

Correspondence

Ventilatory failure in Guillain-Barré syndrome: indications for assisted ventilation in neurological diseases

Sir,—An article by Massam and Jones in your July 1980 issue¹ describes the history of a 14-year-old girl with a Guillain-Barré syndrome from which she recovered without requiring assisted ventilation. The story is an atypical one in its slow deterioration, reaching a trough between the twentieth and thirtieth day of hospital admission. The authors describe a fall in the peak expiratory flow rate (PEFR) to one quarter the predicted value but record normal Paco_2 values, and imply that this made it safe to withhold assisted ventilation. It would, however, be irresponsible to assume that the therapeutic management of this single case has more than anecdotal relevance to the general problems of how to treat ventilatory failure of neurological origin.

The causes of this type of ventilatory failure are usually complex. In some cases of the Guillain-Barré syndrome, and even more so in more obvious cerebral disorders such as encephalitis, there may be a failure of brainstem mechanisms resulting in reduced respiratory drive. Secondly, there may be failure of muscles innervated by the nuclei in the brainstem and the lower cranial nerves (bulbar palsy), resulting in difficulties in swallowing and in failure to cough effectively, which may lead to the aspiration of secretions into the bronchi and lungs. Thirdly, there is paralysis of the respiratory muscles proper—that is, the diaphragm, intercostals, and the accessory muscles. The rate of deterioration may be slow, or it may be catastrophically rapid, and all patients with even a hint of bulbar or respiratory muscle paralysis should be transferred without delay to respiratory intensive therapy units where tracheal intubation and mechanical ventilation can be undertaken as soon as the need arises.

Delays in instituting these measures by the application of some rule of thumb based on measurements of the forced expiratory volume in one second (FEV_1) or PEFR are liable to lead to major problems in management. It is true that some patients whose FEV_1 or PEFR is above 1.0 l or 200 l/min respectively may be capable of maintaining normal partial pressures of oxygen and carbon dioxide in the arterial blood, but in many cases inability to increase tidal volume above a level which is barely adequate to maintain ventilatory requirements may insidiously reduce the ventilation/perfusion ratio in the lungs, and cause progressive arterial hypoxaemia. At this stage, few, if any, of these patients are able to cough effectively, and the retention of secretions, particularly if respiratory infection supervenes, often results in lobar or segmental collapse, and in some cases, sudden respiratory arrest.

Our experience with respiratory failure in 25 adult

patients with polyneuropathy, and nine with myasthenia gravis, has forced us to the conclusion that no patient is "safe," even if the arterial blood gases are normal, when the FEV_1 falls below 1.5 l or the PEFR below 300 l/min. In these circumstances, the patient's ability to cough effectively becomes the principle indication for mechanical ventilation.

The significance we attach to this clinical observation has prompted us to institute mechanical ventilation at an earlier stage than if we were to wait for the FEV_1 or PEFR to fall below any "critical" level or for the arterial blood gas status to deteriorate. If it is delayed too long, the management of mechanical ventilation becomes much more difficult. The chief problem in such cases is that of maintaining a satisfactory PaO_2 even when the minute volume of (oxygen-enriched) ventilation is increased to a level which lowers the Paco_2 to an undesirably low figure and produces a marked degree of respiratory alkalaemia. It then becomes necessary to apply positive end-expiratory pressure to the ventilator circuit, to increase the frequency of periodic manual hyperventilation (to keep the peripheral airways patent) and to ensure that even small amounts of secretion do not accumulate in the larger bronchi, using bronchoscopic aspiration, if necessary, to keep these bronchi clear.

The long-term prognosis in both polyneuropathy and myasthenia gravis is generally good, and a clear understanding of the indications for mechanical ventilation in these diseases is of vital importance. Undue delay may, in some cases, increase the technical and clinical problems for ventilator care. At worst, such a delay can be fatal.

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Reference

- 1 Massam M, Jones RS. Ventilatory failure in the Guillain-Barré syndrome. *Thorax* 1980; 35:557-8.

Sir,—Grant *et al* note the particular points about respiratory function which we made in our paper¹ and add that . . . "It would be irresponsible to assume that the therapeutic management of this single case has more than anecdotal relevance to the general problems of how to treat ventilatory failure of neurological origin."

We agree entirely that it would be wrong to extra-

polate the points we made to cover the management of respiratory failure of neurological origin in general. Having made that point, the authors in their letter then proceed to a dissertation on this broad subject. We would not disagree with most of the points which they make, but would add that they are generally not relevant to our report. Of course, there are many different patterns of relationship between ventilatory failure and blood gas abnormalities, but most of these have not been well documented.

We would suggest that it is Grant *et al* who are offering in their letter rules of "anecdotal relevance"

for the management of this difficult problem. More hard data are required, and that is what we endeavoured to supply.

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Reference

- 1 Massam M, Jones RS. Ventilatory failure in the Guillain-Barré Syndrome. *Thorax* 1980; 35:557-8.

Computers in Critical Care and Pulmonary Medicine announce The Third International Symposium 17-19 June 1981 at Norwalk Hospital, Yale University School of Medicine, Norwalk, Connecticut, USA

The purpose of this meeting is to bring together physicians, biomedical engineers, and computer scientists who are interested in the application of computer technology to the pulmonary function laboratory, critical care unit and monitoring of critically ill patients. The preliminary list of topics for which abstracts are invited include: pulmonary function including spirometry, diffusion, rebreathing techniques and blood gas analysis, exercise physiology, instrumentation for gas analysis, mass spectrometry, pulmonary application of nuclear medicine, monitoring during anaesthesia and postoperative period, critical care monitoring of cardiopulmonary parameters including FRC, lung water, pulmonary perfusion, O₂ and CO₂ transport, modelling of the cardiopulmonary system, critical care information management systems, data acquisition and display, applications for feed-back control of ventilation and cardiopulmonary function.

Abstracts are invited in these areas and early registration is requested. The deadline for abstracts is 1 March 1981. The registration fee will be \$250.00. For further information please write to: Dr Sreedhar Nair, Symposium Chairman, Computers in Critical Care and Pulmonary Medicine, Norwalk Hospital, Yale University School of Medicine, Norwalk, CT 06856, USA.

Paediatric Cardiology 1981

London 16-18 September 1981, sponsored by the British Heart Foundation. The programme will include sessions on: heart rhythm, pulmonary vascular disease, pulmonary atresia with VSD, cross-sectional echocardiography, atrial isomerism, pulmonary atresia with intact septum, assessment after operation of CHD, valve disease and replacement in children, double outlet ventricle. Organising committee: Professor RH Anderson, Dr EA Shinebourne, Professor FJ Macartney, Dr M Tynan. Further information may be obtained from Paediatric Cardiology 1981, Conference Services Ltd, 3 Bute Street, London SW7. Tel: (01) 584 4226, Telex: 916054 JOYBRI.