Etiology and Prevention of Topical Cardiac Hypothermia-induced Phrenic Nerve Injury and Left Lower Lobe Atelectasis during Cardiac Surgery*

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Left hemidiaphragm elevation is frequently noted following cardiac surgery employing topical hypothermia. We speculate that contact of the left phrenic nerve with ice causes nerve injury, resulting in left hemidiaphragm paresis or paralysis and left lower lobe atelectasis. Left diaphragm elevation was noted on postoperative chest x-ray examination of 36 of 60 (60 percent) consecutive patients in whom topical cooling of the heart with a cold slush solution was administered prior to use of a cardiac insulation pad (CIP, Shiley Laboratories, Irvine, California). Following the use of the CIP in a similar group of 60 consecutive patients, only five (8 percent) showed evidence of diaphragmatic elevation. The difference in incidence of diaphragmatic elevation between these two groups is statistically significant (p<0.001). A comparison of postoperative left lower lobe atelectasis prior to the use of the CIP was also statistically significant (p<0.001). There was no significant difference in the aortic cross-clamp time or the volume of intraaortic cardioplegia used in these two groups. The use of topical cardiac hypothermia has been shown to protect the myocardium. Phrenic nerve injury secondary to the use of ice in this method has been documented. The use of a cold solution without ice chips or slush, or the insertion of a CIP prior to the use of topical cardiac hypothermia (when ice chips or slush are used) decreases the exposure of the phrenic nerve to cold injury and decreases the incidence of paresis of the left diaphragm and resultant atelectasis.

Topical cooling of the heart as an adjunct to the infusion of cold cardioplegic solution into the aorta has become an accepted technique to protect the myocardium during open heart surgery.14 Left phrenic nerve paralysis or paresis, and resultant diaphragmatic elevation and left lower lobe atelectasis, following the use of topical cardiac hypothermia has been reported in the literature,7–10 but no method of prevention for these complications has been reported. We have noted a decrease in the incidence of postoperative left diaphragmatic elevation and left lower lobe atelectasis after using a cardiac insulation pad (CIP). The present review was designed to evaluate the efficacy of the CIP in decreasing the incidence of postoperative left diaphragmatic elevation and left lower lobe atelectasis.

Materials and Methods

Our method of employing topical cardiac hypothermia is a modification of techniques previously described.14

A cardiac insulation pad is inserted (Fig 1, view from head of table). The aorta is cross-clamped and cold cardioplegic solution is injected into the root of the aorta while, simultaneously, cold normosol solution is poured over the heart externally. The normosol solution is at a near-freezing temperature and frequently contains ice chips or is of a slushy consistency. Approximately 3,000 ml of normosol solution is used for each application. The myocardial temperature after application is 10–15°C. If the myocardial temperature rises to 20°C while the aorta is cross-clamped, a second or third injection of cardioplegic solution and application of slush is performed.

Retrospectively, preoperative and postoperative chest x-ray examinations were compared from a group of 120 patients who had

**Figure 1. Insertion of CIP into position between the heart and the pericardium.**

Cardiac Hypothermia-induced Phrenic Nerve Injury (Wheeler et al)
cardiac surgery employing cardiopulmonary bypass, intra-aortic cardioplegic solution injection, topical cardiac hypothermia and systemic cooling to 25°C. The patients were divided into two groups: group 1 consisted of 60 consecutive patients undergoing cardiac surgery without the use of the CIP; and group 2 consisted of the next 60 consecutive patients, in whom the CIP was used. The case mix is listed in Table 1. All procedures were performed by the same surgical team consisting of two board certified thoracic surgeons (LJR and JDH). The only change in the operative routine was the insertion of the CIP prior to cross-clamping the aorta in the last 60 patients.

All 120 preoperative chest films were reviewed. None of the patients had elevation of the left diaphragm nor atelectasis prior to surgery. Chest x-ray films were obtained at the time of hospital discharge and again five to six weeks postoperatively. The level of the left diaphragm leaf was compared on preoperative and postoperative films. The left hemidiaphragm was considered elevated if it was above the right diaphragm leaf and had been below this on preoperative film. If the margin of the diaphragm was obscured by pleural effusion or an infiltrate, the position of gas in the stomach was used to estimate the level of the diaphragm. Any minimal or questionable change in the level of the diaphragm was read as normal.

The following data were recorded: type of procedure performed, aortic cross-clamp time, amount of intra-aortic cardioplegia used, degree of diaphragmatic elevation and/or atelectasis on discharge or follow-up chest x-ray film.

RESULTS

The review of postoperative x-ray films are summarized in Tables 2 and 3. The incidence of diaphragmatic elevation in group 1 (without the CIP) was 36 of 60 (60 percent) compared to 5 of 60 (8 percent) in group 2 (CIP was used) (Table 2). Chi-squared analysis indicated that the incidence of diaphragmatic paralysis in group 2 was significantly less (p<0.001) than that of group 1. The x-ray films were then reviewed to determine the incidence of left lower lobe atelectasis in both groups (Table 3). When the CIP was not employed, 34 of 60 (57 percent) patients had left lower lobe atelectasis. In contrast, among patients in group 2, only 13 of 60 (22 percent) developed atelectasis in the postoperative period. Again, using chi-square analysis, the difference proved significant (p<0.001).

Aortic cross-clamp time and volume of cardioplegic solution utilized were compared. These data are summarized in Table 4. By the Student t-test and analysis of variance, mean cross-clamp time was comparable in groups 1 and 2. Within group 2, mean aortic cross-clamp time for the five patients who developed diaphragmatic elevation was 88 min, compared to 69 min for the other 55 patients in this group. This difference was significant (p<0.05), but may be the result of the small number of patients and needs to be further evaluated for a larger group. There was no significant difference between the two groups with respect to the

Table 1—Operations

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
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<tbody>
<tr>
<td>Coronary bypass</td>
<td>48</td>
<td>55</td>
</tr>
<tr>
<td>Valves</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Valves and coronary bypass</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>ASD or VSD</td>
<td>1</td>
<td>0</td>
</tr>
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Table 2—Comparison of Incidence of Diaphragm Elevation with and without Cardiac Insulation Pad

<table>
<thead>
<tr>
<th></th>
<th>Elevated</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 (no pad)</td>
<td>36 (60%)</td>
<td>24 (40%)</td>
</tr>
<tr>
<td>Group 2</td>
<td>5 (8%)</td>
<td>55 (92%)</td>
</tr>
</tbody>
</table>

Table 3—Comparison of Incidence of Atelectasis with and without Cardiac Insulation Pad

<table>
<thead>
<tr>
<th></th>
<th>Atelectasis</th>
<th>No atelectasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 (no pad)</td>
<td>34 (57%)</td>
<td>26 (43%)</td>
</tr>
<tr>
<td>Group 2</td>
<td>13 (22%)</td>
<td>47 (78%)</td>
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</table>

Figure 2. CIP in place and position of the left phrenic nerve.
amount of intraaortic cardioplegic solution employed.

**DISCUSSION**

The anatomic position of the left phrenic nerve on the pericardium exposes the nerve to prolonged hypothermia during topical cooling of the myocardium in cardiac surgery, since it courses along the most dependent portion of the pericardial sac (Fig 2).

The development of atelectasis in 86 to 96 percent of immediate postoperative patients undergoing coronary artery bypass surgery was reported by Good et al.11

Scannell et al11 described four cases of phrenic nerve paralysis in 25 patients who underwent cardiac surgery using topical hypothermia as a method of myocardial protection. He referred to this finding as the "frostbitten phrenic," or a cold injury to the nerve.

Marco et al11 examined the effects of topical cardiac hypothermia on the phrenic nerve in dogs. They showed that direct exposure of the phrenic nerve to both ice chips and a slush solution caused phrenic nerve paresis or paralysis and elevation of the diaphragm. They also demonstrated that this injury resolved within 60 to 90 days.

Irisawa et al12 reported a case of bilateral phrenic nerve paralysis due to the use of topical cardiac hypothermia.

Benjamin et al13 reported a 63 percent incidence of infiltrative or atelectatic change in a group of 122 coronary artery bypass patients where topical cooling of the heart with ice was utilized. These findings were attributed to the effects of hypothermia on the left phrenic nerve. Fifty-five percent (22 of 40) of their group 2 had fluoroscopic documentation of paresis or paralysis of the left hemidiaphragm. This group of patients, and their incidence of elevated diaphragms, is comparable to that of our group 1.

Witte et al14 reported that 93.2 percent of patients undergoing cardiac surgery developed perceivable pathologic changes on routine postoperative chest x-ray examination. Limited motion was noted on the left side in 69 percent with fluoroscopy. They attributed this to direct damage of the left phrenic nerve caused by topical cardiac cooling.

Bjork et al15 were the first to describe the use of a cardiac insulation pad. They used this, however, as an insulator for maintaining the patient's body temperature at 30°C. They did not mention a decrease in postoperative atelectasis nor diaphragmatic elevation.

Topical cardiac hypothermia as an adjunct to the infusion of cold cardioplegic solution into the aorta has become an accepted technique to protect the myocardium during open heart surgery and can be accomplished by several methods.16 These techniques include the use of a cooled physiologic solution, with or without ice chips or slush. Several authors have reported the development of atelectasis with left and/or right phrenic nerve paralysis or paresis, especially when ice chips or slush are used.17,18,19 Due to the retrospective nature of our study, we were unable to absolutely confirm the paresis or paralysis of the left hemidiaphragm with fluoroscopy. However, our reported incidence is comparable to the documented incidence of 55 to 64 percent reported in the literature.13 (Table 5). Some cardiac surgeons use sterile ice slush makers to provide their topical cardiac hypothermia and have reported no evidence of cold injury to the phrenic nerve.18

The decreased number of patients who experience elevated left diaphragm and/or left lower lobe atelectasis after the insertion of an insulation device between the pericardium and the heart in our series tends to substantiate the theory that cold injury to the phrenic nerve from topical hypothermia is the cause of nerve injury and hence, diaphragm paralysis.

Length of exposure time of the phrenic nerve to ice as measured by the aortic cross-clamp time was slightly longer in group 1 (Table 3). Although not significantly different in our series or the series by Benjamin et al.18 it is possible that the time difference could be significant with a larger group of patients. Volume of intraaortic cardioplegia in each group was essentially equal.

Direct surgical trauma to the left lower lobe and/or phrenic nerve is rarely a contributory factor, but must be considered because of the occurrence of this complication prior to the use of topical cardiac hypothermia.17

The efficacy of topical hypothermia in cardiac surgery for myocardial preservation has been well documented and is used by many cardiac surgeons.19 The occurrence of atelectasis and/or elevation of the left leaf of the diaphragm after the use of topical hypothermia has been noted by several authors and is concluded to be the result of a cold injury to the phrenic nerve.17,18 There have also been reported cases of bilateral phrenic nerve paralysis with respiratory compromise.13

We conclude that the major cause of paralysis and or paresis of the left diaphragm and resulting atelectasis of the left lower lobe after the use of a topical cardiac hypothermia is caused by cold-induced phrenic nerve injury. We also have concluded that these complications can be prevented or reduced by the use of a

<p>| Table 5—Incidence of Fluoroscopically Documented Left Diaphragm |</p>
<table>
<thead>
<tr>
<th>Series</th>
<th>Paralysis/Paresis Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benjamin et al13</td>
<td>55</td>
</tr>
<tr>
<td>Witt et al14</td>
<td>69</td>
</tr>
<tr>
<td>Group 1*</td>
<td>60</td>
</tr>
</tbody>
</table>

*Not documented due to retrospective nature of study.
physiologic solution that has been profoundly cooled but has no ice chips or slush in it, or by the insertion of a CIP prior to the use of topical hypothermia if there are ice chips or slush in the solution. This has not been previously reported in the English literature.

REFERENCES
5. Stinson EB. Myocardial preservation. ACS Postgraduate course on Cardiac Surg. 1977; 19-23
9. Benjamin JJ. Cascade PN, Rubenfire M, Wajszczuk W, Kerin NZ. Left lower lobe atelectasis and consolidation following cardiac surgery; the effect of topical cooling on the phrenic nerve. Radiology 1982; 142:11-14