PostScript

LETTERS TO THE EDITOR

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Atopic cough

The correspondence on atopic cough between McGarvey/Morice and Fujimura/Ogawa[1 2] raises a number of very important issues regarding the diagnosis and treatment of chronic cough. These issues warrant careful consideration, not only because of the huge illness burden posed by the frequency of chronic cough but also because issues of terminology and recommended treatment continue to be unclear and irregularly applied.

The ''3Rs'' of chronic cough—rhinitis, reflux and reactive airways (asthma)—have a certain appeal. They are recognised, often repeated (primary research articles were outnumbered by reviews, letters and case reports on chronic cough in 2002–2003), and easily retained in the short term memory of busy clinicians. In clinical practice they are useful. But there are a number of crucial issues that remain to be addressed. The 3Rs frequently coexist in patients with chronic cough, which means there are more diseases than there are patients, and that can't be a good thing. Also, what is the best way to tell if rhinitis/reflux/reactivity is relevant in the patient in front of you? Why do only a subgroup of people with rhinitis/reflux/reactive airways present with chronic cough?

Furthermore, the 3Rs denote a single disease mechanism—namely, activation of the afferent limb of the cough reflex at the site of the disease process (nose, airway, oesophagus, respectively) which is increasingly ignorant of other relevant mechanisms in chronic cough such as eosinophilic inflammation of the airway,[3] extrathoracic airway hyperresponsiveness,[4] oesophageal dysmotility,[5 6] and airway protussive mediator release, possibly a reflection of neurogenic inflammation.[7]

Problems also exist in relation to eosinophilic bronchitis, a descriptive term which indicates the pattern of airway inflammation present. When first described in chronic cough, eosinophilic bronchitis was reported as a disease mechanism and a marker of a good response to corticosteroid treatment.[3] Recently, the term eosinophilic bronchitis has been used as a disease label in chronic cough—that is, a diagnosis in itself.[8–10] In this way, eosinophilic bronchitis has been incorporated into the anatomic-diagnostic protocol as a cause of idiopathic cough to be considered when all other avenues have failed. This is problematic since eosinophilic bronchitis occurs in all three of the ''Rs''[11] and is also present in most patients labelled as having atopic cough. It also ignores the excellent and prompt response to corticosteroid treatment that occurs in eosinophilic bronchitis. It is less useful to consider eosinophilic bronchitis as a disease or a diagnosis of exclusion. Rather, it is a pattern of airway inflammation that is present in a number of common diseases and, when symptomatic, indicates a good response to an accessible treatment (inhaled corticosteroid). After serious diseases have been ruled out, maybe the first approach to chronic cough should be a supervised trial of 'Roids (steroids) and, if that fails, then go for the 3Rs.

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Interrupter resistance

Sly and Lombardi[1] in their recent editorial suggest that interrupter resistance (Rint) measurements are useful in the management of lung disease in young children. We believe this claim needs further consideration.

Rint measurements can be helpful when change following an intervention—such as the administration of a bronchodilator—is greater than its within-occasion repeatability but, for a measurement to be useful for following change with time in the individual, it must have acceptable between-occasion repeatability. In the same issue, Beelen *et al*[2] reported between-occasion variability of 0.38 kPa/l.s (2 SD of the differences between measurements) in 25 healthy children. This figure is similar to that of Chan *et al*[3] who reported 72 measurements in healthy children and 95 measurements in children with stable mild asthma. In the healthy children the between-occasion repeatability was 32% expected for age, but in the asthmatic children this rose to 52%. As a hallmark of asthma is bronchial lability, this is not unexpected. These figures need to be compared with the change expected with treatment. Pao *et al*[4] showed that, in an identical group of asthmatic children, a change in mean Rint of 16% occurred with treatment with inhaled corticosteroids. Although this change was demonstrated in a group of children, it would not be picked up easily in the individual because the between-occasion repeatability of Rint is much greater than the change expected.

Rint seems to be a good tool for research and, for that reason, measurements should be standardised. However, we believe its usefulness for the practising clinician is quite limited as measurements in the individual are not sufficiently reliable on a day to day basis. It is difficult to imagine that further refinement and standardisation of the method will improve this.

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Authors' reply

We thank Drs Dundas and McKenzie for their comments. We agree with them that the interrupter resistance (Rint) is able to detect short term chanes in airway calibre after bronchodilator inhalation. However, we must disagree with their comment that Rint has a poor long term repeatability and their consequent conclusion that Rint is not useful for routine clinical purposes. The long term repeatability (38 days apart) of Rint measurements (2 SD of the difference between two sets of measurements) reported by Beelen *et al*[1] in healthy preschool children was actually 0.37 kPa/l.s in 25 children under field conditions and 0.28 kPa/l.s in 15 children under laboratory conditions. This value is very similar to the long term repeatability