Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Nasal CPAP in Treatment of Persistent Atelectasis

To the Editor:

I read with interest the review article by Mansel and Norman (Chest 1990; 97:1446-52) titled "Respiratory Complications and Management of Spinal Cord Injuries." They emphasize that pulmonary complications, especially persistent atelectasis and recurrent pneumonia of the lower lobes, are a problem. Various forms of therapy, including fiberoptic bronchoscopy and saline lavage, have had limited success rates. Recently, Duncan et al1 and Davies and Grant2 reported resolution of recurrent atelectasis in their patients with nasal continuous positive airway pressure (CPAP) therapy. I attempted this mode of therapy again recently in a 45-year-old Latin American woman who had sustained multiple head injuries in a motor vehicle accident. Her mental status improved rapidly, and she was extubated. She had recurrent pneumonia secondary to persistent collapse of her left lower lobe (Fig 1), and various courses of antibiotics were administered during the next two to three months. Nasal CPAP therapy at a pressure of 10 cm H2O was instituted. The pressure was increased to 12.5 cm H2O a week later. At this pressure, the left lower lobe atelectasis resolved. The duration and optimal pressures of nasal CPAP therapy are unclear at this time. Intermittent use of nasal CPAP for recurrent atelectasis with pressures between 10 and 15 cm H2O for 4 to 12 h/d has been reported.1-3 This form of therapy is simple and noninvasive and may complement fiberoptic bronchoscopy and saline lavage.

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2. Davies CA, Grant IS. Total collapse of lung and CPAP (letter). Anaesthesia 1987; 42:780

To the Editor:

We have reviewed with interest Dr Thommi's letter and appreciate his comments regarding nasal CPAP therapy for recurrent atelectasis. We agree with his assessment that CPAP may be of benefit to patients with persistent atelectasis. Although our experience has been to use positive pressure by face mask, usually on an intermittent basis, a role for nasal CPAP may be useful in some cervical spine injury patients with persistent or recurrent atelectasis. One concern we do have, however, is the inability of a quadriplegic patient to remove a positive pressure mask if complications such as nausea and vomiting or gastric distention were to develop. If positive pressure is to be used in a quadriplegic patient, our preference would be to use it intermittently with monitoring during the treatment period by a respiratory therapist.

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Intensive Management of Severe Head Injury

To the Editor:

We were pleased to read the article by Borel et al (Chest 1990; 98:180-89) on the intensive management of patients with severe head injury. It was a nice review of the problems that can be encountered by the critical care specialist who is caring for neurosurgical patients. We agree with the medical techniques proposed for intracranial pressure control. However, the proposed dose for pentobarbital loading (36 mg/kg) seems to be quite excessive. Most textbooks of neurosurgery and critical care recommend loading doses of 3 to 10 mg/kg.1-3 Although infusions of pentobarbital in doses of up to 15 mg/kg in young healthy patients are well tolerated when the preload is maintained with continuous

Figure 1. X-ray film of the chest showing collapse of the left lower lobe.
infusion of fluid, in our experience, even 5 to 10 mg/kg cannot always be administered due to the development of hypotension and cardiovascular depression during the infusion. This could be explained by the fact that the typical neurosurgical patient requiring pentobarbital coma is significantly dehydrated by fluid restriction, mannitol administration, and diuretic therapy in an attempt to reduce intracranial pressure. In addition, these patients are frequently old, may have some septic process, and may have some degree of myocardial impairment.

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To the Editor:

The concerns expressed by Drs Teba and Weber regarding loading doses of pentobarbital in the 36-50 mg/kg range are well founded. Administering high-dose barbiturate therapy carries all the risks of emergency anesthetic administration to a patient near death. This is probably the reason why most textbooks of neurosurgical and critical care recommend smaller loading doses. Lower loading doses optimize patient safety but may not provide rapid reduction of cerebral oxygen consumption, cerebral electrical activity, and intracranial pressure. Recent reports advocate the use of high loading doses because of the concern for a timely therapeutic response for the selected patient indicators discussed in our review. A loading dose of 28 to 34 mg/kg administered during a 4-h period has been shown to raise serum levels to 30 to 40 µg/ml quickly enough to reduce elevated intracranial pressure and minimize further neurologic injury. Although the relationship between drug concentration and pharmacologic response may be loosely defined in these patients, it is well established that these serum levels are associated with coma, isoelectric EEG, and effective adjunctive therapy for head injury. While we readily acknowledge the difficulty in inducing and maintaining high-dose barbiturate therapy, it is beyond the scope of this report to discuss in detail such administration protocols, which have been discussed elsewhere.

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Air Trapping following Coronary Artery Bypass Surgery

To the Editor:

In a recent report in your journal, Dries et al presented a case of hemodynamic compromise associated with air trapping following coronary artery bypass surgery. This phenomenon was originally described by Weng et al,1 as cited by Dries et al. Over the past few years we have witnessed lung hyperinflation following coronary artery bypass surgery in at least five additional cases, four of which were in women with a significant history of smoking. The observed air trapping was never due to high ventilation rate or prolonged inspiratory time, but was rather due to severe bronchospasm, and resulted in high airway pressure once attempts to close the chest were made. This bronchospasm is very resistant to conventional bronchodilator therapy; therefore, the use of the expiratory retard mode is indicated.2

The differential diagnosis of hypotension occurring following chest closure (ie, whether it is due to pulmonary hyperinflation or to primary cardiac failure) can be greatly simplified by analyzing the effects of the mechanical breath on the arterial waveform. We have recently shown that a state of inadequate preload is characterized, and may actually be quantified, by the amount of the decrease in the systolic blood pressure following a mechanical breath relative to the systolic pressure during apnea (Δ down).3 On the other hand, a state of congestive heart failure is characterized by a near-total lack of a decrease in the systolic blood pressure following a mechanical breath, because the transient decrease in venous return does not affect left ventricular stroke output. Furthermore, an early inspiratory increase in the systolic pressure, denoting a positive cardiovascular effect of the increase in intrathoracic pressure, may appear in congestive heart failure.4

Thus, if hypotension occurs following chest closure, a Δ down of more than 10 percent of the systolic pressure during a short apnea is indicative of inadequate preload due to either absolute hypovolemia or lung hyperinflation, the latter case being usually associated with a high peak inspiratory pressure. If such hypotension is accompanied by a lack of a Δ down, and the systolic pressure is unchanged or somewhat elevated following a mechanical breath, the diagnosis is congestive heart failure.

This is another example of the usefulness of arterial pressure waveform analysis in the hemodynamic assessment of ventilated patients.5

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2 Weng JT, Smith DE, Graybar GB, Kirby RR. Hypotension

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