dyskinesia may result from neuroleptic use but not levodopa use.

Michael W. Rich, MD, and Steven M. Badwany, MD, Summa Health System, Akron, Ohio

Reprint requests: Dr. M. Rich, 75 Arch St Suite 302, Akron, OH 44304

REFERENCES

Tracheal Aspirate Cultures in Long-term Ventilated Patients Who Have Clinical Pneumonia

To the Editor:

I read with interest the article by Rumbak and colleagues (CHEST 1994; 106:531-34) in the August 1994 issue describing the correlation between the tracheal aspirate (TA) and protected specimen brush (PSB) cultures in stable long-term ventilator-assisted patients with the clinical diagnosis of pneumonia. No patient had received antibiotics for the preceding 5 days at the time of bronchoscopy or tracheal aspirate culture.

In our study,1,2 we enrolled 61 acutely ill ventilated patients. Forty-one of them were ventilated for more than 72 h when the clinical diagnosis of pneumonia (new infiltrate on chest x-ray film, fever, leucocytosis, and purulent bronchorrhea) was established. In contrast with the stable patients of Rumbak et al, 31 of the 41 patients already received antibiotic treatment for more than 24 h. Of these patients, 7 of 31 (22%) had a positive PSB culture. In 3 of 7 the TA culture resulted in less information.

When the PSB culture was negative (24 of 31), 34% of the TA cultures were still positive, which reflects colonization. When we look to the group with no antibiotic pretreatment, which resembles the group of Rumbak et al, in 40% the PSB culture was positive with the same or more germs in the TA culture. When the PSB culture was negative, in 33% still the TA culture was positive, which again reflects colonization. So in our experience, as in many other studies, TA is much less reliable.

Probably there are two reasons for the different results by Rumbak et al. First, they have an extreme high yield in the PCB cultures (69%) and the TA cultures (85%) vs 40 and 60% in our study. Second, their conclusion concerns long-term ventilated patients and this probably cannot be applied to the acute ICU patients described in our study, as well as in many others.

Danielle Galdermans, MD, Hans Slabbynck, MD, and Dirk Cooen, MD, FCCP, Department of Pulmonary Medicine; Jan Nagler, MD, Department of Critical Care; and An Mertens, MD, Department of Microbiology, AZ Middelheim Hospital, Antwerp, Belgium

Meditating on Exhalations

To the Editor:

As a pulmonary physiologist I was trained to think about the exhaled air in terms of vital capacity, end-tidal carbon dioxide tension, and positive end-expiratory pressure (PEEP). I teach our technicians in the Pulmonary Laboratory to always be proud when they can extract a reliable set of vital capacities from a nervous or reluctant patient. Yelling and loud capowing heard near a pulmonary laboratory are almost always a good sign of the labors of a conscientious technician.

Obtaining the levels of carbon dioxide at the very end of exhalation, which reflects the amount of carbon dioxide in the arterial blood, is a more sedated affair because we only need a CO₂ meter and a small sample of expired air while the patient breathes quietly through a mouthpiece.

PEEP is a modern therapeutic intervention used in the care of patients in acute respiratory failure; it raises the patient's alveolar pressure at the end of exhalation, keeping more alveoli open for longer periods of time and resulting in a better oxygenation of the patient's arterial blood.

My "expiratory meditations" came about recently while observing a young lady play the Pied-Piper with the flute. She inhaled deeply to inspiratory capacity every so often and delighted the audience with her exhaled air filtered beautifully by the flute's intricate system of valves. I could almost see the musical notes at the very end of her expiratory efforts, made perhaps more melodic by their increase in CO₂ content.

The trumpet player is interesting because he self-administers PEEP while playing, thus raising not only his audience's music appreciation, but his alveolar and arterial oxygen tensions. No wonder trumpet players always looked so enthralled during performance.

I was taught in medical school that one of the causes of chronic obstructive pulmonary emphysema was a life of prolonged trumpet playing. So naturally when as a junior medical student, I was assigned a middle aged man with this disease, I told him, trying my best to make an impression, that I knew he was a musician. He looked shocked as he assented. I then proceeded to tell him that I also knew he had been playing the trumpet for a long time. He then, to my chagrin, answered with a smile, "No, I always have been a drum player."

Wind instruments, including the human voice, are mostly instruments of exhaled air; vital-capacity-musical-devices accustomed to the presence of carbon dioxide in the passing music are a source of enjoyment, meditation, and curiosity to a drum-playing pulmonary physiologist.

Roberto Llanas, MD, FCCP, Miami Heart Institute, Miami Beach, Florida

Erratum

In the May 1995 issue of CHEST, in the article by Hollander et al on page 1237, the banner reading “For editorial comment see page 1189” should have appeared on page 1242 in the article by Keteyian et al.