An oral glucose tolerance test was repeated in June, 1982 and again reproduced symptoms during the fourth hour. Table 1 shows hormonal evaluation results done during the test. A 24-hour Holter monitor covering the period of OGTT did not show a significant change in rhythm, rate or ST segments. Modification of dietary intake and reassurance have been associated with a marked reduction in anxiety and chest pain frequency, with only one episode of chest pain in the last six months.

**Discussion**

Postprandial or "reactive" hypoglycemia has been long recognized as an early manifestation of non-insulin-dependent diabetes mellitus. These patients have normal fasting blood sugar levels, but exaggerated rise in plasma glucose levels with oral glucose loading due to inadequate insulin release. Late hyperinsulinemia due to hyperglycemia early in the test results in hypoglycemia, usually at three to five hours. Reactive hypoglycemia typically results in symptoms of sympathetic-adrenal discharge including palpitations, anxiety, restlessness, hunger, diaphoresis and tremulousness, but the neuroglycopenic symptoms of cerebral origin are usually not present. Our patient clearly met the criteria for diagnosis of reactive hypoglycemia with blood sugar levels falling to nadir values of 40 mg/dl and 27 mg/dl at four hours during two glucose tolerance tests. This hypoglycemia was associated with significant chest discomfort, palpitations, diaphoresis and fatigue similar to the patient's usual symptoms.

The presence of significant risk factors, character of the chest pain with associated symptoms, and apparent relief with nitroglycerin were all suggestive of an ischemic etiology and resulted in extensive evaluation. The absence of ST-segment change on the electrocardiogram with pain, perfusion defects with thallium scan and normal epicardial coronary arteries all lessen the likelihood of ischemia as an etiology for this patient's chest discomfort. Based upon the work of other authors, it is reasonable to presume that the hypoglycemia is accompanied by elevated catecholamine levels. We speculate that in view of the strong family history of chest pain and marked anxiety, the patient manifested similar somatic symptoms in response to catecholamine stimulation.

Although the mechanism of chest pain in this patient is not fully delineated, it clearly correlates with hypoglycemia and has responded to treatment. Thus, patients with atypical chest pain patterns, particularly when associated with diaphoresis, anxiety, and palpitations, may have reactive hypoglycemia which is a potentially treatable etiology.

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**References**


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**Successful Repair of a Transected Phrenic Nerve with Restoration of Diaphragmatic Function**

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This report describes the feasibility of restoring motor function of the diaphragm by early repair of a transected phrenic nerve, and discusses causes and consequences of phrenic nerve damage.

Phrenic nerve damage is the most frequent cause of permanent or temporary paralysis of the diaphragm. Paralysis of the diaphragm is a serious condition, especially in infants and children in whom it causes atelectasis and hypoventilation, necessitating prolonged ventilation with its inherent complications. In adults, vigorous bronchial toilet and intensive respiratory therapy usually overcome the immediate ill effect of diaphragmatic paralysis, leaving the patient with impaired pulmonary function.

Spontaneous recovery of phrenic nerve conduction may occur after several months if the continuity of the nerve is maintained. If the nerve is transected or otherwise irreversibly damaged, the hemidiaphragm becomes permanently paralyzed and, as its muscle atrophies, it acquires a progressively higher position due to the pressure differential between the peritoneal and pleural spaces. In an attempt to restore normal diaphragmatic tone and function, we recently repaired a transected phrenic nerve with a good clinical result.

**Case Report**

A 20-year-old man presented at the North Central Bronx Hospital with a stab wound of the anterior left chest. His pulse was not palpable and blood pressure unobtainable. An anterior thoracotomy was performed in the emergency ward. The pericardium, which was filled with blood, was opened transversely and a hole in the right ventricle was repaired. It was noted that the left phrenic nerve had been transected. The patient recovered uneventfully except for persistent collapse of the left lower lobe despite bronchopulmonary toilet and inhalation therapy.

Thirteen days after his injury, on Dec 12, 1979, the patient was transferred to Montefiore Medical Center for repair of the phrenic nerve. The thoracotomy incision was reopened, and the left lower lobe was found collapsed. Both ends of the transected phrenic nerve were identified and freed of adhesions. Dissection using the Zeiss operating microscope revealed a proximal neuroma that was discontinuous with a distal gloma, both of which were embedded in scar tissue. The two ends were freed of surrounding adhesions and resected until pouting, healthy fascicles were demonstrated. This resulted in a 3 cm nerve gap. Primary repair was rejected, as this would have placed the suture line under tension, thereby lessening the chances of success. A sural nerve graft was harvested from the left leg and laid into place to bridge the created defect. Microsurgical neurorrhaphy was accomplished under high magnification by incor-
FIGURE 1. Expiratory and inspiratory chest roentgenograms 45 days following phrenic nerve repair. The left hemidiaphragm descends minimally in relationship to the 8th rib (marked).

Follow-up radiographic/fluoroscopic examinations revealed some return of diaphragmatic function six months after repair. Thirteen months after repair, there was significant improvement in diaphragmatic motion (Fig 2).

COMMENT

Most cases of acquired phrenic nerve paralysis are iatrogenic. The nerve is most frequently damaged during a variety of thoracic and cardiac operations.1 Most often, only one nerve is damaged, but cases of bilateral phrenic nerve paralysis have been reported.

There are isolated case reports of phrenic nerve damage porating the graft to the proximal phrenic nerve end using three 9-0 nylon epineural sutures, and to the distal end using four sutures in similar fashion. Following restoration of neural continuity, both suture lines were tension-free. The reconstituted phrenic nerve was placed in a bed of local areolar tissue of fine vascularity to provide an optimal milieu for regeneration.

After reexpanding the collapsed lower lobe with positive pressure ventilation, the chest was closed. Mechanical ventilation was continued overnight to help maintain aeration of the left lower lobe. After extubation, the patient was treated vigorously with intermittent positive pressure breathing. He was discharged two weeks later. Chest roentgenograms in inspiration and expiration revealed no motion of the left hemidiaphragm (Fig 1).

FIGURE 2. Expiratory and inspiratory chest roentgenograms 13 months following phrenic nerve repair. There is a distinct increase in motion of the left hemidiaphragm. Motion of the lateral portion is impaired by adhesions between the diaphragm and the chest wall.
following percutaneous punctures of the subclavian\textsuperscript{4} and internal jugular veins,\textsuperscript{5} following intercostal tube drainage\textsuperscript{6} and after penetrating missile wounds of the chest.\textsuperscript{7}

Paralysis of both hemidiaphragms results in reduction of vital capacity, residual volume and total lung capacity which lead to hypoxemia and hypercapnia.\textsuperscript{8} Unilateral diaphragmatic paralysis may be entirely asymptomatic in adults. Studies of regional lung function, however, have demonstrated a reduction in perfusion and ventilation of 19 percent and 20 percent respectively on the affected side. When the diaphragm is paralyzed intraoperatively, atelectasis of the lower lobe is a frequent complication.

Diaphragmatic paralysis in infants is a far more serious problem, often necessitating prolonged mechanical ventilation of the patient.\textsuperscript{9} Many infants cannot be weaned from the ventilator until diaphragmatic plication\textsuperscript{10,11} or even prosthetic replacement\textsuperscript{12} is performed.

Diaphragmatic paralysis should be suspected in any patient who develops ventilatory insufficiency with or without atelectasis following an intrathoracic operation. The diagnosis can be confirmed radiographically by comparing the levels of both hemidiaphragms in inspiration and expiration on standard chest roentgenograms or during fluoroscopy. The paralyzed hemidiaphragm will not move down in inspiration and often will move up in a paradoxical fashion. These changes obviously cannot be demonstrated during mechanical ventilation, and it is therefore important to obtain the roentgenograms during spontaneous breathing. The most sensitive radiologic test is the "sniff" test, in which the paralyzed diaphragm is observed fluoroscopically to move upward during rapid sniffing.

Invasive methods used in establishing the diagnosis of diaphragmatic paralysis include measurement of phrenic nerve conduction,\textsuperscript{13} transdiaphragmatic pressure differential\textsuperscript{14} and contrast peritoneography.\textsuperscript{15}

The treatment of diaphragmatic paralysis will vary according to the patient's age, the degree of ventilatory impairment and the nature of phrenic nerve injury. In those cases in which phrenic nerve continuity is maintained and spontaneous recovery is expected, prolonged ventilatory support may be indicated until diaphragmatic function returns.\textsuperscript{16} A more aggressive approach of early plication of the paralyzed diaphragm has been recommended in infants who are respiratory-dependent.\textsuperscript{17}

Results of nerve repair have been improved by advances in microsurgery. By reestablishing the continuity of a nerve, either directly or with a nerve graft, a route is provided for the regenerating axons which leads to reinnervation of the end organ. The nerve regenerates 1 mm a day\textsuperscript{18} and restoration of motor function can be expected in approximately 75 percent of the cases.\textsuperscript{19}

Repair of a traumatized phrenic nerve is a logical procedure which, to our knowledge, has not been reported prior to this case. Based on our good result, it can be recommended in appropriate cases. When direct repair of the phrenic nerve is not possible, a segment of intercostal nerve can serve as an easily accessible free graft.

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REFERENCES


Recognition and Diagnosis of Apical Hypertrophic Cardiomyopathy*

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Recognition and diagnosis of nonobstructive apical hypertrophic cardiomyopathy is important to begin to understand the natural history and prognosis of such patients. Our experience with three patients indicates that a clue to the recognition of apical hypertrophic cardiomyopathy lies in the striking electrocardiographic repolarization changes consistent with subendocardial ischemia often prompting admission to the coronary care unit. The diagnosis of apical hypertrophic cardiomyopathy in two patients was confirmed by two-dimensional echocardiographic apical views, but due to a technically inadequate echocardiogram, the

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