TABLE 1. Blood Gas Values during CPR

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>PA</th>
<th>A</th>
<th>PA</th>
<th>A*</th>
<th>CV*</th>
<th>A*</th>
<th>CV*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (min)†</td>
<td>2</td>
<td>2</td>
<td>5</td>
<td>6</td>
<td>3–20</td>
<td>3–20</td>
<td>3–20</td>
<td>3–20</td>
</tr>
<tr>
<td>Bicarbonate (mEq)</td>
<td>—</td>
<td>—</td>
<td>50</td>
<td>50</td>
<td>—</td>
<td>—</td>
<td>3–20</td>
<td>3–20</td>
</tr>
<tr>
<td>pH</td>
<td>7.10</td>
<td>6.99</td>
<td>7.60</td>
<td>7.23</td>
<td>7.36</td>
<td>—</td>
<td>7.01</td>
<td>7.27</td>
</tr>
<tr>
<td>Pco2 (mmHg)</td>
<td>26</td>
<td>42</td>
<td>44</td>
<td>110</td>
<td>27.5</td>
<td>76.4</td>
<td>45.7</td>
<td>64.8</td>
</tr>
<tr>
<td>Po2 (mmHg)</td>
<td>148</td>
<td>16</td>
<td>48</td>
<td>13</td>
<td>115.9</td>
<td>17.4</td>
<td>107.9</td>
<td>29.6</td>
</tr>
</tbody>
</table>

A = arterial; PA = pulmonary artery; CV = central venous.  
* Average of values from five patients.  
† Time blood gas drawn after CPR initiated.

they are venous, are not compatible with endotracheal intubation even in the presence of rudimentary CPR. "I can say with absolute certainty there was cessation of oxygen delivery into this woman's lungs." "You would find these blood gases in corpse blood taken at autopsy."

An example of a blood gas in question, drawn from a vessel in the groin, is pH 7.11, Pco2 68 mmHg, and Po2 17 mmHg. Apparently these experts were unaware of several studies that demonstrate these values to be consistent with a typical venous blood gas analysis obtained during CPR. A comparison of simultaneously obtained arterial and venous (central venous or pulmonary artery) gases is presented in Table 1. Venous blood gases obtained during CPR performed according to the current standards show profound hypoxemia and severe hypercarbia in the presence of a properly placed endotracheal tube and proper ventilation.

Anesthesiologists have a right and even a duty to serve as plaintiffs' experts. But our paramount responsibility is to be accurate, reasonable, and noninflammatory. Ignorance of the literature by the above-quoted experts made life miserable for two anesthesiologists who deserved a better fate.

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REFERENCES


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More about the Mechanisms of Diaphragm Displacement during Spontaneous Breathing

To the Editor.—As the topic of diaphragm movement has been extensively discussed in Anesthesiology, we wish to summarize some of our findings and bring to the attention of Anesthesiology readership the controversial issue of diaphragmatic shape/tension relationship. During spontaneous breathing the dependent part of the diaphragm in the supine human is displaced more than the nondependent regions in the craniocaudal direction. The proposed mechanism for this observation is that the vertical hydrostatic gradient in abdominal pressure is not matched by the hydrostatic gradient in pleural pressure over the diaphragmatic surface. This may result in a vertical gradient in transdiaphragmatic pressure, which creates a more passive stretch in dependent diaphragm. A larger displacement of dependent regions is attributed to the more optimal force/length and shape/tension relationships. Both mechanisms were reiterated as important for diaphragm mechanics during spontaneous breathing until Kim et al. reported that the shape changes were unimportant for diaphragm mechanics over the range of a normal tidal breath. Our data again give a significant functional importance to the diaphragm shape/tension properties as well as to the anatomic characteristics. Although there are substantial differences between the shape of the human (elliptical) and dogs (triangular) rib cage, we believe that valuable information can be deduced by studying the dog model.

By using a computer-based blipale videoroentgenographic technique, we determined diaphragm displacement, regional shortening, and shape changes during spontaneous breathing in supine and prone anesthetized dogs from the spatial position of surgically implanted markers in four diaphragm regions (fig. 1). This marker technique has been described elsewhere. Figure 1 shows the actual diaphragm displacement in one dog in two planes during spontaneous breath (see details in fig 1 legend). In the supine position, the costal–ventral (CoV) region is oriented nondependently; the costal–middle (CoM) region is oriented between dependent and nondependent regions; and the costal–dorsal (CoD) and crural (Cr) are dependent regions. In the supine position during spontaneous breathing, the dependent diaphragm (Cr, CoD, CoM) was displaced more in the craniocaudal axis than was the nondependent region (CoV) (table 1). This nonuniform craniocaudal displacement was not affected by the hydrostatic pressure change after the dogs were turned from supine to prone posture. In the prone posture, nondependent (Cr, CoD, CoM) regions displaced again more than the dependent (CoV) region. This is in accordance with Krayer et al. who stated that "the dominant influence on diaphragm motion may be some anatomic difference between the crural and costal diaphragm regions rather than the hydrostatic pressure gradient".  

As a qualitative assessment of change in diaphragm shape we analyzed
FIG. 1. Actual sagittal (xz) and coronal (zy) radiographic projection of 17 diaphragm markers at FRC and at the end of spontaneous breath. Solid lines denote position of markers at FRC. Dashed lines show position of markers at the end of spontaneous breath. Markers labeled 1, 6, 11, and 15 are on the origin of diaphragm at rib cage, and markers labeled 4, 9, 14, and 17 are at diaphragm muscle insertions at the central tendon. Arrows show direction of marker displacement during tidal breath, as they project in a respective plane. Note the large marker displacement in the caudal (−z) direction, and less displacement in the ventral (−x, A) or lateral (+y, B) plane. It is difficult to define the single best plane of marker projection in a region during a tidal breath, as the orientation of plane changes, frequently in all three dimensions, because of the specific, sometimes spiral, orientation of diaphragm muscle bundles. Reference Cartesian coordinate system and rows of diaphragm markers are schematically indicated on the dog's silhouette.

the relationship between shortening and displacement of the diaphragm regions. For this analysis displacement of the marker adjacent to the central tendon was computed relative to the marker near the rib cage (corrected displacement) (fig. 2). We reasoned that if the radius of curvature of the diaphragm increased, i.e., if the diaphragm became straighter with inspiration, that displacement would be less than shortening (fig. 2A). If the curvature did not change, shortening could equal displacement (fig. 2B). Finally, if the radius of curvature of the diaphragm decreases with inspiration, displacement would exceed shortening (fig. 2C). Any other combination outside 2A, 2B, or 2C is excluded by the fact that the central tendon does not shorten or lengthen and that the markers near the chest wall had only minimal displacements away from the midline in the normal tidal volume range. This concept was tested in vivo, and the result of analysis of the displacement/shortening ratios is shown in table 1. Except in the costal-ventral region, displacement always exceeded shortening (P < 0.05), which is consistent with the mechanical concept proposed in figure 2C. In accordance with Laplace's law (which relates membrane tension and curvature to pressure), our data suggest that the regional shape change is an important factor in the diaphragm tension/transdiaphragmatic pressure relationship and thus diaphragm displacement.

The mechanics of the diaphragm remains an enigma and an example of research limitations in answering the question "How does this complex muscle work?". In 1974 Froese and Bryan1 published their classic paper with an ingenious concept of diaphragm displacement based on the simple fluoroscopic observation. Fifteen years later Krayer et al.2 confirmed this finding with expensive computerized three-dimensional scanning technology. Almost thirty years ago Marshall3 developed the concept of the diaphragm "as a part of the sphere" governed by Laplace's law. Kim4 introduced the belief that this concept is unimportant, and that the diaphragm moves uniformly, like a piston. Our work goes back to Marshall's concept, giving credibility to diaphragm shape. Diaphragm mechanics is complex and is not explained by a simple model based on the transdiaphragmatic pressure and the length/tension re-

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**Fig. 2.** Schematic consideration of three hypothetical situations in sagittal (xz) plane that describes the relationship of corrected displacement (Dc) to shortening (Sh) as a potential index of change in regional diaphragm geometry. Our in vivo analysis of Dc/Sh ratios at the end of a spontaneous breath is consistent with the hypothetical situation presented in C, which denotes an increase in diaphragm curvature. The pattern of marker displacement in the costal-middle region in figure 1 (zy plane) resembles the hypothetical situation presented in figure 2C. r_y = Regional radius of curvature at FRC; r_z = regional radius of curvature at the end of spontaneous breath. Reference Cartesian coordinate system is schematically indicated; z axis is cephalocaudal; x axis is vertical (dorsoventral); and y axis (shown on fig. 1) is lateral to mediastinal.
TABLE 1. Absolute Diaphragm Displacements and Ratios Between Corrected Displacements and Shortenings in Four Diaphragm Regions and Two Body Postures

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>Prone</th>
</tr>
</thead>
<tbody>
<tr>
<td>CoV</td>
<td>Displ (nd)</td>
<td>0.72 ± 0.12</td>
</tr>
<tr>
<td></td>
<td>Dc/Sh</td>
<td>1.02</td>
</tr>
<tr>
<td>CoM</td>
<td>Displ (im)</td>
<td>1.19 ± 0.30</td>
</tr>
<tr>
<td></td>
<td>Dc/Sh</td>
<td>1.50</td>
</tr>
<tr>
<td>CoD</td>
<td>Displ (d)</td>
<td>0.91 ± 0.29</td>
</tr>
<tr>
<td></td>
<td>Dc/Sh</td>
<td>1.30</td>
</tr>
<tr>
<td>Cr</td>
<td>Displ (d)</td>
<td>1.06 ± 0.31</td>
</tr>
<tr>
<td></td>
<td>Dc/Sh</td>
<td>1.54</td>
</tr>
</tbody>
</table>

Diopl = absolute displacement expressed as mean ± SD (centimeters); Dc/Sh = corrected displacement/shortening ratio. CoV = costal—ventral; CoM = costal—middle; CoD = costal—dorsal; Cr = crural. Abbreviations in parentheses denote vertical location of the diaphragm region in respective body position: d = dependent; nd = nondependent; im = midheight between dependent and nondependent.

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REFERENCES


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Should a Laryngeal Mask Be Routinely Used in Patients Undergoing Thyroid Surgery?

To the Editor—Recently, Tanigawa et al. described using a combination of a fiberoptic bronchoscope, nerve stimulator, and laryngeal mask for recurrent laryngeal nerve identification and protection in patients undergoing thyroid surgery. Although we have not doubt about the usefulness of being able to assess cord function preoperatively, we do not agree that the technique described should be advocated for widespread use. First, manipulation of the trachea will mean that there is a real risk of the larynx being displaced off the laryngeal mask. This has occurred in our practice on one occasion where the view of the cords was lost, fortunately without loss of airway control. Although it could be argued that the surgeon, directed by a view through the fiberoptic bronchoscope, could reposition the larynx over the mask, this may have to be done under difficult circumstances and may be complicated by a simultaneous risk of disturbing a large pharyngeal mucous plug, which would impair the view and which might compromise the airway on its own account.

Second, we have noticed laryngeal spasm, even though the concentrations of the volatile agent delivered would seem to be more than adequate (enflurane 2% end-tidal). It occurred at some stage or the other in all patients and was often little more than a minor inconvenience. In all patients it appeared to be related to surgical stimulation and stopped within seconds of ceasing surgical manipulation of the larynx. Although we agree that it is eminently feasible to intubate the trachea with a 6-mm endotracheal tube via the laryngeal mask, this may be difficult to perform when the clinical situation is deteriorating and when access to the patients may be restricted because of the nature of the surgery.

Observation of vocal cord function during thyroid surgery is ob-

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