Sonographic Measurement of Diaphragmatic Motion after Coronary Artery Bypass Surgery*

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Forty-eight patients were prospectively evaluated following coronary artery bypass grafting (CABG) in order to determine values for diaphragmatic mobility by sonography, to compare diaphragmatic motion to chest x-ray findings, to relate diaphragmatic motion to pulmonary function tests, and to determine whether use of the left internal mammary artery (LIMA), aortic cross-clamp time, or other clinical variables were predictive of diaphragmatic dysfunction. Mean left diaphragmatic motion was 2.8 ± 1.1 cm (range, 1.0 to 5.7 cm), mean right diaphragmatic motion was 3.9 ± 1.1 cm (range, 1.8 to 6.4 cm), and ratio of left to right motion was 0.74 ± 0.27 (range, 0.19 to 1.4). Forty-one patients had normally positioned diaphragms on the chest x-ray film; four of these had poor mobility by ultrasonography (<1.6 cm). Of the seven elevated left hemidiaphragms on chest x-ray films, three had an excursion of 1.6 cm or more by ultrasonography. The mean FVC for all patients was 59 ± 13 percent of predicted. There was no relationship between diaphragmatic mobility and FVC or negative inspiratory pressure. The diaphragmatic motion in 36 patients having LIMA grafting was similar to those without (2.7 ± 1.2 cm [n = 36] vs. 2.8 ± 0.8 cm [n = 12], respectively). Aortic cross-clamp time and respiratory symptoms also did not correlate with diaphragmatic mobility. Sonography can be used in the evaluation of diaphragmatic motion after CABG and may be more accurate in detecting a poorly mobile diaphragm than is the chest x-ray film. (Chest 1992; 102:1683-86)

Diaphragmatic dysfunction has been reported as a frequent complication of coronary artery bypass grafting (CABG) and has been associated with hypothermic injury of the phrenic nerve.14 Unilateral diaphragmatic dysfunction, almost always involving the left hemidiaphragm, has been reported to occur in 9 percent to 73 percent of patients after surgery.56 The wide variation in the reported incidence of left hemidiaphragmatic dysfunction may be secondary to the variety of methods of assessment and differences in surgical technique, such as the use of pericardial insulating pads.8 Because diaphragmatic elevation may not always be due to diaphragmatic paresis, studies which examine chest x-ray films for diaphragmatic elevation may overestimate the incidence of poor diaphragmatic mobility.7 Studies of phrenic nerve conduction, which are dependent on electrode positioning and tolerated stimulation voltage, may underestimate the functional ability of the diaphragm.

Sonography is able to evaluate diaphragmatic excursion,8 but no previous studies have investigated the usefulness of this technique in evaluating the degree of diaphragmatic motion after CABG. The relationship of pulmonary function tests to diaphragmatic mobility after CABG has also not been reported.

In order to establish normal value for diaphragmatic mobility by sonography after CABG and to determine whether sonographic measurement of diaphragmatic mobility correlated with pulmonary function, chest radiographic findings, and surgical or other clinical variables, we prospectively evaluated 48 patients one week following CABG surgery.

**Materials and Methods**

Patients eligible for the study were patients who had a routine postoperative course after CABG and had been discharged from the cardiothoracic intensive care unit within two days of surgery. Patients requiring intra-aortic balloon pump support, having valve replacement, having an elevated diaphragm on a preoperative x-ray film, or returning to the operating room for correction of postoperative bleeding were excluded. Thirty-six patients who had left internal mammary artery (LIMA) grafts and 12 who did not were randomly chosen from eligible patients for the study.

Surgery was performed via median sternotomy. Myocardial protection during cardiopulmonary bypass included total-body hypothermia to 28°C, topical ice/saline slush, and cold-blood cardioplegia. A pericardial insulating pad to protect the left phrenic nerve was used in all patients.

On the fifth or sixth day after CABG, patients had a frontal and lateral chest roentgenogram in the Radiology Department. Chest roentgenograms were scored as positive for hemidiaphragmatic elevation if a hemidiaphragm was elevated one rib space relative to the other side and as positive for atelectasis if there was lobar collapse. Immediately following the chest x-ray film, patients had sonographic evaluation of diaphragmatic motion by a skilled sonographer (J.G.), working under the supervision of an ultrasound radiologist (R.M.L.). A 3.5-MHz sector scan probe was used for imaging (Acuson 128/10), and the results were archived on film and videotape. Imaging was performed from posterior windows with the patient seated. After searching for an appropriate window, three

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to five measurements were obtained to ensure reproducible results. Diaphragmatic excursion was measured from the end of normal expiration to total lung capacity, and the greatest excursion of the leading edge of the diaphragmatic echo was recorded at an approximate 45° angle to the craniocaudal direction where the diaphragmatic edge was well defined (Fig 1). During sonographic examination, pulmonary function was recorded by a spirometer (Eagle Survey Spirometer; Warren E. Collins Inc). Negative inspiratory force (NIF) was measured by an inspiratory force meter (Boehringer Laboratories).

Information obtained from the medical record included the number of grafts, total bypass time, aortic cross-clamp time, hours on mechanical ventilation, length of hospitalization, and medical history of diabetes. Patients were interviewed regarding the presence of dyspnea or exercise intolerance.

Statistical analysis was by t-test for groups of unequal size. A p value of <0.05 was considered significant.

RESULTS

The following tabulation shows demographic and clinical information and mean values (± SD) for diaphragmatic movement and pulmonary function in 48 patients following CABG:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>62.9 ± 8.0</td>
</tr>
<tr>
<td>M:F ratio</td>
<td>35:13</td>
</tr>
<tr>
<td>LIMA</td>
<td>26±46</td>
</tr>
<tr>
<td>No. of grafts</td>
<td>2.7 ± 0.9</td>
</tr>
<tr>
<td>Aortic cross-clamp time, min</td>
<td>49.3 ± 16.5</td>
</tr>
<tr>
<td>Right hemidiaphragm motion, cm</td>
<td>3.9 ± 1.1†</td>
</tr>
<tr>
<td>Left hemidiaphragm motion, cm</td>
<td>2.8 ± 1.1†</td>
</tr>
<tr>
<td>Ratio of left to right diaphragmatic motion</td>
<td>0.74 ± 0.27</td>
</tr>
<tr>
<td>FVC; percent of predicted</td>
<td>59.5 ± 13.1</td>
</tr>
<tr>
<td>FEV1; percent of predicted</td>
<td>62.8 ± 16.8</td>
</tr>
<tr>
<td>FEV1/FVC%</td>
<td>74.4 ± 9.3</td>
</tr>
<tr>
<td>Negative inspiratory force, cm H2O</td>
<td>62.0 ± 19.2</td>
</tr>
</tbody>
</table>

*p<0.001.

Four patients had diabetes. The mean time from surgery to extubation was 17.2 ± 6.1 h. The mean length of hospitalization after surgery was 7.4 ± 1.5 days.

On the chest roentgenogram, seven patients had left hemidiaphragmatic elevation of greater than one rib space compared to the right side; four had left-sided and two had right-sided pleural effusions of greater size than one rib space. Six patients had left lower lobe atelectasis; only one of these had an elevated diaphragm.

Figure 2 shows the values for left and right hemidiaphragmatic movement and for the ratio of left to right movement for each subject. Table 1 shows variables associated with diaphragmatic elevation on the chest x-ray film.

There was no relationship between diaphragmatic motion and aortic cross-clamp time, total bypass time, number of grafts, sex, or age. The percent of predicted vital capacity, NIF, and symptoms of dyspnea had no relationship to the degree of diaphragmatic motion. No patient had orthopnea.

Those having LIMA grafts had similar left hemidiaphragmatic motion and pulmonary function tests compared to those who did not (left hemidiaphragmatic motion of 2.7 ± 1.2 cm vs 2.8 ± 0.8 cm, and FVC [percent of predicted] of 56.2 percent ± 13.1 percent

![Figure 1: Sonogram of diaphragmatic movement. For longitudinal orientation, cranial is to left, and caudal is to right. Arrows indicate position of diaphragm. A (left panel): position of diaphragm at end of expiration; B (right panel): position at total lung capacity. Total excursion, measured cursor (indicated by plus sign) to cursor, is 2.7 cm.](http://journal.publications.chestnet.org/ on 06/01/2015)

![Figure 2: Values for sonographic measurement of left and right hemidiaphragmatic motion and ratio of left to right hemidiaphragmatic motion for 48 subjects measured at 5 or 6 days following CABG. Horizontal lines represent mean values.](http://journal.publications.chestnet.org/ on 06/01/2015)
vs 63.5 percent ± 12.9 percent, respectively).

**Discussion**

Left diaphragmatic dysfunction often follows CABG surgery, but the true incidence is uncertain. Previous studies have reported values ranging from 9 percent to 73 percent. The wide variation may in part be due to the methods used to study and define diaphragmatic dysfunction, as well as to differences in surgical technique, including the use of pericardial insulation. Studies using phrenic nerve stimulation have reported values of approximately 10 percent. The incidence based on x-ray findings is higher and more variable. This may be so because authors have used various definitions of diaphragmatic dysfunction; for example, hemidiaphragmatic elevation two ribs above the preoperative film, elevation of the left above the right hemidiaphragm, less than 1 cm of movement on inspiratory compared to expiratory films, and hemidiaphragmatic elevation that is "persistent" or "clear cut."

We designed this study to examine sonographic measurement of diaphragmatic motion following uncomplicated CABG and to relate that motion to pulmonary function, surgical variables, medical history, and symptoms. We did not select symptomatic patients in order to avoid bias in the estimate of the frequency of diaphragmatic dysfunction. We also did not measure preoperative diaphragmatic motion. Normal values for right hemidiaphragmatic motion by sonography for an inspiratory capacity breath in normal subjects have been reported (mean of 5.4 cm in men and 4.0 cm in women); and by fluoroscopy, left hemidiaphragmatic motion is usually greater than the right. Since pain and splinting could reduce diaphragmatic excursion independent of phrenic nerve injury, we believed that comparing left hemidiaphragmatic motion (the diaphragm usually involved in dysfunction after CABG) to right would be a better measure of postoperative left hemidiaphragmatic dysfunction than comparison to preoperative values. Also, if ultrasound is used for clinical reasons following CABG (such as in the evaluation of dyspnea or an elevated diaphragm on the x-ray film), preoperative measurements likely will not be available for comparison.

We found that at one week following CABG surgery, 8 of 48 patients had left hemidiaphragmatic motion which was one-half or less than right hemidiaphragmatic motion, and 7 had absolute left hemidiaphragmatic motion of less than 1.6 cm. This incidence of poor diaphragmatic mobility is more in accord with the results for diaphragmatic dysfunction after CABG studied by phrenic nerve pacing than by chest x-ray films and likely reflects more accurately the incidence of left hemidiaphragmatic dysfunction following modern CABG surgery with pericardial protection from hypothermic injury.

Our prospective study did not find an association between LIMA grafting and an increased incidence of left hemidiaphragmatic dysfunction. While Abd et al. reported that 11 of 13 patients with symptomatic diaphragmatic dysfunction had right or left LIMA grafting, so did 96 percent of the patients with CABG in his study, so the preponderance of those with diaphragmatic dysfunction who had LIMA grafting is not unexpected. Moreover, his group of patients was chosen retrospectively from symptomatic patients diagnosed as having diaphragmatic dysfunction, seven of whom had bilateral paralysis.

For our entire group, vital capacity was 59.5 percent of predicted one week after CABG, a value similar to that reported in other studies. We did not find a relationship between diaphragmatic mobility and pulmonary function. A study by Estenne et al. using phrenic nerve pacing 8 to 13 days after CABG, supports this finding. Estenne et al. found that only 1 of 12 patients in their study had diaphragmatic dysfunction, yet all had reductions in vital capacity. A study by Locke et al. which used magnetometry to evaluate chest wall motion after CABG, offers a reason for these observations. Locke et al. found discoordination of rib cage expansion and airflow, and reduced abdominal, sternal, and rib cage motion one week after surgery and concluded that the restrictive defect after CABG is due in part to alterations in chest wall mechanics.

The chest x-ray film has been used to infer diaphragmatic dysfunction after surgery, although it has been suggested that diaphragmatic elevation may relate more to decreased ventilation to the lung base from
splinting and left lower lobe atelectasis than to diaphragmatic paresis. In our study a normally positioned diaphragm on the x-ray film did not exclude diaphragmatic dysfunction (Table 1); 4 of 41 patients with a normally positioned diaphragm on the x-ray film had diaphragmatic excursion of less than 1.6 cm. An elevated diaphragm was more likely to correlate with poor motion by ultrasound, but three of seven elevated diaphragms moved more than 1.6 cm, and one of these moved 3 cm. While a larger sample size could better define the magnitude of the discrepancy between x-ray films and ultrasonographic findings, in our study the x-ray film alone was not definitive.

We could discern no increased dyspnea in patients with unilateral diaphragmatic weakness compared to those without. This may be so because our patients seemed to have good underlying cardiopulmonary function, and at one week after CABG, those with poorly mobile diaphragms as a group did not have different vital capacities than those with more mobile diaphragms; however, unilateral diaphragmatic weakness has been associated with breathlessness, particularly in those with underlying lung disease. We believe that in patients with marginal cardiac or pulmonary function, diaphragmatic dysfunction should be suspected if there is unusual postoperative dyspnea. These patients should have diaphragmatic motion evaluated by sonography, and one should not rely on the position of the diaphragm on the x-ray film, which is not an accurate predictor for diaphragmatic mobility.

In summary, this study has provided sonographic normative values for diaphragmatic motion after CABG that may be useful clinically. We found that 16.7 percent of the asymptomatic patients after CABG had significantly diminished left hemidiaphragmatic motion compared to the right side, and that left hemidiaphragmatic function did not correlate with the restrictive pattern on pulmonary function tests seen after CABG or with respiratory symptoms. We found that the position of the diaphragm on the chest x-ray film does not adequately estimate diaphragmatic motion, since a normally positioned diaphragm does not exclude poor diaphragmatic mobility, nor does an elevated diaphragm always confirm it. We found no association between diaphragmatic motion and the use of LIMA for grafting, aortic cross-clamp time, or other clinical variables. We believe sonography can be a useful technique to investigate possible unilateral diaphragmatic paresis after CABG, as might be suspected in an unusually dyspneic patient or suggested by an elevated left hemidiaphragm on the chest x-ray film.

ACKNOWLEDGMENTS: We thank Dr. Theodore F. Van Zandt, Professor of Radiology, University of Rochester at Rochester General Hospital, for his assistance, and Mrs. Rebecca Anne Cadregari for typing the manuscript. We acknowledge the assistance of Karen Baby, R.N., and the patients' nurses for expediting data collection.

REFERENCES