Time Course of Recovery from Frostbitten Phrenics after Coronary Artery Bypass Graft Surgery

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Bilateral diaphragmatic paralysis developed in a patient after coronary artery bypass graft surgery during which cold cardioplegia was used. The patient’s progress and eventual recovery over an 18-month period is described, with particular reference to chest wall motion and respiratory pressure measurements. (Chest 1991; 99:1112-15)

Frostbitten phrenics,” a term used to describe bilateral diaphragmatic paralysis after cold cardioplegia for cardiac surgery, was coined by Scannell in 1963. Bilateral diaphragmatic paralysis as a result of cold cardioplegia is uncommon and, if not recognized, may result in prolonged mechanical ventilation, repeated respiratory arrest, and even death. The bedside observation of paradoxic (out-of-phase) motion of the rib cage and abdomen suggests severe weakness, paralysis, or dysfunction of the diaphragm and other inspiratory muscles. We used chest wall motion as a means of describing the time course of recovery from frostbitten phrenics. In addition, we present serial follow-up of other pulmonary function tests in a patient who appeared to have complete recovery over an 18-month period.

CASE REPORT

A 56-year-old white man was seen in consultation for evaluation of severe orthopnea 16 days after he had undergone coronary artery bypass grafting. Since his operation, he had noticed shortness of breath when lying supine. He tolerated lying on his side for longer periods than lying supine. He denied cough or paroxysmal nocturnal dyspnea. His exercise tolerance had improved since the operation because of absence of angina, and he had been able to walk an average of two miles daily during the week preceding his follow-up evaluation.

Examination revealed marked orthopnea, rib cage-abdominal paradox during quiet breathing, and decreased breath sounds at both lung bases. Cardiac examination revealed the healing median sternotomy. Chest roentgenograms showed “poor inspiratory effort and atelectasis at both lung bases” (Fig 1). Results of pulmonary function tests included the following values: TLC, 2.79 L (48 percent of predicted); VC, 1.52 L (40 percent of predicted) (Fig 2); FEV1, 1.33 L (44 percent of predicted); and D (65 percent of predicted), single-breath carbon monoxide diffusing capacity. Pulmonary function tests had not been performed before surgery. Fluoroscopic examination showed minimal movement of both hemidiaphragms during deep inspiration. Peak inspiratory pressure was -32 cm H2O (29 percent of predicted), and Pmax was +112 cm H2O (54 percent of predicted) (Fig 3).

Rib cage and abdominal motions during breathing were assessed by using an inductive plethysmograph (Respitrace) with transducer bands applied to the lower rib cage and mid-abdomen. Konno-Mead diagrams of relative motion of the rib cage and abdomen were then generated in the sitting and supine positions. On the 16th postoperative day, the sitting chest wall motion was normal, but abdominal paradox was marked in the supine position (Fig 4). A presumptive diagnosis of bilateral diaphragmatic paralysis secondary to cold cardioplegia was made.

At a follow-up examination on the 60th postoperative day, symptoms were much improved, and the patient no longer had orthopnea. A Konno-Mead diagram now revealed nearly equal contributions of the rib cage and abdominal components to breathing in the sitting position and absence of the previously observed paradoxic motion in the supine position (Fig 5), although both maximal respiratory pressures had changed only minimally. In addition, during exercise in the sitting position to a work load of 100 W on a cycle ergometer, no paradoxic motion developed (Fig 5). At the 120th postoperative day, maximal respiratory pressures had increased somewhat, and VC had increased to 2.13 L (56

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Frostbitten Phrenics after CABG Surgery (Olopade, Staats)
 percent of predicted) in the sitting position; however, there was a 41 percent decrease in VC when the supine position was assumed. A Konno-Mead diagram again revealed a normal pattern of chest wall motion in both sitting and supine positions.

The VC improved gradually and appeared to plateau at the 340th day and onward (Fig 2). The decrease in supine VC was no longer present at the 250th day.

The Pmax increased to near the lower limit of normal by the 156th day and continually improved throughout follow-up. In contrast to the slow improvement of Pmax, there was a more appreciable and rapid improvement in Pmax to a normal value at the 123rd day (Fig 4); thereafter, Pmax increased even further.

**DISCUSSION**

Bilateral diaphragmatic paralysis secondary to bilateral phrenic nerve damage occurs in about 1 percent

Figure 2. Follow-up VC measurements in sitting and supine positions. Note absence of decrease in VC in supine position from 150th postoperative day on.

Figure 3. Maximal inspiratory and expiratory pressures, showing recovery trend during follow-up period.

Figure 4. Konno-Mead diagram of relative motions of rib cage (RC) and abdomen (ABD) at 16th postoperative day, showing normal sitting chest wall motion but marked abdominal paradox in supine position. Inductive plethysmograph was calibrated using isovolume maneuver.

Figure 5. Konno-Mead diagram of relative motions of rib cage (RC) and abdomen (ABD) at 60th postoperative day, showing absence of previously observed paradox in supine position. In addition, exercise on cycle ergometer to work load of 100 W did not lead to development of paradoxic motion.

To 3 percent of patients undergoing coronary artery bypass grafting. Two postulated causes of the phrenic nerve injury are stretch, which may occur during either retraction of the sternum or manipulation of the pericardium, and hypothermia caused by the iced saline solution instilled into the pericardial sac during cold cardioplegia.

The first evidence of phrenic nerve damage in the postoperative period may be difficulty in weaning from mechanical ventilation. A long delay in diagnosis is not unusual because symptoms from bilateral diaphragmatic paralysis may mimic those of congestive heart failure, asthma, pulmonary embolism, pneumonia, or atelectasis. The diagnosis of frostbitten phrenics may be suggested by the bedside observation
of rib cage abdominal paradoxic motion in a patient with marked orthopnea. Our patient's presentation was somewhat unusual in that he was successfully extubated early in the postoperative period without prolonged ventilation and hospitalization. Diaphragmatic paralysis was not suspected in the immediate postoperative period.

Methods for evaluating diaphragmatic function include assessment of movement of the hemidiaphragms by fluoroscopy, assessment of chest wall motion, measurements of VC changes between the sitting and supine positions, maximal respiratory pressures, trans-diaphragmatic pressures, and diaphragmatic electromyography.9-15

Fluoroscopic examination of the diaphragmatic motion during deep inspiration is easily performed, widely available, and noninvasive. Typically, bilateral paralysis is associated with minimal movement of the hemidiaphragms, as was found in our patient. In some patients with bilateral diaphragmatic paralysis, active contraction of the abdominal muscles occurs during expiration, which causes passive upward displacement of the diaphragm. At the onset of inspiration, there is a sudden reduction of expiratory muscle activity, which allows the diaphragm to descend passively. This pattern of diaphragmatic excursion simulates normal breathing and may delay diagnosis.

Konno and Mead7 described a particularly useful means of evaluating chest wall motion by using an x-y oscilloscope to quantify linear (anteroposterior or lateral) chest wall motion. During normal tidal breathing, both rib cage and abdominal components expand and contract, tracing a slightly open counterclockwise loop on the oscilloscope screen. Normally, the rib cage represents approximately 25 percent of the total chest wall expansion in the supine position and 70 percent in the sitting position.14 In our patient, the typical paradoxic motion associated with bilateral diaphragmatic paralysis was documented on the 16th postoperative day; by the 60th day, this type of chest wall motion was no longer present (Fig 4 and 5). Most likely, the abdominal muscles became active during expiration, as described previously. This could have accounted for the disparity between measurements of diaphragmatic strength and motion and the patient's symptoms. Paradoxic motion also can occur in severely hyperinflated patients with chronic obstructive pulmonary disease and during obstructive apnea.15

Measurement of postural changes in VC is a simple screening test for diaphragmatic weakness or paralysis. A decrease greater than 25 percent in VC from the sitting to the supine position suggests diaphragmatic weakness.9,16 The major limitation to this measurement is the inability of some patients to tolerate the supine position.

The PImax and PEmax are estimates of maximal respiratory muscle strength.10 Lung volume and a patient's effort also affect PImax and PEmax values.11 In our patient the low PImax is highly suggestive of bilateral diaphragmatic paralysis. The decreased TLC caused by diaphragm paralysis and the recent median sternotomy may have been reasons, in part, for the decrease in PEmax. Both PEmax and PImax gradually returned toward normal (Fig 3). Maximal respiratory pressure usually is far in excess of that necessary for normal respiratory function, which may explain the early symptomatic improvement despite objective evidence of persistent weakness of the diaphragm.

An electromyographic evaluation by phrenic nerve stimulation is perhaps the best means of detecting diaphragmatic paralysis but also the most technically demanding.6,12 The nerves may be stimulated in the neck with either transcutaneous or fine needle electrodes.12,13 The diaphragmatic evoked potential is then recorded with surface disk electrodes placed over the diaphragm, usually at the seventh interspace close to the costal margin. Diaphragm paralysis is suggested by a prolonged latency or decreased amplitude or both.

The time needed for recovery from frostbitten phrenics is variable. Marco and colleagues17 carried out bilateral phrenic injury in dogs with cold cardioplegia and noted that complete recovery, as defined by a return of phrenic nerve electromyographic responsiveness to normal, took between 28 and 62 days. It has been suggested that patients who sustain mild demyelination of the phrenic nerves with little axonal damage tend to have a more rapid recovery.6,17 Recovery from postcardioplegic diaphragm paralysis occurred over a period of six months, as measured by serial phrenic nerve electromyography, in a series by Markand et al.8 Other studies have shown variations of up to 27 months in the recovery of diaphragm function based on serial spirometry and measurements of transdiaphragmatic pressure.5,9 Accurate prediction of recovery of phrenic nerve function is difficult and seems to depend on the degree of axonal damage. We postulate that our patient's clinical improvement preceded objective documentation of phrenic nerve recovery by several months because of the use of his abdominal muscles for active expiration and subsequent passive inspiration. In addition, he may have recruited other inspiratory muscles.

In bilateral diaphragm paralysis secondary to cold cardioplegia, high diagnostic acuity is necessary to make an early diagnosis and to avoid unnecessary expense while decreasing morbidity and mortality. Serial measurement of maximal respiratory pressures and spirometry provide useful follow-up information. Analysis of chest wall motion by using inductive plethysmography can quantify the physical sign of paradox and may help explain the disparity between...
objective testing results and symptoms in some patients.

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