery disease was present in 12 [39%]. 94% suffered from hypertension, 42% had diabetes mellitus.

Baseline wall motion abnormalities were found in eight patients, however, global ejection fraction was within normal limits. During the study, ejection fraction [EF], increased from 60.71 ± 6.02 to 62.29 ± 5.19%, p = 0.02. Left ventricular end systolic volume [LVESV], decreased from 38.08 ± 13.30 to 35.39 ± 13.32 ml, p = 0.01. Myocardial performance index [MPi] improved, from 0.29 ± 0.07 to 0.26 ± 0.08, p = 0.03. Left ventricular [LV] global longitudinal strain increased from - 19.31 ± 3.17% to - 20.16 ± 3.34%, p = 0.036 due to improvement in regional strain in the apical and antero-septal segments. Twist increased from 18.32 ± 6.61° to 23.12 ± 6.35° p = 0.01, due to improvement in the apical rotation, from 11.76 ± 4.40° to 16.10 ± 5.56°, p = 0.004. Hyperbaric oxygen therapy appears to improve left ventricular function, especially in the apical segments, and is associated with better cardiac performance. If our results are confirmed in further studies, HBOT can be used in many patients with heart failure and systolic dysfunction.

- ## Safety of hyperbaric oxygen therapy in patients with heart failure: A retrospective cohort study

Schiavo S, Brenna CTA, Albertini L, Djaiani G, Marinov A, et al. PLoS One. 2024 Feb 8;19(2):e0293484.

Background Hyperbaric oxygen therapy (HBOT) has several hemodynamic effects including increases in afterload (due to vasoconstriction) and decreases in cardiac output. This, along with rare reports of pulmonary edema during emergency treatment, has led providers to consider HBOT relatively contraindicated in patients with reduced left ventricular ejection fraction (LVEF). However, there is limited evidence regarding the safety of elective HBOT in patients with heart failure (HF), and no existing reports of complications among patients with HF and preserved LVEF. We aimed to retrospectively review patients with preexisting diagnoses of HF who underwent elective HBOT, to analyze HBOT-related acute HF complications.

Methods Research Ethics Board approvals were received to retrospectively review patient charts. Patients with a history of HF with either preserved ejection fraction (HFpEF), mid-range ejection fraction (HFmEF), or reduced ejection fraction (HFrEF) who underwent elective HBOT at two Hyperbaric Centers (Toronto General Hospital, Rouge Valley Hyperbaric Medical Centre) between June 2018 and December 2020 were reviewed.

Results Twenty-three patients with a history of HF underwent HBOT, completing an average of 39 (range 6–62) consecutive sessions at 2.0 atmospheres absolute (ATA) (n = 11) or at 2.4 ATA (n = 12); only two patients received fewer than 10 sessions. Thirteen patients had HFpEF (mean LVEF 55 ± 7%), and seven patients had HFrEF (mean LVEF 35 ± 8%) as well as concomitantly decreased right ventricle function (n = 5), moderate/severe tricuspid regurgitation (n = 3), or pulmonary hypertension (n = 5). The remaining three patients had HFmEF (mean LVEF 44 ± 4%). All but one patient was receiving fluid balance therapy either

with loop diuretics or dialysis. Twenty-one patients completed HBOT without complications. We observed symptoms consistent with HBOT-related HF exacerbation in two patients. One patient with HFrEF (LVEF 24%) developed dyspnea attributed to pulmonary edema after the fourth treatment, and later admitted to voluntarily holding his diuretics before the session. He was managed with increased oral diuretics as an outpatient, and ultimately completed a course of 33 HBOT sessions uneventfully. Another patient with HFpEF (LVEF 64%) developed dyspnea and desaturation after six sessions, requiring hospital admission. Acute coronary ischemia and pulmonary embolism were ruled out, and

an elevated BNP and normal LVEF on echocardiogram confirmed a diagnosis of pulmonary edema in the context of HFpEF. Symptoms subsided after diuretic treatment and the patient was discharged home in stable condition, but elected not to resume HBOT.

Conclusions Patients with HF, including HFpEF, may develop HF symptoms during HBOT and warrant ongoing surveillance. However, these patients can receive HBOT safely after optimization of HF therapy and fluid restriction.