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

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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].

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REFERENCES


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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 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]. Persuing the
latter paper, I noted that the authors never used a flanged mouthpiece nor did they make their $P_{\text{I, max}}$ measurements from functional residual capacity but rather from residual volume. The type of mouthpiece and the way it is used result in large pressure differences obtained during the measurements of $P_{\text{I, max}}$ and $P_{\text{E, max}}$ [5]. Lung volumes also affect these measurements and appropriate reference values should be used [6].

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From the authors:

In response to the question raised by N.G. Koulouris, we confirm that vital capacity (VC) and the respiratory muscle strength were all performed in the seated posture. We agree with N.G. Koulouris that correlations between the supine Borg and the supine respiratory muscle strength values might be better and it would be relevant to verify this hypothesis. However, as a matter of routine, only the VC was performed in both the seated and supine positions.

Measurement of the maximal inspiratory pressure ($P_{\text{I, max}}$) is conventionally easier to obtain from residual volume (RV) and greater inspiratory pressures are obtained at lower lung volumes. However, in the neuromuscular disorders, the recoil pressure of the respiratory system at RV may be a significant fraction of $P_{\text{I, max}}$. The recoil of the chest wall and lungs is equal at the functional residual capacity (FRC). The difference of values obtained from RV and FRC is not important in healthy subjects [1]. In patients with neuromuscular disorders, the advantage of measuring the voluntary inspiratory strength from FRC is that only the force of the inspiratory muscles is assessed and not the negative recoil pressure of the respiratory system. Changing the reference in the text, as demonstrated in a study by Uldry et al. [2], is more suitable. Indeed, we used the predicted values of Uldry et al. [2] which were measured at FRC.

N.G. Koulouris demonstrates that better values of inspiratory strength were obtained with a tube mouthpiece rather than a flanged mouthpiece in healthy subjects [3]. Patients find the flanged mouthpiece easier than the tube explaining its widespread use [1]. In our experience with neuromuscular disorders, especially in amyotrophic lateral sclerosis with bulbar involvement, air leaks were less important with a flanged mouthpiece [4].

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Do $\beta_2$-agonists inhibit capsaicin-induced cough?

To the Editors:

We read with great interest the paper by Freund-Michel et al. [1] in a recent issue of the European Respiratory Journal, because the results are inconsistent with the medical common sense that $\beta_2$-agonists do not have common antitussive property.

The authors showed that a $\beta_2$-agonist, terbutaline (0–3 mg·kg$^{-1}$), dose-dependently inhibited $10^{-4}$ M capsaicin-induced cough in

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