EDITORIAL

Respiratory monitoring: revisiting classical physiological principles with new tools

M.I. Polkey*, R. Farré[#], A.T. Dinh-Xuan[¶]

When interviewing junior doctors wishing to work in a hospital, a common question, at least in the UK, is to ask them to name an important development in respiratory medicine in the past 10 yrs. Since young doctors like results, they often suggest some therapeutic aspect of applied physiology, such as noninvasive ventilation. In fact, we propose that, even in the post-genomic age, the immediacy of physiology never really loses its appeal throughout most doctors' careers. Indeed, we suspect that, increasingly, accurate physiological techniques will acquire greater importance, as we seek to understand and evaluate the effects of interventions that are inspired by our increasing knowledge of the molecular and genetic basis of disease, a concept first highlighted in the *European Respiratory Journal* a decade ago [1].

In this model (fig. 1), clinical measurement or physiology becomes an interpreter between the clinician and the bench scientist. In vivo measurements can be used to quantify the value of existing therapies and, in turn, to suggest new hypotheses to bench scientists. An example of both these processes may be found in research concerning the familiar observation that patients with chronic obstructive pulmonary disease (COPD) have peripheral muscle weakness. This process had been known to clinicians for decades, but careful measurements established, for example, that such patients consume a disproportionate quantity of healthcare budgets [2], and provide a handle to examine underlying structural processes [3]. Working the other way, physiology can interpret hypotheses generated by bench science in vivo. Thus, when corticosteroids are administered to laboratory animals, skeletal weakness occurs [4]. Patients with COPD who take corticosteroids commonly have quadriceps weakness, and so uncritical analysis might lead to the conclusion that corticosteroids cause weakness in clinical practice. In fact, when assessed using careful physiological techniques, it transpires that giving a commonly used steroid dose to COPD patients for 2 weeks does not cause quadriceps weakness [5], leading to a modified hypothesis that, for example, the effects of exacerbation and the steroids given for the exacerbation combine synergistically to cause quadriceps weakness. Another example of the important role that physiology plays in linking basic science and clinical practice is in the field of sleep medicine. Although the symptoms of sleep disturbances were already well known in the past, the mechanisms underlying these sleep disturbances have been and are still being discovered, after systematic study of the patients' physiological variables

Correspondence: M.I. Polkey, Respiratory Muscle Laboratory, Royal Brompton Hospital, Fulham Road, London SW3 6NP, UK. Fax: 44 2073518939. E-mail: m.polkey@rbh.nthames.nhs.uk

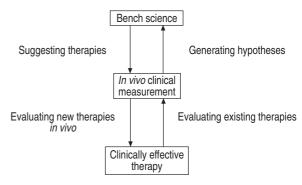


Fig. 1.-Model showing clinical measurement or physiology as an interpreter between the clinician and the bench scientist.

during the night, and thorough assessment of the physiological effects of the application of continuous positive nasal pressure [6]. Further examples of the most recent investigations in clinical physiology during sleep are those which have provided basic scientists with data to test hypotheses concerning the inflammatory processes [7], and cardiovascular consequences associated with respiratory sleep disturbances [8]. These studies could result in future improvements of the conventional preventive and therapeutic strategies.

The coming series focuses on monitoring disease activity in a variety of settings, and was inspired by a successful symposium at the 2003 European Respiratory Society Congress entitled "Translating physiological advances". However, as a result of the editors' generosity with the Journal's pages, we have been able to widen the scope of the series beyond the original symposium. The series includes seven topics: magnetic nerve stimulation; monitoring of dyspnoea; assessment of dynamic hyperinflation; noninvasive monitoring of respiratory function during sleep; noninvasive detection of inflammatory processes within airways and alveoli; noninvasive assessment of pulmonary circulation; and the potential use of telemedicine in respiratory disease. In each case, the authors have sought to explain the underlying physiological principles, beloved of all clinicians, and to demonstrate their clinical applications. We hope that you will find the series stimulating not only in a purely academic sense, but also that you may find possibilities within it to use physiology in order to enhance your clinical and research practice.

References

- Barnes PJ. The fate of respiratory physiology. Eur Respir J 1994; 7: 635–636.
- Decramer M, Gosselink R, Troosters T, Verschueren M, Evers G. Muscle weakness is related to utilization of health care resources in COPD patients. *Eur Respir J* 1997; 10: 417– 423.

^{*}Respiratory Muscle Laboratory, Royal Brompton Hospital, London, UK. "Unitat Biofisica I Bioenginyeria, Facultat Medicina, Universitat Barcelona-IDIBAPS, Barcelona, Spain. "Service de Physiologie-Explorations Fonctionnelles, Centre Hospitalier Universitaire Cochin, Assistance Publique-Hôpitaux de Paris, Université Paris V, Paris, France.

- 3. Bernard S, LeBlanc P, Whittom F, et al. Peripheral muscle weakness in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998; 158: 629–634.
- 4. Nava S, Gayan-Ramirez G, Rollier H, *et al.* Effects of acute steroid administration on ventilatory and peripheral muscles in rats. *Am J Respir Crit Care Med* 1996; 153: 1888–1896.
- 5. Hopkinson NS, Man WDC, Dayer MJ, et al. Acute effect of oral steroids on muscle function in chronic obstructive pulmonary disease. Eur Respir J 2004; 24: 137–142.
- Deegan PC, McNicholas WT. Pathophysiology of obstructive sleep apnoea. Eur Respir J 1995; 8: 1161–1178.
- Ohga E, Tomita T, Wada H, Yamamoto H, Nagase T, Ouchi Y. Effects of obstructive sleep apnea on circulating ICAM-1, IL-8, and MCP-1. J Appl Physiol 2003; 94: 179– 184.
- Lattimore JD, Celermajer DS, Wilcox I. Obstructive sleep apnea and cardiovascular disease. *J Am Coll Cardiol* 2003; 41: 1429–1437.