Role of the Heart in the Loss of Aeration Characterizing Lower Lobes in Acute Respiratory Distress Syndrome

LUIZ M. MALBOUISSON,* CORNELIUS J. BUSCH,† LOUIS PUYBASSET, QIN LU, PHILLIPE CLUZEL, JEAN-JACQUES ROUBY, and the CT Scan ARDS Study Group

Réanimation Chirurgicale Pierre Vians, Department of Anesthesiology, and Department of Radiology, Hôpital de la Pitié-Salpêtrière, University of Paris VI, Paris, France

In the acute respiratory distress syndrome (ARDS), lower lobes appear essentially non-aerated in contrast to upper lobes whose aeration can be preserved in some patients. The aim of this study was to assess the mechanical compression exerted by the heart on lower lobes in patients with ARDS. Fourteen healthy volunteers and 38 patients with ARDS free of left ventricular failure were studied. Cardiorespiratory parameters were recorded and the cardiac dimensions, the pressure exerted by the heart on subjacent lower lobes, and the gas tissue ratio of lower lobes in the supine position were measured using computer tomography and Lungview, a specifically designed software. In patients with ARDS, the heart was larger and heavier than in healthy volunteers. The enlargement of the heart was mainly related to a left cardiac protrusion and the pressure exerted by the left heart on the lower lobes was higher in patients with ARDS than in healthy volunteers (8 ± 3 g · cm⁻² versus 6 ± 1 g · cm⁻², p < 0.01). As a consequence, the faction of gas represented 62% of the left lower lobes in healthy volunteers and 12% only in patients with ARDS. The present study demonstrates that apart from the already known anteroposterior and cephalo-caudal gradients of pressure depending on the lung weight and abdominal pressure, the heart plays an important role in the dramatic loss of aeration characterizing lower lobes of patients with ARDS lying in the supine position.

A cute respiratory distress syndrome (ARDS) is characterized by a decrease in FRC, which predominates in the lower lobes (1). Increased lung weight caused by a high permeability type edema leading to a pressure gradient along the anteroposterior axis (2) and cephalic displacement of the diaphragm secondary to anesthesia and increased intra-abdominal pressure (1, 3, 4) have been proposed as mechanisms explaining the loss of aeration predominating in lower lobes. Early alteration in the surfactant function and the decrease in transpulmonary pressure physiologically observed in the dependent and caudal regions of the rib cage in supine position (5) also likely contribute to atelectasis formation in the lower lobes. In the supine position, the major part of the left lower lobe and a significant part of the right lower lobe are located beneath the heart. U p to now, the influence of the heart on lung aeration in patients with A R D S lying supine has never been specifically addressed. Mechanical compression of intrathoracic structures by enlarged heart has been described in infants with congenital heart disease. Several reports have demonstrated that an enlarged left atrium can compress bronchi and induce left lower lobe atelectasis (6, 7). Clinical studies using radioisotopic ventilation-perfusion techniques performed in supine adults with cardiomegaly showed a decrease in lower lobe’s ventilation predominating on the left side (8, 9) that was reversed in prone position (10, 11). In these studies, the decrease in the ventilation of the lower lobe was caused by a direct lung compression by the heart rather than by a direct compression of the left bronchi. The aim of this study was to evaluate the contribution of the heart to mechanical compression atelectasis of the lower lobes in patients with A R D S.

METHODS

Study Population
Thirty-eight patients with early A R D S as defined by the American-European Consensus Conference (12) were prospectively studied. Inclusion criteria were a PaO₂ < 200 mm Hg at fraction of inspired oxygen (FIO₂) of 1 and the presence of bilateral pulmonary infiltrates. Exclusion criteria were a past history of cardiac disease and/or acute ventricular failure defined either as a cardiac index < 2.5 L · min⁻¹ · m⁻² associated with a pulmonary capillary wedge pressure (PcPcw) > 18 mm Hg and/or a left ventricular ejection fraction < 50%. Informed consent was obtained from the patient’s next of kin. Partial results concerning the computed tomographic (CT) scan of 15 of these 38 patients have been already reported in five studies (1, 13–16). However, Lungview was not available at this time and CT scans of these patients were reanalyzed for the present study.

Fourteen spontaneously breathing volunteers were also studied to assess the physiological influence of the heart on the aeration of the lower lobes in the supine position. Each healthy volunteer was an investigator of the present study and signed a written informed consent. The results concerning 11 healthy volunteers have been partially reported in two recent studies (1, 15).

Study Design
During the study period, all patients were sedated and paralyzed with a continuous intravenous infusion of fentanyl, midazolam, and vecuronium and were ventilated using controlled mechanical ventilation (Cesar V ventilator, Taema, France). All II patients were monitored using a fiberoptic thermoligation pulmonary artery catheter and a radial or femoral arterial catheter. Hemodynamic and respiratory parameters were measured within 24 h of a thoracic CT scan that was performed as an integral part of the respiratory management of each patient.

Hemodynamic and Respiratory Measurements
Throughout the study period, systemic and pulmonary arterial pressures, heart rate (H R), central venous pressure (Pcv), PcPcw, tracheal pressure (Paw) measured at the proximal end of the endotracheal...
tube, gas flow, and tidal volume (VT) measured using a heated and calibrated Hans Rudolph 3700 pneumotachograph (Hans Rudolph, Inc., Kansas City, MO) were recorded at a high sample rate of 100 Hz on a data acquisition and analysis system including MP100 WS data acquisition system (Biopac Systems Inc., Goleta, CA) and a Quadra 610 Macintosh computer (Apple Computer Inc., Cupertino, CA) connected to the analog part of the hemodynamic monitor Merlin (Hewlett-Packard, Palo Alto, CA). Cardiac output was measured using either the thermodilution technique (Explorer SVO2/CO Computer; Baxter SA, Maurepas, France) as previously described (17) or the semicontinuous thermodilution technique (CCO/SVO2/VIP TD catheter; Baxter SA). Systemic and pulmonary arterial blood samples were simultaneously withdrawn within 1 min after the measurement of cardiac output. Arterial pH, PaO2, partial oxygen pressure in mixed venous blood (PvO2), PacO2, hemoglobin concentration, and arterial and mixed venous oxygen saturations were measured using a 1L BGE blood gas analyzer (Instrumentation Laboratory, Norwood, MA) and a calibrated OSM3 hemoximeter (Radiometer Copenhagen, Neuilly-Plaisance, France). Standard formulas were used to calculate cardiac index, pulmonary and systemic vascular resistance indices, stroke work indices, pulmonary shunt, oxygen delivery and consumption. Left ventricular ejection fraction was measured in 33 of the 38 patients by means of transesophageal echocardiography (Hewlett-Packard 77025A ultrasound system; Hewlett-Packard, Andover, MA).

Thoracic CT Scan Protocol

Aquisition of the CT sections. The patients were transported to the Department of Radiology by two physicians, using an Osiris transport ventilator (Taema, France) delivering a PEEP of 1. Electrocardiogram (ECG), systemic arterial pressure, and hemoglobin oxygen saturation were continuously monitored with a Propaq 104 EL monitor (Protocoll System, North Chicago, IL). Images were obtained by a Tomoscan SR 7000 spiral tomograph (Philips, Eindhoven, The Netherlands), with exposures taken at 120 kV and 250 mA s. All images were photographed at a window width of 1,600 Hounsfield units (HU) and a level of ~700 HU. The value of the pitch was 1. CT scans were obtained at end-expiration after disconnecting the patient from the ventilator. The spiral thoracic CT scan consisted of contiguous 10-mm-thick CT sections reconstructed from the volumetric data obtained during a 15-s period of apnea and of high-definition 1.5-mm thin CT sections obtained at intervals of 2 cm that were all selected by means of a thoracic scout view. CT scan acquisitions were performed with patients in the supine position. The 14 normal subjects underwent only a series of 10-mm-thick CT sections that were sufficient for accurately delineating heart, upper and lower lobes. As in patients, CT sections were obtained at the end of a normal expiration (FRC). The reconstructed images were recorded on optical disks for later analysis. All the 10-mm-thick CT sections were used for the calculation of thoracic dimensions.

Figure 1. Schematic representation of a CT section obtained in supine position. RUL = right upper lobe; LUL = left upper lobe; RLL = right lower lobe; LLL = left lower lobe. Each lower lobe was divided in two regions: lower lobe beneath the heart (gray area) and lower lobe located outside the heart limits (white area). The thoracic dimensions measured are represented by the small letters: (a) right anteroposterior sagittal distance; (b) left anteroposterior sagittal distance; (c) maximal thoracic transverse distance; (d) sternovertebral distance. Cardiac dimensions are represented by the left and right protrusions of the heart as indicated by the transverse white arrows superimposed on the cardiac silhouette.

Figure 2. (Upper left panel) Linear regression between the heart weight measured by Lungview and the actual heart weight measured using an electronic scale of four animal’s hearts. The dotted line represents the identity line; the solid line represents the linear regression line. (Upper right panel) Graphic representation of the Bland and Altman method. The solid line represents the mean value; the dotted lines represent 2 standard deviations (SD). (Left lower panel) Linear regression between the heart volume measured by Lungview and the actual heart volume measured from four animal’s hearts. The dotted line represents 2 standard deviations (SD). (Right lower panel) Linear regression between the heart volume measured by Lungview and the actual heart volume measured from four animal’s hearts. The dotted line represents 2 standard deviations (SD).
### HEMODYNAMIC PROFILE OF PATIENTS WITH ARDS

<table>
<thead>
<tr>
<th></th>
<th>Without Shock</th>
<th>Shock</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td>93.6 ± 21.3</td>
<td>100.1 ± 19.2</td>
<td>NS</td>
</tr>
<tr>
<td>Pa, mm Hg</td>
<td>79.9 ± 16.5</td>
<td>79.3 ± 13.7</td>
<td>NS</td>
</tr>
<tr>
<td>Pcv, mm Hg</td>
<td>6.4 ± 3.4</td>
<td>8.2 ± 4.1</td>
<td>NS</td>
</tr>
<tr>
<td>Ppa, mm Hg</td>
<td>26.9 ± 7.7</td>
<td>28.3 ± 6.4</td>
<td>NS</td>
</tr>
<tr>
<td>Ppocw, mm Hg</td>
<td>7.2 ± 3.1</td>
<td>10.2 ± 9.0</td>
<td>0.01</td>
</tr>
<tr>
<td>CI, L·min⁻¹·m⁻²</td>
<td>3.8 ± 1.8</td>
<td>4.0 ± 1.7</td>
<td>NS</td>
</tr>
<tr>
<td>SVWI, g</td>
<td>40.4 ± 17.0</td>
<td>39.7 ± 13.0</td>
<td>NS</td>
</tr>
<tr>
<td>PVRI, dyn·cm⁻¹·m⁻²</td>
<td>1,831.5 ± 917.5</td>
<td>1,649.3 ± 715.7</td>
<td>NS</td>
</tr>
<tr>
<td>LVSWI, g·min⁻¹·m⁻²</td>
<td>40.9 ± 21.7</td>
<td>34.9 ± 9.8</td>
<td>NS</td>
</tr>
<tr>
<td>RVSWI, g·min⁻¹·m⁻²</td>
<td>11.1 ± 5.6</td>
<td>10.8 ± 9.4</td>
<td>NS</td>
</tr>
<tr>
<td>VO₂, mL·min⁻¹·m⁻²</td>
<td>430.4 ± 177.5</td>
<td>467.8 ± 169.7</td>
<td>NS</td>
</tr>
<tr>
<td>VO₂, mL·min⁻¹·m⁻²</td>
<td>118.8 ± 36.0</td>
<td>124.3 ± 47.2</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Definition of abbreviations:** CI = cardiac index; D O₂ = oxygen delivery; HR = heart rate; LVSWI = left ventricular systolic work index; NS = nonsignificant; Pa = mean systemic arterial pressure; Pcv = central venous pressure; Ppa = mean pulmonary arterial pressure; Ppocw = pulmonary capillary wedge pressure; PVRI = pulmonary vascular resistance index; RVSWI = right ventricular systolic work index; VO₂ = oxygen consumption.

All hemodynamic parameters were measured on the same day of the CT scan. Data are expressed as mean ± SD. The variables were compared by an unpaired Student’s t test.

mm-thick sections’ films (parenchymal windows) were analyzed by an independent radiologist (P.C.), who manually delineated the heart contours, the lung parenchyma, and the left and right main fissures. These anatomical delineations were performed by taking into consideration the anatomical landmarks present in the 1.5-mm-thick sections (Figure 3). The number of pixels within the manually delineated heart boundaries was determined in each consecutive CT section where the heart could be identified. For each pixel, the CT number expressed in HU, representing the attenuation coefficient of the chest X-ray by the structure being studied minus the attenuation coefficient of water divided by the attenuation coefficient of water, was measured. The pixels were distributed along 256 compartments located between −56 HU and 200 HU, each compartment representing a 1-HU interval. For each compartment of a known CT number, the volume of the overall heart was computed according to the following formulas:

**Volume of the voxel**

\[ \text{Volume of the voxel} = \text{pixel area} \times \text{section thickness} \]  

**Volume of the compartment**

\[ \text{Volume of the compartment} = \text{number of voxels} \times \text{volume of the voxel} \]  

**Mass of the compartment**

\[ \text{Mass of the compartment} = (1 + \text{CT}/1,000) \times \text{volume of the compartment} \]

The calculation of the mass is based on the tight correlation existing between radiological and physical densities (18, 19). The mass and the volume of the overall heart were computed by adding all the partial masses and volumes of the “cardiac” compartments measured on the consecutive CT sections.

Estimation of the pressure exerted by the heart on the lower lobes. On the CT section located 5 cm below the tracheal carina, the volume and the mass of the left and right cardiac areas located above the lungs were determined (Figure 1). The mean pressure exerted by the heart on the left and right lower lobes was obtained by dividing the cardiac pressure by the volume of the overall heart.

![Figure 3](image-url)
mass located above the left and right lower lobes by the surface of the corresponding main fissura.

Determination of the volumes of lower lobes (gas and tissue). On the CT section located 5 cm below the tracheal carina, the lower lobes were delineated and subdivided into two compartments either located beneath or outside the heart (gray and white areas in Figure 1). On each CT section, the volumes of gas and tissue and the fraction of gas of each compartment were computed using the following equations:

Volume of gas = \((-\text{CT}/1,000) \times \text{Total volume}\) if the compartment considered has a CT number above 0

Volume of tissue = \((1 + \text{CT}/1,000) \times \text{Total volume}\) if the compartment considered has a CT number below 0 or

\[
\text{Volume of tissue} = \text{number of voxels} \times \text{volume of the voxel if the compartment considered has a CT number above 0}\]

\[
\frac{\text{Fraction of gas}}{\text{Volume of gas}} = \frac{\text{Volume of gas}}{\text{Total volume}}
\]

The volume of gas and tissue and the fraction of gas of each compartment were calculated by adding all values determined on each CT section.

Validation of cardiac volume and mass measurements using Lungview. The measurement of the volume and mass of the heart using Lungview was validated by performing the CT scans of isolated heart of four animals (lamb, pig, calf, and beef) with different known volumes (evaluated as the displacement of an equivalent volume of water) and weights (electronic scales) placed within cardboard boxes. Using the Bland and Altman method, the mean bias and precision of cardiac volume measurement were 28 and 50 ml, respectively, and the mean bias and precision of cardiac mass measurement were 217 g and 23 g, respectively. The equations of the linear regressions are shown in Figure 2.

Statistical Analysis

The demographic data, the volume and mass of the heart, and the different lung volumes were compared between normal subjects and patients with ARDS using an unpaired Student's t test. An unpaired Student's t test was also performed to compare the hemodynamic parameters, the heart's volume and mass in patients with ARDS with and without septic shock. The cardiac and thoracic dimensions and the fraction of gas of the parts of left and right lower lobes located beneath and outside the heart were compared between normal subjects and patients with ARDS using a two-way analysis of variance for repeated measures followed by a Student-Newman-Keuls test when indicated. All correlations were made by means of a linear regression analysis. The statistical analysis was performed using SigmaStat 2.03 statistical software (SPSS Inc., San Rafael, CA) and Statview 5.0 (SAS Institute Inc., Cary, NC). All data are expressed as mean ± SD unless otherwise specified. The statistical significance level was fixed at 0.05.

RESULTS

Study Population

Fourteen normal subjects (5 females, 9 males) and 38 patients with ARDS (8 females, 30 males) were studied. The patients were older than normal subjects (56 ± 17 yr versus 33 ± 9 yr, p < 0.001) and their weight was also greater (77.6 ± 12.6 kg versus 67.3 ± 9.1 kg, p = 0.009). However, their height was similar (171 ± 7 cm versus 172 ± 9 cm). ARDS was secondary to postoperative pulmonary infection (n = 17), aspiration pneumonia (n = 7), septic shock from non-pulmonary origin (n = 7), lung contusion (n = 6), and cardiopulmonary bypass.
2.7. The mean delay between the onset of ARDS and the inclusion in the study was 4.2 ± 3.0 days. Thirty-three of the 38 patients studied had a transesophageal echocardiography (TEE) performed within the 48 h preceding or following the transportation to the CT scanner. The mean left ventricular ejection fraction was 62 ± 10%. The lung injury severity score (20) 2.7 ± 0.5; the Pao2, was 91 ± 45 mm Hg with F102, 1, true pulmonary shunt was 47 ± 11%, and the quasi-static compliance measured in ZEEP was 49 ± 28 ml·cm H2O⁻¹.

Twenty-one patients had a septic shock, defined according to reference criteria (21). All patients were treated with norepinephrine at a mean dose of 3.2 mg·h⁻¹. As illustrated in Figure 3, the sternovertebral and the anteroposterior dimensions were significantly increased in patients with ARDS when compared with normal subjects. As shown in Figure 4, the maximal transverse cardiac dimension was greater in patients with ARDS than in normal subjects (p < 0.05) in all CT sections except the most caudal. The enlargement of the patient's heart was related to both a left cardiac protrusion and an anteroposterior enlargement. As shown in Figure 5, the cardiac volume and mass of patients with ARDS were significantly greater than in normal subjects. No differences were observed between patients with and without septic shock.

**Heart-Lung Interdependence**

The pressure exerted by the right and left heart on subjacent lower lobes was significantly greater in patients with ARDS than in normal subjects (Figure 6). As shown in Tables 2 and 3, the overall volumes of left and right lower lobes were significantly reduced in patients with ARDS when compared with normal subjects. This reduction was secondary to a massive loss of gas associated with a significant increase in tissue volume (p < 0.001). In the normal subjects, the fraction of gas of the left lower lobe represented 62% of the overall lung volume and the fraction of gas of the right lower lobe represented 65% of the overall lung volume. These values were respectively 12% and 18% in patients with ARDS (p < 0.001). As shown in Figure 6, the proportion of the left and right lower lobes located beneath the heart was greater in patients with ARDS than in normal subjects, although the difference did not reach statistical significance. The fraction of gas was significantly lower (Figure 7), both in patients with ARDS and normal sub-

---

**Table 2**

<table>
<thead>
<tr>
<th>Volume (mL)</th>
<th>Normal Subjects (n = 14)</th>
<th>ARDS Patients (n = 38)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left lower lobe</td>
<td>50.7 ± 13.5</td>
<td>38.5 ± 16.8</td>
<td>0.018</td>
</tr>
<tr>
<td>Gas volume</td>
<td>31.6 ± 11.2</td>
<td>5.0 ± 6.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Tissue volume</td>
<td>19.1 ± 5.1</td>
<td>33.5 ± 13.6</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LLL segment below the heart</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall volume</td>
<td>26.2 ± 8.9</td>
<td>21.9 ± 9.0</td>
<td>NS</td>
</tr>
<tr>
<td>Gas volume</td>
<td>14.5 ± 6.0</td>
<td>1.2 ± 1.3</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Tissue volume</td>
<td>11.7 ± 4.5</td>
<td>20.8 ± 8.7</td>
<td>0.001</td>
</tr>
<tr>
<td>LLL outside the heart limits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall volume</td>
<td>24.5 ± 11.3</td>
<td>16.6 ± 15.8</td>
<td>NS</td>
</tr>
<tr>
<td>Gas volume</td>
<td>17.1 ± 8.6</td>
<td>4.0 ± 5.9</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Tissue volume</td>
<td>7.3 ± 3.5</td>
<td>13.5 ± 12.2</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Definition of abbreviations: LLL = left lower lobe; NS = nonsignificant.

Data are expressed as mean ± SD. All values were compared by means of unpaired Student's t test.

---

**Table 3**

<table>
<thead>
<tr>
<th>Volume (mL)</th>
<th>Normal Subjects (n = 14)</th>
<th>ARDS Patients (n = 38)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right lower lobe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall volume</td>
<td>65.4 ± 11.4</td>
<td>52.2 ± 16.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Gas volume</td>
<td>42.2 ± 10.4</td>
<td>9.6 ± 9.9</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Tissue volume</td>
<td>23.2 ± 4.1</td>
<td>42.6 ± 13.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>RLL segment below the heart</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall volume</td>
<td>12.7 ± 4.5</td>
<td>10.8 ± 5.3</td>
<td>NS</td>
</tr>
<tr>
<td>Gas volume</td>
<td>7.1 ± 2.8</td>
<td>1.2 ± 1.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Tissue volume</td>
<td>5.6 ± 2.3</td>
<td>9.6 ± 4.9</td>
<td>0.005</td>
</tr>
<tr>
<td>RLL outside the heart limits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall volume</td>
<td>52.6 ± 10.1</td>
<td>41.4 ± 15.5</td>
<td>0.014</td>
</tr>
<tr>
<td>Gas volume</td>
<td>35.1 ± 9.3</td>
<td>8.4 ± 9.1</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Tissue volume</td>
<td>17.6 ± 3.4</td>
<td>33.0 ± 12.8</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Definition of abbreviations: RLL = right lower lobe; NS = nonsignificant.

Data are expressed as mean ± SD. All values were compared by means of unpaired Student's t test.

---

**Thoracic and Cardiac Dimensions**

As shown in Figure 3, the sternovertebral and the anteroposterior sagittal dimensions were significantly increased in patients with ARDS when compared with normal subjects. As illustrated in Figure 4, the maximal transverse cardiac dimension was greater in patients with ARDS than in normal subjects (p < 0.05) in all CT sections except the most caudal. The enlargement of the left lower lobe represented 62% of the overall lung volume.
jects, in the lung areas located beneath the heart in comparison to the lung areas located outside the heart limits on both sides. However, the fraction of gas of the lung area located beneath the heart was more reduced in patients with ARDS than in healthy volunteers: −73% versus −21% (p < 0.001).

DISCUSSION
This study shows that the cardiac volume is increased in patients fulfilling the criteria of ARDS despite the absence of left ventricular failure. As a consequence, the pressure exerted by the heart on the dependent lung regions significantly increases and dramatically reduces the fraction of gas of lower lobes.

Methodological Considerations
Accuracy of CT measurements depend on several factors: the accurate delineation of heart boundaries, the presence of a partial volume effect occurring when the cephalic and caudal parts of the structure being studied are only partially present on the CT section, and motion artifacts related to respiratory movements and heart beats. Respiratory movements were suppressed by acquiring images at end-expiration. Partial volume effect was minimized by limiting the CT section thickness to 1 cm. The cardiac volume and weight of different animals were measured by the software Lungview with an error of 3.5%. Using the same methodology, Breiman and coworkers measured the volume of the spleen preoperatively with an error of 3.6% (22). Despite the high accuracy of volume, weight (18), and diameter measurements from fast spiral CT scan reconstructions, some physiological and methodological limitations must be kept in mind. The tomographic evaluation of heart provides static estimations of ever-changing dimensions of a dynamic structure. The cardiac dimensions were measured during a period of 15 s necessary for acquiring CT images. Depending upon the moment of the cardiac cycle when the section was acquired, the heart volume, weight, and dimensions vary and the data obtained represent mean values of cardiac dimensions. The pressure exerted by the heart on the lower lobes was estimated on one CT section by dividing the mass of the heart zone by the subjacent surface of the main fissura. This estimation is a very simplistic view ignoring the non-orthogonal forces exerted by the heart on the pericardium surface or pulmonary parenchyma, as well as the influence of the heart beats on lung movements and compression (23).

For obvious ethical reasons, the control group was composed of healthy volunteers spontaneously breathing and not of anesthetized patients on mechanical ventilation and free of acute lung injury. It can be hypothesized that the active contraction of the respiratory muscles tends to balance the pressure exerted by the heart on the lower lobes. When the diaphragm is paralyzed during anesthesia, it is likely that the pressure exerted by the heart is no longer counterbalanced and contributes, with the upward shift of the diaphragm, to alveolar collapse of the more dependent lung segments. A control group composed of mechanically ventilated patients would have likely shown a greater loss of aeration in the infracardiac portion of the lower lobes.

Mechanisms of Increased Cardiac Mass in ARDS
In ARDS, several mechanisms may explain the increase in cardiac mass: edema of the cardiac walls, right ventricle dilation secondary to pulmonary hypertension, and hyperkinetic state related to generalized inflammation. Despite a similar height, the patients with ARDS had a weight that was 10 kg greater than the normal subjects. Because no obese patients were included, this difference was likely due to accumulation of extravascular fluid secondary to capillary leak syndrome and substantial fluid loading. In the context of generalized edema related to an alteration in vascular permeability (24, 25), myocardial edema has been shown to be present during septic shock (26). Such a mechanism likely contributed to the in-
creased cardiac mass observed since 55% of the patients had a septic shock associated with their ARDS. A cardiac dysfunction, characterized by a depressed myocardial contractility and cardiac dilation, is also known to occur during sepsis (27, 28). In the present study, left ventricular dysfunction was ruled out and the majority of patients had an elevated cardiac index associated with decreased systemic vascular resistance and pulmonary hypertension. The hyperdynamic hemodynamic profile was likely related to systemic vasodilation and fluid loading (29), and pulmonary hypertension resulted in right ventricular dilation and pooling of blood within right cardiac chambers (30). Very likely, increased cardiac mass and volume were resulting from a combination of these different mechanisms, the importance of which may vary from one patient to another.

Consequences of the Increased Heart Mass on the Lower Lobes

The increase in heart volume resulted in an increase in the transverse dimensions of the left heart and in the proportion of lung parenchyma being located beneath the heart. In normal humans and animals, the pressure exerted by the heart on the lower lobes has a significant influence on the aeration of the subjacent lung. Experimental evidence suggests that the heart is involved in the genesis of the vertical gradient of transpulmonary pressure in physiological conditions (31, 32). Hyatt and coworkers evaluated the distribution of transpulmonary pressure along the vertical axis in head-up dogs (33). The transpulmonary pressure was computed as the difference between the tracheal and the esophageal pressure measured in the esophagus with six balloons placed in series at different heights, from the pulmonary apex to the diaphragm. The physiological vertical gradient of transpulmonary pressure increased when the weight of the heart was increased by replacing blood by an equal volume of mercury. The progressive decline of transpulmonary pressure toward the dependent regions was related to a progressive increase in the pleural pressure secondary to the increase in heart's weight (33).

In patients with ARDS, cardiac mass was increased by 27% in comparison to normal subjects (Figure 8). A grouping with the results of Hyatt and coworkers, this increased cardiac mass likely resulted in an increased pleural pressure in the dependent part of the lungs and contributed to the alveolar collapse observed in lower lobes. A nonincreased pressure exerted by the enlarged heart on the subjacent lung parenchyma was also involved in this loss of aeration. Such a mechanism has been reported in patients with cardiomegaly lying supine in whom a 50% reduction of the lower left lobe ventilation was observed compared with the right side (10). A lehander and coworkers, studying isoxygen ventilation-perfusion lung scans in patients with cardiomegaly, described a 40% reduction of the left lower lobe ventilation in supine position with no concomitant reduction in regional perfusion (11).

Mechanical Factors Compressing Lower Lobes in ARDS

In ARDS, the lower lobes are mechanically compressed by the excessive weight of the edematous lung (2), by the cephalic displacement of the diaphragm (1), and by the increased cardiac mass. The additive effects of these three mechanisms likely explain the dramatic loss of aeration characterizing lower lobes of all patients with ARDS. The increased weight of the edematous lung creates a superimposed gradient of hydrostatic pressure, which leads to a progressive reduction of the transpulmonary pressure in dependent lung areas (2) that is also likely involved in this loss of aeration. Sedation and muscle relaxation suppress the diaphragmatic tonic activity. The weight of the abdominal contents, acting against the flaccid diaphragmatic wall, generates an increase in the abdominal pressure which is predominantly transmitted to the caudal and dependent lung regions, that in turn, leads to a cephalic displacement of posterior regions of the diaphragm (3). This asymmetric upward shift of diaphragm induces an increase in pleural pressure in the most caudal and dependent diaphragmatic regions, contributing to formation of atelectasis in the adjacent region of the lower lobes (34). Although not tested in the present study, the relief of the pressure exerted by the heart on the lower lobes might play a significant role in the beneficial effects of the prone position. In prone position, the weight of the heart is supported by the sternum instead of the lower lobes (34) and the anteroposterior gradient of the fraction of gas is reduced compared with the supine position (36). In the two studies that have evaluated the regional distribution of ventilation in patients with cardiomegaly, the prone position reestablished the normal ventilation of the lower lobes (10, 11).

In conclusion, the present study demonstrates that apart from the already known anteroposterior and cephalocaudal gradients of pressure depending on the lung weight and abdominal pressure, the heart also plays a significant role in the dramatic loss of aeration characterizing lower lobes of patients with ARDS.

References


