different types of QRS complexes which occurred in this symptomatic patient with Wolff-Parkinson-White syndrome type A complicated by complete, or advanced, A-V block in both pathways.

REFERENCES


Transient Unilateral Hypoperfusion of the Lung following Mediastinoscopy*

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Two cases of pulmonary hypoperfusion occurring after mediastinoscopic examination were demonstrated on lung scans. In one case, this finding on the ninth day required a pulmonary angiographic study that yielded normal findings. Repeat lung scans were normal. We propose that localized mediastinal edema or bleeding after mediastinoscopic examination can produce defects of hypoperfusion, and we urge caution in the interpretation of lung scans up to nine days after mediastinoscopic examination.

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The views expressed herein are those of the authors and do not necessarily reflect the views of the US Navy or the Department of Defense.

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CHEST, 73: 2, FEBRUARY, 1978

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ilateral pulmonary hypoperfusion has been attributed to various causes, including bronchial obstruction by carcinoma, adenoma, endotracheal tube, or foreign body, the Swyer-James syndrome, unilateral absence of pulmonary artery, and pulmonary embolus.1-4 This report describes two instances of transient unilateral hypoperfusion of the lung following mediastinoscopic examination. These instances of transient hypoperfusion did not appear to be due to any of the several recognized causes of regional hypoperfusion.

CASE REPORTS

Case 1

An asymptomatic 19-year-old man was referred for evaluation of left hilar adenopathy. He was a nonsmoker. Between 1968 and 1972, the patient had worked on a farm. He denied any other occupational exposures.

The findings from physical examination were normal. The results of routine laboratory studies, including a complete blood cell count and a tuberculin skin test, were normal. The intradermal cutaneous test with histoplasmin produced 20 mm of induration at 48 hours. Serum fungal serologic studies revealed complement-fixation titers of 1:16 to the yeast form and of 1:8 to the mycelial form of Histoplasma. Findings from fiberoptic bronchoscopic examination were normal. Stains and cultures of bronchial washings for bacteria, fungi, and Mycobacterium tuberculosis were negative.

At mediastinoscopic examination, several slightly enlarged lymph nodes were removed from the left hilum. The pathologic examination revealed caseating granuloma; no fungal or acid-fast organisms were seen. A lung scan was done on the ninth postoperative day to evaluate possible effects of the hilar mass on pulmonary perfusion. The lung scan, using macroaggregated albumin labelled with radioactive 99mtechnetium, showed markedly decreased perfusion of the entire left lung (Fig 1). This was initially attributed to mechanical constriction of the left pulmonary artery by the hilar adenopathy. A selective pulmonary angiogram obtained on the 22nd postoperative day showed symmetric perfusion and no evidence of vascular compression. The pulmonary arterial pressure was 22/8 mm Hg, with a mean wedge pressure of 14 mm Hg. A lung scan performed on the 23rd postoperative day was normal (Fig 2). A chest x-ray film and the results of repeat fungal serologic studies were unchanged three months after discharge.

Case 2

A 22-year-old woman initially had a dry cough and recurrent low-grade fevers for one month. The findings from physical examination and routine laboratory data were normal. The tuberculin and histoplasmin skin tests were negative at 48 hours. The results of serum fungal serologic studies were unremarkable. The chest x-ray film showed bilateral hilar and right paratracheal adenopathy. The findings from studies of pulmonary function and from fiberoptic bronchoscopic examination were normal. Stains and cultures of washings with saline solution were negative for bacteria, mycobacteria, and fungi. A premediastinoscopic lung scan with 99mtechnetium-labelled macroaggregated albumin was normal (Fig 3).

At mediastinoscopic examination, several nodes at the level of the right main-stem bronchus were excised. Pathologic examination revealed noncaseating granuloma. Stains
for fungal and acid-fast organisms were negative. On the next day a ventilation scan using radioactive xenon was normal, but a repeat pulmonary perfusion scan demonstrated hypoperfusion of the left upper pulmonary field (Fig 4). A repeat pulmonary perfusion scan obtained 48 hours after mediastinoscopic examination showed equal perfusion of all pulmonary segments. In all three studies the 99mtechnetium-labelled macroaggregated albumin was injected with the patient lying supine. The patient made an uneventful recovery.

**DISCUSSION**

Abnormalities of perfusion on a lung scan represent a nonspecific finding; they may reflect primary vascular disease (vasculitis or pulmonary emboli) or a primary abnormality in ventilation (bronchial obstruction), or a combination of both. Regional hypoventilation secondary to bronchial constriction or mucous plugging is often associated with decreased perfusion. 

Hypoperfusion of a whole lung has been reported after occlusion of main-stem bronchi, intraluminal obstruction, or extraluminal constriction of the major pulmonary vessels. Small hilar bronchogenic neoplasms may produce ipsilateral hypoperfusion far exceeding their ability to obstruct mechanically.

In our case 1, hypoperfusion of the left lung was present nine days after several lymph nodes were removed by a mediastinoscopic procedure. Although no lung scan had been performed before the mediastinoscopic procedure, we believe that, in view of the patient's uneventful recovery, the study on the 23rd postoperative day represented his normal (and preoperative) status regarding pulmonary blood flow. In case 2, normal premediastinoscopic and normal follow-up (48-hour) scans of perfusion adequately established the transient nature of the hyperperfusion of the left lung.

In neither case was vascular compression by hilar adenopathy a likely explanation for the abnormality of perfusion; only small lymph nodes were noted at the time of mediastinoscopic examination, serial chest x-ray films showed no change in the hilar adenopathy, and the angiogram in case 1 showed no extrinsic compres-
sion of the pulmonary artery. Neither patient manifested any symptoms or signs of pulmonary embolism, particularly the symptoms and pathophysiologic changes that might commonly attend the abrupt hypoperfusion of a whole lung. The resolution of the hypoperfusion of the left lung in case 2 was, moreover, considerably more rapid than reported for pulmonary embolism.14-18

Unilateral or regional hypoventilation was most unlikely as the cause of the transient hypoperfusion in these patients, since both had normal findings on bronchoscopic examination, and neither had any interval history, or findings of ventilatory abnormality (eg, inhalation of a foreign body, asthma, bronchitis with heavy secretions). Patient 2 was, in fact, shown to have a normal ventilation scan in conjunction with the abnormal perfusion study.

By exclusion, then, we are led to conclude that the transient regional hypoperfusion observed in these two patients is related in some way to the mediastinoscopic examination which preceded these episodes. The clinical information available provides no guidance as to the likely mechanism. It is conceivable that localized postmediastinoscopic edema or hematoma could cause vascular compression in a low-pressure system such as the pulmonary arterial bed. Bleeding might by itself cause reflex vasoconstriction. Additional studies of patients or experiments in animals may be necessary to elucidate this observation.

We report these two cases primarily for two reasons. First, the possibility that transient abnormalities of pulmonary perfusion after mediastinoscopic examination may not be due to pulmonary embolism should be borne in mind by clinicians called upon to interpret lung scans in the early postmediastinoscopic period. Since the triad of abnormal scan of perfusion, a normal scan of ventilation (as in case 2), and an unchanged chest x-ray film currently represents (short of pulmonary angiographic studies), the most specific indication of acute pulmonary embolism,19 alternatives to the diagnosis of pulmonary embolism may not even receive consideration. Second, mediastinoscopic examination is frequently followed within a few days by thoracotomy and pulmonary resection of variable magnitude. Should such an operation be performed during the time when the patient is subject to significant mediastinoscopically induced hypoperfusion of the opposite lung, the patient may be subject to unsuspected increased operative and postoperative morbidity. In this respect, case 1 demonstrates that the degree of regional hypoperfusion can be quite significant, while case 2 illustrates that the postmediastinoscopic abnormality of perfusion may be contralateral to the side of the nodal biopsy and, therefore, potentially contralateral to the pulmonary lesion requiring pulmonary resection.

REFERENCES
Silent Unilateral Pulmonary Venous Obstruction

Occurrence after Surgical Correction of Transposition of the Great Arteries

James E. Lock, M.D.; Russell V. Lucas, Jr., M.D.; Kurt Amplatz, M.D.; and F. Blanton Bessinger, Jr., M.D.

An 11-year-old girl was found to have completely obstructed left pulmonary veins eight years following corrective surgery for transposition of the great arteries. The patient was acyanotic and asymptomatic. Retrograde flow of arterial blood from the affected left lung accounted for an angiographic appearance that mimicked occlusion of the left pulmonary artery and resulted from a failure of systemic venous development. Pulmonary venous anatomy could only be demonstrated by pulmonary arterial wedge angiographic studies. This experience emphasizes that complete unilateral pulmonary venous obstruction may occur in an asymptomatic patient and underlines the importance of investigating pulmonary venous anatomy in any patient with gross inequality of the distribution of pulmonary blood flow.

Unilateral pulmonary venous obstruction in childhood is usually congenital in origin. The advent of Mustard’s operation for correction of transposition of the great arteries has created a new class of patients who may develop unilateral pulmonary venous obstruction. Pulmonary infiltrates, hemoptysis, and pulmonary hypertension are generally present in both congenital and acquired cases.1,2

We are reporting the findings in a child who developed complete left-sided pulmonary venous obstruction following Mustard’s operation. She has the unusual features of a symptom-free course and retrograde flow of oxygenated blood from the affected lung. Utilizing the hemodynamic and angiographic data, we have speculated on possible physiologic mechanisms and have discussed the practical implications for other children undergoing Mustard’s operation.

CASE REPORT

An 11-year-old girl had had cyanosis in infancy; when she was two years of age, signs of congestive heart failure were noted. The diagnosis of d-transposition of the great arteries with an atrial septal defect, an intact ventricular septum, and modest elevation of the left ventricular pressure (50/0-8 mm Hg), was made. An angiogram demonstrated antegrade flow to both left and right pulmonary arteries (Fig 1A).

At the age of 30 months, the patient underwent Mustard’s operation, and after surgery, she was acyanotic and asymptomatic. When she was six years of age, a routine catheter-