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The adequacy of oxygenation in COPD patients undergoing long-term oxygen therapy assessed by pulse oximetry at home

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The adequacy of oxygenation in COPD patients undergoing long-term oxygen therapy assessed by pulse oximetry at home. P. Śliwiński, M. Łagosz, D. Górecka, J. Zieliński. ©ERS Journals Ltd 1994.

ABSTRACT: It is probable that some daily activities may cause marked falls in arterial oxygen saturation (Sao₂) in patients undergoing long-term oxygen therapy (LTOT), despite good oxygenation at rest.

We estimated the adequacy of LTOT in 34 randomly selected chronic obstructive pulmonary disease (COPD) patients at home by monitoring Sao₂ continuously over 24 h. The subjects were also asked to complete a questionnaire listing frequent daily activities.

Despite almost normal mean Sao₂ (94%) at the beginning of recording (O₂ 2 *l*·min⁻¹, at rest) the subjects studied spent 6.9 h below an Sao₂ of 90%, with minimum Sao₂ of 61%. On average we observed 10 episodes of desaturation in each patient over 24 h, both while breathing air and oxygen. The comparison of Sao₂ recordings and questionnaires revealed the highest number of desaturations during sleep, followed by naps, watching the television, eating, washing and talking.

The oxygen flow rate prescribed, based on blood gas measurements at rest, did not protect 85% of the patients studied from deep falls in ${\rm Sao_2}$ during daily life. An increase of oxygen flow during some activities and during sleep is suggested.

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The benefits of long-term oxygen therapy (LTOT) in patients with chronic obstructive pulmonary disease (COPD) have been reported by a number of investigators [1, 2]. Since the 1980s, domiciliary oxygen therapy has been generally accepted as an important part of the routine treatment of COPD patients with chronic severe hypoxaemia. The main indication for LTOT is a low arterial oxygen tension (Pao₂), usually lower than 55 mmHg (7.3 kPa). The prescribed oxygen flow should increase Pao₂ to at least 65 mmHg (8.7 kPa) [3]. The blood gas tensions used to qualify the patients for treatment are measured at rest. The oxygen flow rate is also adjusted in resting conditions.

In some cases, arterial oxygen saturation (Sao₂) during treadmill/cycle ergometer exercise or during sleep is also assessed [4], but rather to confirm the subject's eligibility for LTOT than to establish the adequate oxygen flow rate.

After the prescription of LTOT, the patients are followed in out-patient clinics or at home, and blood gas measurements are periodically performed at rest. Thus, there is no information about Sao₂ in those patients during their daily activities at home. It is possible that some activities may cause important drops in Sao₂, despite adequate oxygenation while resting.

The purpose of the present study was to estimate the adequacy of LTOT in COPD patients during their usual activities at home, by monitoring Sao₂ continuously over 24 h.

Patients and methods

Thirty four COPD patients, 20 males and 14 females, who were assessed and prescribed LTOT, were studied. They were randomly recruited from a group of 76 patients undergoing domiciliary oxygen therapy. They were at least 6 months on LTOT before the study started. The diagnosis of the disease was based on a history, clinical examination, chest X-ray, electrocardiography (ECG) and pulmonary function tests. The details of the qualification procedures for LTOT have been reported previously [5]. In short, patients had to present with chronic severe hypoxaemia (Pao₂ ≤55 mmHg (7.3 kPa)) to qualify.

Special care was paid to exclude patients suspected of having obstructive sleep apnoea. All subjects completed a Marburg questionnaire and, in case of doubt, overnight pulse oximetry and snoring recordings were performed. All patients were prescribed supplemental oxygen at 2 l-min-1 by nasal prongs, in order to increase Pao₂ above 65 mmHg (8.7 kPa) at rest. Oxygen was delivered by a Healthdyne BX-5000 oxygen concentrator. Each oxygen concentrator was tested for oxygen output before and after monitoring, to ensure that it produced a sufficient concentration of oxygen. In all oxygen concentrators the concentration of O₂ in the gas was above 92% at a prescribed flow. Patients were asked to breathe oxygen as long as possible, but not less than 17 h-day-1, including the entire night.

To minimize the effect of limited activity of the patient due to usage of the oxygen concentrator, and therefore conceivably the degree of desaturation related to activity, the tubing connecting the concentrator and the patient was arranged in such a way that the patient could easily reach any point at home while breathing oxygen.

Arterial oxygen saturation recordings were continuously performed over 24 h by two pulse oximeters (Minolta Pulsox 7) using a finger sensor. The first oximeter was attached to the subject at 9 a.m. and replaced by the second at 9 p.m. To assure good quality of signal from the finger probe, great attention was paid to attaching it well to avoid change in position on the finger. Poor quality recordings due to movement of the probe were characterized by high frequency oscillations, which were easy

to identify and delete from the record. We also introduced a quality index of the recording that estimated the percentage of total monitoring time, when the subject was correctly connected to the pulse oximeter.

The pulse oximeter attached to the patient could be powered either from a battery or an electrical outlet. This allowed the patient to perform outdoor activities, such as walking or shopping. The connection to the electrical outlet, mainly used in the home, was of sufficient length to ensure it did not limit the subject's activity. Patients were asked to spend a day of recording in their usual manner, and to complete a questionnaire covering 24 h divided into 15 min periods. The questionnaire was designed to quantify frequent activities which were important in the patient's daily life. It listed the following activities: washing, dressing, eating, housework, resting or having a nap, reading, watching television, listening to the radio, walking, talking, being angry or upset, experiencing chest pain, dyspnoea or cough, sleep, and breathing without oxygen. The comparison of Sao₂ recordings and questionnaires allowed us to match the activities that caused a marked decrease in arterial oxygen saturation (fig. 1).

The oximeter signal was analysed by means of a computer and specially designed software. Analysis was performed separately for the 12 h periods, 9 a.m. to 9 p.m.

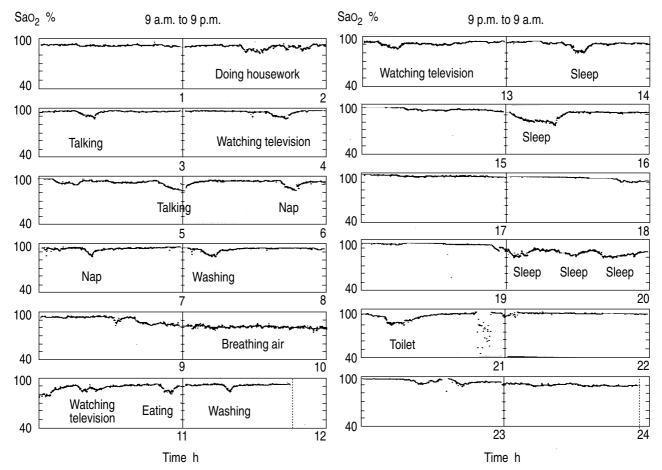


Fig. 1. – Illustration of 24 h arterial oxygen saturation (Sao₂) recording in one representative patient. Falls in Sao₂ during different activities are shown.

("daytime") and 9 p.m. to 9 a.m. ("night"). Sao_2 at the beginning of the study was determined from measurements taken during the first 30 min, whilst the subject was breathing oxygen at rest. A desaturation period was defined as a drop of Sao_2 below 90% for at least 5 min. During the desaturation period, the nadir value of Sao_2 had to fall to 85% or less at least once [6].

Statistical analysis

Differences in mean values were analysed by the Student's t-test for paired data. All reported values are mean±sp.

Results

Baseline characteristics of the 34 patients studied are presented in table 1. Fifteen of them presented with "pink puffer" and 19 with "blue bloated" clinical picture [7]. All subjects had severe airways obstruction, with accompanying blood gas abnormalities. Sao₂ measurements at the beginning of recording, while the subjects were breathing oxygen at the prescribed flow rate, revealed adequate oxygenation in all patients (mean 94±1.8%).

Mean oxygen breathing time of the patients studied on the day of investigation was 19.3 h. This was longer than the average time on oxygen recorded over 1 month (16.2 h).

The quality index of the pulse oximeter recording estimating the percentage of total monitoring time when the subject was correctly connected to the probe, was 99%, indicating that data removed from analysis had only a negligible impact on the overall result.

The results of pulse oximetry obtained during 12 h monitoring periods are shown in table 2. Mean and minimum Sao₂ during both recording periods were almost at the same level, 92±3.2% *versus* 92±3.2% and 59±13.8% *versus* 62±15.4%, respectively. Also, the total time of

Table 1. - Baseline characteristics of 34 COPD patients

Variable		
Age yrs	61±9	
FVC l	2.25±0.9	
FVC % pred	55±16.6	
FEV_1 l	0.69 ± 0.25	
FEV ₁ /FVC %	32±8.2	
Pao, mmHg	52.4±6.6	
kPa	7.0±0.9	
Paco ₂ mmHg	48.7±7.3	
kPa	6.5±1.0	
PreSao, % on O,	94±1.8	
Hct %	46±4.1	

Data are presented as mean \pm sp. COPD: chronic obstructive pulmonary disease; FVC: forced vital capacity; FEV₁: forced expiratory volume in one sec; Pao₂: arterial oxygen tension; Paco₂: arterial carbon dioxide tension; PreSao₂: arterial oxygen saturation during the first 30 min of recording; Hct: haematocrit.

Table 2. - Results of 24 h pulse oximetry at home

Variable	9 a.m. to 9 p.m.	9 p.m. to 9 a.m.	
meanSao, %	92±3.2	92±3.2	
minSao, ² %	59±13.8	62±15.4	
Sao, <90% %	30±26.7	28±27.3	
Sao ₂ <85% %	10±16.1	9.3±12.3	
Sao ₂ <80% %	2.8±5.8	2.9±6.7	
Sao, <75% %	1.0±2.8	1.1±3.1	
Use of O ₂ h	8.7±3.2	10.6±2.0	

Data are presented as mean \pm sp. Sao₂: arterial oxygen saturation. minSao₂: minimum Sao₂; Sao₂<90%, <85%, <80%, <75%: time spent with Sao₂ below 90, 85, 80 and 75%, respectively, (in percentage of the total recording time).

the study that the patients spent below an Sao₂ of 90, 85, 80 and 75% did not significantly differ between daytime and night. The desaturation periods while breathing oxygen lasted 63 min during daytime recording and 84 min during night (NS).

In total, 301 episodes of desaturation were observed (on average 10 episodes in each subject over 24 h), 169 between 9 a.m. and 9 p.m. and 132 between 9 p.m. and 9 a.m. (table 3). One hundred and eighteen desaturation episodes occurred when subjects were breathing air, and 183 when breathing oxygen. The highest number of desaturation periods was observed during sleep (64), followed by having a nap (43), watching the television (38), eating (30), washing (29), and talking (29).

The majority of desaturation periods during sleep was observed in "blue and bloated" patients (47 out of 64). Seventy three out of 135 hypoxic episodes in these subjects, that occurred during other daly activities, were noted while breathing oxygen. In "pink puffers", 50 out of 102 desaturation periods during daily activities were observed when breathing oxygen (table 4).

In five subjects (all "pink puffers"), episodes of desaturation were not observed. In another two patients, such episodes occurred only while breathing air.

Patients breathed oxygen for less hours during the daytime than at night; 8.7 *versus* 10.6 h, respectively, (p=0.002).

Table 3. - Number of desaturation episodes observed in the patients studied during various activities

Activity	9 a.m. to 9 p.m.		9 p.m. to 9 a.m.	
	Oxygen	Air	Oxygen	Air
Time h	8.7	3.3	10.6	1.4
Housework	1	11	0	0
Dressing	0	3	3	7
Dyspnoea or cough	5	2	2	0
Eating	10	10	5	5
Nap or rest	28	6	3	6
Reading	8	9	3	2
Sleep	0	0	60	4
Talking	13	11	0	5
Walking	2	7	0	3
Washing	10	5	5	9
Watching television	22	6	3	7

Table 4. – Number of desaturation episodes in "pink puffers" and "blue bloaters" during sleep and daytime

Patients		Sleep		Other activities	
	n	Oxygen	Air	Oxygen	Air
Pink puffers Blue bloaters	15 19	17 43	0 4	50 73	52 62

Discussion

The main goal of this study was to assess the adequacy of oxygenation in COPD patients undergoing LTOT during their usual activities at home during 24 h.

The surprisingly good quality of pulse oximetry recording was due to the special care paid to optimal attachment of the probe, allowing virtually no change in its position on the finger. Also, the majority of the recordings were obtained when patients were at home and the pulse oximeter was powered from the electrical outlet. This prevented a deterioration of the quality of the signal that we observed sometimes if using a fading battery.

The subjects studied showed a satisfactory Sao₂ (94%) at the beginning of recordings, whilst breathing oxygen at rest. Also, the mean Sao₂ (92%) measured over 24 h seemed adequate. Nevertheless, a detailed analysis of oxygenation revealed a relatively low minimum value of Sao₂ (61%). The total time spent below a saturation of 90% (6.9 h) was much longer than the total time during room air breathing (4.7 h). This means that, on average, our subjects spent 2.2 h a day with a saturation below 90%, despite breathing oxygen. These desaturations were observed during daytime and at night, while breathing air or oxygen. Thus, an almost normal level of Sao₂ determined during a short period of time while the patients were awake and at rest did not guarantee adequate oxygenation in 85% of our subjects during 24 h monitoring.

The question arises as to whether the observed desaturation dips might have been harmful for the patients studied. It has been reported that the extent of nocturnal hypoxaemia is not clinically important [8]. Also, the improvement of exercise tolerance by oxygen breathing may be questionable [9]. On the other hand, there is evidence that interruption of oxygen breathing in COPD patients treated with LTOT results in an immediate increase in pulmonary artery mean pressure (PAP) [10]. Transient nocturnal oxyhaemoglobin desaturation during sleep, accompanied by aggravation of pre-existing pulmonary hypertension, has been commonly observed in COPD patients [11, 12]. Those two findings, together with the well-known correlation between the level of pulmonary hypertension and survival in COPD patients [13, 14], suggest that good oxygenation of subjects undergoing LTOT should be maintained continuously.

There is another occasion when such patients may become more hypoxaemic in stable periods of the disease. It has been shown that even low grade exercise may significantly decrease oxygen saturation and increase PAP [15, 16].

Administration of supplemental oxygen may improve pulmonary circulation by lowering existing pulmonary hypertension as well as by eliminating increases in PAP associated with hypoxaemic episodes [17]. Similarly, supplemental oxygen during exercise protects patients from dangerous desaturations, and may improve exercise performance [18].

The desaturation periods observed during air breathing might have been eliminated by encouraging patients to use oxygen continuously. This is, however, very difficult to achieve in reality, especially for a subject who wants to continue outdoor activities. It seems that hypoxaemic episodes noticed during an oxygen breathing period could have been more easily corrected. The increase of oxygen flow rate above the resting level during some daily activities and during sleep might have improved oxygenation in most cases. The additional amount of oxygen should be established empirically, taking into account the level of Paco, that may limit oxygen flow rate.

The influence of sleep on duration of hypoxaemic episodes can be explained by a variety of factors, such as the aggravation of mismatched ventilation and perfusion [19], or the rapid eye movement (REM) sleep effect [20]. We cannot exclude the possibility that some patients, having misplaced their prongs, breathed oxygen only for some periods during sleep. Several studies have shown that such hypoxaemic episodes are associated with severe cardiac arrhythmias [21, 22], and increases in PAP [11, 12]. Furthermore, Fletcher et al. [23] found significantly better survival in COPD patients who did not desaturate at night compared to those who did. We therefore speculate that the reason for a higher mortality in desaturating patients may be, at least partly, due to longer periods of hypoxaemia at night than in the daytime.

The majority of hypoxaemic episodes during sleep (73%) were noticed in "blue and bloated" patients, although this was only 26% of their total desaturation episodes (table 4). "Pink puffers" suffered mostly from hypoxaemic periods during the daytime (they made up 86% of total desaturations in this group). This is consistent with other studies reporting that nocturnal hypoxaemic episodes occurred more often in "blue bloaters" than in "pink puffers" [20].

It may be surprising that 61% of desaturation episodes occurred when patients were breathing oxygen. These episodes, however, were recorded during a significantly longer time (19.3 h) than those on air (4.7 h). Interestingly, 50% of hypoxaemic periods observed when breathing oxygen occurred during naps or sleep. These two conditions were accompanied by falls in Sao₂ more frequently than others. A surprisingly high number of desaturations was noticed while watching the television. Presumably some of these were also caused by naps.

Although oxygen therapy at the constant flow rate did not completely prevent our subjects from severe desaturations, its efficacy still seems to be high. On average, there was one desaturation episode every 3.06 h when breathing oxygen and one every 1.15 h when breathing air.

We conclude that the prescription of oxygen flow rate based on awake, resting blood gas measurements does not guarantee adequate oxygenation of COPD patients during their daily life. The elevation of oxygen flow rate by an empirically established amount (presumably by $1-1.5\ l\cdot min^{-1}$) before some daily activities and before sleep, to prevent falls in Sao₂, is suggested. However, a high level of Paco₂ and the type of oxygen source may be a useful tool to detect inadequate oxygenation in patients despite LTOT.

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