Oxygen therapy in smokers

Got a match? Home oxygen therapy in current smokers

Y Lacasse, J LaForge, F Maltais

The problems of providing home oxygen therapy to active smokers

The recent death of two patients registered in our respiratory home care programme stimulated this reflection regarding oxygen therapy in current smokers. Both had oxygen dependent chronic obstructive pulmonary disease (COPD) and died from severe burns and inhalation injury that occurred while they were receiving oxygen through their home oxygen concentrator. The inquiries revealed that both were smoking when the accidents happened and that their oxygen concentrator was functioning properly.

Contrary to popular belief, oxygen is not explosive. Rather, oxygen accelerates combustion and is therefore an obvious fire hazard. Several reports underlining the risks of burn in patients who smoke while receiving oxygen have been published.[1] Reports of death are rare, however. Nevertheless, the proportion of active smokers among patients who receive long term oxygen therapy (LTOT) is almost never mentioned in the current literature. In the few reports that courageously and specifically addressed this issue, the proportion reached almost 20%.[2] Such is our own experience.[3] Those who report that none of their patients on home oxygen therapy currently smokes do not know their patients well.

Smokers get home oxygen therapy because physicians prescribe it. Why is that so? An explanation may be that, in the British Medical Council's trial of LTOT in COPD, smoking was not an exclusion criterion even if, at study entry, all patients were ''urged to give up smoking''.[4] It turned out that 37 of the 87 patients (43%) were counted as current smokers. Smoking was not identified as a predictor of mortality in this trial. In the Nocturnal Oxygen Therapy Trial (NOTT),[5] smoking cessation was encouraged but was not required (Dr Tom Petty, personal communication). Although no mention of patients' smoking status was made in the original NOTT paper,[5] a secondary publication indicated that 38% of participants were active smokers at study entry.[6] Hence, these two landmark trials have set precedents that are now accepted in clinical practice. In addition, guidance from official organisations for the prescription of home oxygen therapy in smokers is either totally absent or, at best, rather vague.[7 8]

Before the fatal accidents described above, our institution had requested legal advice from its lawyers regarding the delicate issue of home oxygen therapy in current smokers. Our respiratory home care programme is funded by the Quebec universal medical insurance plan. It delivers care (mainly LTOT and related services) and provides equipment (including oxygen concentrators) to patients with any chronic lung disease. Most of them have COPD. Two questions were asked. What is the responsibility of our programme and that of our institution in case of such accidents? Can we discontinue our services and withdraw our equipment from patients who persist in smoking?

Home oxygen prescription follows a thorough evaluation of the patient to ensure that the treatment received is otherwise optimal and that oxygen therapy is indeed really indicated. This is the physician's responsibility. During this evaluation, patients are asked about their smoking status which they must frankly disclose. This is the patient's responsibility. The physician must inform his/her patient about the fire hazards of home oxygen therapy and must ensure that he/she agrees to comply with the rules of safety. Whatever the patient's smoking status—but especially if the oxygen prescription is maintained despite active smoking—we were advised to provide our patients with written safety instructions on the use of their oxygen concentrator and to ask them to sign a form in which they acknowledge the fire hazards of home oxygen therapy and consent to receive it. The institution is then responsible for providing non-defective equipment that is in accordance with local regulations.

Only physicians can decide to withdraw home oxygen therapy. This decision must not rest on discriminatory grounds. Some may see smoking as a handicap. Withholding or withdrawing oxygen therapy may therefore be considered as a violation of charters of rights in force in most developed countries. The only reason physicians may put forward in order to refuse oxygen therapy is its real contribution to the patient's health and safety. Futile interventions may be declined, as well as those that are associated with unacceptable risks. In this regard, there is sparse but convincing evidence that cigarette smoking determines the severity of secondary polycythaemia in patients with hypoxaemic COPD and that smoking prevents its correction by LTOT.[9] Secondary polycythaemia only represents a surrogate outcome, and this study did not demonstrate that non-smokers live longer than smokers when on LTOT. Nevertheless, this study suggested that the physiological mechanisms by which home oxygen is thought to operate are inhibited by smoking. Unfortunately, the evidence that non-smokers on LTOT fare better than smokers on LTOT will never come from randomised controlled trials.

Despite the above considerations, the provision of home oxygen therapy to active smokers remains a difficult issue. We cannot provide firm recommendations but only suggestions. Firstly, we would not usually screen for resting hypoxaemia in stable patients who smoke. Before drawing arterial blood, the advantages of home oxygen must be balanced against its risks. If it is felt that the patient will not comply with the safety procedures and especially if there is good reason to believe that the patient will smoke while on oxygen, then it is medically justifiable not to prescribe it. Secondly, the indication for home oxygen therapy must be clearly ascertained before it is offered. Although guidelines for LTOT in patients with COPD are universally accepted,[10] inappropriate prescriptions are not unusual.[11] Unfortunately, recommendations of scientific societies regarding the indications for home oxygen in circumstances other than severe daytime hypoxaemia in COPD are often imprecise.[12] In our opinion, indications for home oxygen therapy that are not clearly evidence based should be reconsidered, especially in active smokers—for example, an oxygen prescription to prevent exercise induced desaturation. Thirdly, systematic re-evaluation following the initial prescription of oxygen therapy made within the course of an acute exacerbation of COPD is mandatory. Given the likelihood of smoking resumption following several days of in-hospital abstinence, the decision to discharge the patient with home oxygen therapy is

even more difficult in such circumstances, especially if profound hypoxaemia exists. Fortunately, at least 30% of patients meeting the criteria for domiciliary oxygen after 1 month of apparent stability no longer met the same criteria after an additional 3 months of observation.[13]

In the last 25 years there have been exciting advances in the management of chronic lung diseases. Therapeutic modalities effective in reducing COPD related impairments have received attention, often in randomised trials. Such is the case for home oxygen therapy which is tertiary prevention. Early detection and intervention on individuals at risk for the late consequences of COPD (secondary prevention) and continuing antismoking campaigns (primary prevention) must not be forgotten. Smoking cessation falls into the latter two categories. Otherwise the cost effectiveness of our tertiary prevention interventions may be jeopardised.

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LUNG ALERT

Vitamin D3 and response to glucocorticoids in steroid resistant asthmatics

▲ Xystrakis E, Kusumakar S, Boswell S, *et al*. Reversing the defective induction of IL-10 secreting regulatory T cells in glucocorticoid-resistant asthma patients. *J Clin Invest* 2006;**116**:146–55

This study showed that human IL-10 secreting regulatory T cells (Tregs) inhibit cytokine production from allergen specific Th2 cells in an IL-10 dependent manner. They therefore have the capacity to inhibit the immune response implicated in the pathogenesis of asthma. In steroid resistant asthmatics the failure of T cells to significantly induce IL-10 synthesis in response to dexamethasone was enhanced by the addition of vitamin D3. This restored levels of IL-10 to those seen in steroid sensitive individuals stimulated by dexamethasone alone. Potential mechanisms were explored and it was shown that dexamethasone downregulated glucocorticoid receptor expression, which could be reversed by the addition of vitamin D3. In addition, IL-10 was shown to increase glucocorticoid receptor expression. This suggests potential mechanisms by which poor glucocorticoid responsiveness can be overcome. Oral administration of vitamin D3 in seven steroid resistant asthmatics enhanced the IL-10 response to dexamethasone.

The authors conclude that induction of IL-10 synthesis may contribute to the clinical efficacy of glucocorticoid therapy in asthma. Patients who fail to respond clinically to glucocorticoids also fail to respond ex vivo to induction of IL-10 synthesis and this may be useful as a predictive tool. Induction of IL-10 secreting Tregs in this group of glucocorticoid resistant patients is an appealing therapeutic area. Vitamin D3 enhances IL-10 synthesis in glucocorticoid resistant patients, and there may be potential benefit in administering vitamin D3 in asthmatic patients other than as prophylaxis against glucocorticoid induced osteoporosis.

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