mised by ischemia. In our patient, the stomach was probably herniated no more than eight to 12 hours prior to perforation.

Gastropleural fistula and resultant pneumothorax has previously been reported in traumatic diaphragmatic hernia.7,9,10 There have also been reports of tension pneumothorax due to colopleural fistula in a traumatic diaphragmatic hernia and due to gastropleural fistula in a congenital diaphragmatic hernia.7,9,10 To our knowledge, however, tension pneumothorax secondary to gastropleural fistula following a traumatic diaphragmatic hernia has not been described previously.

The present case emphasizes that a tension pneumothorax can develop in the setting of a traumatic diaphragmatic hernia and a gastropleural fistula. One might expect that it would be difficult for a tension pneumothorax to occur in this setting since, as the pressure in the pleural space rises, air should flow back from the pleura into the stomach. It is likely that the tissues surrounding the gastric perforation created a one-way valve that allowed air to enter the pleural space but prevented its return to the stomach.

Awareness of perforation of strangulated stomach or bowel in a diaphragmatic hernia as a cause of pneumothorax, with or without tension physiology, in a patient with a history of trauma is important so that surgical repair can be undertaken without delay.

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Magnetic Resonance Imaging—The Evaluation of Choice in Residual Shunt after Congenital Heart Disease Surgery?*

Accurate anatomic diagnosis presents a dilemma in patients with residual shunt after corrective surgery for congenital heart disease. We describe a patient who, after atrial septal defect repair, developed dyspnea and central cyanosis despite normal pulmonary arterial pressures and right heart chamber size. A role for early MRI is suggested.

(Chest 1991; 99:249-51)

MET = resting oxygen requirement

The evaluation of patients with recurrent symptoms after congenital heart surgery is frequently complex. Abnormalities related directly to the corrective surgery are frequently added to the residual of the original congenital heart disease. The case presented illustrates an expanded role for MRI in obtaining the high resolution needed to plan subsequent definitive corrective surgery.

CASE REPORT

A 21-year-old white woman was referred to our institution in March 1988, with a five-year history of progressive exertional dyspnea, central cyanosis, and clubbing. Corrective surgery for combined ostium secundum and sinus venosus atrial septal defects had been performed at the age of four years. Dyspnea and cyanosis increased significantly immediately after the patient delivered a premature infant in January 1987. The postpartum work-up included a hemoglobin level of 14.8 g/dl, Po2 of 62 mm Hg, and oxygen saturation of 91 percent. The Po2 on 100 percent oxygen was 82 mm Hg, documenting a right-to-left shunt. An ECG revealed right axis deviation and inverted T waves in the anteroseptal and inferior leads. Chest radiography demonstrated a normal cardiac silhouette with no features of pulmonary hypertension. A ventilation-perfusion scan revealed a V/Q mismatch in the left upper lobe, suggesting a pulmonary embolus or a hypoplastic left pulmonary artery. Normal right and left heart chamber dimensions were present on echocardiography. No residual atrial septal defect was detected. Saline solution contrast injection into the right basilic vein failed to reveal a right-to-left shunt. Oxygen desaturation (87 percent to 74 percent) and central cyanosis occurred at 5 METs during exercise oximetry. Cardiac catheterization in April 1987 demonstrated a low right atrial 3.3-volume percent oxygen step-up and a small net 1.8-L/min right-to-left shunt. There was a striking absence of pulmonary hypertension. A concomitant pulmonary arteriogram revealed an underdeveloped upper branch of the left pulmonary artery. These findings therefore suggested a bidirectional shunt. Physical examination revealed a slender young woman with a BP of 100/70 mm Hg, heart rate of 60 beats per min, a bigeminal rhythm, and mild cyanosis of the lips. Jugular venous and carotid

*From Loma Linda University Medical Center, Loma Linda, Calif. †Department of Cardiology. ‡Department of Internal Medicine. §Department of Radiology.
Reprint requests: Dr. Marais, Loma Linda University Medical Center, 11234 Andersen Street, Loma Linda, CA 92354

CHEST / 99 / 1 / JANUARY, 1991 249
pulses were normal. The chest was clear to auscultation, with a scar from prior thoracotomy. A prominent \( P_2 \) and a grade-2/6 systolic pulmonary outflow tract murmur were appreciated. No lifts or heaves were palpable. The abdomen was normal. Extremities revealed peripheral clubbing and cyanosis without pedal edema.

A tentative diagnosis of partially corrected atrial septal defect with residual shunt was considered. Central cyanosis, in the absence of pulmonary hypertension, suggested the possibility of residual atrial septal defect with a right-to-left streaming shunt, congenital or postsurgical diversion of systemic venous return to the left atrium, or a pulmonary arteriovenous malformation.

Repeat echocardiography at our institution confirmed normal chamber dimensions and wall thickness. Mild mitral valve prolapse, trace tricuspid and mitral regurgitation, and mild pulmonary stenosis with insufficiency were present. No atrial septal defect was detected on green dye injection into the right basilic vein.

Inferior vena caval contrast injection during cardiac catheterization demonstrated preferential opacification of the left atrium and proximal pulmonary veins (Fig 1). Repeated passage of the catheter tip from the inferior vena cava into the left atrium occurred during right cardiac catheterization. Pulmonary arteriovenous malformation was excluded by normal selective pulmonary venous oxygen saturations. Left atrial desaturation was present (oxygen saturation, 87 percent). Normal chamber pressures and vascular resistances were noted. A net 0.2 L/min right-to-left shunt was confirmed. An MRI scan (Magnetom 1 Tesla) was performed with ECG gating and spin-echo technique. The connection between the inferior vena cava and the left atrium was confirmed (Fig 2 and 3).

A residual atrial septal defect with diversion of the inferior vena cava to the left atrium was found at surgery. The septum had been sutured to the eustachian valve lateral rather than medial to the closely approximated orifice of the inferior vena cava. This anomaly was surgically corrected. This is an uncommon, probably underreported, surgical complication.

**Discussion**

The evaluation of residual shunt after corrected congenital heart disease presents the clinician with a complex problem. The diagnostic difficulties are related to the known or undisclosed features of the original abnormality, as well as

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**Figure 1.** Inferior vena caval angiogram. Inferior vena cava connects with both atria, as demonstrated by their simultaneous opacification. Note insertion of hepatic veins.

**Figure 2.** Axial MRI scan at level of right and left atria. Interatrial septum displays inverted Y configuration (arrow). This third chamber represents inferior vena cava sutured into previous atrial septal defect, communicating now with both atria.

**Figure 3.** Sagittal MRI scan at level of left atrium. Inferior vena cava (a) enters postero-inferior aspect of left atrium. Hepatic vein (b) joins inferior vena cava anteriorly.
to the results of corrective surgery. The presence of a right-to-left shunt and central cyanosis in a patient without pulmonary hypertension suggests systemic venous connection to the left atrium,\(^6\) an atrial septal defect with a streaming effect facilitating right-to-left shunt,\(^4\) or a pulmonary arteriovenous malformation. Echocardiography demonstrated many important features of this patient's problem (normal right heart chamber sizes and pulmonary arterial pressures), but failed to define the connection of the inferior vena cava to the left atrium. Pulmonary venous malformation was ruled out by pulmonary arteriography and normal pulmonary vein oxygen saturations. The MRI confirmed the connection of the inferior vena cava to the left atrium demonstrated on inferior vena caval contrast injection.

Magnetic resonance imaging has many advantages in the initial evaluation of congenital heart disease.\(^5,6\) Multiple imaging planes are available which are not routinely accessible by conventional diagnostic modalities. The MRI, in contrast to echocardiography, is independent of the patient's body habitus and chest wall anatomy. It is essentially operator-independent. Excellent images of the mediastinal anatomy and vascular connections to the heart are obtained. The ECG gating avoids image degradation by motion artifact. The flow-void effect on spin-echo T\(_2\) weighted sequences differentiate cardiac chambers (signal) and great vessels (signal void). Blood flow produces intense signal on gradient refocused echoes. Mediastinal fat and fluid-containing structures are identified by intense signal on T\(_1\) vs T\(_2\) weighted sequences, respectively.\(^7\) The role of MRI in congenital heart disease continues to evolve. Current indications include assessment of pulmonary artery agenesis, evaluation of the aorta in coarctation, unravelling of complex anomalies such as tetralogy of Fallot, transposition of the great vessels, single ventricle, and corrected transposition. Disadvantages include the need for ECG gated acquisition imaging, a relatively long acquisition time, difficulties in imaging patients with pacemakers, and expense. The MRI in this patient accurately demonstrated the relationship between the hepatic vein, inferior vena cava, and the atria—clearly revealing the diversion from the inferior vena cava to the left atrium.

We suggest the early use of MRI in patients with undiagnosed residual abnormalities after congenital heart surgery. The excellent resolution of the relationship between the great vessels and the heart adds significantly to the data obtained from echocardiography and cardiac catheterization.

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**Pneumocystis Carinii Pneumonia**

**Rare Cause of Hemoptysis**

D. A. Neville Mascarenhas, M.B.B.S., M.D.;†
Vinamath P. Vasudevan, M.D., F.C.C.P.A
and Kamini P. Vaidya, M.D.‡

**Pneumocystis carinii** pneumonia is a frequent manifestation of the acquired immunodeficiency syndrome (AIDS). It commonly presents with nonproductive cough, fever, and dyspnea. We report this case of *P. carinii* pneumonia presenting with hemoptysis, since to the best of our knowledge, hemoptysis has not been reported to be a presenting manifestation of *P. carinii* pneumonia. Autopsy revealed multiple lung cavities. ([CHEST](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21623/))

| BAL = bronchoalveolar lavage; TMP = trimethoprim; SMX = sulfamethoxazole; INH = isoniazid; PZA = pyrazinamide |

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*From the Departments of Medicine, Pulmonary Medicine, and Pathology, Woodhull Medical and Mental Health Center, Brooklyn, NY.
†Resident, Department of Medicine.
‡Assistant Clinical Professor of Medicine.
§Attending Pathologist.

The most commonly recognized pulmonary pathogen in patients with acquired immunodeficiency syndrome (AIDS) is *Pneumocystis carinii*, which causes pneumonia at least once in 50 to 90 percent of all patients. The clinical

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