non-invasively or the need to protect the airway despite the use of manually assisted coughing. It has been demonstrated that non-invasive systems can almost invariably adequately ventilate the subject and it is a defective swallow or cough that usually leads to the need for a tracheostomy[4] apart from the convenience of being ventilated through this route if respiratory support is needed virtually or completely continuously.

Complications of the tracheostomy itself were infrequent, but almost one-third of the patients required admission for treatment of a chest infection despite their tracheostomy. There was only one death from a respiratory infection in this series and overall 78.9% of patients survived for 1 year. Interestingly, nine patients died suddenly raising the possibility that there is a defect in sympathetic activation in response to stress as part of the neurological deficit in motor neuron disease.

These findings present an optimistic view of tracheostomy ventilation, but quite frequent psychiatric assessments were required, mainly for anxiety and depression. It could be argued that depression is a feature of motor neuron disease and the limitations that it causes occur even without a tracheostomy. A control group would be required to assess whether tracheostomy actually leads to more or less anxiety and depression. Their incidence and severity appeared to be related more to the degree of social support available, the coping capacity of the individual and the loss of independence rather than to the more direct physical effects of the tracheostomy itself. There is also no doubt that tracheostomy ventilation requires more assistance from the family and carers than non-invasive systems. Its impact on the carers and family needs to be carefully assessed and further studies of this are required.

Although there was a shared approach to the decision to start tracheostomy ventilation, this report gives no data about why 38 subjects refused this treatment or why the other 38 accepted it. Previous studies have shown that young males are more likely to accept a tracheostomy than older females.[5 6] This may be related to a common perception that a tracheostomy is a life-saving operation in contrast to the apparently more modest effect of an improvement in the quality of life that can be achieved by non-invasive ventilation.[7] The individual's values such as autonomy and communication, and fears such as of choking and of dying are also important in deciding whether or not to undergo a tracheostomy.[7] Advanced directives have not proved as useful in practice as was anticipated and do not appear to have been of value in this study. It is hard for the individual to anticipate how he or she will feel at the time that a tracheostomy is required. This may not seem attractive early in the natural history of the condition but later, when faced with the situation, many people will take up the offer of a life-saving treatment rather than agreeing to imminent death.

There is no simple answer to when or whether tracheostomy ventilation should be offered to those with motor neuron disease. Whether or not the chance of an increased survival outweighs the intrusiveness of the treatment depends on how the patient values the various aspects of the quality and quantity of life and assesses the impact of the illness and its treatment on family, friends and carers. While some of these issues are primarily medical, others depend on the individual patient's perceptions. It is only when both doctor and patient understand each other's points of view that the correct decisions will be made.

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Muscle mass and strength in obstructive lung disease: a smoking gun?

Samantha S C Kon, William D-C Man

Over the past 2 decades, there has been increasing interest in the extrapulmonary manifestations of chronic obstructive pulmonary disease (COPD). This is supported by the clinical observation that patients show significant improvements in functional performance and health-related quality of life with pulmonary rehabilitation in the absence of lung function changes.[1] Furthermore, the forced expiratory volume in 1 s is an imperfect predictor of mortality in severe patients with COPD,[2] and when forced expiratory volume in 1 s is corrected after double lung transplantation, peak exercise remains only approximately 50% of predicted up to 1–2 years after surgery.[3] This limitation in exercise and functional capacity has led to a particular focus upon the skeletal muscle compartment. Certainly, a surprisingly high proportion of patients with COPD terminate exercise complaining of muscle effort.[4] Cross-sectional studies have typically demonstrated muscle weakness[5] and reduced endurance,[6] particularly of the lower limbs, in COPD compared with age-matched controls. This is corroborated by biopsy findings of muscle fibre atrophy and muscle fibre shift from type I to type II fibres.[7] Skeletal muscle dysfunction seems to be clinically relevant in COPD, as

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loss of skeletal muscle mass and strength are associated with poor health status,[8] increased healthcare use[9] and even mortality,[2 10] independent of lung function parameters.

Debate continues as to the relative importance of systemic and local factors in the aetiology of skeletal muscle dysfunction in COPD. Potentially relevant factors include blood gas abnormalities, corticosteroids, nutritional depletion, anabolic–catabolic hormone imbalance, systemic or local inflammation, oxidative stress, genetic susceptibility and reduced daily physical activity.[7] Studies in patients with COPD have been either cross-sectional or longitudinal without an adequate control group, and it has been difficult to tease out the influence of confounding factors.

In this issue of *Thorax*, van den Borst and colleagues[11] present data from the Health ABC study. This longitudinal study was designed to document the extent of change in body composition (including muscle mass, fat mass and bone mineral composition) in older men and women, identify possible clinical conditions that accelerate these changes and examine the health impact of these changes on strength, endurance, disability and weight-related diseases of old age. In older people, as in COPD, sarcopaenia (loss of muscle mass and strength associated with ageing) is independently associated with disability and mortality.[12] The investigators retrospectively used baseline lung function criteria to divide the Health ABC cohort into those with obstructive lung disease (OLD) and controls. Patients with restrictive spirometry were excluded from analysis. The controls were then further divided according to self-reported cigarette smoking status. This study is therefore both novel and welcome in that it provides longitudinal data about decline in body composition and physical functioning in both patients with presumed COPD and well-matched (smoking, never-smoking and formerly smoking) controls. Although some of the longitudinal data are conflicting between the genders, the overall message is that downward trajectories in body composition and physical function are largely comparable between those with OLD, current-smoking controls and never-smoking controls. The implication is that neither COPD nor smoking accelerates sarcopaenia.

However, caution is required in the interpretation of the results. First, the diagnosis of OLD was based on spirometry rather than a clinical diagnosis, and almost a quarter of the women in this group were never-smokers. Second, the men with OLD seemed to have significantly slower decline in lung function than the never-smoking controls, which raises doubt as to whether the group with OLD is representative of COPD. Lastly, the study population was selected on the basis of age (between 70 and 79 years) with good baseline functioning and hence subject to survivor bias. This cohort is therefore unlikely to be representative of either older adults or the patients typically seen by healthcare professionals. However, the most striking observation in this study was the reduced baseline difference in body composition and physical functioning in the OLD and smoking control groups compared with the former-/never-smoking controls. There may be multiple contributing factors such as early life developmental influences, less-than-optimal diet, certain medications, hospitalisations or reduced daily physical activity, but the data might also suggest that smoking causes a common and early insult to the skeletal muscles.

Could smoking be the unifying aetiological factor for COPD-related skeletal muscle dysfunction? In support of this, smoking causes a reduced proportion of type I fibres, type I and IIa fibre atrophy in the soleus muscle[13] and type IIa and IIb fibre atrophy in the extensor digitorum longus of rats.[14] In humans, increased quadriceps muscle fatigability has been observed in young healthy smokers.[15] Smokers without COPD also have vastus lateralis biopsy evidence of oxidative fibre atrophy and increased glycolytic capacity.[16] Seymour *et al*[17] recently demonstrated that 8% of healthy subjects with normal lung function have evidence of significant quadriceps weakness with smoking history an independent risk factor. Longitudinal studies are required to explore this relationship further. The differential effects of acute and chronic cigarette smoke exposure on skeletal muscle have also not been clarified. Furthermore, interventional (smoking cessation) studies with a focus on the effect upon skeletal mass and functioning would be of particular interest. The current study in *Thorax*[11] demonstrated no differences in body composition and physical functioning between former smokers and never smokers at baseline. If smoking does indeed induce an early insult to the skeletal muscles, these data provide hope that the damage is potentially reversible.

A confounding factor for studies in body composition and physical functioning is reduced daily physical activity. Smoking, for example, is negatively associated with physical activity levels.[18] Although technologies to objectively measure energy expenditure and physical activity are rapidly improving, activity monitors remain expensive, time-consuming and not suitable for routine clinical use. Unsurprisingly, there is a paucity of long-term longitudinal studies examining the relationship between physical activity and body composition, particularly in populations before the development of COPD or sarcopaenia. At a cross-sectional level, objective or subjective measurements of physical activity at a single time point cannot hope to quantify decades of sedentary lifestyle. Although van den Borst and colleagues[11] found no difference in subjective physical activity levels at baseline between the groups, quadriceps weakness was observed in the group with OLD and current-smoking controls in the presence of preserved handgrip strength. These data are corroborated by non-volitional muscle strength studies in patients with COPD, supporting the localisation of skeletal muscle dysfunction to the underused muscles of ambulation (ie, quadriceps) with preservation of strength in the adductor pollicis and diaphragm.[5]

Existing data no longer support the concept that skeletal muscle dysfunction is a manifestation of severe COPD but rather a problem that can exist before the onset of symptoms. Apart from this current study,[11] Seymour *et al*[17] demonstrated that a substantial proportion of patients with COPD in Global Initiative for Obstructive Lung Disease stages 1 and 2, or with an MRC dyspnoea score of 1 or 2, had significant quadriceps weakness (28% and 26%, respectively). These data are further supported by Watz and colleagues[19] who, using objective activity monitoring, demonstrated that only 26% of patients with Global Initiative for Obstructive Lung Disease stage 1 had a physical activity level considered 'active'. Whether one believes that smoking induces a direct early insult to the muscle or whether it is merely contributing to physical inactivity, van den Borst's study raises the question (and challenge) of how to identify asymptomatic people at risk of declining body composition and physical functioning and whether we should be providing effective interventions, such as pulmonary rehabilitation, at a much earlier stage than present.[20]

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Action plans for COPD self-management. Integrated care is more than the sum of its parts

M D L Morgan

The past few years have seen significant changes in attitude in many countries towards the care of people with chronic obstructive pulmonary disease (COPD). These changes have been driven by a greater understanding of the disease and the nature of its impact. Although there have been no new therapies that can modify the course of airway obstruction, there have been considerable advances in the way that we can lessen the impact of the disease on both the patient and the health community. There has clearly been a desire to improve the lives of individual people with COPD, but the major catalyst for change has probably been the desire of commissioners to reduce the cost of unnecessary hospital admissions resulting from exacerbation. To this end, the development of admission avoidance schemes has stimulated integrated community care programmes and importantly the involvement of the patient through a process known as self-management. The constituents of published self-management programmes vary quite widely, but most contain an action plan in the form of a set of instructions to inform the patient how to recognise an exacerbation and act accordingly. This is usually presented in a written format but can be pictorial if there are language difficulties or literacy issues.[1] If they are also armed with treatment in the form of steroids and antibiotics, then it would be hoped that prompt action by the patient would then attenuate the serious exacerbation that would otherwise result in a hospital admission. The article by Trappenburg[2] in this month's journal sheds further light on the individual effectiveness of the action plan on the outcome of acute exacerbations of COPD.

In spite of the fact that early treatment may improve the outcome of exacerbations, the introduction of stand-alone action plans and access to medication for patient-initiated use has been largely disappointing.[3] The data from the most recent systematic review covering five trials indicate that although they increase recognition and steroid/antibiotic use, they have no impact on the use of healthcare resources. In particular, no reduction in hospital admissions, emergency department visits or GP attendances was evident.[4] It seems that people with action plans recognise exacerbations and take more therapy without affecting the outcome. This does not seem to make a lot of sense when you appreciate that they will get essentially the same treatment when they do end up in the hospital. This year, two papers in *Thorax* have shed some light on this puzzle. In the first paper, Bischoff *et al*[5] explored the effect of adherence to written action plans on the recovery from exacerbation in 143 patients who had 288 exacerbations. The written plan was only followed in 40% of the exacerbations, but where patients

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