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ORIGINAL PAPER

# The effect of hyperbaric oxygenation therapy on myocardial function

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**Abstract**
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.

**Keywords** Hyperbaric oxygenation · Echocardiography · Cardiac function

## Introduction

Hyperbaric oxygen therapy (HBOT) includes the inhalation of 100% oxygen at pressures exceeding 1 atmosphere absolute in order to enhance the amount of oxygen dissolved in the body tissues. During HBOT treatment, the arterial $O_2$ tension typically exceeds 2000 mmHg, and levels of 200–400 mmHg occur in tissues [1]. HBOT has been applied worldwide mostly for chronic non-healing wounds and for diving accidents. In recent years, there is growing evidence on the regenerative effects of HBOT. It is now realized, that the combined action of both hyperoxia and hyperbaric pressure, leads to significant improvement in tissue oxygenation while targeting both oxygen and pressure sensitive genes, resulting in improved mitochondrial metabolism with anti-apoptotic and anti-inflammatory effects [2–18]. Moreover, these genes induce stem cells proliferation, augmented circulating levels of endothelial progenitor cells and angiogenesis factors, which induce angiogenesis and improve blood flow in the ischemic area [2–8]. However, in human studies, which were mainly focused on brain regeneration and neuroplasticity, it had been shown, that these effects require prolonged hyperbaric series of 40–60 sessions [6, 19–21].

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The effect of hyperbaric oxygen environment on cardiac function was mostly evaluated during and after a single exposure. Moleant et al. reported on acute negative hemodynamic effects in healthy volunteers after one hyperbaric session [22]. Frobert et al. showed as well that a single exposure to normobaric hyperoxia worsens systolic myocardial performance in healthy male volunteers [23]. Unlike single exposure to hyperbaric environment, the protocol used to induce regeneration of non-healing wounds and certain types of brain injuries have different physiological effects including repeated intermittent increase of the oxygen levels to high and back to normal. An intermittent increase of oxygen concentration can induce many of the mediators and cellular mechanisms, that are usually induced during hypoxia, but without the hazardous hypoxia—termed "hyperoxic-hypoxic paradoxes" [2]. The intermittent hyperoxic exposure during HBOT can increase HIF-1α, matrix metalloproteinases activity, VEGF, stem cells proliferation, elevated circulating levels of endothelial progenitor cells, those induce angiogenesis and improve perfusion in the ischemic region [2–9].

Using the "regenerative" protocol, HBOT can increase the number of proliferating and differentiating satellite cells and the number of regenerated muscles fibers, and promote skeletal muscle isometric strength [24–28]. Both animal and human studies have demonstrated the beneficial effects of HBOT on mitochondrial function [15–18]. In a study on rats with normal mitochondrial function, HBOT increased the production of adenosine triphosphate in muscle tissue compared to a control group [29]. Moreover, in a clinical study by Li et al., it was demonstrated that HBOT may improve myocardial blood perfusion, reduce inflammation and vascular endothelial dysfunction, and further improve myocardial microcirculation in patients after the implantation of drug-eluting stents [30]. Aparci et al. evaluated the changes following a series of 10 hyperbaric sessions in diabetic patients [31]. The authors concluded that HBOT can improve myocardial diastolic function, as reflected by reduction of the E wave deceleration time, when applied repetitively. However, ejection fraction, did not change following 10 sessions [31]. Thus, one can speculate that the relatively new regenerative HBOT may have beneficial effect on cardiac performance, however it has not yet adequately investigated in human subjects.

The aim of this study was to investigate for the first time in humans the effect of the regenerative repetitive HBOT protocol on cardiac function.

## Materials and methods

The study was performed as a prospective single-blinded trial conducted at the Sagol Center for Hyperbaric Medicine and Research and at the Department of Cardiology at Shamir Medical Center between 2017 and 2018. 31 consecutive unselected patients underwent a 60-day course of hyperbaric oxygenation therapy for the purpose of potential improvement in cognitive impairment. The study was approved by the local Helsinki committee and each participant signed informed consent.

In all subjects, echocardiography examination was performed before the first, and at least 3 weeks after the last hyperbaric oxygen therapy. All echocardiography exams were performed using Vivid E9, (General Electric; Horten, Norway) with a standard transducer of 1.7–4 Hz. The frame rate during echocardiography examinations was greater than or equal to 40 frames per second. Comprehensive transthoracic echocardiography examinations were performed according to the latest recommendations on chamber quantification [32]. Briefly, linear, volumetric and Doppler measurements were performed. Biplane left atrial volume index, LAVi, was calculated according to the formula $8/3\pi[(A1)(A2)/(L)]$ normalized for body surface area, A1 and A2 is an area of the left atrium obtained from apical 4- and 2-chamber views respectively, and L is the shortest vertical size of the left atrium. Left ventricular mass index, LVMi was calculated according to the formula $0.8 \times 1.4[(IVS + PW + LVID)^3 - LVID^3] + 0.6$ g normalized for body surface area. Relative wall thickness, RWT, was calculated as (2 left ventricular posterior wall thickness)/(left ventricular internal diameter at end diastole). Standard echocardiographic views were acquired: parasternal long and short axis at 3 levels: basal, mid-ventricular and apical, apical 4-chamber, 2-chamber, and 3-chamber views. Diastolic function was assessed according to the current recommendations [33], E wave amplitude, A wave amplitude, E/A ratio, E wave deceleration time, tissue Doppler E′ septal velocity [E′s], tissue Doppler E′ lateral velocity [E′l]. Restrictive filling pattern was diagnosed when the deceleration time of E diastolic velocity was $< 160$ ms, $E/E' < 14$, and $E/A \geq 2$. Myocardial performance index, MPi, was calculated as (ICT + IRT)/ET, where ICT—isovolumic contraction time, IRT—isovolumic relaxation time, ET—ejection period. The sum ICT and IRT was obtained by subtracting of ET from the interval between cessation and onset of mitral inflow velocity [34]. All echocardiography examinations were then transferred to the EchoPAC workstation (Version 202), for further off-line speckle-tracking imaging analysis. Speckle-tracking imaging analysis was performed by M.L., and 20 studies were examined independently by both M.L. and Z.V. for inter- and intra-observer variability.

For the speckle tracking imaging analysis, the aortic valve closure on the apical long axis view served as a reference for end systole and was verified by aortic Doppler flow recorded from the apical five-chamber view. An 18-segments model and a standard protocol of speckle tracking imaging analysis were used [35]. Regional peak systolic longitudinal strain

was measured in each of the 18 segments and global longitudinal strain was obtained. Radial strain was obtained at basal, mid-ventricular and apical levels of the left ventricle, and basal and apical rotation was calculated. To obtain maximal apical rotation, short-axis views of the apex were acquired with a maximal caudal deviation of the transducer. LV twist (torsion) angle was calculated as an absolute apex-to-base difference in LV rotation [36]. Right ventricular strain was calculated from the apical 4-chamber view [37, 38]. The right ventricular endocardial border was traced manually including the septum at the end-systolic frame. Then global right ventricular strain was obtained. Biplane Left ventricular EF was calculated using speckle tracking imaging from 2 apical views, 4-chamber and 2-chamber. Left ventricular peak untwisting velocity was measured from short axis views according to the recent recommendations [39]. Diastolic strain rate of the left ventricle and of the right ventricle were calculated from apical views [40].

HBOT treatment protocol included 60 daily sessions, 5 days per week. Each session included 90-min exposure to 100% oxygen, at 2 absolute atmospheres with 5 min of air breaks every 20 min. The 5 min air breaks reduce the risk for oxygen toxicity and enable more oxygen fluctuations which induce the so called "hyperoxic-hypoxic" paradox and physiological effects. All HBOT sessions were performed in a multiplace hyperbaric chamber (Starmed 2700, HAUX-Life Support-GmbH, Germany) at the Sagol Center for Hyperbaric Medicine and Research.

During statistical analysis, continuous data were expressed as means ± standard deviations. The normal distribution for all differences was tested using the Kolmogorov–Smirnov test. Two-tailed, dependent *T* test was used to compare post and pre-HBOT changes. Categorical data were expressed in numbers and percentages and compared by chi-square tests. Univariate analysis was performed using $\chi^2$/Fisher's exact test (where appropriate) or to identify significant variables ($p < 0.05$).

## Results

Demographic and clinical data are presented in Table 1**.** 31 consecutive patients underwent echocardiographic examination before and after the 60-day course of hyperbaric oxygenation therapy. The mean age was 70 ± 9.5 years, 28 [90%] were males. All were coherent and active. Coronary artery disease was present in 12 patients [39%], 3 had undergone coronary artery bypass grafting [CABG], and 9—percutaneous coronary interventions [PCI], 8 [26%] were after myocardial infarction. 29 [94%] suffered from hypertension, 13 [42%] had diabetes mellitus, 15 [48%] had a history of cerebral vascular accident. The 2nd echocardiographic examination was done within 6 months after the first, at least 3 weeks after the last session of HBOT.

**Table 1** Demographic data

| | |
|---|---|
| Age | 70 ± 9.5 |
| Males, % | 28 [90%] |
| Coronary artery disease | 12 [39%] |
| Myocardial infarction | 8 [26%] |
| PCI | 9 [29%] |
| CABG | 3 [10%] |
| Hypertension | 29 [94%] |
| Diabetes mellitus | 13 [42%] |
| Smoker | 4 [13%] |
| Renal failure | 6 [19%] |
| CVA | 15 [48%] |
| Malignancy in the past | 2 [6%] |
| Atrial fibrillation | 5 [16%] |
| Hyperlipidemia | 19 [61%] |
| Respiratory diseases[a] | 5 [16%] |

*PCI* Percutaneous coronary interventions, *CABG* Coronary artery bypass grafting, *CVA* cerebral vascular accident

[a]Chronic obstructive lung disease, asthma, obstructive sleep apnea

Conventional echocardiography data are presented in Table 2, Video 1(A, B). Baseline wall motion abnormalities were found in 8 patients, however, global ejection fraction was within normal limits. During the study, LVEF increased from 60.71 ± 6.02 to 62.29 ± 5.19%, $p = 0.02$. Left ventricular end—systolic volume decreased from 38.08 ± 13.30 to 35.39 ± 13.32 ml, $p = 0.01$. Myocardial performance index improved, from 0.29 ± 0.07 to 0.26 ± 0.08, $p = 0.03$. The parameters of diastolic function did not change significantly. There was a trend to higher E/A ratio, $p = 0.08$, and longer E wave deceleration time, $p = 0.07$. There were 6 (19.4%) patients with pseudo-normal filling pattern and 3 (9.7%) patients with restrictive filling pattern before the HBOT. After the HBOT, there were 7 (22.6%) patients with pseudo-normal filling and 2 (6.5%) patients with restrictive filling.

Speckle tracking imaging results appear in Table 3 and Fig. 1a–d. LV global longitudinal strain increased from − 19.31 ± 3.17 to − 20.16 ± 3.34%, $p = 0.036$. Regional strain increased significantly in the apical and the anteroseptal segments. Twist increased from 18.32 ± 6.61° to 23.12 ± 6.35° $p = 0.01$, as can be seen, due to better apical rotation, that increased from 11.76 ± 4.40° to 16.10 ± 5.56°, $p = 0.004$. Right ventricular [RV] global strain increased from − 19.22 ± 4.45 to − 20.24 ± 4.60%, $p = 0.02$. Peak untwisting velocity, diastolic strain rate of the left ventricle and right ventricle did not change significantly.

Intra-observer variability was evaluated in 10 patients (20 echo exams) and was < 2% [ML]. Inter-observer variability

**Table 2** Conventional echocardiography

| | 1st study | 2nd study | *p* value |
|---|---|---|---|
| EF, % | 60.71 ± 6.02 | 62.29 ± 5.19 | 0.02 |
| LVEDV, ml | 96.39 ± 27.83 | 92.83 ± 28.52 | 0.13 |
| LVESV, ml | 38.08 ± 13.30 | 35.39 ± 13.32 | 0.01 |
| LVEDVi, ml/m$^2$ | 48.51 ± 11.17 | 46.69 ± 11.64 | 0.13 |
| LVESVi, ml/m$^2$ | 19.14 ± 5.64 | 17.71 ± 5.58 | 0.01 |
| LVMi, g/m$^2$ | 126.28 ± 28.59 | 118.56 ± 29.21 | 0.30 |
| RWT | 0.43 ± 0.08 | 0.42 ± 0.06 | 0.28 |
| IVS, cm | 1.20 ± 0.18 | 1.17 ± 0.19 | 0.16 |
| PW, cm | 0.98 ± 0.12 | 0.95 ± 0.13 | 0.14 |
| LAVi ml/m$^2$ | 31.11 ± 7.95 | 31.65 ± 7.40 | 0.13 |
| E, cm/s | 65.87 ± 16.48 | 66.10 ± 17.37 | 0.46 |
| A, cm/s | 74.81 ± 20.51 | 72.55 ± 20.25 | 0.16 |
| E deceleration, msec | 200.63 ± 64.33 | 217.17 ± 71.68 | 0.07 |
| E′s, cm/s | 6.61 ± 1.65 | 6.60 ± 1.77 | 0.40 |
| E′l, cm/s | 8.10 ± 1.97 | 8.4 ± 3.00 | 0.18 |
| E′ mean, cm/s | 7.31 ± 1.70 | 7.65 ± 2.42 | 0.18 |
| E/A | 0.93 ± 0.28 | 1.01 ± 0.47 | 0.08 |
| E/E′ | 9.65 ± 3.22 | 9.46 ± 4.97 | 0.44 |
| RAVi, ml/m$^2$ | 21.96 ± 7.67 | 22.66 ± 7.79 | 0.17 |
| PAP, mmHg | 27.37 ± 4.99 | 27.70 ± 5.46 | 0.38 |
| MPI | 0.29 ± 0.07 | 0.26 ± 0.08 | 0.03 |

*EF* ejection fraction of left ventricle, *LVEDV* left ventricular end diastolic volume, *LVESV* left ventricular end systolic volume, *LVEDVi* left ventricular end diastolic volume index, *LVESVi* left ventricular end systolic volume index, *LVMi* left ventricular mass index, *RWT* relative wall thickness, *IVS* interventricular septum thickness, *PW* posterior wall thickness, *LVID* left ventricular internal diameter, *RAVi* right atrial volume index, *PAP* pulmonary artery pressure, *MPI* myocardial performance index

was evaluated in 10 patients (20 echo exams) and was ≤ 2%. [ML, ZV].

## Discussion

In the current study, the cardiac effect of prolonged HBOT protocol, used for potential regenerative purpose, was evaluated, for the first time, to assess its effect on cardiac function. The current results indicate that prolonged HBOT protocol increases left ventricular and right ventricular systolic function, and improves myocardial performance. It should be noted that the 2nd echocardiography examination post HBOT was performed at least 3 weeks after the last therapeutic session. Thus these significant changes represent a potentially sustainable structural–functional change rather than an immediate temporary change.

Most available information related to the hyperbaric environment and cardiac function in humans is referring to short term exposures. A short term exposure to the hyperbaric environment can increase peripheral vasoconstriction, increase afterload, induce bradycardia [41–43] and decrease systolic LV function [2]. A reduction in cardiac output after a single exposure to moderate level hyperbaric oxygenation was demonstrated in rats and was coordinated by a baroreflex-mediated mechanism triggered by vasoconstriction [42]. Acute pulmonary edema following hyperbaric oxygenation, is very rare, with an incidence of 0.02–0.1%. Risk factors are a low ejection fraction (< 35%) and severe aortic stenosis [44, 45]. A single exposure to hyperbaric oxygenation was associated with some changes in diastolic parameters: increase of E/A ratio and prolongation of deceleration time [46], but a short series of 10 repetitive sessions of hyperbaric oxygenation caused shortening of E wave deceleration time without changes of systolic parameters [31].

Unlike a single exposure to high pressure mentioned above, the newly used regenerative protocols of HBOT have the potential induce beneficial effects. The protocol used in the current study includes repeated 90 min daily exposures (60 sessions, 5 days per week) to lower pressures, while every 20 min there is a 5 min air break. This is a relatively new protocol used in humans to induce regenerative processes in non-healing wounds and certain brain injuries [6, 19–21]. In the current study diastolic parameters that included conventional echocardiography parameters and advanced speckle tracking parameters (diastolic strain rate, peak untwisting velocity) did not change significantly following hyperbaric oxygenation therapy. However, using advanced cardiac analysis, our results showed, for the first time in humans, a small but significant increase in left ventricular ejection fraction following hyperbaric oxygenation therapy and higher global and regional left ventricular strain. The ejection fraction increased due to a decrease in the systolic volume of the left ventricle, which reflects improved myocardial contractility. The improvement in apical strain led to a more intense apical rotation and, as a result, an increase in twisting angle. Global right ventricular strain demonstrated a small but significant improvement similar to the global left ventricular strain.

Along with normal aging there is a decrease in cardiac mitochondrial functions [47]. One of the most intriguing effects of HBOT relates to improving mitochondrial functions demonstrated in both animal and human studies [15–18, 29, 48]. Improved mitochondrial functions may explain cardiac beneficial effects in the current study cohort of "normal aging population". According to the mitochondrial theory of aging, cardiac mitochondria are not only altered by oxidative stress but also produce more antioxidative enzymes [49]. Oxidation of fatty acids in mitochondria is reduced with aging and results in an increased amount of circulating free fatty acids, that can be increased by the ischemic stress too. Cytochrome C release is increased by aging, leads to deletions in mitochondrial

**Table 3** Speckle tracking imaging results

| | 1st study | 2nd study | *p* value |
|---|---|---|---|
| RVGS[a], % | − 19.22 ± 4.45 | − 20.24 ± 4.60 | 0.02 |
| Twist, ° | 18.32 ± 6.61 | 23.12 ± 6.35 | 0.01 |
| Rotation apical, ° | 11.76 ± 4.40 | 16.10 ± 5.56 | 0.004 |
| Rotation basal, ° | − 6.80 ± 5.16 | − 6.98 ± 4.16 | 0.44 |
| Peak untwisting velocity, °/s | − 100.92 ± 67.23 | − 113.82 ± 46.67 | 0.52 |
| LVGS[b], % | − 19.31 ± 3.17 | − 20.16 ± 3.34 | 0.036 |
| Antero-basal strain, % | − 15.69 ± 2.99 | − 14.46 ± 3.46 | 0.06 |
| Mid anterior strain, % | − 18.35 ± 3.96 | − 17.54 ± 4.0 | 0.15 |
| Antero-apical strain, % | − 24.96 ± 7.35 | − 25.88 ± 6.02 | 0.25 |
| Infero-basal strain, % | − 14.50 ± 4.32 | − 15.62 ± 4.92 | 0.06 |
| Mid inferior strain, % | − 18.62 ± 4.36 | − 19.88 ± 4.86 | 0.04 |
| Infero-apical strain, % | − 26.69 ± 6.55 | − 27.81 ± 5.76 | 0.19 |
| Latero-basal strain, % | − 15.65 ± 4.17 | − 15.38 ± 3.93 | 0.40 |
| Mid lateral strain, % | − 17.23 ± 3.37 | − 17.27 ± 4.38 | 0.48 |
| Latero-apical strain, % | − 21.42 ± 6.35 | − 24.12 ± 6.19 | 0.007 |
| Septo-basal strain, % | − 12.38 ± 4.06 | − 11.62 ± 7.28 | 0.29 |
| Mid septal strain, % | − 18.35 ± 3.19 | − 19.35 ± 4.47 | 0.12 |
| Septo-apical strain, % | − 24.50 ± 5.30 | − 27.88 ± 4.39 | 0.002 |
| Antero-septal basal strain, % | − 15.62 ± 3.61 | − 17.08 ± 4.08 | 0.04 |
| Mid antero-septal strain, % | − 20.88 ± 3.68 | − 22.96 ± 4.79 | 0.02 |
| Antero-septal apical strain, % | − 24.42 ± 5.30 | − 27.85 ± 5.79 | 0.006 |
| Postero-basal strain, % | − 15.29 ± 5.09 | − 15.35 ± 4.96 | 0.48 |
| Mid posterior strain, % | − 17.54 ± 3.76 | − 17.62 ± 3.76 | 0.46 |
| Postero-apical strain, % | − 21.88 ± 4.81 | − 24.50 ± 5.24 | 0.02 |
| Global circumferential strain, % | − 19.23 ± 6.00 | − 19.81 ± 5.16 | 0.38 |
| Basal circumferential strain | − 12.76 ± 4.31 | − 13.43 ± 6.26 | 0.36 |
| Mid-ventricular circumferential strain | − 16.38 ± 5.03 | − 17.26 ± 6.96 | 0.27 |
| Apical circumferential strain | − 24.95 ± 8.13 | − 26.85 ± 11.39 | 0.25 |
| LV diastolic strain rate, 1/s | 0.90 ± 0.29 | 0.93 ± 0.44 | 0.36 |
| RV diastolic strain rate, 1/s | 0.96 ± 0.38 | 0.98 ± 0.41 | 0.45 |

[a]Right ventricular global strain

[b]Left ventricular global strain

DNA and promotes apoptosis [50]. The amplification of reactive oxygen species in the mitochondria is associated with left ventricular hypertrophy, which results in increased myocardial oxygen demand and decreased coronary perfusion pressure due to compression of coronary microcirculation. The mismatch in oxygen supply–demand induces relative ischemia in the hypertrophic heart [51]. The better availability of oxygen to the mitochondria of myocytes and the easing of the contraction process initiated by HBOT may explain the improvement in systolic function observed in our study. To the best of our knowledge, the effect of repeated HBOT exposure on cardiac function in the "standard aging population" has not yet been investigated. We emphasize that it is not clear when during the course of therapy improvement starts. We observed a sustained effect after the completion of the entire course of 60 sessions. Does improving systolic contraction means improving heart function? Improvement in myocardial performance index which reflects the effectiveness of the total systolic and diastolic function of the left ventricle may indicate the entire positive effect of hyperbaric oxygenation on the heart.

## Limitations

There are several limitations: this was not a randomized study, but rather an observational prospective evaluation, on a limited number of participants. Clearly, a study on a larger and more diverse cohort is recommended to further evaluate the effect of HBOT on cardiac function. Nevertheless, these observations are intriguing, and we believe—original, and should be further pursued.

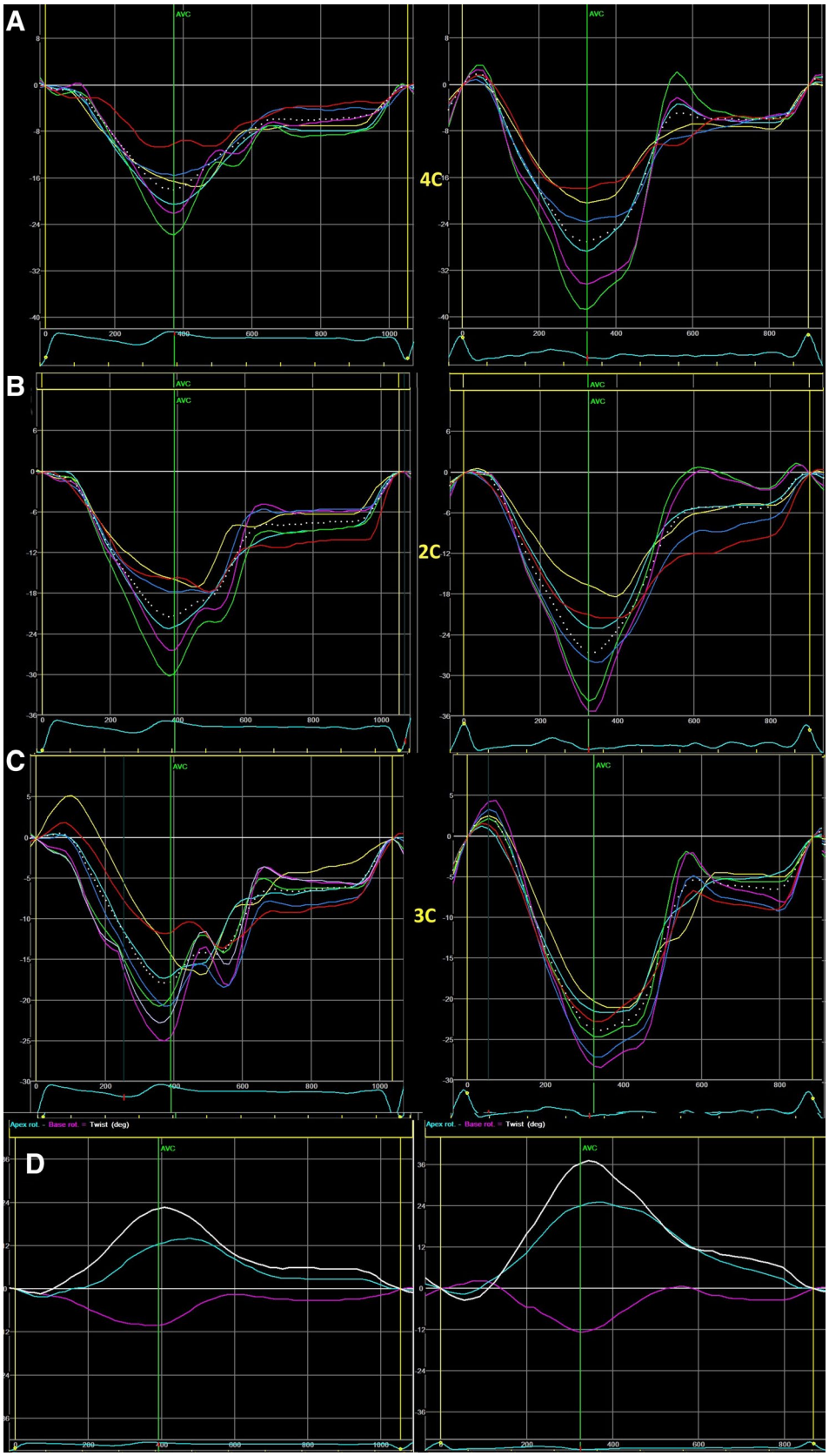


**Fig. 1** Speckle tracking imaging results of a 68-year old healthy man before [Left image] and after hyperbaric oxygenation therapy [Right image]. **a** Apical 4-chamber view, regional longitudinal strain: yellow—septal basal, bright blue—mid-septal, green—septo-apical, purple—latero-apical, blue—mid-lateral, red—lateral basal. Dotted curve—global strain from the 4-chamber view. In all the segments regional strain after hyperbaric oxygenation is higher, especially apical strain [green and purple curves]. **b** Apical 2-chamber view, regional longitudinal strain: yellow—infero-basal, bright blue—mid-inferior, green—infero-apical, purple—antero-apical, blue—mid-anterior, red—antero-basal. Dotted line—global strain from the 2-chamber view. The regional strain is higher after hyperbaric oxygenation therapy, especially in the apical segments. **c** Apical 3-chamber view, regional strain: yellow—postero-basal, bright blue—mid-posterior, green—postero-apical, purple—antero-septo-apical, blue—mid-septal, red—antero-septal basal. Dotted line—global strain from the 3-chamber view. The regional strain is higher in the apical and septal segments after hyperbaric oxygenation therapy. **d** Twist and rotation of the left ventricle, obtained from the short axis basal and apical views. White curve—twist, blue—apical rotation, purple—basal rotation. After hyperbaric oxygenation therapy twist is higher primarily due to higher apical rotation

## Conclusions

Our results indicate that hyperbaric oxygenation led to an improved systolic contraction of the left ventricle, a better systolic contraction of the right ventricle and, finally, improved myocardial performance. If our results are confirmed in further, larger studies, HBOT may potentially be used in the future in patients with heart failure and systolic dysfunction.

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### Compliance with ethical standards

**Conflict of interest** There is no conflict of interests.

**Ethical approval** Our study was approved by the local Helsinki Committee and was done in accordance with the ethical standards of the local Helsinki Committee.

**Informed consent** Informed consent was obtained from all individual participants included in the study.

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