chylothorax case KS was orally. The administration was our observation. The incidence of AIDS, we might see more cases of chylothorax secondary to pulmonary KS.

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REFERENCES

Mexiletine for Paroxysmal Atrial Fibrillation

To the Editor:

Although the effectiveness of Vaughan Williams class 1A (and recently class 1C) antiarrhythmic drugs for paroxysmal atrial fibrillation (PAF) prevention has been well established, as far as we know the effectiveness of class 1B drugs has only been reported in a case by Slater et al.1 In their report, mexiletine was given to four patients with nodal tachycardia, reportedly restoring sinus rhythm in all. The authors also happened to find that the injection of mexiletine in a patient with nodal tachycardia mixed with sinus rhythm and atrial fibrillation not only terminated nodal tachycardia but also restored sinus rhythm.

With the exception of this report, we have not found any other concerning the effects of mexiletine on atrial fibrillation in humans.

We have recently experienced a case similar to that of Slater. In our case, a patient who had confirmed frequent ventricular prematurity contractions with mixture of PAF (as measured by Holter ECG) was successfully treated by oral mexiletine. This experience led us to examine the effects of mexiletine therapy on PAF in more patients.

Mexiletine was given orally to two women and four men (mean age 36.8 years, range 28 to 75) with PAF; three patients with idiopathic PAF, two with ischemic heart disease and one with valvular heart disease. These patients showed more than one episode of PAF, accompanied by subjective symptoms, in a week. Numbers of atrial prematurity contractions (APC) and occurrence and duration of PAF were monitored by Holter ECG before and after mexiletine administration (450 to 600 mg/day).

Complete disappearance of PAF was seen in three patients, and more than 50 percent decrease in the episodes of PAF and APC was observed in one patient. The remaining two patients showed no response to the drug. The number of APC decreased by 52 ± 14 percent (p<0.01) in all the patients who were given mexiletine orally. Serum concentration of mexiletine was 1.20 ± 0.5 µg/ml.

These four patients have since been successfully treated by oral mexiletine therapy for three to 16 months with no recurrence of PAF.

Among the four patients who reacted to the drug, injection of mexiletine (2 mg/kg) to one patient restored sinus rhythm from atrial fibrillation on three different occasions in a week. However, injection of mexiletine did not have effects on PAF in the remaining three patients.

Results indicated that mexiletine was effective on the prevention of PAF in more than 50 percent of the patients with a history of frequent PAF episodes. Our interpretation is that mexiletine's action probably decreased the numbers of APC (which could have triggered atrial fibrillation) and possibly increased atrial fibrillation threshold, as reported for ventricular fibrillation. The action of converting atrial fibrillation to sinus rhythm does not seem as potent in mexiletine as in class 1A and 1C drugs, judging from the fact that mexiletine (when given intravenously) was effective only in one of four patients. Nevertheless, the mechanisms by which mexiletine prevents PAF could be elucidated.

An interesting finding is that class 1B drugs (ie, lidocaine) have thus far been considered ineffective on atrial arrhythmias, whereas mexiletine—classified under the same class—did show its effectiveness on such arrhythmias. Mexiletine might very well have a different action than that of drugs belonging to the same category. Recently, there has been a report in which mexiletine showed somewhat different electrophysiologic effects than those of lidocaine on the calcium currents of guinea-pig cardiac cells. Singh and Vaughan Williams reported that mexiletine reduced the maximum driven frequency and conduction velocity, but increased electrical threshold in isolated rabbit atrium.2

We would like to conclude that mexiletine might be a first choice drug for PAF prevention because its effectiveness was demonstrated in more than 50 percent of the patients in our experience, and because no significant adverse effects were recorded. However, the prevention mechanisms of mexiletine remain to be elucidated.

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Spontaneous Pneumothoraces in AIDS Patients Receiving Aerosolized Pentamidine

To the Editor:

Spontaneous pneumothoraces occur not infrequently in patients with acquired immunodeficiency syndrome (AIDS) who have con-
Iodine Determination in Amiodarone Lung

To the Editor:

Amiodarone is an iodine derivate, and its accumulation in the pulmonary parenchyma has been documented. Iodine has also been found in the alveolar macrophages. We present a case where iodine quantification in the pulmonary tissue was carried out, with lung biopsy thoracotomy (LBT). A 69-year-old man had an amiodarone intake during 15 months of 200 mg/day. He developed symptoms of progressive respiratory insufficiency that required ICU treatment. Chest radiology displayed diffuse bilateral alveolar-interstitial pattern. Most common causes of diffuse pulmonary infiltrates were discarded through data obtained with Swan-Ganz catheterization, bronchofiberscopy, bacteriologic study with Barlett aspiration catheter, RAL cell count, virologic and serologic studies (Table 1). Pulmonary scanning with Ga 67 showed mild diffuse hypercapitation in posterior lower segments of left lung. Biopsic sample was carried out in this area (LBT). Our pathologic findings correlated with data reported by Marchilinsky. Mineralogic quantification with spectrophotometry was performed. Iodine concentration found was 2.1 x 10. This determination proved to be negative in a necropsic analysis carried out in a patient without respiratory disease and no history of amiodarone intake, and of same age as the reported case.

We conclude that spectrometric determination is useful in evaluating iodine levels in lung tissue. Amiodarone-treated patients display iodine-recognizable levels in lung tissue samples.

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Table 1—Hemodynamic Data

<table>
<thead>
<tr>
<th></th>
<th>Before equalization</th>
<th>After equalization</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO</td>
<td>2.8 L/min</td>
<td>4.1 L/min</td>
</tr>
<tr>
<td>WP</td>
<td>19 mm Hg</td>
<td>13 mm Hg</td>
</tr>
<tr>
<td>CVP</td>
<td>24 cm H2O</td>
<td>16 cm H2O</td>
</tr>
<tr>
<td>BP</td>
<td>80/50 mm Hg</td>
<td>130/70 mm Hg</td>
</tr>
<tr>
<td>PVR</td>
<td>450 dyn/secmm³</td>
<td>400 dyn/secmm³</td>
</tr>
<tr>
<td>SVR</td>
<td>2200 dyn/secmm³</td>
<td>960 dyn/secmm³</td>
</tr>
</tbody>
</table>

CO = cardiac output; WP = wedge pressure; CVP = central venous pressure; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance