Effective Management of Pericardial Neoplasia

Malignant invasion of the pericardium is always serious and, unfortunately, increasingly detected. Once the cell type is identified, physicians have an array of therapeutic options (condensed in Table 1).

In this issue of CHEST (see page 1412), Dr. Martinoni and colleagues have very carefully followed up their series of patients with various malignancies who were treated successfully with intrapericardial thiotepa. There were no complications from either the therapy or from the pericardiocentesis, which was necessary in every case. Absence of complicated taps may be ascribed both to echocardiographic monitoring and the very large size (749 ± 230 mL [± SD]) of the effusions. Before pericardiocentesis all patients were quite ill, mainly due to weakness and dyspnea; 56% were in New York Heart Association heart failure class III and 28% in class IV owing to cardiac tamponade in the majority of patients.

Thiotepa is both an antineoplastic and a sclerosing agent. Unlike tetracycline, it induces no chest pain, no myelosuppression, and no ECG changes. Of the many intrapericardial therapies, only tetracycline has appeared to have significantly positive effects, at least in the short run, yet it is strictly a sclerosant, suppressing effusions without antineoplastic effects (indeed, indwelling pericardial tubes and catheters can provoke pericardial sclerosis without necessitating chemical sclerotherapy). However, thiotepa is almost an ideal agent based on its performance: significant effects on several kinds of malignancy because of its antineoplastic properties, as well as suppression of effusions. In two patients dying on the 22nd and 25th days after the procedure, at autopsy there was no evidence of pericardial effusion.

It will be important to learn the results of long-term follow-up in the series by Martinoni and colleagues, since the pericardium itself was effectively treated and all patients were receiving systemic chemotherapy, especially in those with metastatic cancer and particularly certain cancers (eg, breast, lung), in whom survival has been months to a year. Perhaps the combination of local effectiveness with systemic antineoplasia will now improve that grim outlook.

<table>
<thead>
<tr>
<th>Pericardiocentesis</th>
<th>Pericardiocentesis Plus Intrapericardial Tetracycline or Chemotherapeutic Agents</th>
<th>Subxiphoid Pericardiectomy</th>
<th>Pleuropericardial Window</th>
<th>Pericardectomy/Resection</th>
<th>External Beam Irradiation</th>
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</thead>
<tbody>
<tr>
<td>Advantages</td>
<td>Relieves tamponade</td>
<td>Relieves tamponade</td>
<td>Can evaluate extent of disease</td>
<td>Can evaluate extent of disease</td>
<td>Noninvasive</td>
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<tr>
<td>Local anesthesia</td>
<td>Local anestheisa; lower recurrence than pericardiocentesis alone; controls most malignant effusions</td>
<td>Local anestheisa; yields fluid for study; yields pericardial biopsy; permits examination of pericardial space</td>
<td>Excellent for diagnosis; can be done by balloon catheter</td>
<td>Excellent for diagnosis</td>
<td>Nondiagnostic</td>
</tr>
<tr>
<td>Disadvantages</td>
<td>Very frequent recurrence</td>
<td>Occasionally recurrences</td>
<td>General anesthesia unless by balloon</td>
<td>General anesthesia</td>
<td>Tamponade unchanged significantly less if thoracoscopic</td>
</tr>
<tr>
<td></td>
<td>No biopsy</td>
<td>Sometimes logistically difficult</td>
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</table>

*Adapted from Spodick1 with permission.
Earlier detection should also improve therapeutic success. Yet, primary and secondary pericardial malignancies can elude detection, at least until a clinically significant pericardial effusion appears. While 50% of pericardial effusions in patients with malignancy anywhere else in the body are “sympathetic”—presumably autoimmune—with no signs of pericardial metastases,1 diagnostic success is also influenced by knowledge of malignancy elsewhere. However, it is not rare for a benign or malignant tumor to first appear as pericardial effusion with or without cardiac tamponade. Advances in diagnosis, systemic chemotherapy, and treating pericardial lesions intrapericar-
dially should continue to improve the situation.

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References

Does Benign “Primary Snoring” Ever Exist in Children?

The American Academy of Pediatrics1 has recommended that all children who snore be evaluated. The best test to investigate whether snoring is a health risk is the nocturnal polysomnogram. But there are many variations in what are considered to be normal or abnormal polysomnograms in children. The article by Li and colleagues in this issue of CHEST (see page 1467) is an example of the problems associated with the use of standard guidelines derived from criteria that were initially developed for adult sleep-disordered breathing (SDB). In their study, on 2 nights of polysomnography in children, these authors indicated that “We classified an indi-
vidual with normal obstructive apnea index but with nocturnal snoring for > 4 nights per week as suffering from primary snoring.” They argue that the absence of consensus did not allow the selection of any other definition of SDB. Many questions are raised by this study, which relate to the following: (1) the definition of the children’s group (ie, is age sufficient to define grouping?); (2) the use, for research purposes, of an obstructive apnea index when we know that SDB in children is rarely associated with sleep apnea; (3) can we expect good research outcome from usage of limited equipment for home recording?; and finally, (4) does “primary snoring” really exist?

Regarding age, age grouping is important in children, but to ignore the occurrence of puberty that leads to enlargement of tongue and mucosa, particularly the nasal mucosa, is a problem. We systematically use Tanner staging,2 particularly for patients between 9 and 15 years of age. Even if Tanner staging involves a subjective component, this allows us to place teenagers who are in Tanner stage 5 in the “young adult scoring criteria” group. In considering the effect on sleep and on the size of the upper airway, we integrate Tanner staging in the formation of our subgroup of patients who are 10 to 14 years old. A 10 or 11-year-old patient who is in Tanner stage 1 is not the same as one who is in Tanner stage 2 or 3. Age is also important in that, by 2 years of age, >98% of children in our normative database have a respiratory rate during any stage of sleep of < 20 breaths/min. This allows us to calculate easily the number of 30-s epochs with elevated respiratory rates. As breathing frequency × tidal volume = minute ventilation, tachypnea during sleep that is associated with snoring is a polysomnographic indication of abnormal breathing, which was distinctly shown in an outcome study looking also at clinical complaints.3,4 But age subdivision is also critical during the prepuberty years, as 60% of the adult craniofacial features will be developed by 4 years of age, and 90% by around 12 years of age.5 The investigation of craniofacial growth patterns is important, and the impairment of skeletal maxillo-mandibular growth is critical when considering a diagnosis of SDB. The impairment of nasal breathing leads to noisy breathing, increases in respiratory effort, mouth breathing, and further abnormal skeletal development that can impact on upper airway size. This skeletal change occurs much before the puberty-related soft-tissue enlargement.

With regard to the choice of the apnea index as the sole selection criterion, abnormal breathing during sleep has been linked, for example, to sleepwalking, sleep terror, bedwetting, anxiety, phobia, aggressive behavior, inattention, hyperactivity, school difficul-