been recognized recently in Japan that man also acquires the infection by eating sliced raw flesh ("sashimi" in Japanese) of wild boars.1 Norimatsu and colleagues2 reported 136 cases of human paragonimiasis and emphasized that most of the patients did not eat freshwater crabs but ate sliced raw flesh of wild boars. Miyazaki and his associate3 showed that the pig and wild boar could serve as paratenic hosts of P. westermani experimentally and concluded that the above-mentioned outbreak of human paragonimiasis was due to the custom of inhabitants eating sliced raw flesh of wild boars.

Sharma,4 in an editorial in the same issue, described three questions to ask patients from overseas. I would like to add one more question: Do you love raw meat? (Or have you ever eaten anything unusual or raw?)

**Hiroshi Kawane, M.D., F.C.C.P.,
Kawasaki Medical School,
Karasaki City, Okayama, Japan**

Reprint requests: Dr. Kawane, Dept. of Medicine, Kawasaki Medical School, Karasaki City, Okayama 701-011, Japan

**REFERENCES**


**To the Editor:**

Dr. Hiroshi Kawane's comment is extremely important. Readers of Chest, internists and other pulmonologists should add boar meat sashimi to the list of sources of Paragonimus westermani.

**Om P Sharma, M.D., F.C.C.P.,
University of Southern California Medical Center, Los Angeles**

**Theophylline-induced Ventricular Tachycardia in a Patient with Chronic Lung Disease**

**Sensitivity to Verapamil**

**To the Editor:**

A 65-year-old man with chronic pulmonary emphysema was admitted for acute respiratory distress. Immediately after aggressive theophylline therapy was started for persistent bronchospasm, monomorphic ventricular tachycardia (VT) with a left bundle branch block QRS morphology appeared, following frequent ventricular premature beats (Fig.1). An intravenous dose of lidocaine (75 mg) failed to terminate the VT. Subsequent intravenous verapamil (3 mg) prolonged the cycle length and terminated VT following a ventricular premature beat (Fig.). Plasma theophylline level was 58.0 μg/ml (therapeutic range 10 to 20 μg/ml).

Verapamil has not generally been considered effective in the management of VT with a few exceptions (ie, idiopathic sustained left ventricular tachycardia). A recent report1 revealed that atrial tachycardia was sensitive to verapamil in a patient with theophylline therapy. Our observation showed that VT in a theophylline-toxic patient was also sensitive to verapamil. This is consistent with a previous experimental report in dogs.2

According to our observation, intravenous verapamil is considered a drug of choice for theophylline-induced VT.

**Akiko Taniguchi, M.D.;
Tohru Ohe, M.D., and
Katsuro Shimoru, M.D.,
National Cardiovascular Center,
Osaka, Japan**

Reprint requests: Dr. Ohe, Division of Cardiology, National Cardiovascular Center, 5-7-1 Fujishirodai, Suita, Osaka, Japan 565

**REFERENCES**

History of Oxygen

To the Editor:

I read with the interest the letter by Dr. Zanetti (CHEST 1989; 95:706) in which he pointed out that Professor Robin credited HBOers with the first use of 100% oxygen over 100 years before it was discovered by John Priestly in 1772. According to Astrup and Severinghaus, oxygen was first discovered (isolated) by Joseph Priestly in 1774. Incidentally, there is some evidence to suggest that oxygen was independently discovered by Carl Wilhelm Scheel two years before Priestly, but his findings were not published until after Priestly's findings.1

John Salyer, R.R.T.,
Educational Coordinator,
Pediatric Respiratory Care,
Rainbow Babies and Childrens Hospital,
Cleveland

REFERENCE
1 Astrup E, Severinghaus JW. The history of blood gases, acids and bases. Copenhagen: Munsksgaard 1986; 36-49

To the Editor:

I appreciate the further enlightenment on the discovery of oxygen given by Mr. Salyer and stand corrected on Joseph Priestly's first name. Another reference,1 however, does list 1772 as the date of publication of his famous work, Observations on Different Kinds of Air.

I hope, however, that Mr. Salyer and others did not misunderstand the main point of my letter—that hyperbaric oxygen therapy cannot be 300 years old, as stated by Dr. Robin. The first practical use of 100 percent oxygen in a hyperbaric chamber dates back to the 1930s. Hyperbaric oxygen is not to be confused with hyperbaric air therapy, a centuries old worthless technique which Dr. Robin was perhaps alluding to in his historic comment and which has no relevance to the present debate on HBO.

Claude L. Zanetti, M.D.,
Chicago

REFERENCES

Tracheal Trauma

To the Editor:

I enjoyed reading the recent report of Abbey et al on "Massive Tracheal Necrosis Complicating Endotracheal Intubation."1

In 1981, Dr. John Burke and I reported two similar cases from the Massachusetts General Hospital. Both our patients had severe tracheal damage secondary to low-pressure, high-volume endotracheal tube cuffs which were monitored carefully against over-inflation. The findings in these patients suggested that, in the presence of tracheal inhalation injury or severe tracheal infection, strict monitoring of pressures—though essential—does not preclude tracheal trauma from a low-pressure cuff.

Physicians and nurses should not feel complacent about the use of any endotracheal cuff, regardless of that cuff's pressure characteristics and particularly in the presence of tracheal inhalation injury, tracheal infection, or other complicating factors. Though cuff pressures of 20 to 25 mm Hg are unlikely to cause severe injury, there is no arbitrarily safe level of cuff pressure in these patients.

Mark R. Katlic, M.D., F.C.C.P.,
Chief of Thoracic Surgery,
Wilkes-Barre General Hospital,
Wilkes-Barre, Pennsylvania

REFERENCES
1 Abbey NC, Green DE, Cicale MJ. Massive tracheal necrosis complicating endotracheal intubation. CHEST 1989; 95:459-60

Cryosurgery, Electrocautery and the Laser

To the Editor:

I have read the article of Cavaliere et al (CHEST 1988; 94:15-21) concerning the now well-known laser therapy. The color illustrations are particularly interesting. However, in the introduction the authors say that "endoscopic application of electrocautery and cryosurgery, though effective, may damage the normal tissue." We have no experience with electrocautery, but we have been using cryosurgery for the last four years and have treated about 200 patients. We have found that cryotherapy has no damaging effect on normal tissue. Would the authors like to expand their statement?

Normal tissue will be damaged only when the cryoprobe is placed against it for a sufficient period. This may be indicated in the treatment of genuine tracheal stenoses. After the slough has parted, the underlying mucosa appears normal and without a retractorial scar. The lack of tissue alteration is a useful consequence in the treatment of tracheal or bronchial stenoses which require the association of cryotherapy and surgery because the sutures may then be effected without any problem. Indications for these two techniques are often the same, except perhaps in the case of an emergency (cryosurgery has a delayed effect). The techniques may be complementary and we think that they should not be systematically compared or opposed.

J. P. Homasson, M.D., F.C.C.P.,
Centre Hospitalier Specialise
en Pneumologie,
Chestly-Larve, France

To the Editor:

Dr. Cavaliere used a reference—my publication—to state that cryosurgery was not a precise therapeutic modality. I certainly agree that any therapy which involves poorly defined tissue freezing, necrosis and subsequent slough is not precise and may destroy normal as well as pathologic tissues. Cryosurgery is also a tissue

CHEST / 96 / 4 / OCTOBER, 1989 / 959