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Letters to the Editor

Dear Editor

Treadmill exercise duration and dyspnea recovery time in chronic obstructive pulmonary disease: effects of oxygen breathing and repeated testing

We read the report by Marques-Magallanes *et al.* (1), recently published in this *Journal,* with interest and the conclusion with concern. The aim of the study was to ascertain the effect of oxygen, compressed air and air breathing, on dyspnea and recovery time following repeated treadmill exercise in COPD. The compressed air and oxygen were delivered through a low-flow face mask (M/C mask) with a fixed flow of 10 l min^{-1}. The authors suggest that this type of mask delivers a fractional inspired oxygen concentration (FIO_2) of approximately 40%. COPD patients have been shown to achieve peak tidal inspiratory flow rates of up to 360 l min^{-1} during exercise (2). A flow rate of 10 l min^{-1} delivered by the mask would therefore be considerably less than the inspiratory flow rate of the patient at peak exercise. The consequences of not matching patient and device flow rate during the recovery period are reduced oxygen concentration due to air entrainment (3) and a further increase in inspiratory muscle work as the patient overcomes the resistance of the mask. This is demonstrated by the increased recovery time of the compressed air breathing with the mask compared to breathing air without a mask. Although dyspnea was measured using VAS scores, these data were omitted from the results. These data may have provided useful information in interpretation of symptoms in relation to the three tests. The authors quite rightly suggest that ventilatory parameters during exercise are most closely related to dyspnea (2) and specifically (*in that paper*) to peak tidal inspiratory flow rate. Inspiratory flow rates are determined by the mechanical properties of the lungs and chest wall and the intensity of the simulation of the inspiratory muscles and their strength. Muscles fatigue more rapidly when inspiratory flow rates are high (4). The authors recognized the possibility of muscle fatigue during and following exhausting exercise and speculated this as a reason for shorter endurance time with the second and third tests. In this study the load on the inspiratory muscles was increased during the recovery period rather than relieved. It is recognized that oxygen alone is not responsible for relief of dyspnea but a combination of relief of hypoxia and inspiratory load. The effect of this has been demonstrated in studies using CPAP (5). The authors conclude that oxygen administration during exercise recovery has no effect on reducing dyspnea recovery time and thus should not be recommended as intermittent treatment for post-exercise dyspnea. The patients in this study were severely compromised achieving only 80–90 s of treadmill walking. Meticulous attention to oxygen delivery may provide different results. It would be of considerable importance to see if the results of this study were replicated supplying an appropriate oxygen concentration and adequate flow rates before such recommendations are implemented.

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Reply to the Editor

We acknowledge the comments and concerns of Dodd *et al.* regarding the methodology and conclusions of our study. There is no doubt that entrainment of air when using 'low flow' oxygen delivery systems produces an element of uncertainty regarding the inspired oxygen concentration (FIO_2) which is achieved. Also, we agree that a low flow mask offers some resistance to entrainment which might increase the work of breathing. This mechanism might tend to increase recovery time offsetting any beneficial effect of oxygen. However, the reality is that this is how patients are inclined to use supplemental oxygen. Furthermore, some

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physicians are inclined to recommend its use in his fashion. In describing the methodology of our study, we specifically state that 'the experimental conditions were intended to reflect the clinical circumstances under which patients take supplemental oxygen, accepting that an absolutely constant $F\dot{I}O_2$ could not be guaranteed'. Our conclusions are therefore practical and based on how we believe oxygen is commonly used.

We agreed that it would be interesting to test the effect of an assured, constant $F\dot{I}O_2$ on dyspnea recovery time avoiding any increase in respiratory muscle work. Such a study would require a high flow or reservoir oxygen delivery system with application of positive pressure such as CPAP and the likelihood of such systems gaining approval for clinical practice is remote. Even if meticulous attention to these factors revealed a positive effect of supplemental oxygen on post-exercise dyspnea, we believe that such an effect is likely to be small. Of far greater consequence, as shown in our study, is the effect of prior exercise, and presumably pre-existing metabolic factors such as lactic acidosis or respiratory muscle fatigue, in determining the duration of dyspnea recovery. Patients whose breathlessness scores had returned to baseline between 20 and 30 min after exhausting walking exercise were unable to exercise again for the same duration, indicating that although they felt they had recovered symptomatically, physiological recovery was incomplete.

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Dear Editor

Of Bourne, badgers, and a bovine 'TB alert'

In response to the global upswing of tuberculosis, a new initiative 'TB Alert' was launched by two Liverpool doctors, P. Davies and B. Squire, on 5 June at the Royal Society of Tropical Medicine & Hygiene, London. Mainly focused on human TB, attention was also drawn to the worsening bovine TB epidemic in British cattle, already increased by 45% over last year, and my warning of a risk to man, particularly via unpasteurized bulked milk (1,2), and of the value of cross-fertilization of ideas. The new Bourne badger/cattle initiative launched on 17 August (3) belatedly recognizes this risk, whilst a further possible link with another *Mycobacterium* (*M. paratuberculosis*), which is not invariably killed by pasteurization, is noted as a cause of Johne's scouring disease in cattle and perhaps Crohn's bowel disease in man (4). Ubiquitous in cattle, sheep, goats, pigs and horses, this may be one cause of false-positive 'TB reactors' in cattle (5). In fact, there is an exact parallel between the difficulty in diagnosing and treating drug-resistant TB in man early enough to prevent spread and the inadequacies of current cattle test/trace procedures: the skin test is only approximately 80% sensitive, thus missing perhaps one in five TB carriers, whilst in late herd eradication situations up to 80% of reactors may be later found to have TB false-positive results (due to *M. avium, M. paratuberculosis,* etc.).

Progress in science is most rapid where often very simple 'right questions' yield 'right answers', and is greatly hampered by asking the 'wrong questions' or by what Charles Darwin described as 'false facts' (1). Koch discovered the tubercle bacillus in 1882, but then did great damage by claiming that bovine TB was of little relevance to man. It took a Royal Commission a decade or so to re-establish the risk of unpasteurized milk in 'scrofulous' children particularly, and it is ironic that pasteurization is still not mandatory in law in the U.K. (apart from in Scotland).

Sadly, a very quick and simple resolution to 'The Great Badgers and Bovine TB Debate' is being overlooked as politically incorrect (2), and instead the Bourne/Krebs 'scientific' badger cull (3,6) is unlikely to resolve matters since it raises the wrong questions, will be costly, impractical, and the statistics 'won't be able to cope with so many bodge factors' (7). This is political expediency pursued to the point of absurdity', as an ex-Ministry vet said over a decade ago, whilst the last independent scientific review then found what Bourne will 're-discover' after 5 years; that badger culls are simply a waste of money because they do not work (8,9). The debate arising from this initiative has at least highlighted the simple flaws in the Krebs/Bourne review. It is worth noting where the 191-page Krebs Report is weak (6): it does recognize that cattle are infectious at any stage of the disease; at the early microscopic or non-visible lesion (NVL) or late gross visible lesion (VL) stage which could be reached within a month (pages 100, 104) (1,2, 10,11). However, despite noting very briefly that spill-over from cattle to badgers and other wildlife might occur (pages 24, 152–155), it fails to appreciate the implications of this pivotal flaw in the protocol for deciding 'Attribution of source' of herd breakdowns (pages 59, 90, 163), as well as the flaw in claims that badger culls 'work' (page 30). The claim that badger TB is of respiratory aetiology is repeated (page 45), even though it often starts as a cervical lymphadenopathy of the submandibulars, i.e. dietary 'scrofula' from eating invertebrates from under cow pats, or as in the 1950s in Swiss badgers, feeding on roe deer carrion (1). Lastly, Krebs noted, but did not explain, the dramatic rise of TB in new Midlands and Welsh herds, and was not made aware of the rise in incidence of cattle TB, even though such data can be found in the Ministry of Agriculture, Fisheries and Food (MAFF) annual Badger and Animal Health Reports ('data unavailable', pages 13, 56, 141, 156).

It will be necessary to 're-discover' the basics of cattle TB before any progress can be made towards eradication, with cattle TB as bad today as it was in the early 1960s, before badgers became 'implicated' (page 141). The classic studies note that cattle are infectious whether NVL or VL, so TB can spread inexorably within and between herds, forming clusters of herds or TB 'pockets'. The test/slaughter policy brought the incidence of cattle TB down from Midlands