Physical examination revealed the following: blood pressure 120/78 mm Hg; pulse, 72 and regular; heart, normal; and mild wheezing in the lung. Results from ECG, chest radiograph, blood gases, and routine blood tests were within normal limits. Doppler echocardiography showed a normal heart. Ergometry induced NSVT in the second minute of the Bruce protocol.

Cardiac angiography revealed normal coronary arteries and normal left ventricular function. Electrophysiologic study was planned but the patient refused further investigation.

**DISCUSSION**

The use of coughing as a tool for conversion of VT has been known for many years. It has been suggested that coughing could help as a form of “cardiac massage,” due to the intrathoracic pressure it induces.

We et al. suggest there are several advantages to cough-induced cardiac compression over external massage: the procedure is simple and can be self-induced; it does not lead to traumatic complications or possible damage to ribs or sternum; and it can be carried out anywhere and in any position.

Sakai and Mori. reported a case of “schlucktachycardia” in 1926. Omari et al. were the first to report a case of cough-induced tachycardia. To the best of our knowledge, this is the first report of NSVT precipitated by cough.

While coughing, the intrathoracic pressure becomes elevated and can increase to 450 mm Hg. The linear air velocity is 50 to 120 m/s and can rise as high as 280 m/s, approaching the speed of sound. The amount of air expired during a cough ranges normally from 1 to 3 L. From these values, one can estimate the amount of kinetic energy generated as approximately 1 to 25 J. Thus, strictly on the basis of energy considerations, it may be feasible for cough to produce sufficient mechanical energy to cause cardiac depolarization. We found no common pathophysiologic mechanism to convincingly explain the precipitation of NSVT during cough and exercise. However, it is possible that increased air linear velocity in both exercise and coughing could stimulate the larynx, causing reflex NSVT.

**REFERENCES**


**Pediatric Lung Transplantation for Graft- Versus-Host Disease Following Bone Marrow Transplantation**

Steven R. Boas, M.D.; Blakeslee E. Noyes, M.D.; Geoffrey Kurland, M.D.; John Armitage, M.D.; and David Orenstein, M.D.

Nine years after receiving a bone marrow transplant for aplastic anemia, a 14-year-old girl with severe pulmonary disease associated with graft-versus-host disease received a double lung transplant. Subsequent to lung transplant, her lung function improved dramatically (FEV₁ increasing from 20 to 73 percent predicted normal, residual volume decreasing from 316 to 130 percent predicted normal values). The patient is currently well 15 months after transplant, while receiving immunosuppression consisting of FK506 and azathioprine. Double lung transplantation may offer a therapeutic option for the treatment of graft-versus-host pulmonary disease in selected patients.

(Chest 1994; 105:1584-86)

| BMT=bone marrow transplantation; GVHD=graft-versus-host disease |

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Lung transplantation has become an accepted form of therapy for end-stage pulmonary disease in adults and children. Pediatric patients have undergone lung transplantation for a variety of diseases, including cystic fibrosis, idiopathic pulmonary fibrosis, primary pulmonary hypertension, and congenital heart disease.

Bone marrow transplantation (BMT) has been used for a variety of hematologic disorders, including aplastic anemia, acute and chronic leukemias, and immunodeficiencies. Graft-versus-host disease (GVHD) is a complication of BMT in 40 to 60 percent of transplant recipients. Pulmonary interstitial fibrosis has been found in 20 percent of patients within 12 months after BMT. Airway obstruction, especially obliterative bronchiolitis, has been reported in 11 to 17 percent of BMT patients, with chronic GVHD preceding almost all of the cases reported. This pulmonary involvement by GVHD carries a mortality rate of approximately 50 percent. Medical therapy using increased immunosuppression with corticosteroids, antithymocyte globulin, nonspecific γ-globulin, cyclosporine, or FK506 offers promise, but also is associated with substantial toxic reactions and treatment failure. Therefore, alternative therapies for severe pulmonary disease following GVHD are needed. Calhoun et al reported successful single lung transplantation in an adult with pulmonary fibrosis after BMT. We now report lung transplantation as treatment in a child with severe pulmonary GVHD.

CASE REPORT

A 14-year-old white girl was initially referred to our institution for evaluation for lung transplantation in 1990. Eight years before, she had been diagnosed as having aplastic anemia and received an HLA identical, mixed lymphocyte culture compatible BMT from her sister. Prior to BMT, she received a combination of oral thio guanine and intravenous cytarabine and cyclophosphamide as immunosuppression. Complete engraftment took place by the 14th day after BMT, and long-term immunosuppression was continued with oral azathioprine. Acute GVHD developed 2 weeks after BMT and she was treated with a course of methylprednisolone. Her clinical course was complicated by adenoviral pneumonia 2 months after BMT with the development of respiratory failure requiring mechanical ventilation and tracheostomy 4 months after BMT. Her subsequent course was further complicated by pneumococcal and staphylococcal sepsis treated successfully with intravenous antibiotics.

Over the next 3 years, she developed dyspnea, orthopnea, wheezing, peripheral cyanosis, and exercise intolerance. Pulmonary function testing 3½ years after BMT showed an FVC less than 30 percent of predicted and an oxyhemoglobin saturation of 90 percent during exercise (Table 1). Pulmonary function testing 3 months later indicated a mixed restrictive and obstructive defect. Her chest radiographs showed increased interstitial markings and an echocardiogram 9 years after BMT was consistent with pulmonary hypertension. She was treated with nitroglycerin and supplemental oxygen. Despite these measures, she remained debilitated with chronic cough and exercise limitation.

At the time of referral to our center for lung transplant evaluation, the physical examination revealed a well-nourished girl with moderate dyspnea and a respiratory rate of 36 breaths/min. The thorax had a normal anteroposterior diameter and there were intercostal retractions. Inspiratory and expiratory crackles and an occasional wheeze were heard on auscultation. Cardiac examination revealed a normal S1 and a prominent split of S2. There was 2+ clubbing of the digits. A complete blood cell count was normal. Echocardiography demonstrated a mildly dilated right ventricle without tricuspid regurgitation, a moderately dilated pulmonary artery, and early closure of the pulmonary valve consistent with pulmonary hypertension. The right ventricular ejection fraction by a multiple gated acquisition scan was 35 percent. Chest radiograph revealed a right upper lobe bulla as well as emphysematous changes and increased interstitial markings consistent with fibrosis. Pulmonary function testing showed both restriction and very severe obstruction.

She underwent double lung transplant 15 months after the initial evaluation. Pathologic study of her native lungs revealed diffuse interstitial and focal parenchymal fibrosis (with compensatory emphysema) as well as bronchiolitis obliterans, consistent with GVHD of the lung.

Following the transplant, her immunosuppressive regimen included FK506, azathioprine, and prednisone. Episodes of acute cellular rejection were diagnosed by transbronchial biopsy specimens on the ninth and 21st postoperative days. Both of these episodes were treated successfully with 3-day courses of methylprednisolone. She was discharged from the hospital 1 month after transplant on a regimen of FK506, azathioprine, prednisone, acyclovir, and trimethoprim-sulfamethoxazole.

The patient has done well in the 15 months since hospital discharge and is currently in school without limitation. Results of pulmonary function tests performed 15 months after transplant (Table 1) were virtually normal.

DISCUSSION

Within the past decade, lung transplantation has become a therapeutic option for the treatment of end-stage pul-

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Table 1—Pulmonary Function Before and After Lung Transplantation

<table>
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<th>Date</th>
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<th>12/89</th>
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</table>

DL-transplant

*Pulmonary function tests from patient. Bone marrow transplantation was performed in February 1982 and double lung (DL) transplantation was performed in August 1981. FEV₁=forced expiratory volume in 1 s (percent predicted); MMEFR=maximal mid expiratory flow rate; TLC=total lung capacity; VC=vital capacity; Dco=diffusing capacity components; SaO₂=oxygen saturation; NA=not available.
monary disease in adults and children. Pulmonary GVHD following BMT has been a common and serious problem, often with an inexorable downhill course, despite dramatic medical treatment.

Calhoon et al recently reported the case of an adult who received a single lung transplant because of severe pulmonary fibrosis following allogeneic BMT. We present a patient who received a BMT for aplastic anemia and then developed a decline in her pulmonary function over the ensuing 4 years. She eventually was left with severe restrictive and obstructive pulmonary disease with a long-term oxygen requirement and exercise intolerance. She has done well in her 15 months following double lung transplantation.

We believe that this case demonstrates the potential efficacy of lung transplantation in children for the treatment of severe pulmonary disease associated with chronic GVHD. Double lung transplantation may offer a therapeutic option for the treatment of GVHD.

REFERENCES

Heimlich Valve Treatment and Outpatient Management of Bilateral Metastatic Pneumothorax*

Peter Van Hengel, M.D.; and Jan H. A.M. Van de Bergh, M.D.

A 51-year-old man was treated for bilateral pneumothorax secondary to pulmonary metastases from malignant fibrous histiocytoma. After failure of regular therapy, Heimlich flutter valves were used producing disappearance of the bilateral pneumothorax and adequate outpatient management. (Chest 1994; 105:1556-87)

Spontaneous bilateral pneumothorax complicating metastatic sarcoma has been reported before, especially in children. In case of recurrence of the pneumothorax, chest tube drainage alone most often fails. Bleomycin or tetracycline pleurodesis seems to be more successful. The chest tube with one-way valve described by Heimlich provides a good alternative to standard chest tube drainage and suction.

We describe a patient in whom bilateral pneumothorax due to metastatic malignant fibrous histiocytoma was effectively treated with the Heimlich flutter valve after failure of repeated attempts to achieve chemical pleurodesis.

CASE REPORT

A 51-year-old man suffered from a malignant fibrous histiocytoma of the left thigh with pulmonary metastases. Just before chemotherapy (epidoxorubicin) was to have been started, the patient was examined elsewhere for a pneumothorax on the left side. Chest tube drainage and tetracycline pleurodesis resulted in complete expansion of the lung. Three weeks thereafter, shortly after the first dose of chemotherapy, the pneumothorax relapsed and again drainage and tetracycline pleurodesis followed.

Four days later, the patient was admitted to our hospital with complaints of shortness of breath. The chest x-ray film revealed bilateral pneumothorax (Fig 1). A computed tomographic scan showed, in addition to one large hilar mass on the right, multiple small metastases and bullous changes especially near the pleura on both sides. Bilateral chest tubes were inserted and connected to suction. Because of the poor reaction to tetracycline that ensued, bleomycin pleurodesis was carried out after 4 (right side) and 9 (left side) days of drainage. Unfortunately, again pneu-

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