elimination from 5 to 2 mm Hg/s. The patient died three days later of septic shock caused by Pseudomonas pneumonia.

Auto-PEEP is attributed to high minute ventilation, short expiratory time, small diameter of endotracheal tubes, and, more often than not, elevated airway resistance. It is normally a function of small airway tone, mucous plugging, and dynamic collapse. While bronchomalacia is a common disorder in COPD patients, the weakness of the posterior wall of the trachea to such an elevated degree is exceptional. Moreover, to our knowledge it has not been described as a source of this amount of auto-PEEP. The response to external PEEP suggests an internal pneumatic stabilization component.

Rafael Fernández, M.D., Francisco Baigorri, M.D., Lluís Blanch, M.D., Albert Martín, M.D., and Antonio Artigas, M.D., Hospital de Sabadell, Sabadell, Spain

References

Foreign Bodies in the Tracheobronchial Tree

To the Editor:

In the August 1991 issue of Chest, Drs Marik and Ballhausen described a patient who aspirated a mercury thermometer into the right lower bronchus. The authors stated that they were unaware of any earlier similar reports. I wish to bring to the authors’ attention a nearly identical case described in the article reporting on our experience with 66 foreign bodies in the tracheobronchial tree.

Figure 3 in that article is remarkably similar to the one shown in the communication by Drs Marik and Ballhausen. There is, however, one noteworthy difference in the management of our patients: We elected to extract the thermometer through a bronchotomy, rather than resecting the lobe, thus saving the normal lung parenchyma. Among our 66 patients, there were only six instances in which resection of lung tissue was necessary (four lobectomies, two segmentectomies); the resections were done only in cases of a long-standing obstruction (up to 12 years in case 2), with destruction of the distal parenchyma.

Dov Weissberg, M.D., F.C.C.P., Department of Surgery, E. Wolfson Medical Center, Holon, Israel

References

Alternation between Atrial Flutter and Atrial Fibrillation

To the Editor:

I read with interest the article by Tunick et al., which appeared in the January 1992 issue of Chest. The frequency of coexistence of atrial flutter in patients with atrial fibrillation, especially in those with periods of sinus rhythm (20/24 [83 percent]), and its significant association with the use of a type 1A antiarrhythmic drug are not unexpected.

In 1956 I reported that atrial flutter was a frequent finding (51 percent) in 115 patients with atrial fibrillation during administration of quinidine, a type 1A antiarrhythmic agent. The more frequently the electrocardiograms were recorded during quinidine therapy, the more frequently the atrial flutter was observed. It is therefore not surprising that a much higher incidence was seen with Holter monitor recording, as reported by Tunick et al. After all, the two arrhythmias share a similar macro-reentrant mechanism, fibrillation being due to random reentry and flutter being due to nonrandom localized reentry.

Tsung O. Cheng, M.D., F.C.C.P., Division of Cardiology, George Washington University Medical Center, Washington, DC

References

656 Communications to the Editor

Downloaded From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21652/ on 06/27/2017