Changes in left ventricular ejection fraction during REM sleep and exercise in chronic obstructive pulmonary disease and sleep apnoea syndrome

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ABSTRACT: Nine patients, 4 with chronic obstructive pulmonary disease (COPD) and 5 with obstructive sleep apnoea syndrome (OSAS) were monitored during sleep, rest and exercise. Left ventricular ejection fraction (LVEF) was investigated using gated equilibrium ^{99m}technetium ventricular scintigraphy during rapid eye movement (REM) sleep, during exercise, and during wakeful rest. Control wakeful rest periods used for comparison with a study state (either REM sleep or exercise) were always selected during the same circadian segment as that state. Myocardial stress thallium-201 scintigraphy was performed during, and 4 h after, exercise, and results were compared to a daytime rest period. Several patients had myocardial hypoperfusion despite a normal electrocardiographic (ECG) treadmill test. During REM sleep, all patients exhibited a significant change in LVEF (>5%) compared to wakefulness. During exercise, 5 subjects increased their LVEF normally (>5%) and 4 (1 COPD, 3 OSAS) decreased it. All patients had a similar change (increase or decrease) during REM and at maximal exercise. Our results suggest that REM sleep in COPD and in OSAS can produce a myocardial stress as great as that produced by exercise. We conclude that REM sleep, like exercise, is a state in which morbidity may become higher and that it may account for mortality in COPD and OSAS patients with compromised myocardial circulation.
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Exercise and rapid eye movement (REM) sleep are two states during which oxyhaemoglobin desaturation may occur in chronic obstructive pulmonary disease (COPD) [1, 2]. Sleep apnoeas occurring in obstructive sleep apnoea syndrome (OSAS) also lead to oxygen desaturation [3]. In patients with COPD, REM sleep oxyhaemoglobin desaturation has been shown by indirect measure to stress the flow reserves of the coronary circulation. The demand for coronary blood flow in this state can be transiently as great as during maximal exercise [4]. Chronobiological studies have suggested that in these subjects, sleep is associated with an increased risk of death [5]. In patients with OSAS, recent studies have shown an increased mortality [6–8] although the risk of dying during sleep is still disputed.

To investigate whether REM sleep myocardial stress may be similarly present in OSAS as in COPD, we have studied the changes in left ventricular ejection fraction (LVEF) during quiet wakefulness, exercise and REM sleep.

Methods

Population

Nine patients gave informed consent to be studied. Participants did not have history or clinical evidence of ischaemic heart disease or left ventricular dysfunction and must have had previous documentation during sleep of a clear drop in oxygen saturation (Sao_2) of at least 20% compared to wakeful supine Sao_2 measurements. The 9 subjects included 5 male OSAS patients with a mean age of 52±6 yrs and 4 COPD patients, 3 men and 1 woman, with a mean age of 58±14 yrs. Anthropometric and spirometric data are presented in table 1.

Table 1. – Anthropometric, arterial blood gas, and spirometric data for 4 patients with COPD and 5 patients with OSAS

	COPD	OSAS	p
Age yrs	58±14	52±6	NS
Height cm	164±6	173±3	NS
Weight kg	70±14	96±14	<0.01
Pao$_2$ mmHg	63±8	77±16	NS
Paco$_2$ mmHg	44±4	41±6	NS
pH	7.40±0.02	7.41±0.04	NS
Sao$_2$ %	91±3	92±7	NS
FVC	68±9	79±15	NS
FEV$_1$	47±9	83±18	NS
FEV$_1$/FVC	54±12	80±3	<0.05
TLC	88±21	72±7	NS

Data are mean±SD. Spirometric data are expressed as percentage of predicted values, except the FEV$_1$/FVC ratios. Blood gas: awake. Pao$_2$, Paco$_2$: arterial oxygen and carbon dioxide tensions, respectively; Sao$_2$: oxygen saturation; FVC: forced vital capacity; FEV$_1$: forced expiratory volume in one second; TLC: total lung capacity; COPD: chronic obstructive pulmonary disease; OSAS: obstructive sleep apnoea syndrome.

Protocols

Subjects were their own controls and were monitored during daytime wakefulness and nocturnal sleep. LVEF was investigated by gated equilibrium ^{99m}technetium ventricular scintigraphy during rest, exercise, REM sleep, and supine quiet wakefulness.

Myocardial stress thallium-201 scintigraphy was performed during daytime exercise in all subjects.

Nocturnal sleep studies

Subjects underwent a minimum of 2 nights of polygraphic monitoring. The first night was a non-invasive baseline recording of sleep and respiration. LVEF was determined on night 2, the "experimental night". Electroencephalogram (EEG) with C3/A2-C4/A1-C3/C4-01/02 of the 10/20 electrode placement system, electrooculogram (EOG), chin electromyogram (EMG), and electrocardiogram (ECG) with a modified V2 lead were recorded continuously on a Van Gogh 5,000 (Sweden) polygraph. Respiration was monitored with uncalibrated inductive plethysmography; air flow was measured by buccal and nasal thermistors; and Sao$_2$ with a Biox-Ohmeda 3700 ear oximeter. Blood pressure (BP) was continuously monitored through a humeral arterial line (Seldicath 4 F) connected to a Hewlett-Packard 78205D pressure transducer.

LVEF was measured by gated equilibrium ^{99m}technetium ventricular scintigraphy (see below). Subjects first received training in adapting to the position of the Gamma camera (Sopha Medical-Buc-France) near the chest; during sleep, they were supported in a fixed postion relative to the camera. Some subjects required 1 night of training prior to data collection. LVEF was measured during a long REM period in the second half of the night.

Wake studies

Quiet wakefulness. Subjects were awakened during the last half-hour of nocturnal data collection just after the REM measurement period, and wake measurements were made with patients maintaining their sleeping position. The wake period was thus in the same circadian time segment as the REM sleep period.

Exercise testing. Following a standardized rest period, subjects performed a symptom-limited treadmill exercise using a supine cycle ergometer (Monarch-Sweden). Workloads were increased by 25 watt increments every 2 min. Tests were interrupted when incapacitating dyspnoea developed or, in the absence of symptoms, when heart rate (HR) reached 85% of the predicted maximal value for age and gender. We continuously monitored cardiac function with a 12-1ead ECG and monitored HR and Sao$_2$. Arterial blood gases were measured with the subjects at rest before and at the end of the test. LVEF was assessed during rest before testing and at the last level of the exercise test, with gated equilibrium ^{99m}technetium scintigraphy.

A few days later, subjects returned during the same circadian period and were allowed to reach the same level of exercise under identical experimental conditions. Myocardial thallium-201 scintigraphy was performed at maximum exercise and 4 h later.

Scintigraphy techniques

LVEF measurement. 5 cc of venous blood was sampled immediately before sleep onset and during the daytime rest period. Red blood cells were labelled, using 1,110 MBq (30 mCi) of ^{99m}technetium for sleep studies and 740 MBq (20 mCi) for exercise testing studies, before reinjection. Measurements were made using the conventional equilibrium method. The Gamma camera was placed at a 45 degree left oblique anterior position in relation to the subject. The camera was coupled to a computerized memory-retrieval system that allowed an ECG-gated automatic analysis. To determine LVEF, 16 images per cardiac sample were sampled. The means for 2 min epochs [9, 10] were calculated from the data. This segment of time, which was the smallest compatible with the technique, was used for sleep and exercise studies. Rest and wake allowed 5 min period LVEF determination, leading to highly reproducible measurements [11]. In our laboratory, the LVEF normative value at supine rest during quiet wakefulness was 60±8%, and the normal increase with maximum effort was ≥5% [11].

Myocardial scintigraphy was performed using 11 MBq (3 mCi) thallium 201 injected at maximal exercise. Spectrophotometric imaging over 180 degrees was analysed at the time of injection and 4 h later, to investigate coronary redistribution [9, 10].

Data analysis

For the subjects with OSAS, we were careful to select 2 min REM sleep segments without marked intrusion of another sleep stage or significant arousal. To evaluate the impact of apnoea and Sao$_2$ drop on LVEF in OSAS using a technique with 2 min resolution, we selected segments with the least amount of EEG sleep change that included the longest apnoeic events (at least two) with concomitant Sao$_2$ drops and upward evolution with resumption of ventilation. In subjects with COPD, we selected 2 min segments with stable sleep EEG. LVEF results were expressed as the mean of 3±1 two minute epochs.

All the values are expressed as mean±SD. LVEF results were compared with the Wilcoxon signed-rank test, and other comparisons were made with the unpaired t-test.

Results

Patients had a classic profile during baseline nocturnal sleep recordings [12]. OSAS patients slept a mean of 389±69 min. Their mean respiratory disturbance index (RDI), defined as the number of apnoeas and hypopnoeas per hour of sleep, was 64±20. The lowest measured Sao$_2$ was 50±15%. COPD patients slept a mean of 257±69 min. Their mean RDI was 0; their lowest Sao$_2$ was 55±13% (table 2).

Tables 3 and 4 present the individual Sao$_2$, HR per minute, and mean arterial blood pressure (mABP) data obtained during quiet wakefulness and REM sleep, and during pre-exercise rest and exercise when LVEF was calculated in COPD and OSAS patients. Table 5 presents the mean LVEF measurements expressed as a percentage.

Table 2. – Sleep architecture, apnoea index and minimum Sao$_2$

	COPD	OSAS	p
Sleep latency min	34±21	15±23	NS
TSP min	412±63	446±62	NS
TST min	257±69	389±69	<0.05
Time awake % TSP	37±18	12±11	NS
Stage 1 % TSP	10±6	3±2	NS
Stage 2 % TSP	72±15	81±5	NS
Stage 3 and 4 % TSP	2±2	0	NS
REM sleep % TSP	10±9	16±5	NS
Apnoea index	0	64±20	NS
Minimum Sao$_2$ %	55±13	50±15	NS
Initial Sao$_2$ %	82±0.4	89±0.4	NS

TSP: total sleep period (time from sleep onset to final awakening); TST: total sleep time (TSP minus time of intervening wakefulness); REM: rapid eye movement. For other abbreviations see legend to table 1

Table 3. – Changes in Sao$_2$, heart rate and mean arterial blood pressure during sleep and exercise in 4 patients with chronic obstructive pulmonary disease

Patient no.		Awake	REM	Rest	Exercise
1	Sao$_2$	90	67±5	91	86
	HR	64	65±11	87	140 (91%)
	mABP	101	120±11	110	133
2	Sao$_2$	89	78±1	94	86
	HR	75	71±4	59	155 (94%)
	mABP	93	97±9	93	113
3	Sao$_2$	91	81±7	90	98
	HR	64	61±8	65	130 (68%)
	mABP	65	63±7	90	150
4	Sao$_2$	76	59±4	90	86
	HR	98	106±5	111	149 (100%)
	mABP	95	93±4	96	133

Data are mean±SD. Heart rate (HR) is per minute, percentage of maximal HR is in brackets. Mean arterial blood pressure (mABP) is in mmHg. Sao$_2$ is a percentage. REM: rapid eye movement; Sao$_2$: oxygen saturation.

Sleep studies

Sao$_2$ dropped significantly in COPD patients during REM sleep compared with quiet supine wakefulness monitored during the same circadian segment. Heart rate increased and mean ABP generally decreased, despite individual differences. In OSAS patients, similar overall findings were noted, although LVEF evolved differently than in COPD. When the delta change between the two measurements was calculated, 3 of 4 COPD patients and 2 of 5 OSAS patients significantly increased their mean LVEF, while the other patients decreased.

Daytime studies

When pre-exercise, rest, and exercise data points were analysed, the mean HR of patients with COPD increased to 88±7% of maximum HR; overall there was a mean fall in Sao$_2$ of 4% (patient 3 showed a rise of 8%) and an increase in mean ABP, although it did not reach significance. Subjects with OSAS also increased mean HR to 76±12%, increased mean Sao$_2$ by 1%, and significantly increased mean ABP. ECG monitoring during exercise showed no abnormality in COPD or OSAS patients.

The LVEF changes associated with exercise compared with the pre-exercise rest period are presented in table 5. LVEF units also varied by more than 5% [11] compared with rest, and the changes were highly significant (p=0.008).

Comparison between REM sleep and exercise

In order to determine the exact myocardial stress associated with REM sleep, we compared the LVEF changes in both conditions. Subjects who increased their

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Table 4. – Changes in Sao_2, heart rate and mean arterial blood pressure during sleep and exercise in 5 patients with obstructive sleep apnoea syndrome

Patient no.		Awake	REM	Rest	Exercise
5	Sao_2	91	71±18	94	98
	HR	68	63±9	61	125 (75%)
	mABP	102	115±20	106	166
6	Sao_2	90	75±26	92	93
	HR	75	70±11	68	125 (64%)
	mABP	120	153±26	113	126
7	Sao_2	86	73±0.7	81	80
	HR	72	71±7	81	155 (88%)
	mABP	152	141±20	116	159
8	Sao_2	95	88±8	97	98
	HR	71	65±8	71	144 (90%)
	mABP	106	118±19	90	145
9	Sao_2	93	70±11	96	94
	HR	76	77±19	78	125 (74%)
	mABP	121	142±27	122	160

Data are mean±SD. Heart rate (HR) is per minute, percentage of maximal HR is in brackets. Mean arterial blood pressure (mABP) is in mmHg. Sao_2 is a percentage. Sao_2: oxygen saturation; REM: rapid eye movement.

Table 5. – Changes in LVEF during REM sleep and exercise

Patient no.	Wake	REM	n	REM-Wake	Rest	Exercise	Exercise-Rest
COPD							
1	41±2.0	54	1	+13	41±2.1	47	+0.6
2	42±1.8	49±0	2	+07	57±1.7	61	+04
3	51±2.2	62±4.6	4	+11	57±2.1	71	+14
4	29±2.3	20±2	3	-09	47±1.8	42	-05
OSAS							
5	65±1.7	50±12	3	-15	72±1.3	59	-13
6	56±1.9	63±5.7	4	+07	72±1.5	79	+07
7	55±2.4	62±3.8	3	+07	69±2.0	76	+07
8	44±2.3	29±5.6	4	-15	54±2.1	50	-04
9	59±1.6	53±2.8	2	-06	90±2.2	70	-20

Mean LVEF is expressed as percentage±SD for 3 wake periods and for n REM periods. LVEF: left ventricular ejection fraction; REM: rapid eye movement; COPD: chronic obstructive pulmonary disease; OSAS: obstructive sleep apnoea syndrome.

LVEF during exercise as compared with rest likewise increased it in REM sleep compared to quiet wakefulness. Similarly, subjects who decreased LVEF during exercise also decreased LVEF during REM sleep. The statistical significance, when percentage of change was considered, was the same with exercise and REM sleep (p=0.008), and there was no statistical difference between REM sleep and exercise changes.

Myocardial stress thallium scintigraphy showed abnormalities in four subjects (patients no. 1, 2, 4 and 8), representing anteroseptal and inferior hypoperfusion without myocardial redistribution after four hours, except in patient no. 1, who revealed an unsuspected and evolving coronary disease. Patients no. 4 and no. 8 exhibited an abnormal ejection fraction during exercise as well as during REM sleep.

Conclusions

It has been many years since the cardiovascular effects related to apnoeas were first reported. They include oscillations in blood pressure related to intrathoracic pressure changes [13–15] and cardiac output changes during the apnoeic event as shown by the thermodilution technique [16].

In COPD, there are classically no systematic mABP changes associated with nocturnal falls in Sao_2, and increases in HR in relation to oxygen desaturation are still in question.

LVEF increase during exercise corresponds to a physiological response and reflects myocardial stress. We used this global index of contractility to reflect similarly myocardial stress associated with REM sleep. The

technique had previously been thoroughly investigated. An increase greater than 5% in LVEF units normally occurs in control subjects after exercise [9–11]; but in patients with coronary heart disease [17], valvulopathies [18, 19], or certain cardiomyopathies [20] LVEF units were found to be unchanged or even decreased. Investigation of the LVEF with exercise accurately evaluated global heart performance, independent of the cardiac disease involved. Myocardial stress thallium scintigraphy during exercise is a highly sensitive indicator of coronary ischaemia [9, 10, 21, 22]. However, the short duration of sampling for data analysis in technetium scintigraphy is a drawback when studying patients with obstructive sleep apnoea, as 2 min epochs are required. This is not an issue during REM sleep in COPD patients, as a steady-state with a marked and relatively stable drop in Sao_2 can exist during each 2 min epoch. In OSAS patients, however, there are successions of apnoeas with resumption of ventilation and regular cyclic arousals and hypoventilation. Although LVEF was easy to calculate, and its overall evolution during REM sleep easy to evaluate in COPD patients, it was more difficult in OSAS patients. However, our strict protocols eliminated much of the variance that was due to state changes and cyclical variation of the breathing pattern in OSAS, although all of the variance could not be completely eliminated because desaturation and return to normal saturation occurred during the 2 min scintigraphic analysis. Still, the results are of great importance for a better understanding of the risks incurred by our patients during REM sleep.

Our study is the first to systematically investigate LVEF changes in patients with breathing disorders during sleep, and it indicates that changes noted in LVEF are similar during exercise and REM sleep. The idea that REM sleep could be as stressful as exercise in patients with coronary heart disease has previously been entertained but has never been clearly demonstrated in any disease entity. It is interesting to note that in our patient population, despite a 12-1ead ECG and a treadmill test for those with OSAS, no patient was identified with coronary heart disease; however, myocardial stress thallium scintigraphy showed that 4 of 9 subjects had myocardial hypoperfusion, possibly a factor in the increased mortality recently reported for OSAS and COPD patients [6–8]. Using the HR systolic BP product as an indirect method to reflect myocardial oxygen consumption, SHEPARD et al. [23] have described intermittently high demands for myocardial blood flow related to apnoea and to lowest Sao_2 in OSAS. The same authors [4] also used indirect methods to calculate that the oxyhaemoglobin desaturation during sleep stresses the flow reserve of the coronary circulation in COPD patients.

Of interest is the difference in overall change in the subjects' LVEF, both during exercise and during REM sleep. Two subjects, patient no. 4 (COPD) and patient no. 8 (OSAS), who showed a decrease in LVEF compared with the rest, had myocardial hypoperfusion, while patient no. 9 did not, indicating that the overall decrease in LVEF cannot be linked solely to the myocardial problem seen in patients no. 4 and no. 8. Several factors may be involved, including obesity, duration of the disease, and the associated hypoxaemia experienced by the patients. However, none of these had significant influence. This finding should be confirmed in a larger population.

The change in LVEF during REM sleep was well documented; we expressed the results as a mean of several 2 min epochs, taking great care to eliminate segments with long arousal intrusion or sleep stage changes and performing measurements only during a well-established REM period lasting long enough to avoid state changes (i.e. an early morning REM period, considering the usual human sleep structure). Also, instead of comparing a quiet wake period at sleep onset, which would not have been in the same circadian time segment as the experimental REM period, and in which the subject would not have been kept supine for several hours prior to the measurement, we waited until almost the end of the experimental REM period to awaken the subject for quiet-rest-wakefulness measurements. Similarly, the measurements between pre-exercise quiet-rest-wake and exercise-wake were performed in close proximity, once again taking into account the circadian timing, with subjects monitored in the supine position. There was a systematic difference in the absolute values of the two wake measurements, but this was not competely surprising, considering the circadian timing of the two rest periods and the amount of rest/sleep prior to each measurement. The important variable is the delta change between rest and REM sleep on the one hand, and rest and exercise on the other, and their directions of change.

Our findings may encourage similar investigations of other types of patients, on whose health status REM sleep may also have a negative impact.

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P.A. Levy, C. Guilleminault, D. Fagret, J.M. Gaio, Ph. Romand, C. Bonnet, C.M. Pison, J.E. Wolf, B. Paramelle. Modifications de la fraction d'éjection ventriculaire gauche pendant le sommeil REM et l'effort, dans les bronchopneumopathies chroniques obstructives et dans le syndrome d'apnée du sommeil.

RÉSUMÉ: Neuf patients, dont 4 atteints de bronchopneumopathie chronique obstructive (BPCO) et 5 atteints de syndrome d'apnée obstructive du sommeil (OSAS) ont été suivis pendant le sommeil, le repos et l'effort. La fraction d'éjection du ventricule gauche (LVEF) a été examinée par une scintigraphie ventriculaire à l'équilibre au technetium 99m, au cours du sommeil à mouvement oculaire rapide (REM), au cours de l'effort, et pendant le repos éveillé. Le périodes de contrôle en repos éveillé, utilisées pour comparaison avec l'état faisant l'objet de l'étude (soit le sommeil REM, soit l'effort), ont toujours été sélectionnées pendant le même segment circadien que l'état étudié. La scintigraphie d'effort myocardique au thallium 201 a été réalisée pendant et 4 heures après l'effort, et les résultats ont été comparés à une période de repos pendant la journée. Certains patients avaient une hypoperfusion myocardique malgré un test d'ECG normal au tapis roulant. Pendant le sommeil REM, tous les patients ont développé une modification significative de LVEF (>5%), par comparaison avec la période d'éveil. Pendant l'effort, 5 sujets ont augmenté leur LVEF normalement (>5%) et 4 (dont 1 BPCO et 3 OSAS) l'ont diminuée. Chez tous les patients, l'on a noté une modification similaire (accroissement ou diminution) au cours du REM et à l'effort maximal. Nos résultats suggèrent que le sommeil REM dans les BPCO et l'OSAS peut produire un effort myocardique aussi important que celui déclenché par l'effort. Nous concluons que le sommeil REM, comme l'effort, est un état dans lequel la morbidité pourrait augmenter, et ceci pourrait rendre compte de la mortalité chez les patients atteints de BPCO et d'OSAS dont la circulation myocardique est compromisé.
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