tific observations, the creation of intermediate care beds may end up having similar overutilization and eventual shortages as full critical care units. It is essential that admission to these units be based on criteria uniformly applicable to patients based on clinical requirements regardless of payer mix, age, or other potential biases that could create a "two-tier" intensive care structure.

Despite certain cautions, graded care options in the care of medical and respiratory patients offer considerable opportunities to cost-effectively improve the process of care for a population of patients with critical illness. Planning for such units should be based on scientific observations of the impact of such units and monitoring techniques on inpatient morbidity and mortality, as well as the operational issues of staffing and finance. Health care systems should view these units as useful resources to manage critical care facilities, maintaining access to limited critical care beds and securing appropriate monitoring and therapy for high-risk patients.

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Critical Illness Polynueuropathy

Adequate respiration depends upon the integrity of two systems: a gas exchange mechanism (the lungs), and a pump which ventilates the lungs. The pump consists of four major components: the chest wall, muscles of respiration, central control mechanisms within the brain stem, and the peripheral nerve supply to the muscles of respiration. Failure of any of the four pump components can lead to ventilator failure requiring intubation and mechanical ventilation.

Pump failure secondary to loss of normal peripheral nerve supply to the respiratory muscles has occurred in a variety of conditions, including relatively acute onset amyotrophic lateral sclerosis, chronic spinal muscular atrophy, Guillain Barre syndrome, and neurolgic amyotrophy. In the early 1980s, a new syndrome of peripheral nerve dysfunction, leading to pump failure requiring prolonged ventilator support, was recognized. Critically ill, septic, ICU patients, often with multiorgan failure, were noted to have marked difficulty in weaning from the respirator, with laboratory findings consistent with an acute peripheral neuropathy. Since the original reports, Bolton and coworkers have published a series of articles which help define the electrophysiologic and clinical aspects of what has been termed critical illness polyneuropathy.

In this issue, Witt et al (see page 176) report the results of a prospective study of critical illness polyneuropathy. Out of 1,167 admissions to the intensive care unit, 324 patients were in the ICU for more than five days and at risk of developing sepsis with multiorgan failure. Forty-three (13.2%) of the 324
patients met the criteria for multiple system failure. Of the 43 patients, 30 had either clinical or electrophysiologic evidence of a polynuropathy. All had evidence of an encephalopathy.

The characteristics of the neuropathy are similar to those previously reported. Nerve conduction velocities are normal, with normal F-waves, indicating preservation of the myelin sheath around the larger diameter, more rapidly conducting nerve fibers. Decreased amplitude of the compound muscle action potential coupled with spontaneous activity on needle electrode examination, are indicative of an axonal neuropathy, and help differentiate it from acute Guillian-Barré syndrome.

The syndrome is predominantly a disease of older age with a mean age of 64. Males and females are affected equally. It tends to develop after prolonged time in the ICU, with the mean onset occurring after 28 days in the ICU. There is significant mortality, as would be expected when multiple organs are involved, with 20 of the 43 patients dying. Of those individuals who did survive, the prognosis for significant improvement from the neuropathy was quite good.

While the article by Witt et al helps clarify the frequency and clinical aspects of the syndrome, the cause of the neuropathy remains unclear. Of many variables considered, the presence of elevated serum glucose, lowered serum albumin levels, and time in the ICU correlated most closely with the development of the syndrome. These are probably simply indicators of the severity of the patient’s illness.

The role of superimposed muscle dysfunction, occurring concomitantly with the denervation and contributing to the pump failure, is likewise unclear. In patients with denervating diseases, such as ALS and chronic spinal muscular atrophy, muscle catabolism is accelerated.14 In the setting of sepsis and neuropathy, it is likely that this is further accelerated, resulting in greater difficulty in weaning the patient from the respirator, and prolonging ICU time.15,16

Future studies will need to address the variety of complex, metabolic and potentially toxic events occurring in critically ill, septic patients which result in significant damage to both central and peripheral nervous systems. In the meantime, Witt et al are to be commended for their efforts to further define this syndrome. When clinicians are faced with an ICU patient who is difficult to wean from the ventilator, it is advisable to obtain an EMG to look for the polynuropathy which may be underlying the pump failure.

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The Challenge of Pulmonary Hypertension

The authors of the article "The Effects of Phenylephrine on Right Ventricular Performance in Patients with Pulmonary Hypertension," (Chest 1999;98:1102[Nov]) are to be congratulated for this definitive study of the effects of phenylephrine infusion on patients with pulmonary hypertension. In their study, phenylephrine increased pulmonary arterial pressure, raising end-diastolic pressure of the right ventricle, and produced a fall in cardiac output. Clearly any effect of raising right ventricular coronary perfusion pressure was negated by these effects.

Unfortunately, Rich and colleagues attempted to directly apply an experimental design constructed to