1) wiping the insertion tube with a 20 percent povidone iodine scrub solution.
2) suctioning iodine scrub solution through the biopsy channel and cleaning the channel with the cleaning brush.
3) suctioning sterile water through the channel.
4) suctioning freshly activated gluteraldehyde through the channel and soaking the entire insertion tube for 10 minutes.
5) rinsing the insertion tube with 200 ml of sterile water.
6) air drying the biopsy channel.

Repeat cultures of the biopsy channel yielded heavy growth of both Serratia and Pseudomonas. The insertion tube was then resoaked in gluteraldehyde for an additional 20 minutes, and cultures were still positive. Only after an additional 45 minutes in gluteraldehyde were the cultures negative.

Because many of the patients in our Burn Center have either colonized or are infected with antibiotic-resistant organisms, and our recent experience has demonstrated that recommended methods of disinfection may not be effective, we are now sterilizing the fiberoptic bronchoscope in ethylene oxide gas after every use.

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To the Editor:

Since the time of our report in Chest, we have continued to routinely culture the bronchoscope just prior to each procedure and have never grown any organisms of significance. However, because of our concern about such organisms as Serratia and Pseudomonas, we generally have also been sterilizing the fiberoptic bronchoscope in ethylene-oxide gas after every use. About the only time it is not sterilized after use is when there are several procedures in a row in one day and we usually try to be careful to use the bronchoscope only on people with fairly normal lungs and avoid using it on a patient with potentially contaminated lungs and then on someone else with similar lungs. We have not identified any problems related to gas sterilization except that it is very important to educate central supply about this delicate instrument and be sure they do not handle it roughly. As a matter of fact, on some occasions, heavy instruments were set on top of it and the bronchoscope was damaged. We have learned now to package it wrapped in very bulky towels to avoid that problem. Since the experience of 1975, I have seen no further patients in whom the bronchoscope was even suspected of being the mode of transmission for a nosocomial infection.

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Treatment of Refractory Lobar Atelectasis in Amyotrophic Lateral Sclerosis with PEEP

To the Editor:

The following patient with chronic neuromuscular disease illustrates resolution of left lower lobe atelectasis with PEEP after comprehensive respiratory therapy and flexible fiberoptic bronchoscopy failed.

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CASE REPORT

A 64-year-old paraplegic, ventilator-dependent woman with known amyotrophic lateral sclerosis was admitted because of increased dyspnea and production of thick sputum. She had been stable at home with a permanent tracheostomy breathing room air using a Thompson pressure regulated ventilator continuously at night and intermittently during the day.

On admission, physical and radiographic signs of left lower lobe atelectasis were demonstrable (Fig 1). The Po2 was 42 and PCO2 42 on ambient air.

Despite a program of continuous mechanical ventilation, comprehensive respiratory and physical therapy, and therapeutic flexible bronchoscopy, the atelectasis persisted for one week. Accordingly, the patient was placed on a Bennett MA-1 ventilator with 6 cm of PEEP. Within 48 hours, there was resolution of atelectasis and improvement in arterial blood gas to previous levels.

DISCUSSION

The use of respiratory therapy and flexible bronchoscopy is well established for the prevention and treatment of lobar atelectasis.1-3 The successful use of PEEP in postoperative lobar atelectasis has been described recently.4 PEEP was effective in resolving refractory left lower lobe atelectasis in a patient with amyotrophic lateral sclerosis.

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Bilateral Carotid Body Resection in Asthma:
Vulnerability to Hypoxic Death in Sleep

To the Editor:

The report by Chang et al (Chest 1978; 73:652-656) on the danger of carotid body resection as a form of therapy for asthma and the response by Winter requires additional comment. Chang et al emphasized the loss of crucial warning signals of acute hypoxemia. Their patient had three episodes of cyanosis and disorientation which they attributed to the loss of the ventilatory response to acute hypoxemia. This patient did not show the anxiety, restlessness and other premonitory symptoms and signs usually associated with acute hypoxemia. These authors concluded that carotid body resection was both unnecessary and potentially dangerous. In defense of the procedure, Winter criticized the Chang report by stating that some of his patients describe an awareness of hypoxia and by pointing out that subjects may survive despite a blunted ventilatory response to hypoxia, either occurring congenitally or in adaptation to altitude. Winter goes on to promote the procedure in treatment of asthma and other forms of chronic airflow obstruction.

One of the most crucial roles of the carotid bodies has been ignored by physiologists and clinicians and receives no mention in this latest round of the carotid body resection saga, viz: Is hypoxia capable of causing arousal from sleep? Recently it was pointed out that, in the context of sleep, the ability to arouse may be the most important response to a respiratory stimulus. Hypoxia is a powerful arousal-promoting stimulus; this response, like the ventilatory response, is mediated by the carotid bodies. The arousal response does not simply parallel the ventilatory response and in fact should be regarded as a separate reflex. For example, in the unanesthetized dog, hypoxia caused a brisk ventilatory response in wakefulness, nonrapid eye movement (NREM) sleep and rapid eye movement (REM) sleep; however, there was a sharp difference in the ability of hypoxia to cause arousal in the two major sleep states. In NREM sleep, hypoxia rapidly led to arousal on multiple tests in 4 dogs (SaO2 87.5 ± 2.6 percent mean ± SE), whereas a much greater level of desaturation (70.5 ± 3.4 percent) was required to produce arousal from REM sleep. This is likely the explanation of why the patients with obstructive sleep apnoea have longer apneas and larger drops in saturation in REM sleep. Thus, the arousal response to a respiratory stimulus can be depressed, despite a sustained ventilatory response. The converse may also be true. The ventilatory response to hypercapnia is abolished during the phasic component of REM sleep, yet the arousal response is sustained. The significance of these studies is that knowledge of the ventilatory response to a respiratory stimulus in the awake state tells us little about the ventilatory or arousal responses to the same stimulus in the sleeping state. However, if the carotid bodies or nerves are damaged, then both the ventilatory and arousal responses will be markedly reduced. But if a reduction in the hypoxic ventilatory response is caused by a central defect, or a defect on the output side (spinal cord, phrenic and intercostal nerves, respiratory muscles, or lung mechanics), then the arousal response may be well sustained. Conversely, a selective defect in arousal responses may occur leaving an otherwise intact ventilatory response.

There are a number of reasons why the arousal response to hypoxia is of particular importance. First, the state of wakefulness involves a powerful neural drive to breathing in addition to any respiratory drive provided by hypoxia or hypercapnia. For the asthmatic, waking from sleep, which provides additional drive to the respiratory muscles, may be the crucial event which prevents hypoxemic central nervous system depression and death. Second, arousal from sleep will initiate a behavioral as well as ventilatory response to the stimulus. For example, if an individual obstructs the nasal and oral airways by rolling prone onto the pillow, then a vigorous ventilatory response to the asphyxia in the presence of a closed airway would be useless; arousal and a change in position would be critical. Similarly, hypoxic arousal in the sleeping asthmatic may provide a life-saving behavioral response, i.e., taking further treatment or seeking medical help.

How is sleep relevant to asthma? There is no doubt that asthma frequently worsens during the night. There is evidence that most unexpected deaths occur at night. Nocturnal asthma has been related to the sleep-state per se and might result from autonomic instability during REM sleep. Whatever the mechanism, the defenses against the ensuing hypoxemia are vital. Destruction of the carotid body removes one of the most important defenses and undoubtedly predisposes the asthmatic to "unexpected" nocturnal death. Recent studies on the control of breathing in sleep give strong support to the view of Chang et al that such a procedure in an asthmatic is potentially life-threatening.

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