



CORRESPONDENCE

Panic attacks in COPD and the somato-psycho-somatic feedback

To the Editors:

In the March 2010 issue of the *European Respiratory Journal* I encountered two articles on chronic obstructive pulmonary disease (COPD) which gave me an incentive for a comment.

In the excellent review by MACKLEM [1], the role of a high respiratory rate in severe COPD is discussed, in particular with its consequence of dynamic hyperinflation and increased work of breathing. The author also addressed the need of proper instruction for the patients to avoid rapid breathing and to increase tidal volume (slow, deep breathing) whenever possible.

The other article by LIVERMORE *et al.* [2] deals with panic attacks in COPD and how to successfully avoid them by cognitive behavioural therapy (CBT).

What links these two papers is a propensity for rapid breathing, which is an inevitable feature of high-intensity exercise and most likely, but not so easy to assess, in panic disorders.

What we can observe here is what one could call a somato-psycho-somatic feedback. Emotional stress (with the urge to sigh and hyperventilate) as well as exercise (beyond aerobic threshold) in patients with severe COPD can raise the respiratory rate to an extent that dynamic overinflation ensues. This, in turn, can progress to a dramatic reduction in inspiratory capacity, further increase of shallow breathing at an even higher frequency, with eventually neuromechanical uncoupling [3] and the development of a kind of intrinsic positive end-expiratory pressure. Then, when patients excessively use their emergency inhaler to no avail, they will experience panic and mortal fear. In a situation like this, pump failure of respiration with CO₂-retention would be common.

COPD patients, who are suspected of having panic attacks, eagerly confirm such a mechanism when alerted to the possible links between panicking and dyspnoea on the aforementioned occasions. As stated in the review by MACKLEM [1], there seems to be a close relationship of respiratory rate and rise in carbon dioxide tension. Hence, a hypercapnic reaction could throw COPD (Global Initiative for Chronic Obstructive Lung Disease stage 3–4) patients off the track. When properly informed, most of them consider training themselves to counteract these mechanisms by slow pursed-lip breathing, maybe supported by a small dose of oral lorazepam.

I have occasionally encountered such patients who had been treated with high-dose systemic steroids and bronchodilators, reminding me of patients with vocal cord dysfunction or hyperventilation syndrome who were mistaken for asthmatics.

In conclusion, panic attacks as well as physical exercise can lead to rapid breathing in severe COPD patients with the

consequence of dynamic hyperinflation. A dangerous somato-psycho-somatic feedback cycle can occur in patients who, in the state of maximal overinflation, loss of inspiratory capacity and neuromechanical uncoupling, would panic even more because their bronchodilator will not give them any relief.

In general, in dyspnoea on exercise or panic disorders, the emergency use of bronchodilators in COPD patients should be discouraged. Instead, patients should be convinced of the importance of slow and deep breaths, even during exercise. Optimally, they should foresee the point when they would lose control over their breathing and pause until they regain the ability of slower breathing.

F. Kummer

Correspondence: F. Kummer, Karlsgasse 9/11, Vienna 1040, Austria. E-mail: fkummer@aon.at

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From the author:

Although most of the medical literature is written by professional investigators, we can learn a great deal from the wisdom of expert clinicians. Such is the case with F. Kummer's correspondence. This is not to say that F. Kummer is not a professional investigator; PubMed lists at least 92 of his publications going back to 1965. But today he writes of his clinical experience in teaching patients with COPD how to cope with panic attacks. These occur with emotional stress. Wisely, he discourages the use of bronchodilators while telling his patients to regain control of their breathing and to breathe slowly and deeply. When they do this the work of breathing decreases, breathing becomes easier and panic is relieved. We

should hear more from expert clinicians like F. Kummer. It is important for them to share their wisdom with us.

P.T. Macklem

Correspondence: P.T. Macklem, Meakins Christie Laboratories, McGill University, Medicine, Montreal, QC, Canada. E-mail: peter.macklem@gmail.com

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From the authors:

In our original article we outlined the type of somato-psychosomatic feedback cycle occurring in panic attacks in chronic obstructive pulmonary disease (COPD) that F. Kummer has further elaborated in his correspondence. In fact, this type of feedback cycle is described in the most widely accepted theory of panic attacks and panic disorder in the physically healthy, the cognitive (or “catastrophic interpretation”) model [1], and our recent research has supported the applicability of this model to panic in COPD [2–4]. We agree with F. Kummer that some of the most helpful advice respiratory physicians can give their patients with COPD to prevent them entering the feedback cycle of panic is to use “pursed-lip breathing” to help manage their dyspnoea, and to avoid excessive use of short-acting β -agonists.

We would caution that for individuals with COPD who have already begun to experience panic attacks, for those who have fully developed panic disorder, and even those who are psychologically vulnerable to panic, this sensible advice in isolation will unfortunately not be a sufficient intervention. However, the type of brief, evidence-based psychological intervention that we have reported, when added to comprehensive pulmonary rehabilitation programmes, can help COPD

patients to self-manage their own care more effectively [5], treat panic attacks when already present and prevent the development of panic attacks or panic disorder. A more extended psychological intervention will be required for patients who have fully developed panic disorder [3], but without the inherent risks of benzodiazepine medications in the elderly: dependence, cognitive impairment and falls [6].

N. Livermore*, L. Sharpe[#] and D.K. McKenzie[†]

*Liaison Psychiatry, The Prince of Wales Hospital, Randwick, [#]School of Psychology, University of Sydney, and [†]Respiratory and Sleep Medicine, Prince of Wales Hospital, Sydney, Australia.

Correspondence: N. Livermore, Liaison Psychiatry, The Prince of Wales Hospital, Barker St, Randwick, NSW, 2031, Australia. E-mail: Nicole.Livermore@sesiahs.health.nsw.gov.au

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Inspiratory muscle strength and Borg dyspnoea score

To the Editors:

I read with great interest the paper of JUST *et al.* [1]. They demonstrated that the Borg dyspnoea scale, particularly assessed in the supine posture, is a useful noninvasive predictor of voluntary respiratory muscle strength tests in patients with amyotrophic lateral sclerosis and has good sensitivity and specificity; however, there are a few points that need clarification.

It is known that the pressure values obtained during voluntary respiratory muscle strength tests (maximum sniff and maximal inspiratory and expiratory pressure ($P_{I,max}$ and $P_{E,max}$, respectively)) are significantly lower in the supine posture [2].

Thus, it may be more appropriate to correlate the supine Borg with the supine values of those tests, instead of the ones obtained seated. Such a relationship may increase the predictive value of the Borg scale.

It is not clear whether the vital capacity (VC) and the rest of the respiratory muscle strength tests were performed in the upright (standing) or seated posture. At least VC values are expected to be higher in the upright position [3].

In the methods, under the “Pulmonary function tests” section, it is stated that “Static mouth pressure was measured using a flanged mouthpiece [...] at functional residual capacity, as previously described” by BLACK and HYATT [4]. Perusing the