Editorials

Home care ventilation

The report of Sivak et al[1] illustrates that selected patients can be treated successfully with long-term mechanical ventilatory support in the home. The social, psychological, and financial considerations in the selection of patients; the evaluation of their responses to treatment; and the clinical and symptomatic effects of treatment are well documented in the report. We discuss the physiological criteria most useful in selecting patients for such treatment and in evaluating their responses.

The outstanding characteristic of the patients reported by Sivak et al[1] is the absence of chronic obstructive lung disease (with the exception of a single patient in group III). This is a most important consideration, since respiratory insufficiency or frank respiratory failure can be viewed from the physiological perspective of inadequacy of either the lungs or of the respiratory pump mechanism. In the former case, lung disease greatly increases the burden on the respiratory pump mechanism, and respiratory insufficiency (related to the respiratory muscles) can be viewed as an instance of high-output failure. Indeed, the work of Gibson et al[2] has shown that the mechanical output of the respiratory muscles in such patients is greater than normal, despite mechanical compromise of their efficiency. The clinical observation of marked tachypnea and use of accessory muscles of respiration further attests to a greater than normal output of their ventilatory control systems.

In contrast, patients with neuromuscular disease (such as those in groups II, IV, and V) demonstrate inadequate function of the respiratory pump mechanism as a result of limitation in the transmission of impulses from the nervous system (either centrally or peripherally) to the respiratory muscles and their subsequent conversion to mechanical energy. Such patients manifest low output respiratory failure, from the perspective of the respiratory muscles. Patients with defects in the skeletal system, such as those in group I, may often present a special case of high output respiratory muscle failure. The muscles themselves are driven to produce large mechanical forces. However, the restrictive nature of the skeletal defect prevents these large muscle forces from moving the respiratory system sufficiently to produce adequate ventilation. Consequently, adequate ventilation is inhibited. Patients with ankylosing spondylitis have a similar physiological problem, and may often manifest respiratory muscle fatigue as reported by Tsanaclis and Grassino.[3]

The clinical rationale for treatment is closely related to physiological limitation as outlined by Sivak et al[1] and most dramatically manifested in the responses of group I patients in contrast to those of group II. Whereas both groups showed substantial benefit from treatment, the former group achieved in large part the stipulated goal of rehabilitation by therapeutic support. In contrast, effective therapeutic support of the latter group could not achieve rehabilitation in the face of progressively advancing neurological disease.

With the single exception of the patient in group III, all the patients reported by Sivak et al[1] present a problem in documentation of progressive physiological deterioration. This is because the design of most clinical pulmonary func-

tion testing procedures relies upon a basic adequacy of the respiratory muscle pump itself. Abnormalities in test results then reflect the extent of disease of the lung or airways. However, when there is inadequacy of respiratory muscle function, one would not expect most pulmonary function test results to clearly indicate the extent or progression of the state of respiratory muscle function. In such conditions, the most appropriate index of the need for mechanical assistance to ventilation is the clinical symptomatology, as used in the report of Sivak et al.[1]

One particular physiological problem that may arise with respiratory muscle formation is illustrated by patients 23 and 24, namely the occurrence of diaphragmatic paralysis following multiple intrathoracic surgical procedures. In such cases, fibrous tissue may obscure the phrenic nerves, which may then be severed during the surgical procedure. Indeed, inadvertent severing of one or both phrenic nerves may occur during any thoracic surgical procedure, especially in small children, and should always be considered when a patient with otherwise normal lungs presents extraordinary difficulties in weaning from mechanical ventilation during the immediate postoperative period. It is especially important to recognize this complication early in the postoperative period, as prompt surgical reconnection of the severed nerve will restore diaphragm function completely. The physiological and clinical manifestations of diaphragmatic paralysis are reviewed by Loh et al.[4]

In conclusion, it appears that technological developments for providing mechanical ventilatory assistance are now at a sufficiently advanced state that the provision of such therapy is entirely appropriate in the patient's home. Patients with neuromuscular or musculoskeletal disease may often benefit substantially from such therapy and may thereby be permitted to live with a significantly improved quality of life, avoiding many of the prolonged hospital stays they might otherwise be subjected to. Knowledgeable physicians, nurses, and home care therapists can satisfy a true social obligation to such patients by thoughtfully evaluating and, when appropriate, instituting therapy with home care ventilatory support.

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References

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Influence of HLA, A, B, and DR antigen matching in transfused cadaver renal transplant patients

The two most important histocompatibility systems in man are the ABO blood group and the HLA complex.[1] The rules governing transplantation with regard to the ABO system are the same as those for blood transfusion (the donor must be compatible with the recipient). In most instances, transplants performed across the ABO barrier will result in immediate graft failure. HLA antigen inheritance (of Class I HLA A, B, and C and Class II HLA D/DR antigens) obeys basic genetic principles when the antigens are codominant. Therefore, siblings can be (1) HLA-identical and have a 90% one-year graft survival; (2) half or haploidentical and have a 60% to 70% one-year graft survival; and (3) share no HLA antigens and have a 45% to 50% one-year graft survival. Matching of Class I and II HLA antigens is of unquestioned importance to the survival of renal allografts from living-related donors; its predictive value in cadaveric transplantation has been the subject of much debate during the past decade.

Ideally, as many Class I and II HLA loci should be matched (to avoid incompatibilities) in order to obtain maximum graft survival. Because of the extreme polymorphism of the HLA antigens, this may be difficult to accomplish. To increase the chances of obtaining well-matched donor-recipient pairs, regional and national organ-matching and distribution services have been set up to pool the prospective recipients from one region into a single group.

Historically, the relevance of matching for the Class I HLA A and B antigens has been questionable. In 1968, sequential analyses of North American data from P. I. Terasaki's laboratory initially