Eur Respir J
1992, 5, 1035–1036

EDITORIAL

Breathlessness perception in airways obstruction

P.W. Jones*

It has been known for many years that there are variations between patients in terms of their perception of breathlessness during airways obstruction. This has led to the concept of "poor perceivers" [1]. Poor perception probably has at least two components; the most widely recognized is diminished awareness of acute exacerbations of obstruction, but a second component is failure to recognize chronic changes. The presence of these two patterns can be clearly seen in the patients studied by RUBINFIELD and PAIN [1]. Subsequent studies have sought to address this problem by measuring breathlessness and a range of physiological variables associated with airflow limitation during the acute induction of airways obstruction or bronchodilatation [2–6]. Tests of the perception of external resistances have also been carried out [7–9]. The common finding is of wide variation in breathlessness perception and awareness of added resistances.

No clear overall pattern has emerged with regard to factors responsible for these differences. RUBINFIELD and PAIN [1] were unable to identify any characteristics that distinguished perceivers from poor perceivers. They interpreted their later methacholine studies [3] as indicating that perception of acute exacerbations increased with deteriorating airways obstruction. In contrast, BURDON *et al.* [2] found less dyspnoea during histamine induced bronchoconstriction in patients with a lowered baseline forced expiratory volume in one second (FEV_1) compared to patients with normal function. Studies using external resistances have produced mixed results. Asthmatics and normals exhibited similar perception of added loads [7, 8] but in chronic obstructive airways disease (COAD) patients, perception was reduced compared to both normals and asthmatics [8]. Attempts have been made to relate dyspnoea perception to bronchial responsiveness. BURDON *et al.* [2] found that patients with high responsiveness to histamine perceived less dyspnoea than less responsive subjects. Whilst this observation was statistically significant, the scatter in the data is considerable and one data point appears to be particularly influential. An alternative interpretation may be that less responsive subjects experienced more systemic effects due to histamine because a higher dose was needed and this may have influenced them. In support of this conclusion, a study using methacholine found no correlation between the provoking concentration producing a 20% fall in FEV_1 (PC_{20}) and breathlessness [9].

In this edition of the Journal, NOSEDA *et al.* [10] present another study in this field. Two groups of patients were selected, one with high reversibility (asthmatics) and the other with low reversibility (COAD patients). A large placebo response was observed with nebulized saline in most of the asthmatics and in half of the COAD patients. In the latter, the average improvement in dyspnoea with saline was 40% of the maximum change recordable using the chosen scale. The asthmatics showed wide interindividual variation in breathlessness estimation, but these variations all appeared to belong within the same population. This was not the case with the COAD patients, in whom two very distinct patterns were found. One group, already mentioned, were those who had a placebo response to saline, and a second group was highly unresponsive. The patients who registered improved breathlessness with saline (the asthmatics and half of the COAD patients) also showed significant improvement with bronchodilator. These results are not explicable purely in terms of physiological change within the lungs. Following bronchodilator, some measures of airways function were improved in patients who indicated significantly improved breathlessness, but these changes tended to be patchy and there was no correlation between them and changes in dyspnoea. Furthermore, changes in airways function could not account for the large improvements in dyspnoea with saline.

This is yet another study that has failed to clearly identify mechanisms responsible for differences in dyspnoea perception in airways disease. Why this persisting failure? One reason may be the wide variation in breathlessness perception known to be present in normal individuals [11, 12]. In patients with established disease, it may not be possible to identify these inherent differences and this may confound attempts to identify effects due to disease. Despite this problem, it is possible to generate hypotheses concerning dyspnoea detection in disease, based on existing evidence. The literature suggests a pattern to which patients with more severe fixed airways obstruction perceive acute changes in airways obstruction less well than milder asthmatics. Several papers reviewed here contain in their discussions the suggestion that disease duration and the experience

* Division of Physiological Medicine, St George's Hospital Medical School, Cranmer Terrace, London SW17 0RE, UK.

of breathlessness may modify subsequent estimates of breathlessness. This proposal invokes the operation of a non-linear system, since the output of the system (breathlessness) feeds back to modify the process by which it is perceived. The operating characteristics of such a system would change continuously, so that it may not be possible to predict breathlessness from airways measurements. This hypothesis does, however, allow the prediction that breathlessness perception may be greater in patients experiencing wide swings in airways obstruction and dyspnoea. Support for this suggestion comes from two studies in which daily peak expiratory flow rate (PEFR) and dyspnoea at rest were measured over a period of days. The quality of breathlessness perception correlated poorly with asthma severity, but size of daily variation in PEFR [13] and bronchodilator response [5] were the best correlates. It has also been observed, in a study on the detection of external resistances, that asthmatics showed much wider variations in perception compared to normals - both lesser and greater degrees of sensitivity [7]. The hypothesis that prior experience may modulate subsequent estimates of dyspnoea has been tested and confirmed in normals [14, 15]. There are no comparable studies in disease, but in a recent study, dyspnoea first increased then recovered in two patients during their initial response to antigen challenge, but it did not change during the subsequent late response [6].

Breathlessness, like pain, has both magnitude and emotional components. There is evidence that normal subjects may reliably distinguish between these two [12]. It is not known which is more important in disease. Dyspnoea is critical to patients with airways obstruction, it limits activity and impairs "quality of life". It also provides a warning of deterioration. This presents a paradox for the physician. Morbidity, even of mild asthma, remains high despite vigorous and enthusiastic treatment. Toleranceis, therefore, an appropriate response on the patient's part. On the other hand, over-tolerance may lead to failure to appreciate life-threatening asthma. Even with a better understanding of the development of dyspnoea, we will still be left with this problem.

References

1. Rubinfield AR, Pain MCF. – Perception of asthma. *Lancet,* 1976; i: 882–887.
2. Burdon GW, Juniper EF, Killian KJ, Hargreave FE, Campbell EJM. – The perception of breathlessness in asthma. *Am Rev Respir Dis,* 1982; 126: 825–828.
3. Rubinfield AR, Pain MCF. – Conscious perception of bronchospasm as a protective phenomenon in asthma. *Chest,* 1977; 72: 154–158.
4. Wolkove N, Dajozman E, Colacone A, Kriesman H. – The relationship between pulmonary function and dyspnoea in obstructive lung disease. *Chest,* 1989; 96: 1247–1251.
5. Peiffer C, Toumi M, Razzouk H, Marsac J, Lockhart A. – Relationship between spontaneous dyspnoea and liability of airways obstruction in asthma. *Clin Sci,* 1992; 82: 717–724.
6. Turcotte H, Corbeil F, Boulet L-P. – Perception of breathlessness during bronchoconstriction induced by antigen, exercise and histamine challenge. *Thorax,* 1992; 45: 914–918.
7. Burki NK, Mitchell K, Chaudhary BA, Zechman FW. – The ability of asthmatics to detect added resistive loads. *Am Rev Respir Dis,* 1978; 117: 71–75.
8. Gottfried DSB, Altose MD, Kelsen SG, Cherniak NS. – Perception of changes in airflow resistance in obstructive pulmonary disorders. *Am Rev Respir Dis,* 1981; 124: 566–570.
9. Yamamoto H, Inaba S, Nishimura M, Kishi F, Kawakami Y. – Relationship between the ability to detect added resistance at rest and breathlessness during bronchoconstriction in asthmatics. *Respiration,* 1987; 52: 42–48.
10. Noseda – *Eur Respir J,* 1992: 5: 1043–1053.
11. Adams L, Chronos N, Lane R, Guz A. – The measurement of breathlessness induced in normal subjects: validity of two scaling techniques. *Clin Sci,* 1985; 69: 7–16.
12. Wilson RC, Jones PW. – Differentiation between the intensity of breathlessness and the distress it evokes in normal subjects during exercise. *Clin Sci,* 1991; 80: 65–70.
13. Peiffer C, Marsac J, Lockhart A. – Chronobiological study of the relationship between dyspnoea and airway obstruction in symptomatic asthmatic subjects. *Clin Sci,* 1989; 77: 245–252.
14. Wilson RC, Jones PW. – Influence of prior ventilatory experience on the estimation of breathlessness during exercise. *Clin Sci,* 1990; 78: 149–153.
15. Jones PW, Oldfield WLG, Wilson RC. – Breathlessness in humans during exercise over a six month period following four weeks at an altitude of 4,000 metres. *J Physiol (Lond),* 1990; 430: 90p.