subpopulation at risk for ventricular arrhythmias, Aitken and Martin reported that in 54 consecutive patients with serum theophylline levels ranging from 39 to 78 mg/L, there was no correlation between toxic side effects and underlying disease, patient's age, or serum theophylline level. Sessler and Cohen tempered their conclusions by noting that "further work is needed to confirm these observations in larger studies of similar design," but we feel that their conclusions may be misinterpreted and cause the reader to believe that it is acceptable not to monitor all patients who have an elevated serum theophylline level.

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To the Editor:

Bender and colleagues contend that our conclusions that ventricular fibrillation (VF) and cardiac arrest are rare complications of theophylline toxicity" may be misinterpreted and cause the reader to believe that it is acceptable not to monitor all patients who have an elevated serum theophylline level." Clearly, selected cardiac arrhythmias, including not only VF and cardiac arrest but also supraventricular tachyarrhythmias and ventricular tachycardia (VT), are serious treatable complications of toxicity, and ECG monitoring plays an important role in their timely recognition. Although our data suggest that such clinically significant arrhythmias may be less common than has been generally appreciated, the sample size was modest. We intentionally avoided making recommendations for withholding ECG monitoring, and until reliable criteria to identify patients who are at very low risk for such arrhythmias are developed and validated, we support the use of ECG monitoring for all toxic patients.

We stand by our conclusions, drawn from our experience as well as the literature, that VF and cardiac arrest are rare complications of theophylline toxicity. While Bender et al seek to demonstrate that ventricular tachyarrhythmias are in fact fairly common, the literature they cite is biased toward a higher prevalence of serious toxicity than is likely to be observed in most clinical settings. Paloucek and Rodvold culled all cases of theophylline toxicity published in the English-language literature from 1975 to 1985. Since most cases that are the subject of a case report or small series represent instances of severe or unusual toxicity rather than routine cases, the overall prevalence of serious manifestations reported in that cumulative review is likely to be excessive. Hall et al described four episodes of VT among 22 consecutive cases of toxicity from intentional overdose. This report and others document that ventricular tachyarrhythmias and cardiac arrest occur primarily in acute overdose patients who have very high serum theophylline concentrations (STCs) (> 100 mg/L).

Although acute overdoses may account for the majority of theophylline toxicity cases reported to poison control centers, this population represents a relatively small percentage of toxic patients encountered in clinical series. Furthermore, cases reported to poison control centers are generally more likely to have severe, unusual, or problematic manifestations, whereas mild toxicity following chronic theophylline overmedication is more likely to go unreported.

In a recent series of 116 consecutive patients who presented in the emergency departments of our hospitals with an STC >50 mg/L, only 12 percent had ingested an acute overdose, and nearly half had mild toxicity following chronic overmedication. Clinically significant ventricular tachyarrhythmias (one with VF preceded by VT and another with VF) were limited to two patients who had STCs >100 mg/L following acute overdose. In another series, none of the 51 consecutive patients with STC >39 mg/L were reported to have ingested an intentional overdose, and serious arrhythmias were limited to one VT and one "presumed fatal arrhythmia." Therefore, from the perspective of the clinician (as opposed, perhaps, to poison center personnel), available evidence suggests that ventricular tachyarrhythmias and cardiac arrest are quite uncommon.

Bender et al extrapolate the findings of Aitken and Martin, who found no correlation between "side effects" and underlying disease, age, or STC, in an attempt to refute our observation that patients with frequent or complex ventricular premature beats (VPBs) tend to have advanced age and heart disease. We commented that the few well-documented cases of sustained VT reported in the literature occurred among older patients with heart disease and/or very high STC. We made no attempt to develop a predictive index for identification of patients with VPBs or VT. Rather, we concluded that VPBs (which typically do not require therapeutic intervention) are common among theophylline-toxic patients when prolonged ECG recording is performed and that the presence of ventricular ectopy during toxicity may largely reflect underlying conditions such as chronic heart and lung disease, as has been previously demonstrated for nontoxic patients.

Bender et al imply that since Aitken and Martin found no correlation between underlying illness, age, and STC and side effects, serious toxicity, such as clinically significant arrhythmias, occurs unpredictably in all patients with theophylline toxicity. Aitken and Martin studied a specific population: middle-aged to elderly men (veterans) with very high STCs (>39 mg/L), none of whom were reported to have experienced an acute intentional overdose; thus, extrapolation of their results to other groups should be undertaken with caution. For example, whereas other studies also demonstrate a poor correlation between STC and severity of illness for patients with chronic theophylline overmedication, a recent report of the data on adult and pediatric patients with chronic overmedication demonstrated a significant correlation between advanced age and the presence of life-threatening events. Additionally, in a series of 65 consecutive toxic pediatric patients, none had ventricular tachyarrhythmias or cardiac arrest. Finally, there is compelling evidence of a strong correlation between marked STC elevation (>100 mg/L) and high risk of life-threatening events, including ventricular tachyarrhythmias, for patients with acute overdose. Thus, a variety of factors, or a combination of factors, may influence the risk of developing clinically significant arrhythmias.

Telemetry beds are in short supply in many hospitals. Thus, it is important to strive to identify the populations of theophylline-toxic patients in whom clinically significant cardiac arrhythmias are expected (and in whom close monitoring is therefore mandatory) and those in whom such arrhythmias are highly unlikely to occur (and in whom ECG monitoring might therefore be omitted). In addition to ventricular tachyarrhythmias, supraventricular tachyarhythmias are important manifestations of toxicity, which are probably more common, may be poorly tolerated hemodynamically, and may necessitate antiarrhythmic therapy. Although VPBs are frequently considered to be serious or life-threatening complications of toxicity, such ectopy rarely requires therapeutic intervention or
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 overmedication.

Developing reliable predictive indices, particularly for the chronic overmedication 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.

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Complications of Thoracostomy

To the Editor:

The case report by Meisel et al,1 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 hydropneumothorax, 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.

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.

Trocars 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.

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REFERENCE

To the Editor:

We read with interest the case report by Meisel et al,1 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 (e.g., 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.

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REFERENCE

To the Editor:

The report by Dr Meisel and colleagues1 brings to our attention yet another complication of pleural intubation using the trocar-type chest tube. Several years ago we reported a similar complication,