surprisingly, reduced values were found in 11 out of 30 women, though these would have been considered normal if compared with normal ranges for female patients. We therefore find it hard to agree that diaphragmatic myopathy is the cause of the shrinking lung syndrome of systemic lupus erythematosus.

Dr Gibson quotes two papers which appear to suggest that patients with isolated bilateral diaphragm weakness or paralysis can develop hypercapnia or hypoventilation, but we do not believe that these contradict our paper, in which we found no evidence of chronic hypercapnia or nocturnal hypoventilation in six patients with longstanding bilateral diaphragm paralysis who had otherwise normal lungs, respiratory muscles, and chest wall.[3] Most of the patients reported in Newsom Davis's original paper[4] had diffuse weakness of the respiratory muscles as well as the diaphragm. The two papers quoted by Dr Gibson are case reports: in the first the patient was nursed supine but recovered fully when allowed to adopt a more upright posture.[5] In the second the patient had undergone a recent thoracotomy, which is likely to have impaired rib cage and chest wall movements.[6] By contrast we described patients with proved bilateral diaphragm weakness who have remained otherwise well for up to 10 years.

CLARE M LAROCHE
MALCOLM GREEN
Respiratory Muscle Laboratory, National Heart and Lung Institute, and Brompton Hospital, London

1 Laroche CM, Mulvey DA, Hawkins P, Walport M, Strickland B, Moxham J, Green M. Diaphragm strength in the "shrinking lung" syndrome of systemic lupus erythematosus. *Q J Med* 1989;**265**:429–39.
2 Wilcox PG, Stein HB, Clarke SD, Pare PD, Pardy RC. Phrenic nerve function in patients with diaphragmatic weakness and systemic lupus erythematosus. *Chest* 1988;**93**:352–8.
3 Laroche CM, Carroll N, Moxham J, Green M. Clinical significance of severe isolated diaphragm weakness. *Am Rev Respir Dis* 1988; **138**:862–6.
4 Newsom Davis J, Goldman M, Lok L, Casson M. Diaphragm function and alveolar hypoventilation. *Q J Med* 1976;**45**:87–100.
5 Sandham JD, Shaw DT, Guenter CA. Acute supine respiratory failure due to bilateral diaphragmatic paralysis. *Chest* 1977;**72**:96–8.
6 Kreitzer SM, Feldman NT, Saunders NA, Ingram RH. Bilateral diaphragmatic paralysis with hypercapnic respiratory failure. *Am J Med* 1978;**65**:89–95.

AUTHOR'S REPLY Drs Laroche and Green take me to task over two points where their own data or interpretations differ from those of other authors.

The likely contribution of diaphragmatic weakness to the "shrinking lung" syndrome of systemic lupus erythematosus is supported by four clinical studies (refs 70–74 in my article) and by the only published clinicopathological study of the diaphragm in this condition (ref 72). The reasons for the different results obtained by Laroche and others[1] are not clear but one factor may be patient selection. They specifically excluded patients with "generalised muscle weakness due to coexistent polymyositis" (criteria not stated), whereas other authors made no such exclusions, using only a descriptive clinicophysiological and radiographic definition of the "shrinking lung" syndrome. It is likely that other factors are important in restricting lung expansion in some patients but the evidence overall still favours diaphragmatic weakness as the major factor in most cases of the "shrinking lung" syndrome as usually defined.

The second controversial area concerns the role of bilateral diaphragmatic paralysis in the development of hypercapnia and nocturnal hypoventilation. I would agree that bilateral diaphragmatic paralysis is not necessarily associated with *daytime* hypercapnia. I also agree that the patients reported by Newsom Davis *et al* (ref 10) had generalised respiratory muscle weakness and, as I emphasised in my article, this is the usual setting in which bilateral diaphragmatic paralysis is seen. The evidence against *nocturnal* hypoventilation as a consequence of bilateral diaphragmatic paralysis per se is, however, less convincing. Studies in normal subjects suggest that such patients would be most vulnerable during rapid eye movement (REM) sleep, when the other respiratory muscles are likely to be inhibited. Such an effect was indeed well documented in one patient with apparently "pure" bilateral diaphragmatic paralysis who had gross hypoventilation and periods of "central" apnoea during REM sleep.[2] Unfortunately, detailed sleep studies were not reported by Laroche *et al* (ref 11), but it is noteworthy that the two of their six patients with the longest periods of REM sleep showed decreases in arterial oxygen saturation as large as 20% and 27%. Clearly, more studies are desirable but the rarity of such "pure" cases makes this difficult to achieve. Meanwhile my conclusion that, in the presence of normal function of other respiratory muscles, bilateral diaphragmatic paralysis has less profound consequences than previously described remains a fair summary of the published data.

G J GIBSON

1 Laroche CM, Mulvey DA, Hawkins P, *et al*. Diaphragm strength in the "shrinking lung" syndrome of systemic lupus erythematosus. *Q J Med* 1989;**265**:429–39.
2 Stradling JR, Warley ARH. Bilateral diaphragm paralysis and sleep apnoea without diurnal respiratory failure. *Thorax* 1988;**43**:75–7.

NOTICES

International symposium on respiratory psychophysiology

The 10th International Symposium on Respiratory Psychophysiology will be held at the University of Amsterdam on 21 and 22 September 1990. The conference will include workshops, limited to 25 participants each. Submission of abstracts relevant to the symposium themes for oral or poster presentations is invited. For further information please contact Dr B Garssen, Department of Medical Psychology, Academic Medical Centre, Meibergdreef 15, 1105 AZ Amsterdam, The Netherlands.

British Sleep Society

The second annual scientific meeting of the British Sleep Society will be held in Leeds on 24–26 September 1990. The programme will include sessions on narcolepsy, parasomnias, developmental aspects of sleep disorders, measures of wakefulness, methodology, sleep in the postoperative period, and the pharmacology of sleep disturbances. Free papers will be presented both by poster and verbally and abstracts are invited. For further information please contact Dr I Hindmarch, University Department of Psychology, Leeds LS2 9JT (fax 0532 421639) or Dr CD Hanning, Sleep Disorders Clinic, Leicester General Hospital, Leicester LE5 4PW (0533 584602 (direct line); fax 0533 737991).