impacts outcome adversely. The frequency of progression from VPBs to sustained VT appears to be low, but confirmatory work is needed. Although current evidence suggests that children are at low risk and that acute overdose patients with high STCs are at high risk of developing significant arrhythmias, the majority of toxic patients are adults with chronic overdosage.

Developing reliable predictive indices, particularly for the chronic overdosage group, will require further study. Previous reports have been limited by small sample size, lack of prospective continuous ECG monitoring, and/or incomplete information as to whether tachyarrhythmias were sustained or transient, required therapeutic intervention, and/or were hemodynamically significant. As we did in our previous study, we urge further study of the prevalence of and risk factors for clinically significant cardiac arrhythmias in theophylline toxicity.

Curtis N. Sessler, M.D.,
Medical College of Virginia,
Richmond, and
Marc D. Cohen, M.D.,
Indiana Heart Physicians,
Indianapolis

REFERENCES
1 Sessler CN, Cohen MD. Cardiac arrhythmias during theophylline toxicity: a prospective continuous electrocardiographic study. Chest 1990; 98:672-78
7 Atten ML, Martin TB. Life-threatening theophylline toxicity is not predictable by serum levels. Chest 1987; 91:10-14

Complications of Thoracostomy

To the Editor:

The case report by Meisel et al, which appeared in the September 1990 issue of Chest, is to be commended for describing a complication of grave importance from a very commonly performed procedure. Unfortunately, the report fails to emphasize two extremely critical points.

First, the decision as to the position of placement of the chest tube must be based on several different aspects of the clinical as well as the radiologic findings. Proper evaluation of the chest radiograph should include looking for areas of adhesions. A region where the lung is expanded in the face of a hydro pneumothorax, such as could occur with localized adhesions, is certainly one of these potential danger points. This should cause one to consider placement of the tube in a different area and/or with a different technique.

The second, and certainly most important, point relates to the treatment the patient receives after the chest tube was placed in a major cardiovascular structure. Clearly, the only life-saving treatment in such a situation is clamping the chest tube and immediate emergency thoracotomy or sternotomy, preferably in the operating room if the patient is stable, prior to removal of the chest tube. The case report failed to mention or recommend this proper course of action. Moreover, it did not point out that removing the chest tube prior to thoracotomy is likely to be a fatal maneuver.

Trocac chest tubes per se are not dangerous if used correctly in appropriate situations by skilled operators. Knowing how to properly handle the complications is vital for anyone performing tube thoracostomy. As always, judgment is of paramount importance.

Charles F. Reuben, M.D., F.C.C.P.,
Department of Thoracic and Cardiovascular Surgery,
St. Joseph's Hospital,
Milwaukee

REFERENCE

To the Editor:

We read with interest the case report by Meisel et al, in which they describe a complication of trocar thoracostomy, namely, perforation of the right atrium. This prompted us to report a recent case in which pulmonary artery perforation occurred during thoracostomy.

A 63-year-old man underwent a left-sided pneumonectomy in 1972 because of squamous cell carcinoma. In 1982 left-sided empyema due to a bronchopleural fistula occurred. In October 1989 the patient was admitted again with dyspnea, a productive cough, and high spiking fever. His cough was characteristically posture dependent. A chest radiograph showed an air-fluid level in the left hemithorax suggestive of empyema. Needle aspiration failed because of heavy, thickened pleura. During an attempt to drain the cavity with a trocar system, pulsating blood came through the tube. The trocar was left in situ so that its location could be shown to the surgeon and further blood loss could be prevented (blood for cross matching was taken from a side channel), and an emergency sternotomy was performed. The tube was found to be in the central part of the pulmonary trunk. The tube was removed, and the 0.8-cm defect was closed with monofilament sutures. Postoperative recovery was uneventful. No further attempts were made to drain the cavity. At present the patient is well without complaints suggesting empyema.

Although not stated in surgical textbooks, it is our opinion that in case of accidental injury of a main vessel with a sharp instrument (eg, a trocar), it is mandatory to leave this instrument in place to prevent exsanguination. This long-taught clinical adage is confirmed by our experience with this patient.

K. W van Kraalingen, M.D.,
J Stam, M.D.,
and J. Rauwerda, M.D.,
Free University Hospital,
Amsterdam, The Netherlands

REFERENCE

To the Editor:

The report by Dr Meisel and colleagues brings to our attention yet another complication of pleural intubation using the trocar-type chest tube. Several years ago we reported a similar complication,
which occurred when a pleural drain was unintentionally placed in the hepatic vein. However, our patient survived the complication. Once blood appeared in the drain and the complication was recognized, the drain was clamped, not removed, and the patient was taken to the operating room. There, with the patient under general anesthesia, the tube was removed and the bleeding was controlled.

Unintentional placement of a pleural drain in a major blood vessel does not necessarily lead to exsanguination. Prompt clamping of the drain will stop almost any bleeding. The drain should be removed only after the patient has been anesthetized and is ready for a thoracotomy, with a sufficient amount of blood prepared for transfusion.

Doc Weissberg, M.D., F.C.C.P.,
and Yoram Fintsi, M.D.,
Tel Aviv University,
E. Wolfson Medical Center,
Holon, Israel

REFERENCES


To the Editor:

I welcome Dr Reuben's comments, with which I fully agree. A region where lung parenchyma is hyperinflated in the presence of hydropneumothorax may certainly signify local adhesions. However, identification of such a region on a less-than-optimal x-ray film of a patient with a diseased lung and a rib cage deformity of such an extent is almost impossible.

As for the second point made by Dr Reuben and the remarks made by Dr van Kralingen and colleagues and by Drs Weissberg and Fintsi, who have encountered similar complications, we must emphasize that thoracostomy was performed on the spot, immediately after it became clear that resuscitative efforts were unsuccessful in stabilizing the rapidly deteriorating patient. This may have been the situation due to a nonsealing laceration of the right atrium. We fully support the long-taught clinical adage that an instrument penetrating a blood vessel should not be removed anywhere except in the operating room under direct vision.

Simcha Meisel, M.D.,
Department of Cardiology,
Sapir Medical College,
Kfar-Saba, Israel

Intravenous Etoposide Therapy and Intractable Hiccups

To the Editor:

The therapeutic effectiveness of etoposide in the treatment of small cell lung cancer is well established. However, its clinical toxicity is sometimes very troublesome. For the first time, I recently encountered a patient who developed intractable hiccups after receiving intravenous etoposide therapy.

A 71-year-old man was diagnosed as having small cell carcinoma of the lung and hilar adenopathy (T2N2M0, clinical stage III), based on the findings from chest roentgenography, chest computed tomography, and a transbronchial lung biopsy. The patient received combination chemotherapy with 100 mg of cisplatin and a total dose of 300 mg of etoposide in three fractions (Fig 1). From day 2 the patient complained of gradually increasing hiccups, which were clearly associated with two more treatments with etoposide. The patient did not respond to standard management, including irritation of the nasopharynx and the use of a dopamine antagonist (metoclopramide, 50 to 70 mg/day) and an antiepileptic drug (clonazepam, 1.5 to 6.0 mg/day). On day 9, however, continuation of symptomatic therapy produced resolution of the intractable hiccups, as a result of reduction of the etoposide reaction.

It is known that several drugs, such as diazepam, alpha-methyl-dopa, and dexamethasone, induce an intractable type of hiccups, which are reflexive spasmodic contractions of the diaphragm with sudden inspiration abruptly terminated by a variable audible closure of the glottis. However, little information about etoposide-induced intractable hiccup is available. Although hiccups seem to be a relatively minor problem, the patient had significant discomfort with prolonged episodes. Intractable hiccups should be added to the list of potential adverse effects of intravenous etoposide therapy.

Shigenobu Umeki, M.D., Ph.D., F.C.C.P.,
Kawasaki Medical School,
Okayama, Japan

Reprint requests: Dr Umeki, Division of Respiratory Diseases,
Department of Medicine, Kawasaki Medical School,
377 Matsushima, Karashiki, Okayama 701-01, Japan

REFERENCES


Chest Wall Vascular Malformations

To the Editor:

A 32-year-old woman's gynecologist auscultated a "murmur" in her chest. The patient's past history was positive for a spontaneous right pneumothorax 14 years previously. This resolved without treatment. A second spontaneous pneumothorax on the right occurred four years after the first, and was treated with an anterior thoracostomy tube. The results of the current physical examination were unremarkable except for a two-component vascular sound in the posterior right side of the chest medial to the scapula. A harsh systolic bruit was followed by a soft venous hum that continued through cardiac diastole. Both sounds diminished with the Valsalva maneuver. A two-view chest x-ray examination was normal. Selective

Figure 1. Clinical course. CDDP = cis-diamminedichloroplatinum (cisplatin).