to COPD then it must be associated with a commensurate physiological abnormality, which is a reduction in after-bronchodilator forced expiratory volume in one second (FEV1) or at least abnormality of the flow/volume loop. In medico-legal practice, I have been seeing large numbers of subjects managed principally in general practice with COPD or symptoms wrongly attributed to it. Too frequently, limitation of exercise cannot be explained by the objective reduction in pulmonary function. My previous clinical practice leads me to believe that this is not confined to any particular group of patients and certainly not related to litigation. The vast majority of patients have access to appropriate medication and use it in at least the prescribed dose, so there is little room for improvement in the pharmaceutical approach. Nevertheless there is much perceived and real morbidity. The tragedy is that much of the associated disability is not only accepted too passively, but is also unnecessary.

The diagnosis may be made on the basis of no pulmonary function tests or tests of poor quality. Often, the mere presence of cough and sputum or an industrial history leads to the diagnosis, which is then indelibly reproduced in the notes without thought as to its validity, even if subsequent pulmonary function tests are normal. Too often, a history of breathlessness is accepted at face value as objective evidence of disability due to respiratory disease. The perceived level of exercise limitation probably does reflect actual activity, but is rarely objectively confirmed either explicitly or opportunistically as, for example, by comparison with performance on the Bruce protocol. Overdiagnosis is only part of the problem. Many of these subjects do have mild COPD as demonstrated by minor changes in FEV1 or the shape of the flow/volume loop, but with disproportionate exercise limitation. The clinician accepts the diagnosis, maybe rightly, but attributes disability directly to it, usually wrongly.

The problem arises because breathlessness is incorrectly regarded as the prime symptom reflecting impairment associated with cardiorespiratory dysfunction. This, however, is not the case. It is exercise limitation itself that is the proper measure of cardiorespiratory dysfunction. If cardiorespiratory dysfunction is the direct cause of this exercise limitation then it must be associated with changes in carbon dioxide or acid-based balance causing fatigue or the legs to give out. This is well recognised in athletes who accept breathlessness as incidental. It should apply equally to those with COPD, particularly when the disease is mild. In practice it is the dislike of breathlessness itself and the consequent vicious circle of increasing perceived breathlessness, exercise restriction and muscle weakness that is the prime cause of disability in COPD and not the actual impairment of lung function. Whether or not the COPD itself contributes to the perception of breathlessness is immaterial.

Management must involve acceptance by the patient of the true cause of disability, a message often difficult to convey in these days of patient autonomy, followed by rehabilitation to reverse the vicious circle. The approach might include: 1) taking history, directed first at the extent of disability and then at the associated symptoms, with the least reliable, breathlessness, last in the list; 2) objective assessment of exercise tolerance in all cases; 3) an absolute requirement for a physiological confirmation of the diagnosis by spirometry, including flow/volume loops (most unhelpful in this respect is the concept of stage 0 COPD as cough and sputum; it should be normal FEV1 with abnormality of the flow/volume loop); 4) development of tables of optimal rather than average exercise tolerance against FEV1 % predicted, stratified by the shape of the flow/volume loop; and 5) immediate intervention with low-tech rehabilitation in primary care, with the triple benefit of preventing unnecessary morbidity, ensuring that those whose disease does subsequently decline are already in the best position to cope with it, and reducing the impact of COPD as comorbidity in other conditions.

By addressing the underlying problem of inability to cope with breathlessness, this physiological approach might substantially reduce the burden of COPD, which is indeed excessive, but not always for the reasons suggested by RENNARD *et al.* [1] and DEKHUIJZEN [2].

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### References

1. Rennard S, Decramer M, Calverley PMA, *et al.* Impact of COPD in North America and Europe in 2000: subjects' perspective of Confronting COPD International Survey. *Eur Respir J* 2002; 20: 799–805.
2. Dekhuijzen PNR. The Confronting COPD International Survey: patients hardly know they have COPD. *Eur Respir J* 2002; 20: 793–794.

*From the authors:*

We are pleased to respond to the comments made by C.K. Connolly. We completely agree with the need to dissociate disability from dyspnoea in the clinical assessment of the chronic obstructive pulmonary disease (COPD) patient. Dyspnoea can arise from many inputs, and, while it correlates with airflow limitation, it does so relatively poorly. Dyspnoea can limit performance. We agree with the suggestion that evaluation of exercise performance can assist in the evaluation of COPD patients. Muscle weakness, however, is a better predictor of disability in COPD than airflow, and this weakness may be due not only to detraining, but also to the inflammatory processes and circulating cytokines that characterise some COPD patients. Because of this latter point, we disagree with C.K. Connolly's suggestion that disability in COPD must be related to the measurable forced expiratory volume in one second (or flow/volume loop) abnormalities.

Clearly the chronic obstructive pulmonary disease patient's clinical status reflects not only the impaired airflow, but also other pulmonary and systemic aspects of the disease. We doubt the disease is underdiagnosed; our survey, in fact, focused on diagnosed cases. Among those individuals, we have little doubt that it is underevaluated. C.K. Connolly's suggestion that more aggressive assessment of objective measures in the chronic obstructive pulmonary disease patient is needed is one with which we wholeheartedly agree. More aggressive identification of the undiagnosed individuals will also be important.

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