

## Recommendations for long term oxygen therapy (LTOT)

Report of an SEP (European Society of Pneumology) Task Group

I have read the recommendations for long term oxygen therapy (LTOT) from the SEP (Levi Valensi P, *Eur Respir J*, 1989, 2, 160–164) with great interest and some surprise. The recommendations deal mainly with the correction of the haemodynamic consequences of hypoxaemia. Certainly the correction of tissue hypoxaemia, for example in the central nervous system (CNS) and the kidneys, resulting from LTOT is important for the beneficial effect on neuropsychologic functioning, polycythemia, and probably quality of life and survival.

The patient selection criteria for the MRC and NOTT studies were not restricted to stable state COPD with  $P_{aO_2} < 7.3$  kPa (55 mmHg) as might be concluded from the recommendations. Both studies included patients with  $P_{aO_2} < 8$  kPa (60 mmHg) if they had one or more episodes of ankle oedema (MRC) or had oedema, polycythemia or P pulmonale on ECG (NOTT).

For patients with COPD and  $P_{aO_2}$  7.3–8.7 kPa (55–65 mmHg) haemodynamic measurements, sleep recording and/or exercise testing are recommended by the SEP task group. In table 2 (SEP Task Group) sleep recording during room air and oxygen breathing is recommended for snoring obese patients. As an alternative arterial blood gas analysis 4 hours after the onset of sleep, "as quickly as possible with apnoea detection" is suggested. For the detection of patients with the sleep apnoea syndrome, who need other forms of treatment than LTOT, and 20% of whom are not overweight, this seems a questionable recommendation.

The improvement of nocturnal hypoxaemia and its haemodynamic consequences with oxygen are mentioned. However, the role of nocturnal hypoxaemia in the development of persistent pulmonary hypertension and cor pulmonale is not known. Several studies show that at least in COPD and the sleep apnoea syndrome nocturnal hypoxaemia without daytime hypoxaemia does not lead to cor pulmonale or to an increased mortality rate [1, 2]. MIDGREN *et al.*, showing that mean  $SaO_2$  during sleep is closely related to mean  $SaO_2$  during wakefulness, is referred to, but in the wrong context [3]. A more extensive discussion of the aims of the investigations and the LTOT

in patients with  $P_{aO_2} > 8$  kPa (60 mmHg) is needed.

In France and Sweden approximately one third of the patients treated with LTOT suffer from chronic hypoxaemia from causes other than COPD, with late sequels of tuberculosis, interstitial fibrosis and kyphoscoliosis as the most common diagnosis [4, 5]. In the Swedish retrospective survey longer mean treatment times after the start of LTOT were found among patients with kyphoscoliosis and sequels of tuberculosis than among COPD patients, indicating that these patients may benefit from LTOT, and these diagnoses could be mentioned among the controversial indications for LTOT. Further investigations of the effect of LTOT in patients with chronic hypoxaemia from other causes than COPD, if possible controlled randomized trials, are certainly needed.

### References

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