Thoracicabdominal Mechanics during Resuscitation Maneuvers*

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The importance of intrathoracic pressure in generating blood flow during cardiopulmonary resuscitation has recently been emphasized. The purpose of this study was to investigate the factors involved in generating intrathoracic pressure. Studies were performed in anesthetized paralyzed dogs with the circulation intact. Balloon-tipped catheters were placed in the abdomen and esophagus for measurement of intra-abdominal and intrathoracic pressures and cannula placed in the airway for airway pressure. The following four maneuvers were studied: (1) chest compression with open airway; (2) chest compression with closed airway; (3) pulmonary inflation to transpulmonary pressure (TP) of 30 cm H$_2$O (TP = 30); and (4) chest compression plus pulmonary inflation (TP = 30). We found that under static conditions, chest compression alone produced small positive intrathoracic pressures (9 ± 8 cm H$_2$O), but these could be increased by closing the airway pressure (18 ± 6 cm H$_2$O) or inflating the lungs (15 ± 7 cm H$_2$O). The combination of inflating the lung and compressing the chest produced the highest intrathoracic pressure (48 ± 15 cm H$_2$O; p < 0.001). The pressure developed was highly variable and the distribution of pressures within the thorax was not uniform. As the intrathoracic pressure became large, a pressure gradient developed from thorax to abdomen, and the diaphragm everted; this pressure gradient could divert blood from the brain.

The original concept of resuscitation proposed that chest compression produced blood flow by a "cardiac pump" mechanism, in which the ventricles are squeezed between the sternum and the vertebral column. Various observations during cardiac arrest have shown that other mechanisms are also operative. There is minimal difference in pressure between intrathoracic arteries and veins during chest compression. Arterial pressure and consciousness can be maintained during cardiac arrest by coughing alone, without chest compression. Furthermore, Weisfeldt and co-workers have shown that arterial pressure and carotid flow were higher in both dogs and humans when chest compression and airway closure or when simultaneous chest compression and ventilation were added to standard cardiopulmonary resuscitation (CPR). These data suggest that the thorax acts as a unit, the "thoracic pump." When the thorax is compressed, the pressure in the whole intrathoracic vascular compartment is raised, and blood flows out of this high-pressure compartment to the rest of the body. In this model the heart acts as a conduit, and the critical variable is intrathoracic pressure. Debate exists as to the more important factor in CPR but it seems clear that increased intrathoracic pressure can at least augment blood flow.

Little is known about the factors involved in generating intrathoracic pressure during CPR and the mechanical characteristics of the various components of the thoracoabdominal compartment. Our goals were to investigate under static conditions the distribution of thoracoabdominal pressure produced by various manipulations which may be useful in CPR and to examine the interaction of the thorax and abdomen during these maneuvers.

Materials and Methods

Preparation

Ten dogs (20 to 30 kg) were anesthetized with pentobarbital, paralyzed with pancuronium, and placed in a 90° V-shaped animal board. The dogs were intubated with cuffed endotracheal tubes. Thin-walled balloons, 5 cm in length and 3 cm in circumference, attached to air-filled polyethylene catheters were used for pressure measurements in the esophagus and the abdomen. The balloons had an unstressed volume of 3 ml, and measurements were performed with 1 ml in the balloons. They were placed in the lower and upper esophagus and in the abdomen. The upper esophageal balloon was 10 cm above the center of the lower esophageal balloon. The lower esophageal balloon was positioned just above the esophagogastric junction. The position of the balloons was verified by the characteristic drop in pressure during inspiration in the esophagus with simultaneous rise in pressure in the abdomen. The intra-abdominal balloon was placed in the upper peritoneal cavity just under the

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diaphragm via laparotomy, and the incision was tightly closed in layers. The balloons were connected to transducers (Hewlett-Packard or Validyne), and the output was recorded on an eight-channel recorder (Hewlett-Packard).

A 7F A2 multipurpose catheter was inserted into the right atrium by the external jugular vein and connected to a transducer (HP ± 100).

To verify that the lower esophageal balloon was an accurate reflection of intrathoracic pressure, we made measurements of intrapleural pressure from two additional sites in three dogs. A flat balloon constructed of two square pieces of 3 × 3-cm rubber dental dam glued airtight around the edges and attached to an air-filled polyethylene catheter was placed via a thoracotomy in the left lateral pleural space, at the midchest position and at approximately the same level as the esophageal balloon in the transverse and sagittal plane. This balloon has zero pressure over a range of 0.5 to 5 ml, and measurements were made with 1 ml in the balloon. Another simultaneous measurement was made with an air-filled polyethylene catheter placed in the right pleural space via thoracotomy at the same approximate position of the esophageal balloon. Care was taken to minimize the residual pneumothorax during closure of thoracotomies. An example is shown in Figure 1. Pressures recorded during all of the maneuvers we tested were identical from these two pleural sites and the lower esophageal balloon.

Airway pressure was obtained from a 19-gauge needle inserted into the rubber tubing connecting the endotracheal tube to the animal respirator. This was connected by a large-bore polyethylene tubing to a transducer. The airway pressure line and the lower esophageal line were also connected to a second transducer to obtain transpulmonary pressure.

**Procedure**

The following four maneuvers were performed: (1) chest compression with an open airway, displacing the sternum 7 cm in a ventral-todorsal direction (V-D); (2) the same degree of chest compression with a closed airway; (3) pulmonary inflation without chest compression to a transpulmonary pressure of approximately 30 cm H2O, as calculated by airway minus lower esophageal pressure; and (4) pulmonary inflation to a transpulmonary pressure of 30 cm H2O after chest compression (7 cm V-D) with a closed airway. Transpulmonary pressure of 30 cm H2O was used because with inflation pressures greater than this, there is little further change in pulmonary volume and a greater risk of barotrauma.

The chest compression was performed with a specially constructed wooden frame lever which could displace the sternum a desired amount and then be fixed. The pressure was applied to the lower one-third of the sternum by a rectangular wooden plate approximately 15 × 10 cm; contact between the chest wall and plate was padded by an air-filled bladder. Side-to-side movement of the dog was kept to a minimum by the deep V-shaped animal board.

The maneuvers were performed on live paralyzed anesthetized dogs with a sustained pressure for 10 to 15 seconds. No resuscitation was performed, as the dogs did not have arrested circulations.

Results are presented as the mean ± standard deviation. An analysis of variance was performed to measure the variance in the population, and a paired t-test was used to compare the differences between maneuvers when the F value was significant.

**Results**

The mean pressures of the airway, lower esophagus, and abdomen for each maneuver performed on the ten dogs are listed in Table 1. Upper esophageal pressures could be obtained in only eight dogs; these were usually at least a few centimeters of water lower than the pressure in the lower esophagus, but the difference was only significant for chest compression with open airway (Table 2). With chest compression and an open airway, as used in standard CPR, the mean lower esophageal pressure was 9 ± 8 cm H2O. When chest compression was performed with a closed airway, the intrathoracic pressure was significantly higher, 18 ± 6 cm H2O (p < 0.01). Pulmonary inflation to a transpulmonary pressure of 30 cm H2O produced a lower esophageal pressure of 15 ± 7 cm H2O, which was not significantly different from chest compression with an open airway or compression plus a closed airway. Chest compression with a closed airway and inflation to a transpulmonary pressure of 30 cm H2O produced the highest intrathoracic pressure (48 ± 18 cm H2O), which was significantly greater than the other maneuvers (p < 0.001).

The rise in intrathoracic pressure with each maneuver produced a rise in intra-abdominal pressure, but

<table>
<thead>
<tr>
<th>Maneuver</th>
<th>Airway Pressure, cm H2O</th>
<th>Esophageal Pressure, cm H2O</th>
<th>Abdominal Pressure, cm H2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest compression</td>
<td>0</td>
<td>9 ± 8</td>
<td>4 ± 3</td>
</tr>
<tr>
<td>Chest compression and</td>
<td>16 ± 5</td>
<td>18 ± 6†</td>
<td>6 ± 3†</td>
</tr>
<tr>
<td>airway closure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary inflation</td>
<td>45 ± 6†</td>
<td>15 ± 7</td>
<td>6 ± 4</td>
</tr>
<tr>
<td>Pulmonary inflation and</td>
<td>77 ± 20‡</td>
<td>48 ± 18‡</td>
<td>11 ± 6‡</td>
</tr>
<tr>
<td>chest compression</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Table values are means ± SD (n = 10).
†Compared to chest compression alone, p < 0.01.
‡p < 0.001.
cm H₂O was added to the chest compression, intrathoracic pressure was much higher, and transdiaphragmatic pressure became more negative. This time, electrical stimulation of the diaphragm produced an increase of the esophageal and airway pressure (Fig 3B).

The right atrial pressures are plotted against simultaneous lower esophageal pressures in Figure 4. The right atrial pressure correlated with the esophageal pressures obtained by chest compression and open as well as closed airways, but not with the esophageal pressure obtained by inflation.

**DISCUSSION**

The main findings of this study are as follows: (1) under static conditions the intrathoracic pressure developed during ventral-to-dorsal chest compression in dogs was very small with an open airway; however, the intrathoracic pressure became greater when the upper airway was closed or the lung was inflated, particularly when both of these preceded the chest compression; (2) the pressure developed was highly variable among animals; (3) the distribution of pressure within the thorax was not uniform; and (4) as the intrathoracic pressure became large, a pressure gradient developed from the thoracic to abdominal cavity, and the diaphragm everted.

Since an important part of CPR is the development of intrathoracic pressure, we analyzed the factors involved in generating this pressure. We used esophageal pressure as a measure of intrathoracic pressure. It is possible that the rise in esophageal pressure was due to direct compression of the esophagus by the mediastinum, rather than a true rise in intrathoracic pressure; however, we believe that this explanation is unlikely because the pressures of a flat balloon and air-filled catheter located at the same transverse plane as the lower esophageal balloon were similar to the pressure recorded in the lower esophageal balloon (Fig 1).

The esophageal pressure increased even during chest compression with an open airway (Table 1). An external force applied to the chest wall will be transmitted to the intrathoracic cavity in an amount dependent upon the stiffness of the chest wall. This force will raise the pressure in airways, but this pressure should be dissipated as air is displaced from the chest. Thus, there must have been some airway closure to trap air in the alveoli and produce positive intrathoracic pressure. Indeed, there was a mean transpulmonary pressure of 8 cm H₂O; however, the airway closure was not complete because significantly higher intrathoracic pressures were generated by directly closing the airway. The difference between upper and lower esophageal pressure was most likely because the force was applied to the lower part of the thorax.

<table>
<thead>
<tr>
<th>Maneuver</th>
<th>Lower Esophageal Pressure, cm H₂O</th>
<th>Upper Esophageal Pressure, cm H₂O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest compression</td>
<td>7 ± 8</td>
<td>3 ± 4</td>
</tr>
<tr>
<td>Chest compression and</td>
<td>17 ± 5</td>
<td>14 ± 8</td>
</tr>
<tr>
<td>airway closure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary inflation</td>
<td>16 ± 8</td>
<td>11 ± 3</td>
</tr>
<tr>
<td>Pulmonary inflation and chest compression</td>
<td>44 ± 18</td>
<td>38 ± 15</td>
</tr>
</tbody>
</table>

*Table values are means ± SD (n = 8).
†p<0.05 for lower compared to upper.

**Table 2—Esophageal Pressures during Maneuver**

![Figure 2. Transdiaphragmatic pressure (y axis) plotted for each maneuver (CC open, chest compression with open airway; CC closed, chest compression with closed airway; TP 30, inflation to transpulmonary pressure of 3 cm H₂O; and CC TP 30, chest compression plus pulmonary inflation). Transdiaphragmatic pressure is defined as abdominal pressure minus thoracic pressure. In normal respiration, this is positive number, and abdominal pressure is higher than thoracic pressure; but in almost every maneuver studied, thoracic pressure was higher than abdominal pressure, so that transdiaphragmatic pressures were almost always negative.**

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The esophageal pressures produced by chest compression with an open airway were very small and would not be very effective in generating blood flow if intrathoracic pressure is an important determinant of flow in CPR. Significantly higher intrathoracic pressures were obtained by inflating the lungs to a transpulmonary pressure of 30 cm H₂O or by compressing the chest with the airway closed. A striking finding was that the esophageal pressure produced by pulmonary inflation was equal to that produced by chest compression: namely, at low pulmonary volumes, chest compression was not more effective in developing high pleural pressure than the inward elastic recoil of the chest wall at high pulmonary volumes. Furthermore, when the chest wall was compressed at high pulmonary volumes, there were even larger changes in...
esophageal pressure.

When analyzing how these esophageal pressures were generated, it is useful to think of the thorax and abdomen as one unit. This is more correct in the upright posture where subdiaphragmatic abdominal pressure is identical to supradiaphragmatic pleural pressure, and transdiaphragmatic pressure is equal to zero. In the supine position at FRC, the nondependent part of the diaphragm is flaccid, and transdiaphragmatic pressure equals zero, but the lower parts of the diaphragm are probably under passive tension due to hydrostatic abdominal pressure; however, to a useful approximation, the abdominal and thoracic cavities may be regarded as a single unit when the diaphragm is not actively contracting or developing passive tension.

The transmission of an applied force across the wall of an elastic structure, such as the thoracic wall, is dependent upon the elastic recoil properties of the structure and its volume. Thus, the efficiency of an external applied force in producing changes in pleural pressure will be maximized by increasing the stiffness of the elastic structure (elastic recoil of the thoracoabdominal unit) and by minimizing changes in volume. The volume displacement can easily be minimized by closing the airway prior to compression. Thus, the esophageal pressure with a closed airway was double that with an open airway. The elastic recoil properties of the thoracoabdominal unit can be increased by applying a counterpressure to the abdominal wall, the most compliant part of the system. We studied the effect of abdominal compression in a preliminary fashion by manually applying 14 to 18 kg of pressure to the abdominal wall. Indeed, this maneuver produced a further increase of esophageal pressure by about 100 percent of that without abdominal compression. It has been demonstrated in dogs that carotid flow improves when abdominal compression is added to CPR techniques.

The greatest change in esophageal pressure was observed when the lung was inflated almost to total lung capacity (transpulmonary pressure, 30 cm H2O) and the chest was compressed. This maneuver maximized the efficiency of the applied forces because the overall chest wall and pulmonary volume displacement was minimized. The only two ways that the volume could be displaced was laterally in the thorax or downward through the diaphragm. It is unlikely that much volume was displaced laterally. Furthermore, ventral-to-dorsal compression of the thorax actually reduces pulmonary volume further by a natural rotation of the ribs about their articulations. It is also clear that at high pulmonary volumes the diaphragm became quite stiff, for the important drop in pressure was from thorax to abdomen and not across the abdominal wall. The diaphragm actually descended and everted, and the passive tension of the diaphragm opposed the transmission of the high intrathoracic pressures into the abdomen. Under these conditions the thoracic cavity and abdomen no longer functioned as a single unit. A rise in intrathoracic pressure without an equal rise in intra-abdominal pressure is undesirable during CPR, for a gradient will exist from thorax to abdomen, which could divert blood from the brain; this could easily be corrected by applying a force to the abdomen.

Another factor contributing to the greater pleural pressure produced by chest compression at a transpulmonary pressure of 30 cm H2O is the resistance of the lung to the distorting forces. It is harder to distort the lung at high pulmonary volumes. Agostoni et al used the term, pneumatic rigidity, for this resistance of the lung to changes in shape at volumes close to total lung capacity. Thus, if the lungs become stiffer at high pulmonary volumes, chest compression at high pulmonary volumes will be more effective in raising pleural pressure.

In all four maneuvers performed, the distribution of pressure within the thorax was not uniform. The upper esophageal balloon generally showed smaller changes in pressure than the lower esophageal balloon. Furthermore, the right atrial pressure did not correlate with the esophageal pressure when the lungs were inflated (Fig. 4). In addition, even though all animals were treated uniformly, the variability among animals was substantial. The uneven distribution of pleural pressure within the thorax is most likely due to uneven chest wall shapes, so that the chest wall compression was not identical in all dogs. Differences in abdominal compliance may also explain the large variability in esophageal pressures. Although we have no clear evidence to support this proposition, it is worth mentioning that the dog that had the lowest increases in intrathoracic pressure had recently delivered puppies and had a very lax abdominal wall.

The uneven distribution of pressure along the esophagus presents a potential problem in using lower esophageal pressure as the intrathoracic pressure measurement for calculating transpulmonary pressure (distending pressure of the lung). The apices of the lung will be surrounded by lower pleural pressure, but the airway pressure will presumably be identical throughout the bronchial tree. Therefore, the transpulmonary pressure will be much higher in the upper lung, and the risk of barotrauma will be underestimated.

Another corollary of the uneven distribution of pressures among and within animals is that compressive forces applied to the most compliant region of the chest wall (that is, the abdomen) should result in larger and more uniform changes in thoracoabdominal pressure. This compressive force would most logically be applied by a circumferential belt, rather than be limited to the ventrodorsal plane used in the prelimi-
nary fashion in this study.

Admittedly, our results cannot be directly extrapolated either to human or animal CPR. Cardiopulmonary resuscitation is a dynamic process, while all of our measurements were made in the static situation. We believe that static pressure applications of the thorax are responsible for the low intrathoracic pressures that we have found in all maneuvers except chest compression with pulmonary inflation. We have hypothesized that during dynamic situations the abdominal viscera probably support the diaphragm transiently as it resists the forces applied to it during chest compression, and, thus, larger intrathoracic pressures are produced. In four dogs, we applied repetitive manual chest compressions at a frequency of one per second against an open airway and with a closed airway and produced esophageal pressures that were double those we found with sustained chest compression.

The mechanism of blood flow during cardiopulmonary resuscitation is still controversial, but it would seem that increased intrathoracic pressure cannot but help increase blood flow out of the thorax. Simple modifications of the standard technique, such as closing the airway, inflating the lungs, and abdominal compression will increase intrathoracic pressure.

REFERENCES