Communications to the Editor

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Chagas’ Disease

To the Editor:

In reference to the very interesting report by Spira and colleagues,1 which appeared recently in CHEST, I would like to add another illness to Dr. Spira’s list: Chagas’ disease. This is an infection, endemic in Central and South America, mostly in the central heartlands of Brazil, caused by a protozoan, Trypanosoma cruzi. In its chronic stage, it causes a well-known myocardiopathy, as well as often very prominent esophageal and colonic lesions, the “megas.” Its bronchopulmonary manifestations have been recently and extensively reviewed.2 In its chronic stage, it causes a well-known myocardiopathy, as well as often very prominent esophageal and colonic lesions, the “megas.” Its bronchopulmonary manifestations have been recently and extensively reviewed.2 T cruzi infects wild animals, such as armadillos, racoons, and rodents, as well as humans. Transmission is by the blood sucking beetles of the Triatominae subfamily, which live in the cracks of mud-walled housing; by night, they bite their victims, usually in uncovered areas, mostly the face, originating their vulgar name of “barbeiros” (barbers). In the United States, the disease may be seen in immigrants3 or be acquired by congenital or blood transfusion transmission.4 Chagas’ disease may course with an acute phase, seen mostly in children, with fever and inflammation at the inoculation site. Occasionally, there is severe homolateral palpebral inflammation (Roñãa’s sign). The chronic stage is usually marked by the myocardial lesions, as well as the esophageal and colonic dilatations. These produce severe symptoms (dysphagia, constipation) and typical radiologic and endoscopic findings. The myocardiopathy is characterized by arrhythmias and bundle-branch blocks, usually with severe and intractable heart failure at the late stages. The anatomic basis for all of these lesions is fundamentally similar. There is a gradual destruction of nerve cells in the digestive tube walls and of myocardial myofibrils by T cruzi Leishmania forms. The lungs may be affected secondarily to heart disease (congestion, thromboembolism, and amiodarone fibrosis) or to esophageal disease (aspiration pneumonia). More specific, rare and even more rarely significant, is the bronchopat- thy, which is the reason why Chagas’ disease should be included in Dr. Spira’s list. It is a veritable, if macabre, experiment in neurophysiopathology, perpetrated by nature. Essentially, it appears that the destruction of Leishmania bodies and the rupture of microcysts may denervate the bronchial walls. Lima Pereira, as noted in the study by Bethlem and colleagues,2 showed bronchial wall ganglia with a decrease in the number of neurons and with residual nodules, as well as ganglionitis, periganglionitis, and extensive denervation.

The ensuing bronchomotor functional disturbances have been extensively studied. In summary, a decrease in maximum expiratory flow rate has been observed by some, though inconsistently,4 but not by others.2,5 Pharmacologic challenges have been inconclusive; nifedipine may cause a fall in the maximum expiratory flow at very low lung volumes.2

Aside from the very challenging neurophysiopathologic model, the clinical impact of these bronchial lesions is certainly small. In 250 autopsies, Koeberle6 found 13 cases of bronchiectasis, as opposed to 69 cases of megacolon and 61 of megasphagnum. The dilatation may extend to the trachea, which does not happen in other diseases that course with bronchiectasis. CT and high-resolution CT studies should help to better evaluate these interesting lesions.

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Possible Diaphragmatic Ischemia Following Harvesting of the Internal Mammary Artery

To the Editor:

Harvesting the internal mammary artery is a standard part of the majority of coronary artery bypass operations, but it is
associated with an increased incidence of postoperative pulmonary complications. The use of bilateral mammary arteries increases this risk appreciably. Leaving the ipsilateral pleura unopened does not prevent collapse/consolidation development. The explanations vary, none of which truly explain the excessively high risk of collapse/consolidation after harvesting the internal mammary artery.

The internal mammary artery gives off two terminal branches: the superior epigastric and the musculophrenic. The musculophrenic artery runs around the costal margin of the diaphragm and supplies the lower intercostal spaces and the diaphragm. Thus, disconnection of the distal end of the mammary artery for coronary grafting may impair the blood supply to the diaphragm, intercostal muscles, and the phrenic nerve. The relatively ischemic diaphragm thus may not contract in response to stimulation of the phrenic nerve. A temporarily paralyzed diaphragm becomes flaccid and raised just as in phrenic nerve injury, and increases the chance of pulmonary collapse/consolidation. The ischemia is obviously only temporary until the preformed collaterals open up. Thus, the raised left hemidiaphragm after harvesting the left internal mammary artery may not be secondary to overlying collapse/consolidation or phrenic nerve palsy, but rather a primary event with collapse/consolidation being a secondary event.

The importance of the internal mammary artery was demonstrated in a swine model that is being developed for other experiments; it was utilized to demonstrate the extensive blood supply to the diaphragm. Crystal violet dye was injected into the left atrium, after the left internal mammary artery had been harvested and disconnected distally. The left hemidiaphragm demonstrated virtually no uptake of the dye, compared with the right side. Direct injection into the internal mammary artery resulted in the whole ipsilateral hemidiaphragm taking up the crystal violet dye virtually immediately (Fig 1).

Obviously the data are preliminary and could have been bolstered by phrenic nerve tests, local tissue oxygen saturation, nicotinamide adenine dinucleotide/nicotinamide adenine dinucleotide (reduced forms), ratios and videofluoroscopy of the diaphragms postoperatively. The aim of this small study was not to be exhaustive and comprehensive, but merely to draw the cardiac surgeon’s attention to another possible reason that pulmonary complications after harvesting the internal mammary artery may be due to diaphragmatic ischemia.

Now that the internal mammary artery is being used in pulmonary transplantation for bronchial artery revascularization, diaphragmatic complications could become more important in an already critical field.

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Giant Schwannoma of the Posterior Mediastinum

To the Editor:

We read with interest the article by Strollo and colleagues on primary mediastinal tumors; we fully support the descriptions of schwannoma symptoms, locations, diagnosis, and treatment. These neoplasms are the most common mediastinal neurogenic tumors; they are usually benign and slow growing and frequently arise from a spinal nerve root, but may involve any thoracic nerve.

Recently, a 48-year-old woman was admitted to our hospital because of dyspnea at rest, productive cough, and a referred vague right chest pain of 10 years’ duration. Physical examination showed dullness on percussion and decrease of breath sounds in the right hemithorax. A chest radiograph showed extensive radiopacity involving most of the right hemithorax. A chest CT scan showed a huge, well-circumscribed, heterogeneous tumor with punctate calcifications in the posterior mediastinum (Fig 1).

Figure 1. Crystal violet staining of the diaphragm. Dark areas represent perfusion with crystal violet and pale areas represent relatively ischemic areas. Orientation: viewed from below, left hemidiaphragm uppermost, anterior to the left.

Figure 1. CT scan of the chest showing a heterogeneous tumor arising from the paraspinal sulcus, compressing the lung and displacing the mediastinum with the subocclusion of the main right bronchus.
Complete resection of the mass and bilobectomy, because of the middle and lower lobe destruction, due to extensive compression by the tumor, was performed via thoracotomy. Histologic findings revealed a well-circumscribed schwannoma arising from an intercostal nerve root.

In spite of the large volume of the mass, our patient did not experience any frank paresthesia or nerve pain to confirm the statements of Swanson\textsuperscript{2} and Gale and colleagues,\textsuperscript{3} even for larger schwannomas.

Our case showed that the indolent and slow growing nature of these tumors can lead to a dangerous underestimation of the case.

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Squamous Cell Carcinoma With Unknown Origin

Metastasis to Mediastinal Lymph Nodes

To the Editor:

The case report by Blanco and colleagues in CHEST (September 1998)\textsuperscript{1} was of great interest to me. In the issue, the authors claimed the first English-language report describing metastasis of a squamous cell carcinoma of unknown origin to mediastinal lymph nodes. However, Holmes and Fouts,\textsuperscript{2} in 1970, analyzed 686 patients with a diagnosis of metastatic cancer of unknown primary origins. As their Table 2 demonstrates,\textsuperscript{2} one patient was diagnosed histologically with squamous cell carcinoma, and the principle sites of metastases were the mediastinal lymph nodes.

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