NOCTURNAL OXYGEN DESATURATION IN PATIENTS WITH CONGESTIVE HEART FAILURE

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INTRODUCTION

It's well known that sleep disorder breathing may occur in many diseases. Modifications of the respiratory pattern during sleeping were reported in patients with Congestive Heart Failure (CHF) (1) even if the consequences that such alterations may cause in these subjects are not well known. The most important alterations that compromise the quality of sleep of these patients are presence of "arousals", cardiac rhythm disturbance, oxyhemoglobinic desaturations and the cyclic alterations of ventilation known as "Cheyne-Stokes breathing" or as "periodic breathing" (1,2). Polysomnography is the most complete diagnostic method to evaluate sleep disorder breathing. However, the use of this technique is limited by its high costs, need of highly qualified staff, short compliance of patients and finally relatively long times of reading and evaluation of polysomnographic tracing. The aim of our study was to verify if evaluation of nocturnal arterial oxyhemoglobin saturation obtained by a simple pulse-oxymetry, may provide significative data about ventilatory pattern of patients affected by CHF. This technique is widespread, much better tolerated and less expensive.

MATERIALS AND METHODS

We studied 10 subjects (8M, 2F), mean age 71.4±12.4 yrs, mean body mass

index (BMI) of 24.3±3.0 Kg/m^2 admitted to sub-intensive cardiological therapy unit (Institute of Cardiovascular Diseases, University of Bari). All patients were admitted with diagnosis of congestive heart failure due to left ventricular insufficiency caused by ischemic, hypertensive, or idiopathic cardiopathy (class 3rd - 4th according to New York Heart Association).

Patients were selected according to clinical history, clinical observation and instrumental examinations. Mean PaO_2, in awake patients, was 77.1±13.7 mmHg, mean $PaCO_2$, in awake patients, was 36.9±3.4 mmHg, mean SaO_2, in awake patients, was 95.4±2.7%.

The ejection fraction of left ventricle (EFV) estimated by M-mode echography, showed a mean value of 30.4±8.2% (range 20-45%).

The selected patients were not affected by primitive lung diseases (according to clinical history, respiratory function tests and standard chest X ray), nor neurological diseases, and they didn't show history of sleep disorders such as narcolessia and/or sleep apnoea. All patients were in stable clinical condition (examination was performed 34 days after acute event) and they didn't take hypnotic or sedative drugs, nor oxygentherapy. Diuretics, vasodilators, antihypertensives, anthiarithmics and digitalis drugs were administered.

Nocturnal pulse-oxymetry has been performed by a pulse-oxymeter (Pulsox 7/Minolta) provided with a digital probe at a sliding speed of 24 cm/h.

Baseline arterial oxygen saturation (SaO_2 B) was measured in ambient air, during wake, while patients were in supine position, before sleeping. We measured the following indexes:

- Mean nocturnal arterial O_2 Saturation (SaO_2 M)
- Minimum value of SaO_2 measured during sleep (Nadir)
- Total sleep time (TST)
- Number of desaturation episodes/h (ODI): an arterial desaturation episode has been defined as a decrease of SaO_2 B for at least 4%.
- Total sleep time (TST) spent with a SaO_2<90% ($TST_{SaO2<90\%}$).

Furthermore, we verified if the tracings showed a typical sinusoidal trend

with sequences of "fall-rise" of SaO_2 values, collected in "wave-trains" different from those of patients with Sleep Apnoea Syndrome (SAS) and Chronic Obstructive Pulmonary Disease (COPD) and compatible with periodic breathing.

RESULTS

In our selected sample of 10 patients, no subject was obese (BMI<27 Kg/m^2) (tab. 1). All patients showed clinical and instrumental signs of CHF due to left ventricular insufficiency (Class 3rd - 4th according to NYHA) in relatively stable condition (3-4 days after the acute event), caused by ischemic or hypertensive or idiopathic cardiopathy.

Mean arterial blood gas values were within normal limits; the EFV confirmed the existence of left ventricular failure (range 20-45%).

The analysis of pulse-oxymetric indexes (tab. 2) shows that mean SaO_2 B, in our sample, was considerably over 90%. Similarly, SaO_2M was over 90%, except for one patient who showed a value of 89.73%.

Tab. 1 - Characteristics of the sample (values are expressed as mean ± SD).

Number of subjects	10
Sex	2F / 8M
Age (years)	71.4 ± 12.4
BMI (Kg/m)	24.3 ± 3.0
PaO_2 (mmHg)	77.1 ± 13.7
$PaCO_2$ (mmHg)	36.9 ± 3.4
SaO_2 (%)	95.4 ± 2.7
EFV	30.4 ± 8.2

Tab. 2 - Pulse oxymetric indexes.

	mean ± SD
SaO_2B (%)	95.3 ± 1.1
SaO_2M (%)	93.0 ± 1.7
Nadir (%)	83.9 ± 5.2
ODI	15.7 ± 18.4
$TST_{SaO2<90\%}$	7.8 ± 12.4

All the patients presented nocturnal desaturation episodes, but only in two subjects, the ODI values (61.3/h and 34.8/h respectively), and the typical trend of oxymetric tracing, suggested an Overlap Syndrome (defined as "Sleep Apnoea Syndrome in presence of cardiopathy"), in absence of remarkable anamnestic data.

Mean Nadir was significantly low (83.9%; range 77-92%), showing a significant decrease of nocturnal SaO_2 even in patients without obstructive sleep apnoea (ODI<15/h).

In these patients, the tracing presented a "fall-rise" sequence of SaO_2 (range of 3-7%) lasting from 36 to 72 seconds (mean value 66 sec).

These sequences are collected in "wave-trains" which occur repeatedly from 3 to 7 times during night (fig. 1), constituting from 14% to 71% of TST (mean value 26.5±15%).

Finally, the patients spent only 7.8% of TST with SaO_2 values below 90%, and in a clear hypoxaemic state.

In this regard, we have to consider that our patients started from high SaO_2 B values and that consequently the decreases of PaO_2, during sleep, did not influence dramatically nocturnal SaO_2.

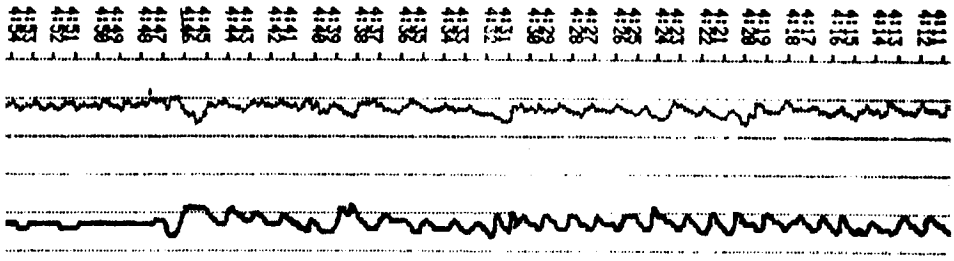

Figure 1 - Wave trains of nocturnal SaO_2 oscillations (a fall-rise sequence) in a patient with congestive heart failure (upper line: Heart Rate; lower line: SaO_2%).

DISCUSSION

Many authors reported the development of significant and sometimes severe nocturnal desaturations in patients with congestive heart failure,

although oxygenation values were within normal limits during wake (1-3). In this study we used a simple pulse-oxymetry: our preliminary data confirm these findings. All our patients showed significant desaturation events overnight.

Nevertheless, the trend of nocturnal saturimetric tracing in these subjects is different in comparison with other kind of patients studied previously.

In COPD patients with a $SaO_2 \geq 90\%$, when awake, nocturnal pulse-oxymetry shows occasional falls of SaO_2, often steep and deep and a gradual increase of SaO_2 at lower level than the basal values.

Nocturnal tracing of SAS subjects is characteristic with deep, steep and frequent SaO_2 falls, and subsequent increases of SaO_2 up to values similar (or higher) than the basal values, with a homogeneous and comprehensive trend defined as "tooth of saw".

In 80% of our sample, we have found very regular, sometimes deep oscillation (as periodic changes of nocturnal SaO_2), concerning about 25% of TST which, according to literature data, may be referred to breathing disturbances such as periodic or Cheyne-Stokes breathing, as showed by various authors with polysomnography.

Some authors (3-4) hypothesised that the possible consequences of the cyclic hypoxaemia during sleep may be harmful for the long-term prognosis of patients affected by heart failure.

In fact, arterial desaturations, which may sometimes be severe, can make a myocardial insufficiency worse and can cause an increase of oxygen consumption (5).

Moreover, there may be an increase of pulmonary arterial (6) and systemic arterial pressure (3,7).

In our sample, $TST_{SaO2<90\%}$, was 7.8%, showing a mild nocturnal hypoxaemia, according to Davies et al. (8) who have found in their population, a $TST_{SaO2<90\%}$ of about 6% with a sudden fall of SaO_2 during sleep.

The mechanism causing nocturnal hypoxaemia in these patients could be referred to:

- the decreased pulmonary compliance and the supine position which reduces the pulmonary volumes;
- the occurrence of periodic breathing that, together with the supine position, causes hypoxaemia during REM sleep;
- the decreased EFV which reduces markedly the venous mixed PaO_2 (9,10).

The degree of nocturnal hypoxaemia in these cardiopathic patients is similar to the subjects affected by SAS (11) and COPD (12).

In addition, our study shows the validity of pulse-oxymetry as a screening method on patients (not necessarily pneumopathic) at risk of nocturnal respiratory disturbances.

According to the obtained instrumental data and in relation to the particular trend of nocturnal oxymetric tracing, it is possible to consider the utility of nocturnal (and/or diurnal) oxygentherapy in these patients.

The efficacy of nocturnal oxygentherapy in order to improve sleep architecture, to reduce the number of hypoxaemic events and also of periodic breathing episodes during night, was demonstrated by controlled studies (4), but the role of oxygentherapy on long-term prognosis of these patients has not been verified.

Furthermore, the pulse-oxymetry can select patients to examine with more sophisticated methods as polysomnography, in order to perform a complete diagnostic examination.

In conclusion, our study shows that patients affected by congestive heart failure, even if in a stable clinical condition and with a PaO_2 within normal values, present more or less severe disturbances of nocturnal SaO_2, with periodic and regular sequence of SaO_2 increase-decrease that may be referred to ventilatory troubles such as periodic breathing or Cheyne-Stokes breathing verified by polysomnography.

In these patients the pulse-oxymetry may be considered an efficacious, simple, cheap and well tolerated method of evaluation of noctural SaO_2 behaviour. Further studies are requested to verify the utility of nocturnal oxygentherapy on long-term prognosis of these patients.

The aim of our study was to evaluate the modifications of the respiratory pattern during sleeping in patients with congestive heart failure (CHF) by a simple pulse-oxymetry. We studied 10 subjects (8M/2F), mean age 71.4±12.4 yrs, admitted to sub-intensive cardiological therapy unit, with diagnosis of CHF due to left ventricular insufficiency by ischemic, hypertensive or idiopathic cardiopathy, when in a stable clinical condition. All patients presented arterial blood gas values within normal limits. The ejection fraction of left ventricle showed a mean value of 30.4±8.2% (range 20%-45%). Nocturnal pulse-oxymetry was performed by pulse-oxymeter (PULSOX 7 Minolta) provided with a digital probe at a sliding speed 24 cm/h. Our data showed that all patients presented nocturnal desaturation episodes (mean oxygen desaturation index 15.7±18.4). In two patients, we found an "Overlap Syndrome" (obstructive sleep apnoea in presence of cardiopathy). In other patients pulse-oxymetry showed a typical sequence of "fall-rise" basal O_2 saturation lasting from 36 to 72 seconds, collected in "wave trains" which were present from 14% to 70% of total sleep time compatible with periodic breathing. In conclusion, our study shows that patients affected by CHF, even if in stable condition and with a PaO_2, within normal values, present more or less severe disturbances of nocturnal SaO_2, with periodic and regular sequences of SaO_2 fall-rise that may be referred to ventilatory troubles such as periodic breathing or Cheyne-Stokes breathing. In these patients the pulse-oxymetry may be considered an efficacious, simple, cheap and well tolerated method.

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