How to Cite:
Abu Shabana, S. M., Elshal, A. M., Elhadidy, T., Shalabi, N. M., Gomaa, G. F., & Saleh, A. M. (2022). Impact of nocturnal hypoxia and hypercapnea on global myocardial performance in patients with sleep related breathing disorders. *International Journal of Health Sciences*, *6*(S9), 2565–2577. https://doi.org/10.53730/ijhs.v6nS9.12985

Impact of nocturnal hypoxia and hypercapnea on global myocardial performance in patients with sleep related breathing disorders

Shaimaa M. Abu Shabana
Chest Medicine specialist, Mansoura chest hospital, Egypt
Email: dr.sh.shabana@gmail.com

Abdallah M Elshal
Department of cardiology Medicine, Faculty of Medicine, Mansoura University, Egypt

Tamer Elhadidy
Department of chest Medicine, Faculty of Medicine, Mansoura University, Egypt

Nesrien. M. Shalabi
Department of chest Medicine, Faculty of Medicine, Mansoura University, Egypt

Gamal F Gomaa
Department of cardiology Medicine, Faculty of Medicine, Mansoura University, Egypt

Abdelbaset M. Saleh
Department of chest Medicine, Faculty of Medicine, Mansoura University, Egypt

Abstract---Background: Sleep related breathing disorders (SRBDs) are disorders caused by a decline or interruption in breathing during sleep and can be complicated by cardiovascular complications. Objective: The aim of this study was to evaluate the impact of chronic intermittent hypoxia and nocturnal hypercapnia on global myocardial performance utilizing different modes of echocardiography in patients with SRBDs. Methods: This a case control study was conducted on 142 adult subjects who attended to outpatient clinics and Mansoura university sleep center, Egypt during the period study from august 2016 till august 2018. Results: There were statistically significant higher BMI and NC in patients with OSA, OHS groups versus the control group ($p<0.001$). Comparing OSA& OHS groups with control group, statistically significant difference regarding each of MPI, GLS, TAPSE (mm) and grade of DD by TDI ($p<0.05$), GLS, TAPSE (mm) and grade of DD by TDI respectively. Speckle tracking echocardiography

International Journal of Health Sciences ISSN 2550-6978 E-ISSN 2550-696X © 2022.
Manuscript submitted: 9 May 2022, Manuscript revised: 18 July 2022, Accepted for publication: 27 August 2022

showed statistically significant differences all over the 17 segment of the LV and the most affected segments are Inferolateral, Inferior and Inferoseptal and showed that basal segments more affected than mid and apical segments.Conclusion: there is cardiovascular comorbidity associated with chronic intermittent hypoxia and nocturnal hypercapnia in patients with OSA and OHS.

***Keywords*---**intermittent Hypoxia, nocturnal Hypercapnia, Global Myocardial Performance, Sleep Related Breathing Disorders.

Introduction

Sleep-related breathing disorders are conditions of abnormal and difficult respiration during sleep, including chronic snoring and sleep apnea. Some sleep-related breathing disorders have limited health impact, but others can have serious consequences because of their potential effects on sleep and the balance of oxygen and carbon dioxide in the blood (Kakarla, 2021).

Data on the prevalence of sleep-disordered breathing (SDB) in the African general population are scarce, and a better understanding is urgently needed. The BeSAS study provides the first large-scale objective evaluation of SDB prevalence and associated factors in Africa. The high prevalence of SDB identified should stimulate the development of public health policies to prevent and treat this condition in African countries (Wachinou et al., 2022). Both in general population and in those with cardiovascular diseases (CVD), OSAS is 2 to 3 times more common in men than in women and in older adults than in the young (Albuquerque et al, 2012).

The role of capnometry in adult polysomnography has been more limited, usually for the quantitative assessment of sleep hypoventilation syndromes (Iber etal, 2007) or for the measurement of end-tidal CO2 (ETCO2) as asignal for the detection of airflow obstruction in sleep apnea (Weihu etal, 2011). Few studies are available to assess the impact of chronic intermittent hypoxia with nocturnal hypercapnia on global myocardial performance in patients with Sleep-related breathing disorders and this is the aim of this study.

Subjects and Methods

This a case control study was conducted on 142 adult subjects who attended to outpatient clinics and Mansoura university sleep center, chest department at Mansoura University Hospital, Dakahlia, Egypt during the period study from august 2016 till august 2018. After chest department committee approval, the study protocol was approved by Institution Review Board (IRB) code number: MD/16.07.45, Mansoura University. Informed written consent was obtained from each participant sharing in. Confidentiality and personal privacy respected in all levels of the study. Collected data not used for any other purpose.

The studied subjects were divided into three groups: OSA group: Included 92 patients who had symptoms consistent with OSA and confirmed as OSA with

polysomnography (AHI≥5 events/hour). OHS group: Included 20 patients who had symptoms consistent with SRBDs and confirmed as OHS with polysomnography and exclusion of other causes of alveolar hypoventilation. Control group: Included 30 subjects who were free from SRBDs symptoms, and their AHI were <5 events/hour.

Inclusion criteria: Age: ≥ 18 Years' old, Sleep-related breathing disorders: Patients in this study was diagnosed as have SRBDs after full night polysomnography according to American Academe of sleep Medicine **(AASM manual, 2014).**

Exclusion criteria: patients with chronic pulmonary diseases (COPD, BA, IPF…) and smokers, patients with any cardiac diseases as heart failure, rheumatic heart diseases, coronary artery disease and myocardial infarction (MI), patients with active infectious disease, patients with serious co-morbidity such as chronic or decompensated liver disease, chronic kidney disease, malignancy and thromboembolic disease, patients with neuromuscular disorders or with previous trauma to the head and neck and kyphoscoliosis and pregnancy.

Methods
All subjects included in this study underwent:

Full history taking including: A detailed medical history was taken according to a specifically designed protocol which included demographic data (name, age, sex and special habits particularly smoking status). Attention was given to history suggestive of SRBDs: Night symptoms as snoring, chocking and witnessed apnea, bad dreams and nocturia, and daytime symptoms (morning headache, awakening dry mouth and excessive daytime sleepiness. Medical history of any comorbid diseases. **Physical examination including:** Anthropometric measures: included body-mass index (BMI), neck circumference, Systolic and diastolic blood pressure, respiratory rate, heart rate and neck veins and lower limbs examination. Upper airway examination was done and included: **Tonsillar examination:** Tonsil size is graded from 0 to 4**, Friedman tongue position (FTP):** graded from I to IV**, Modified Mallampati score (MMP):** graded from I to IV **(NGC, 2014). Scales and Questionnaires:** From clinical assessment patients were given score according to these questionnaires**: Epworth Sleepiness Scale (ESS), berlin Questionnaire (BQ) and STOP-Bang Questionnaire.**
Laboratory investigation: Morning Arterial blood gases (ABGs), complete blood count (CBC), thyroid function (TSH & free T4), lipid profile, liver function tests and serum creatinine.

Electrocardiography (ECG)

Radiological study: Chest x-ray.
Pulmonary function test: Spirometry was performed by (smart PFT CO) manufactured by medical equipment Europe-Hammet Burg- Germany.
Polysomnography (PSG): Full night attended Polysomnography using SOMNO screen™ plus, SOMNO medics, Randersacker, Germany, SN: 6357
Capnometry for end-tidal CO2 (ETCO2): The etCO2 values were transferred to the SOMNO screen™ using an adapter. The DOMINO software displayed a

numerical value in mmHg In addition, a CO2 waveform (capnogram) was displayed. **Echocardiography:** All patients underwent transthoracic echocardiography using of Vivid E9 XD clear Dimensions ultrasound machine, manufactured in 2008 by General Electric Company in Norway, using M5S transducer. The procedure was performed with the patient breathing quietly and lying in the left lateral position. Two-dimensional-2D, M-mode echocardiography, Pulsed-wave Doppler (PWD), Tissue Doppler Imaging (TDI) and speckle tracking were used to assess LV morphology and function.

Pulsed-wave Doppler (PWD) echocardiography was carried out to assess LV diastolic function. In the apical 4-chamber view, with the sample volume placed at the mitral valve leaflet tips, transmitral flow velocities (early-E and late-A diastolic filling velocities, DT-deceleration time, E/A ratio) were recorded.

Speckle tracking echocardiography

Speckle tracking was done by Automated Function Imaging (AFI) which is a software tool that systematizes 2D speckle tracking after obtaining real time apical views including apical four, three and two chambers' views to measure in real-time regional as well as global longitudinal strain of the myocardial wall.

Ethical considerations

All the candidates participated in this study were enrolled voluntary with having the right to freely withdraw from the study at any time without any affection on the provided medical service.

Statistical analysis

Data were analyzed using the Statistical Package of Social Science (SPSS) program for Windows (Standard version 21). The normality of data was first tested with one-sample Kolmogorov-Smirnov test. The two groups were compared with Student t test for parametric data and Mann Whitney test for non-parametric data. Pearson correlation: correlate quantitative data (parametric). Spearman correlation: correlate quantitative data (non-parametric).
Results

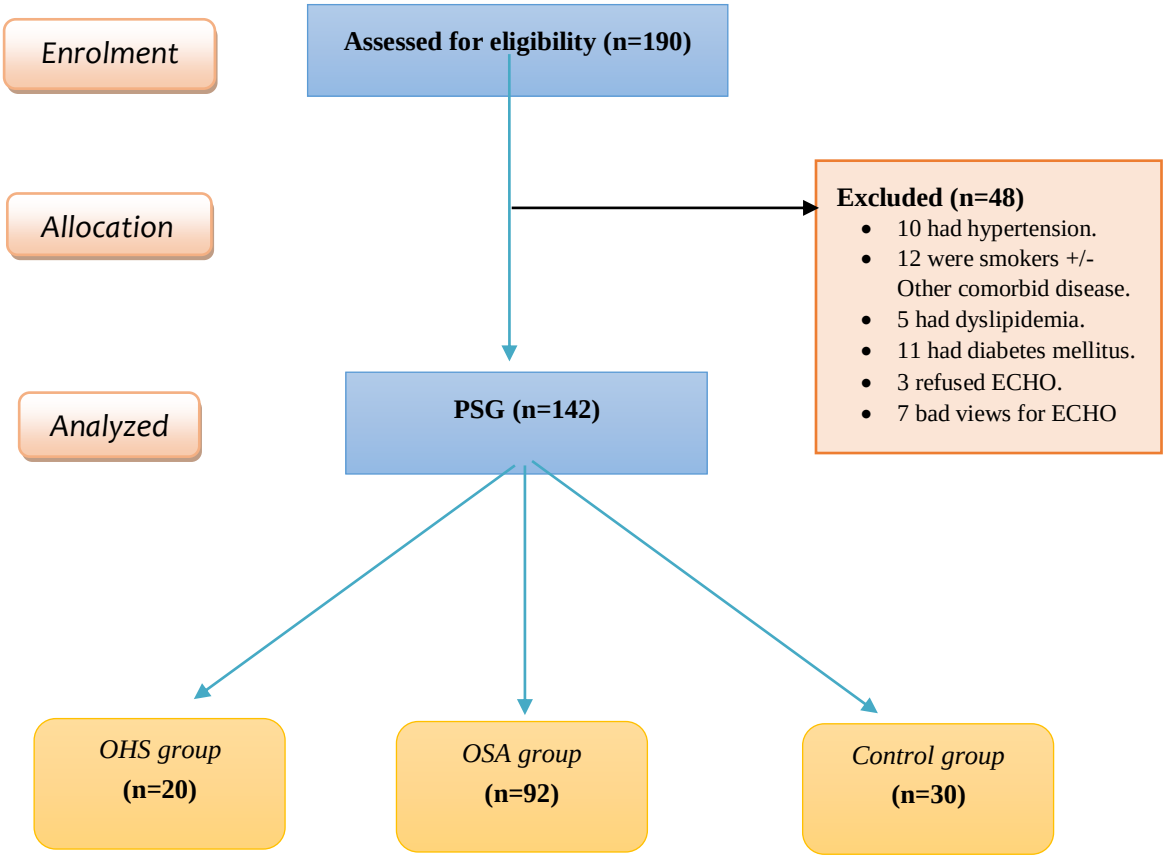

Figure (1): Flowchart of the studied patients and healthy groups.

TABLE (1): correlation between ABG and studied groups. First, when comparing OSA group with control group, statistically significant differences were found between the two groups **regarding SaO2, PCO2** ($p<0.05$) And HCO3 ($p<0.001$). Second, when comparing OHS with control group, statistically significant differences were found between the two groups regarding each of PCO2 and HCO3 ($p<0.001$).

Table (1): Blood gases among studied groups

ABG	OSA group (n=92)	OHS group (n=20)	Control group (n=30)	Test of significance		
				P1	P2	P3
PH	7.38±0.02	7.38±0.03	7.38±0.03	t=0.825 p=0.411	t=0.112 p=0.912	t=0.567 P=0.572
PCO2	41.57±3.66	41.96±3.21	38.96±2.61	t=3.61 p=0.001*	t=3.62 p=0.001*	t=0.442 P=0.66
PO2	82.55±7.29	84.42±2.73	84.25±2.71	t=1.24 p=0.217	t=0.222 p=0.825	t=1.12 P=0.263

SaO2	95.50±1.31	96.10±1.07	96.17±1.08	t=2.51 p=0.013*	t=1.38 p=0.172	t=1.91 P=0.06
HCO3	24.64±2.19	25.02±1.27	23.45±1.58	t=2.74 p=0.007*	t=3.72 p=0.001*	t=0.752 P=0.454

t: student t- test, ***** statistically significant (if p<0.05), **OSA**: obstructive sleep apnea, **OHS:** obesity hypoventilation syndrome, PO**2:** Partial pressure of oxygen, **PCO2:** partial pressure of carbon dioxide, **HCO3:** bicarbonate., **p1:** compare OSA with control group, **p2:** compare OHS with control group, **p3:** compare OSA with OHS group

Table (2) displays correlation between AHI and O2 and CO2 parameters. According to the current results, statistically significant and positive correlations were found between AHI and each of number of desaturations <90%, number of desaturations <80%, average desaturation, ODI, average CO2 REM, Average CO2 NON-REM **(P ≤0.001),** On the other hand, there were statistically significant and negative correlations between AHI and each of basal SpO2, average SpO2 (p<0.05). AND minimum SpO2 **(P ≤0.001).**

Table (2): Correlation between AHI and O2 and CO2 parameters

	AHI	
	r	P value
Basal SpO2	-0.256	0.014*
Minimum SpO2	-0.578	≤0.001*
Average SpO2	-0.241	0.021*
No of desaturation <90%	0.704	≤0.001*
No of desaturation <80%	0.367	≤0.001*
Average desaturation	0.608	≤0.001*
ODI	0.710	≤0.001*
average CO2 REM	0.239	0.008*
Average CO2 NON-REM	0.326	≤0.001*
MAX. CO2 REM	0.173	0.057
MAX. CO2 NON-REM	0.168	0.064
Max. Co2 sleep	0.146	0.109
MIN. CO2 REM	0.166	0.067
MIN. CO2 non-REM	0.051	0.580

Table (3): when comparing OSA group with control group, statistically significant differences were found between the two groups regarding each of MPI, GLS, TAPSE (mm) and grade of DD by TDI (p<0.05) and when comparing OHS group with control group, statistically significant differences were found between the two groups regarding each of GLS, TAPSE (mm) and grade of DD by TDI (P≤0.001)

Table (3): ECHO data among studied groups

ECHO data	OSA group (n=92)	OHS group (n=20)	Control group (n=30)	Test of significance		
				P1	P2	P3
EF %	66.95±6.36	70.15±5.20	68.16±6.57	t=0.89 p=0.37	t=1.13 p=0.263	t=2.09 p=0.038*
MPI	0.48±0.12	1.21±0.04	0.39±0.02	t=4.01 p≤.001*	t=1.29 p=0.201	t=2.03 p=0.045*
GLS %	-16.65±6.06	-15.55±1.69	-19.13±0.51	t=1.83 p=0.027*	t=10.93 p≤.001*	t=0.99 p=0.324
TAPSE mm	19.34±2.54	14.95±1.14	21.00±1.85	t=3.28 p=0.001*	t=12.99 p≤.001*	t=7.54 p≤.001*
Grade of DD by PWD Normal I II III	 20 (21.7%) 64 (69.6%) 8 (8.7%) 0 (0%)	 4 (20.0%) 16 (80.0%) 0 (0%) 0 (0%)	 5 (16.7%) 25 (83.3%) 0 (0%) 0 (0%)	x^2=3.48 p=0.18	x^2=0.09 p=0.764	x^2=2.01 p=0.37
Grade of DD by TDI: Normal I II III	 14 (15.2%) 51 (55.4%) 11 (12.0%) 16 (17.4%)	 3 (15.0%) 9 (45.0%) 8 (40.0%) 0 (0%)	 5 (16.7%) 25 (83.3%) 0 (0%) 0 (0%)	x^2=11.66 p=0.009*	x^2=14.61 p=.001*	x^2=11.4 p=0.01*

x2: Chi square test**, *** statistically significant (if p<0.05), **OSA:** obstructive sleep apnea hypopnea, **EF:** ejection fraction, **MPI:** Myocardial performance index, **DD:** diastolic dysfunction**, GLS**: Global Longitudinal Strain, **TAPSE:** Tricuspid annular plane systolic excursion. **PWD:** Pulsed-Wave Doppler. **TDI**: Tissue Doppler imaging. P1: compare OSA group with control, P2: compare OHS group with control, P3: compare OSA group with OHS group

Table (4): show Comparison between the studied groups as regarding myocardial segmental affection with significant differences between the studied groups all over the 17 segment of the LV according to AHA classification but the most affected segments are **Inferolateral**, **Inferior** and **Inferoseptal ,** and showed that basal segments more affected than mid and apical segments.

Table (4): Comparison between the study groups regarding left ventricular segmental affection:

ECHO data	OSAH group (n=92)	OHV group (n=20)	Control group (n=30)	Test of significance		
				P1	P2	P3
Basal Anterosep <-16 (Normal) >-16 (affected)	-14.14±2.81 30 (32.6%) 62 (67.4%)	-14.60±1.9 8 (40.0%) 12 (60.0%)	-20.10±2.05 30 (100%) 0 (0%)	t=10.67 p≤.001*	t=9.53 p≤.001*	t=0.693 p=0.490

Basal Anterior <-16 (Normal) >-16 (affected)	-17.98±2.82 69 (75.0%) 23 (25.0%)	-16.55±1.31 17 (85.0%) 3 (15.0%)	- 20.46±1.13 30 (100%) 0 (0%)	t=4.66 p≤.001*	t=11.20 p≤.001*	t=2.21 p=0.029 *
Basal AnteroLat <-16 (Normal) >-16 (affected)	-19.50±2.18 92 (100%) 0 (0%)	-18.75±0.71 20 (100%) 0 (0%)	- 20.90±1.49 30 (100%) 0 (0%)	t=3.26 p=.001*	t=5.98 p≤.001*	t=1.51 p=0.133
Basal InferoLat <-16 (Normal) >-16 (affected)	-9.16±6.21 3 (3.3%) 89 (96.7%)	-9.70±4.49 0 (0%) 20 (100%)	- 18.66±1.02 30 (100%) 0 (0%)	t=8.31 p≤.001*	t=10.56 p≤.001*	t=0.365 p=0.715
Basal Inferior <-16 (Normal) >-16 (affected)	-7.54±5.34 0 (0%) 92 (100%)	-9.05±3.54 0 (0%) 20 (100%)	- 17.86±0.68 30 (100%) 0 (0%)	t=10.52 p≤.001*	t=13.31 p≤.001*	t=1.20 p=0.232
Basal Inferosep <-16 (Normal) >-16 (affected)	-16.76±2.88 71 (77.2%) 21 (22.8%)	-14.90±1.37 9 (45.0%) 11 (55.0%)	- 18.70±0.59 30 (100%) 0 (0%)	t=3.64 p≤.001*	t=13.43 p≤.001*	t=2.80 p=0.006 *
Mid Anterosep <-16 (Normal) >-16 (affected)	-17.60± 1.35 90 (97.8%) 2 (2.2%)	-16.60±1.18 19 (95.0%) 1 (5.0%)	- 20.50±1.57 30 (100%) 0 (0%)	t=9.73 p≤.001*	t=9.44 p≤.001*	t=3.07 p=0.003 *
Mid Anterior <-16 (Normal) >-16 (affected)	-17.98±2.82 69 (75.0%) 23 (25.0%)	-16.55±1.31 17 (85.0%) 3 (15.0%)	- 20.46±1.13 30 (100%) 0 (0%)	t=4.66 p≤.001*	t=11.20 p≤.001*	t=2.21 p=0.029 *
Mid Anterolat <-16 (Normal) >-16 (affected)	-19.50±2.18 92 (100%) 0 (0%)	-18.75±0.71 20 (100%) 0 (0%)	- 20.90±1.49 30 (100%) 0 (0%)	t=3.26 p=.001*	t=5.98 p≤.001*	t=1.51 p=0.133
Mid InferoLat <-16 (Normal) >-16 (affected)	-10.40± 5.22 7 (7.6%) 85 (92.4%)	-11.20±3.17 0 (0%) 20 (100%)	- 18.66±1.02 30 (100%) 0 (0%)	t=8.58 p≤.001*	t=12.03 p≤.001*	t=0.655 p=0.514
Mid Inferior <-16 (Normal) >-16 (affected)	-10.31±3.67 1 (1.1%) 91 (98.9%)	-10.60±2.79 0 (0%) 20 (100%)	- 19.36±0.99 30 (100%) 0 (0%)	t=13.31 p≤.001*	t=15.78 p≤.001*	t=0.327 p=0.745
Mid Inferosep <-16 (Normal) >-16 (affected)	-16.91±2.94 71 (77.2%) 21 (22.8%)	-17.60±1.09 20 (100%) 0 (0%)	- 18.70±0.59 30 (100%) 0 (0%)	t=3.28 p=.001*	t=4.58 p≤.001*	t=1.02 p=0.308
Apical Anterior <-16 (Normal) >-16 (affected)	-19.21±1.98 89 (96.7%) 3 (3.3%)	-18.55±0.75 20 (100%) 0 (0%)	- 20.46±1.13 30 (100%) 0 (0%)	t=3.31 p=.001*	t=6.61 p≤.001*	t=1.45 p=0.149

Apical Lateral <-16 (Normal) >-16 (affected)	-12.00±5.11 25 (27.2%) 67 (72.8%)	-18.45±2.83 18 (90.0%) 2 (10.0%)	-20.40±0.96 30 (100%) 0 (0%)	t=8.93 p≤.001*	t=3.48 p=.001*	t=5.45 p≤.001*
Apical Inferior <-16 (Normal) >-16 (affected)	-12.22± 3.71 13 (14.1%) 79 (85.9%)	-11.35±3.08 0 (0%) 20 (100%)	-19.16±1.34 30 (100%) 0 (0%)	t=9.99 p≤.001*	t=12.29 p=.001*	t=0.985 p=0.327
Apical Septum <-16 (Normal) >-16 (affected)	-17.60± 1.35 90 (97.8%) 2 (2.2%)	-16.60±1.18 19 (95.0%) 1 (5.0%)	-20.50±1.57 30 (100%) 0 (0%)	t=9.73 p≤.001*	t=9.44 p≤.001*	t=3.07 p=0.003 *
Apical Apex <-16 (Normal) >-16 (affected)	-18.77±3.46 85 (92.4%) 7 (7.6%)	-18.45±2.18 19 (95.0%) 1 (5.0%)	-20.90±1.49 30 (100%) 0 (0%)	t=3.26 p=.001*	t=4.71 p≤.001*	t=0.398 p=0.691

P1: compare OSAH and control groups
P2: compare OHV and control groups
P3: compare OSAH and OHV groups

Discussion

In our result, the mean age in OSA group was 43.60±8.58 years, and in OHS group was (38.65±8.39) while in control group it was 41.23±10.05 years. The majority of subjects in both OSA and control groups (69.6% of OSA group, and 66.7% of control group) were males while OHS group were mainly female (70%). Statistically highly significant differences were found between the studied groups as regards BMI, and NC **(p<0.001).**

The prevalence of OHS has been reported to be higher in men; however, among patients referred to the sleep disorders clinic, OHS was more prevalent in women than men (BaHammam et al, 2016). The delay in identifying OHS in women was linked to a worse and more advanced consequence of the disease (Palm et al, 2016).

In this concern, several previous studies demonstrated higher BMI and NC in OSA patients. For instance, Kang et al., (2014) found that patients with OSA had a significantly higher BMI (26.8±3.9 vs. 23.1±2.9 kg/m2 in patients without OSA, P < 0.001) and significantly higher NC than patients without OSA (38.1± 3.6 vs. 34.4±3.2 cm, respectively, P < 0.001). Also, a study done by Kim et al., (2015) demonstrated significant higher BMI in OSA patients versus non OSA patients (26.0 vs. 24.2 kg/m2, respectively, P= 0.0204) and significant higher NC in OSA patients (38.7 ±3.0 vs. 35.6 ±3.3 cm in non OSA patients, P< 0.001).

In the current study, there was a significant difference between OSA and control groups regarding SaO2, PCO2 and HCO3. Also, significant differences were found between OHS and control group regarding each of PCO2 and HCO3. The presence of chronic hypercapnia in OSAS has previously been attributed to a variety of factors including underlying lung disease, presence of coexisting central hypoventilation, impaired ventilatory response to CO2, and obesity (Berger et al.,

2001). Additionally, Eskandari et al., (2017) found a significant change in arterial blood gases in higher AHI severity classes. Mean pO2 decreased with approximately 0.5 kPa from mild to severe OSA patients ($p < 0.001$), mean pCO2 increased slightly in patients with severe OSA ($p= 0.046$) and HCO3- was positively correlated with AHI. While, pH values did not change along with severity class of OSA

The results of a pervious study revealed a direct correlation between the OSA severity based on the AHI and SpO2 parameters (Wali et al., 2020). Polysomnography is the gold standard examination method for the diagnosis of OSA. ODI (which is the mean number of desaturation events that occur within an hour) is another parameter used for increasing the diagnosing and grading the quality of PSG. Several studies have reported a strong correlation between AHI and ODI (Temirbekov ET AL., 2018)

Consistent with our results; Kawata et al., (2007) demonstrated that fourteen percent (168 of 1,227 patients) exhibited daytime hypercapnia and these patients had higher AHI values compared with normocapnic patients, and logistic regression analysis showed that only AHI was a predictor of daytime hypercapnia. Also, Kepez et al., (2009) revealed significant lower nocturnal mean oxygen saturation with higher AHI. Also, similar to our results; Haruki et al., (2009) demonstrated significant differences as regard lowest O2 saturation and saturation time < 90% between OSA patients and control group. Also; a pervious study by Arias et al., (2005) revealed significant higher saturation time < 90% and significant lower mean nocturnal SaO2 and minimum SaO2 in OSA patients versus control group.

The present study showed that there was a statistically significant and positive correlations between AHI and each of GLs% and grade of DD by TDI ($P\leq0.001$). TAPSE (mm) and grade of DD by PWD ($p<0.05$). In particular, an analysis of the global longitudinal strain (GLS) is a novel index for the assessment of the LV systolic function and subtle deteriorations (Reisner et al., 2004).

Moreover, Deng et al., 2021 also found that severe OSAS patients had slight LVH. Also, IVS and LVPW diameters, and LVM and LVMI, were slightly higher in patients with severe OSAS, while they were within normal limits in patients with mild and moderate OSAS. This study does not explain the reason for LVH; however, it could be caused by high blood pressures and/or nocturnal hypoxemia.

Al Otair and his colleagues found positive correlation between PaCO2 and LVDD and negative correlation between PaO2 and LVDD in OSA patients but without significant difference (Al Otair et al., 2018). These data may partly depend on the patient selection and use of only conventional Doppler, which is not accurate for detecting early diastolic dysfunction in patients with a normal ejection fraction (EF) (Ommen et al., 2000).

In accordance with the current results, Cho et al., 2012 showed that only the GLS was identified as the best index to demonstrate an association between global LV dysfunction and the severity of OSA independently of the BMI, diabetes, and

hypertension. This myocardial dysfunction assessed by the GLS and Tei index improved after the surgical modification of the airway, and additionally there was a reduction in the LV filling pressure. In the present study, a significant difference found between OSA and control groups regarding each of MPI, GLS, TAPSE (mm) and grade of DD by TDI. Also, significant differences were found between OHS and control groups regarding each of GLS, TAPSE (mm) and grade of DD by TDI. While a significant difference was found between OSA and OHS groups regarding each of EF %, MPI, TAPSE (mm) and grade of DD by TDI.

In this concern, previous studies assessed right ventricular MPI by conventional PWD echocardiography. For instance; Duman et al., (2008) found that right ventricular MPI measured by PWD was elevated in children with adenotonsillar hypertrophy (ATH), but these authors did not confirm the presence of OSAS by polysomnography (PSG). Rather, they used the OSAS severity score. Additionally, they did not investigate left ventricular MPI. Using PWD, Chan et al., (2009) also observed a higher right ventricular MPI in children with moderate to severe OSAS (confirmed by PSG) than in those with mild OSAS and those without OSAS.

Consistent to prior reports; *Deng et al., 2021* also showed that the values of IVS and LVIDs were higher in patients with severe OSAHS than those without OSAHS or those with less severe OSAHS. Furthermore, they also observed that the LVM and LVMI-height were also significantly higher in patients with severe OSAHS. These findings strongly indicate that presence of OSAHS is significantly associated with LV remodeling.

Moreover, *Deng et al., 2021* also found that severe OSAS patients had slight LVH. Also, IVS and LVPW diameters, and LVM and LVMI, were slightly higher in patients with severe OSAS, while they were within normal limits in patients with mild and moderate OSAS. This study does not explain the reason for LVH; however, it could be caused by high blood pressures and/or nocturnal hypoxemia.

A statistically significant differences were observed between the studied groups all over the 17 segment of the LV according to AHA classification but the most affected segments are Basal InferoLateral (96.7%), (100%) in both OSA and OHS respectively, Basal Inferior (100%) in both groups OSA and OHS, Basal Inferoseptal by (55.0%) in OHS group, Mid InferoLateral (92.4%), (100%) in both OSA and OHS respectively, Mid Inferior (98.9%), (100%) in both OSA and OHS respectively, Apical Lateral (72.8%) in OSA group and Apical Inferior (85.9%), (100%) in both OSA and OHS respectively. Basal Anteroseptal segment also affected in both OSA and OHS (67.4%), (60.0%) respectively. and showed that basal segments more affected than mid and apical segments.

Conclusion

There is Impact of chronic intermittent hypoxia and nocturnal hpercapnia on myocardial performance in OSA & OHS patients assessed by different modes of echocardiography as regard TAPSE , GLS% AND subtle diastolic dysfunction .
This study not only assess the impact of chronic intermittent hypoxia and nocturnal hypercapnia in patients with sleep related breathing disorders (OSA

and OHS) on global myocardial performance but also shed the light on main segmental myocardial affection through utilizing speckle tracking which showed that inferolateral, inferior and inferoseptal regions mostly affected than other regions and basal segments of them mostly affected than mid and apical segments

References

AASM (2014): American Academy of Sleep Medicine. International classification of Sleep Disorders, 3rd edn. American Academy of Sleep Medicine, Darien, IL.

Al Otair HA, Elshaer F, Elgishy A, Nashwan SZ, Almeneessier AS, Olaish AH and BaHammam AS (2018): Left ventricular diastolic dysfunction in patients with obesity hypoventilation syndrome. Journal of Thoracic Disease; 10(10): 5747-5754.

Albuquerque FN, Calvin AD, Sert Kuniyoshi FH et al. (2012): Sleep-disordered breathing and excessive daytime sleepiness in patients with atrial fibrillation. Chest; 141:967–973.

Arias MA, García-Río F, Alonso-Fernández A, Mediano O, Martínez I, Villamor J (2005): Obstructive sleep apnea syndrome affects left ventricular diastolic function: effects of nasal continuous positive airway pressure in men. Circulation. 2005 Jul 19;112(3):375-83.

BaHammam AS, Pandi-Perumal SR, Piper A, Bahammam SA, Almeneessier AS, Olaish AH and Javaheri S (2016): Gender differences in patients with obesity hypoventilation syndrome. J Sleep Res. 2016 Aug; 25(4):445-53.

Berger KI, Ayappa I, Chatr-amontri B, Marfatia A, Sorkin IB, Rapoport DM and Goldring RM (2001): Obesity hypoventilation syndrome as a spectrum of respiratory disturbances during sleep. Chest. 2001 Oct 1;120(4):1231-8.

Chan JY, Li AM, Au CT, Lo AF, Ng SK, Abdullah VJ and Wing YK (2009): Cardiac remodelling and dysfunction in children with obstructive sleep apnoea: a community based study. Thorax, 64(3), 233-239

Cho KI, Kwon JH, Kim SM, Park TJ, Lee HG and Kim TI (2012): Impact of obstructive sleep apnea on the global myocardial performance beyond obesity. Echocardiography, 29(9), 1071-1080.

Deng M, Huang YT, Xu JQ, Ke X, Dong YF and Cheng XS (2021): Association Between Intermittent Hypoxia and Left Ventricular Remodeling in Patients with Obstructive Sleep Apnea-Hypopnea Syndrome. Frontiers in Physiology, 11, 1808.

Duman D, Naiboglu B, Esen HS, Toros SZ and Demirtunc R (2008): Impaired right ventricular function in adenotonsillar hypertrophy. The international journal of cardiovascular imaging, 24(3), 261-267.

Eskandari D, Zou D , Grote L, Schneider H, Penzel T and Hedner J (2017): Independent associations between arterial bicarbonate, apnea severity and hypertension in obstructive sleep apnea. Respiratory Research; 18:130.

Haruki N, Takeuchi M, Nakai H, Kanazawa Y, Tsubota N, Shintome R, Lang RM, Otsuji Y (2009): Overnight sleeping induced daily repetitive left ventricular systolic and diastolic dysfunction in obstructive sleep apnoea: quantitative assessment using tissue Doppler imaging. European Journal of Echocardiography.1;10(6):769-75.

Iber, C. (2007). The AASM manual for the scoring of sleep and associated events: Rules. Terminology and Technical Specification .

Kakarla S (2021): Sleep Related Breathing Disorders. J Sleep Disor: Treat Care 10:1.

Kang HH, Kang JY, Ha JH, Lee J, Kim SK, Moon HS, Lee SH (2014): The associations between anthropometric indices and obstructive sleep apnea in a Korean population. PLoS One ;9(12):e114463.

Kawata N Tatsumi K, Terada J, Tada Y, Tanabe N, Yuichi T and Kuriyama T (2007): Daytime hypercapnia in obstructive sleep apnea syndrome. Chest; 132 (6): 1832-8.

Kepez A, Niksarlioglu EY, Hazirolan T, Ranci O, Kabul HK, Demir AU, Kaya EB, Kocabas U, Aytemir K, Sahin A, Tokgozoglu L (2009): Early myocardial functional alterations in patients with obstructive sleep apnea syndrome. Echocardiography ;26(4):388-96.

Kim SE, Park BS, Park SH, Shin KJ, Ha SY, Park J, Park KM (2015): Predictors for presence and severity of obstructive sleep apnea in snoring patients: significance of neck circumference. Journal of Sleep Medicine. Dec 31;12(2):34-8.

National Guidelines Clearinghouse (NGC) (2014): Management of obstructive sleep apnoea/hypopnoea syndrome in adults. A national clinical guideline. Available from: http://www.guideline.gov/content.aspx?id=3878

Ommen SR, Nishimura RA, Appleton CP, Miller FA, Oh JK, Redfield MM and Tajik AJ (2000): Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: a comparative simultaneous Doppler-catheterization study. Circulation, 102(15), 1788-1794.

Palm A, Midgren B, Janson C, Lindberg E. (2016): Gender differences in patients starting long-term home mechanical ventilation due to obesity hypoventilation syndrome. Respir Med.110:73-8.

Reisner SA, Lysyansky P, Agmon Y, et al (2004): Global longitudinal strain: A novel index of left ventricular systolic function. J Am Soc Echocardiogr, 17: 630– 633.

Temirbekov, D., Güneş, S., Yazıcı, Z. M., & Sayın, İ. (2018): The ignored parameter in the diagnosis of obstructive sleep apnea syndrome: the oxygen desaturation index. *Turkish archives of otorhinolaryngology*, *56*(1), 1.Wali SO, Abaalkhail B, AlQassas I, Alhejaili F, Spence DW, Pandi-Perumal SR (2020): The correlation between oxygen saturation indices and the standard obstructive sleep apnea severity. Annals of thoracic medicine. Apr 1;15(2):70.

Wachinou, A.P., Houehanou, C., Ade, S., Totah, T., Berger, M., Solelhac, G., Amidou, S., Fiogbe, A.A., Alovokpinhou, F., Lacroix, P. and Preux, P.M., (2022): Prevalence of sleep-disordered breathing in an African general population: The Benin Society and Sleep (BeSAS) study. *The Lancet Respiratory Medicine*.

Weihu C, Jingying Y, Demin H, Yuhuan Z and Jiangyong W (2011): End-tidal carbon dioxide concentration monitoring in obstructive sleep apnea patients. Am J Otolaryngol. 32:190-3.