previously documented observations or if they have any data to support their contention that these factors *directly* affected the development of postoperative atrial arrhythmias in their series.

We also have some questions regarding the method by which various components of the conduction system were actually "preserved." With respect to the crista terminalis, it is not clear whether the authors are referring to preservation of longitudinal or transverse cardiac conduction. Their comments imply that avoidance of transverse anatomic disruption of the crista terminalis constitutes "preservation" of this structure. In their surgical technique the authors describe performing a longitudinal incision along the sulcus terminalis. In that the sulcus terminalis represents the medial epicardial edge of the crista terminalis, we would assume that such an approach, through either creating or closing the incision, could create conduction block in the vicinity of the crista terminalis and may also alter preferential longitudinal conduction along it, factors that have both been implicated in the pathogenesis of intraatrial reentrant tachycardias in a variety of experimental investigations[4, 6] and clinical studies.[5] In addition, we wonder whether the authors could clarify the superior extent of the atriotomy used. As we interpret the location of the atriotomy used, if extended proximally enough, such an incision would have the potential of causing direct injury to the sinoatrial node, which is located along the rostral portion of the crista terminalis. If, as in the Senning operation, the atriotomy was not made along the sulcus terminalis but rather anterior to it on the right atrial free wall, these problems would be avoided.

Finally, we caution the conclusions that this particular technique prevented the development of postoperative atrial arrhythmias. The follow-up in this small series was very short. Arrhythmia-free survival in this patient population correlates to the length of follow-up, perhaps, as the authors recognized, related to the cumulative effects of prolonged atrial distention and stretch.[7] It is only with intermediate and late-term follow-up that any meaningful conclusions in this regard can be reached.

Sanjiv K. Gandhi, MD
Burt I. Bromberg, MD
Charles B. Huddleston, MD
Department of Surgery
Division of Cardiothoracic Surgery
Washington University School of Medicine
St. Louis Children's Hospital, Suite 5W24
One Children's Place
St. Louis, MO 63110

REFERENCES

1. Hashimoto K, Kurosawa H, Tanaka K, Yamagishi M, Koyanagi K, Shinichi I, et al. Total cavopulmonary connection without the use of prosthetic material: technical considerations and hemodynamic consequences. J Thorac Cardiovasc Surg 1995;110:625-32.
2. Balaji S, Gewillig M, Bull C, deLeval MR, Deanfield JE. Arrhythmias after the Fontan procedure: comparison of total cavopulmonary connection and atriopulmonary connection. Circulation 1987;84(Suppl):III162-7.
3. Kürer CC, Tanner CS, Norwood WI, Vetter VL. Perioperative arrhythmias after the Fontan repair. Circulation 1990; 82(Suppl):IV190-4.
4. Rodefeld MD, Bromberg BI, Schuessler RB, Boineau JP, Cox JL, Huddleston CB. Atrial flutter after lateral tunnel construction in the modified Fontan operation: a canine model. J Thorac Cardiovasc Surg 1996;111:514-26.
5. Olgin J, Kalman J, Fitzpatrick A, Epstein L, Lesh MD. Role of right atrial endocardial structures as barriers to conduction during human type I atrial flutter. Activation and entrainment mapping guided by intracardiac echocardiography. Circulation 1995;92:1839-48.
6. Boineau JP, Schuessler RB, Mooney CR, Miller CB, Wylds AC, Hudson RD, et al. Natural and evoked atrial flutter due to circus movement in dogs: role of abnormal atrial pathways, slow conduction, nonuniform refractory period distribution, and premature beats. Am J Cardiol 1980;45:1167-81.
7. Fontan F, Kirklin JW, Fernandez G, Costa F, Naftel DC, Tritto F, et al. Outcome after a "perfect" Fontan operation. Circulation 1990;81:1520-36.

12/8/71448

Reply to the Editor:

Our conclusion regarding the prevention of postoperative arrhythmias was based on clinical evidence, Holter electrocardiograms, and p trigger–signal averaged electrocardiograms (see Table V in the article). Those data all negatively supported the prevalence of atrial arrhythmias after the repair with our technique. However, we do not have other data that Dr. Gandhi and associates probably consider important factors in the development of atrial arrhythmias.

For the atrial incision, we made a longitudinal incision along the sulcus terminalis in the right atrial free wall, *at least 2 to 3 cm away from the crista terminalis,* as in the Senning operation. Thus we assume that the incision could not create conduction block in the vicinity of the crista terminalis and could not alter the longitudinal conduction along it. The superior incision of the atriotomy used in our technique never approaches the sinoatrial node, because it extends beneath the medial aspect of the superior vena cava. Therefore, injury to the sinoatrial node is always preventable.

Kazuhiro Hashimoto, MD
Hiromi Kurosawa, MD
Department of Cardiovascular Surgery
Jikei University School of Medicine
3-25-8, Nishishinbashi, Minatoku
Tokyo, Japan

12/8/71446

Intrapulmonary benign fibrous tumor of the pleura

To the Editor:

We read with great interest the communication of Aufiero and associates[1] in the August 1995 issue of the Journal concerning intrapulmonary benign fibrous tumor of the pleura.

We recently admitted to our institution a 38-year-old woman with a large mass in the left lung, which had been discovered accidentally on a chest roentgenogram ob-

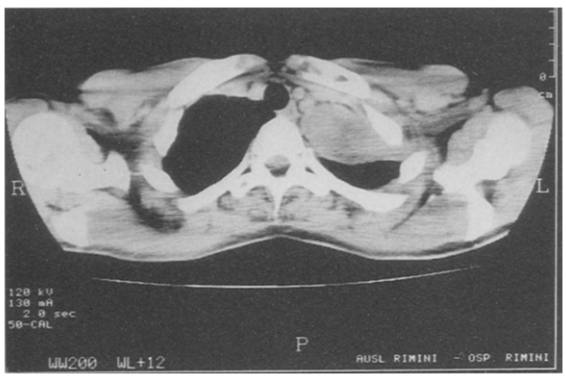

Fig. 1. Chest computed tomographic scan showing the extrapulmonary apical mass on the left side. *R,* Right; *P,* posterior; *L,* left.

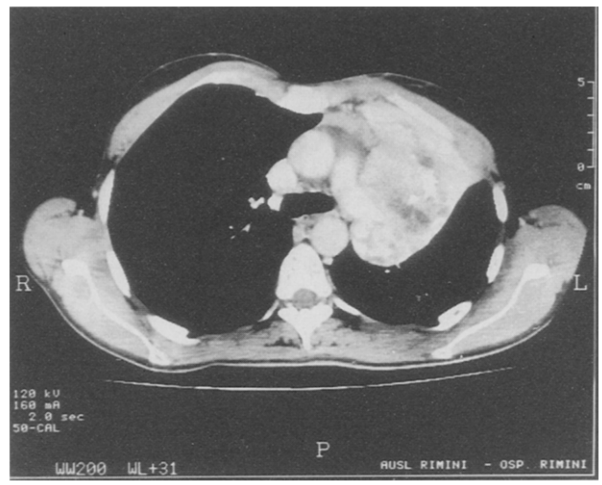

Fig. 2. Chest computed tomographic scan at a lower level revealing the tumor of the left upper lobe with involvement of the left pulmonary artery. *R,* Right; *P,* posterior; *L,* left.

tained for a motor vehicle accident. A chest computed tomographic scan revealed two distinct lesions, one related to the apical parietal pleura (Fig. 1) and the other involving diffusely the upper lobe of the left lung with compression of the left main pulmonary artery (Fig. 2). A review of her past roentgenograms showed the apical extrapulmonary lesion but no signs of parenchymal involvement at least 8 years before admission. A left pneumonectomy was necessary inasmuch as the left main pulmonary artery appeared involved by the tumor and its nature could not be determined by the intraoperative histologic examination. The mass arising from the apical parietal pleura, which did not involve the lung, was excised as well. Grossly, both lesions appeared circumscribed with a smooth surface, and a cut section revealed dense, whorled fibrous tissue. The intrapulmonary mass did not reach the visceral pleura. Histologic study of both tumors disclosed proliferation of spindle cells with a very low mitotic rate of less of 1 mitosis per 10 high-power fields and a scattered storiform pattern. The spindle cells were separated by abundant collagen. The pulmonary artery was found to be compressed but not infiltrated. Immunohistochemically, both tumors were positive for vimentin and negative for actin and cytokeratin. A diagnosis of benign fibrous tumor of the pleura was made. The specimens were sent for review to a pathologist at another institution, who confirmed the diagnosis.

Intrapulmonary benign fibrous tumors of the pleura have rarely been described,[1, 2] and none of the above was associated with a second distinct extrapulmonary lesion. Whether the multiple metachronous neoplasms in our case were due to the exposure to hypothetical predisposing risk factors creating a favorable environment for a multifocal growth, to the propagation by contiguity, or by blood-borne metastases, indicating an aggressive biologic behavior despite the benign histologic pattern, remain merely speculative.

The atypical localization (from the parietal pleura, intralobar, inverted growth into the parenchyma) and the large size, which have been associated more frequently with malignancy,[3] warrant an aggressive attitude toward this tumor, regardless of the histologic findings. Therefore, we recommend excisional biopsy and radical resection in all the cases of intrapulmonary fibrous tumors of the pleura, concurring with the conclusions of Aufiero and associates.[1]

Alessandro Brunelli, MD
Armando Sabbatini, MD
Giovanbattista Catalini, MD
Aroldo Fianchini, MD
Department of Thoracic Surgery
University of Ancona Medical School
Ancona, Italy

REFERENCES

1. Aufiero TX, McGary SA, Campbell DB, Philips PP. Intrapulmonary benign fibrous tumor of the pleura. J Thorac Cardiovasc Surg 1995;110:549-51.
2. Yousem SA, Flynn SD. Intrapulmonary localized fibrous tumor: intraparenchymal so-called localized fibrous mesothelioma. Am J Clin Pathol 1988;89:365-9.
3. England DM, Hochholzer L, McCarthy MJ. Localized benign and malignant fibrous tumors of the pleura: a clinico-pathologic review of 223 cases. Am J Surg Pathol 1989;13:640-58.

12/8/71725