The Prone Position Eliminates Compression of the Lungs by the Heart

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The prone position improves gas exchange in many patients with ARDS. Animal studies have indicated that turning prone restores ventilation to dorsal lung regions without markedly compromising ventral regions. To investigate a potential mechanism by which this might occur, the relative volume of lung located directly under the heart was measured in the supine and prone positions in seven patients. Four axial tomographic sections between the carina and the diaphragm were analyzed (Sections 1 through 4). When supine, the percent of the total lung volume located under the heart increased from 7 ± 4% to 42 ± 8%, and from 11 ± 4% to 16 ± 4% in Sections 1 through 4, in the left and right lungs, respectively. When prone, the percent of left and right lung volume located under the heart was ≤ 1 and ≤ 4 %, respectively, in all four sections (p < 0.05 for each section, supine versus prone). Although a large fraction of the lung, particularly on the left, is located directly under the heart in supine patients, and would be subject to the compressive force resulting from heart weight, almost no lung is located under the heart when patients are prone and the compressive force of the heart is directed towards the sternum.

The beneficial effects of the prone position on lung function were first postulated in 1974 (1), demonstrated in patients with the acute respiratory distress syndrome (ARDS) a few years later (2, 3), and confirmed more recently in a number of institutions throughout the world (4–8). We investigated the mechanism by which this improvement might occur and found that turning prone had limited effects on the distribution of regional perfusion (9) but markedly improved dorsal lung ventilation and, accordingly, also improved dorsal lung ventilation-perfusion relationships, with minimal if any compromise of ventral lung ventilation or ventral ventilation-perfusion relationships (10). The implications of these findings are that reversible air-space closure occurs in dorsal lung regions when patients with ARDS are supine, and that turning prone sufficiently alters dorsal lung transpulmonary pressures to reverse this closure without shifting the air-space closure to the ventral regions.

A number of factors could contribute to this differential ability of the prone position to alter dorsal lung transpulmonary pressures, including, among others, the compressive effects of consolidated lung (11, 12), direct transmission of the weight of abdominal contents to caudal regions of the dorsal lung (1, 13, 14), and direct transmission of the weight of the heart to the regions lung located beneath it (15–22). The purpose of this study was to determine the fraction of lung that might be subjected to the weight of the heart when patients are in the supine versus the prone position.

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METHODS

At Denver Health Medical Center high-resolution chest computed tomography (HRCT) is routinely performed in both the supine and the prone positions when the study is ordered to evaluate patients for possible interstitial lung disease. Patients are asked to hold their breath after taking a full inhalation while sections 1 mm thick are obtained 10 mm apart with the patients supine, and 20 mm apart with them prone. Twenty consecutive HRCTs performed between May 1, 1998 and July 11, 1998 were reviewed. All seven scans that were interpreted as showing no parenchymal lung disease were analyzed.

Four sections from the prone scan were chosen such that they would be approximately evenly spaced from just caudal to the carina, to just cephalad to the most cephalad portion of the diaphragm. These were matched as closely as possible in the cephalad-caudal plane to four sections from the scans obtained in the supine position. The cardiac and pleural margins were traced on 10 by 10 mm/cm graph paper. The size of the HRCT images (i.e., six sections printed on each $14 \times 17\frac{1}{4}$ film) was such that the 1 cm marker on each image represented 4 mm on the graph paper. Perpendicular lines were drawn from the right and left lateral cardiac margins to the dependent chest wall. In each section the relative volume of lung parenchyma that could be affected by heart weight was determined by counting the number of square millimeter boxes located medial to these lines in each hemithorax, and expressing this as a fraction of the total number of square millimeter boxes present in each hemithorax (Figure 1 [supine] and Figure 2 [prone]).

Data are presented as mean ± SD. Results from the four sections obtained in the supine and prone positions were compared using Student's paired *t* test; $p < 0.05$ was considered to be significant.

RESULTS

The study population consisted of four men and three women with a mean age of 49 ± 18 yr (range, 28 to 73 yr). The cardiothoracic ratio measured on standard posterior-anterior chest roentgenograms was 0.48 ± 0.05 (range, 0.42 to 0.54). None had any history of cardiac disease.

When the patients were supine, from 7 to 42% of the left lung, and from 11 to 13% of the right lung were located under the heart in the four sections analyzed (Table 1; Figures 1 and 2, *a* through *d*). The percent of the left lung that was under the heart increased progressively and considerably from the subcarinal section (Section 1) to the section located just cephalad to the most cephalad portion of the diaphragm (Section 4). The percent of the right lung that was under the heart also increased in the more caudal sections, but less consistently, and to a much smaller extent.

When the patients were prone ≤ 1% of the left lung and ≤ 4% of the right lung were located under the heart in any section ($p < 0.05$ for each of the four sections, supine to prone, Table 1; Figures 1 and 2, *e* through *h*).

DISCUSSION

The important findings of this study were that (*1*) in the supine position, a considerable fraction of both lungs is located underneath the heart and, as such, would be subjected to the compressive force resulting from the weight of the heart and the

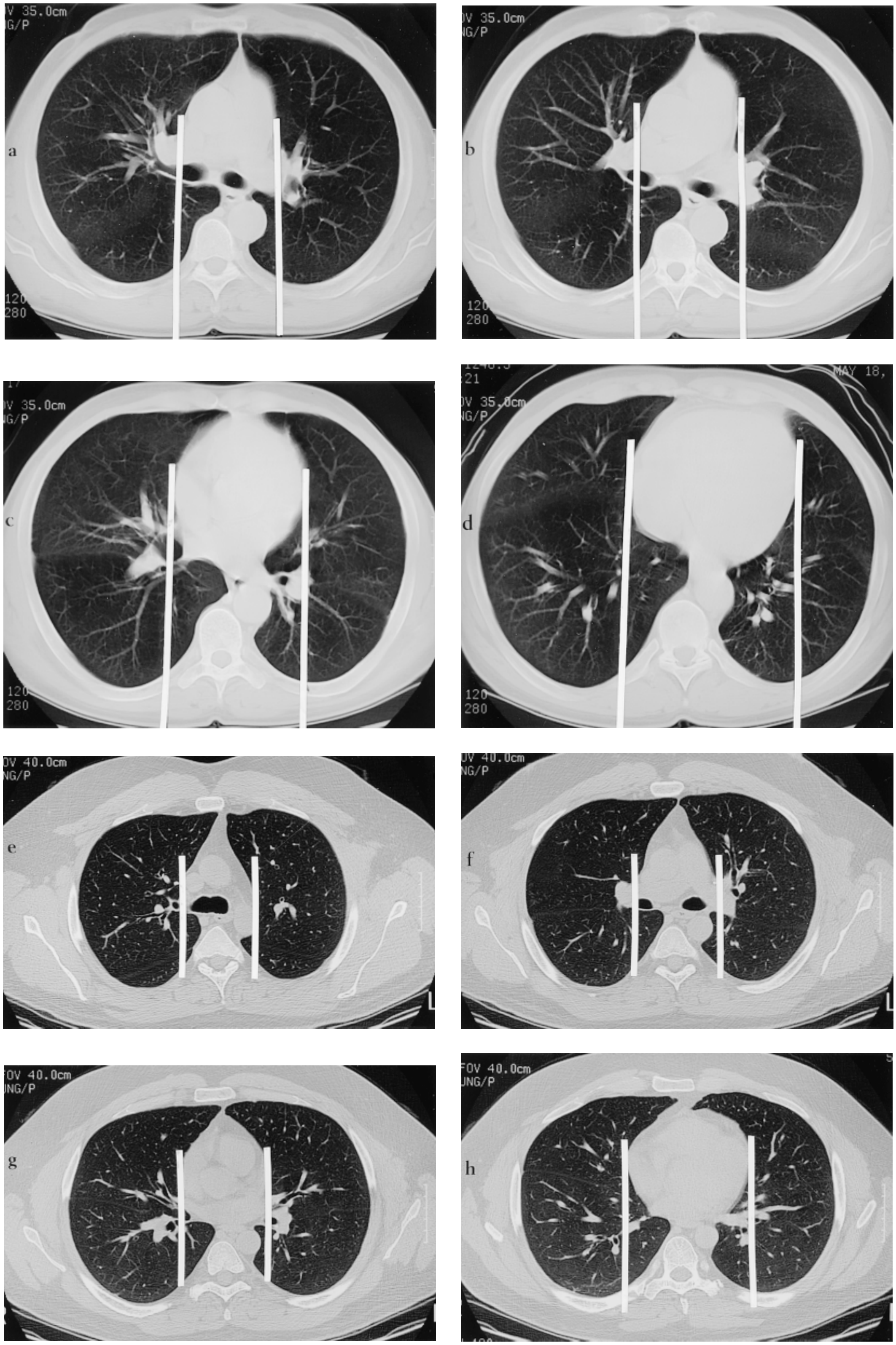

Figure 1. Computerized chest tomograms of two representative supine patients. Sections 1 through 4 (*a* through *d*, and *e* through *h*, for the two patients, respectively) were selected to be relatively evenly spaced between the region located just caudal to the carina and that located just cephalad to the most cephalad portion of the diaphragm. Areas of lung medial to the perpendicular lines were quantified as being under the heart.

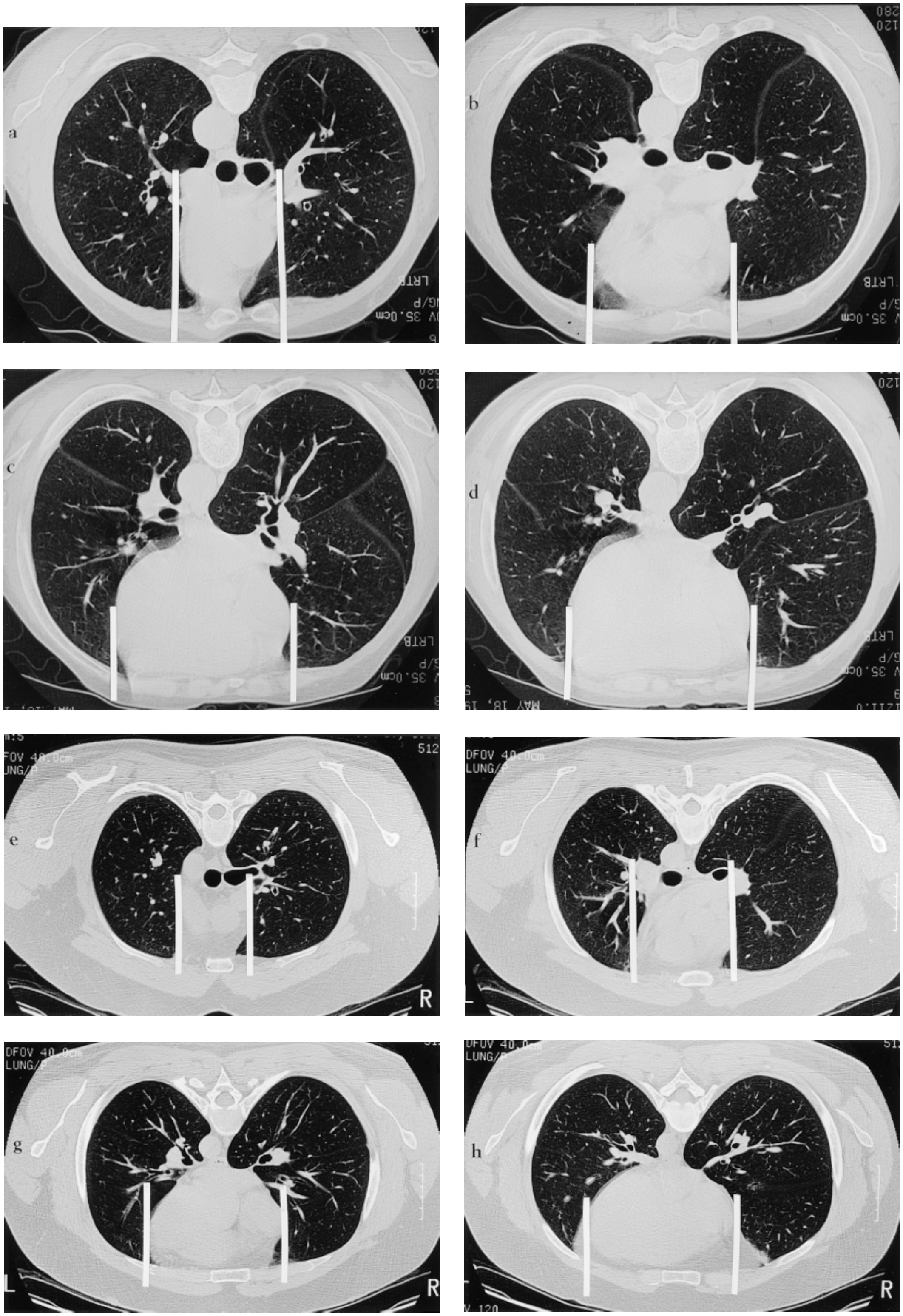

Figure 2. Computerized chest tomograms of the same patients in Figure 1, but obtained in the prone position. Sections 1 through 4 (*a* through *d*, and *e* through *h* for the two patients, respectively) were again selected to be relatively evenly spaced between the region located just caudal to the carina and that located just cephalad to the most cephalad portion of the diaphragm, at similar intervals as the sections displayed in Figure 1. Areas of lung medial to the perpendicular lines were again quantified as being under the heart.

blood contained therein; and (*2*) in the prone position, only a very small fraction of either lung would be similarly affected.

Critique of Methods

Although all patients were instructed to inhale fully prior to obtaining HRCT, radiology technicians made no specific effort to assure that patients actually achieved total lung capacity (TLC), and the scans were more likely obtained at a volume that was somewhat less than TLC.

An actual volumetric measurement of TLC and of the volume of lung that was located under the heart could have been obtained from full chest spiral tomograms. The questions being asked in this study, however, did not warrant the cost and radiation exposure associated with obtaining full lung tomograms in the supine and prone positions in a new study population. By examining selected sections of standard HRCTs that had previously been obtained for clinical indications, all costs and additional radiation exposure were obviated.

Because the distance between sections was 10 mm in the supine position and 20 mm in the prone position, Sections 1 through 4 in the two positions could not be aligned to precisely the same anatomic location. Given the distance between the sections, however, the maximum misalignment possible was less than 20 mm.

The mean predicted blood-free heart weight for the seven patients included in this study was 305 g (23). Assuming that the normal cardiac output is 6 L, that the normal resting heart rate is 75 beats/min (giving a stroke volume of 80 ml), and that the normal left (and right) ventricular stroke volume is > 55 to 60%, the combined left and right ventricular end-diastolic blood volume would be 267 to 290 ml, such that the *in vivo* heart volume of our patients was approximately 600 ml. We found that approximately 90% more lung was located under the heart in the left, compared with the right hemithorax (although the difference ranged from 42 to 162% in the four slices) (Table 1). This suggests that the heart volume is, on average, distributed approximately 0.66:0.33 (i.e., 400 g:200 g) to the left versus the right chest, respectively. The mean predicted TLC of our patients was 6.2 L. Assuming this is divided 0.45:0.55 between the left and the right lung, the heart should occupy approximately 14% of the left hemithorax and 6% of the right at TLC. These percentages are clearly not evenly distributed throughout each hemithorax, as the heart is confined to perhaps the most dependent 50% of each. Accordingly, the heart might occupy as much as 28 and 12% of the lower half of the left and right hemithoraces, respectively. This estimate is quite similar to the average of 25 and 14% from the four lung slices that we measured volumetrically (Table 1).

As a check on the reliability of our methods we quantified the relative volume of lung located under the heart, as a percent of the volume of lung present in *all* the sections of the full lung CT scans obtained at near TLC in two of the patients whose data are included in Table 1 (examining 10 and 11 CT sections, respectively). We found that approximately 10 and 13% of the right lung, and 16 and 20% of the left lung were located under the heart in the supine position in the two patients, respectively; again quite close to the theoretical estimates noted above.

The volume of lung located under the heart at, or near, total lung capacity (TLC) may *under*estimate the percent that would be found if the patients were studied at functional residual capacity (FRC), or at the end of inhalation during a normal tidal breath. At TLC all alveoli are at their maximum volume and the lung tissue volume/unit area should be constant throughout the lung. At any lung volume below TLC alveoli in dorsal lung regions will be less distended than those located in ventral regions, and alveoli in lung regions located under the heart would be even less distended, thereby increasing the volume of lung that would potentially be affected by the weight of the heart. Alternatively, the lungs descend relative to the heart as TLC is approached, possibly *increasing* the volume of lung subjected to compression over that which exists at FRC. Additional studies would be needed to determine which of these factors was predominant.

Heart-Lung and Lung-Heart Interactions

The idea that the heart can affect regional lung distention and that the lung can affect the transmural pressures of the heart dates to 1947 when Brookhart and Boyd (15) noted that "the dog's heart produces deformation of the adjacent lung, raising pressure on the external surface of the heart above the pressure existing between the lung and the wall of the thorax." Shortly thereafter a number of other investigators suggested

TABLE 1

RELATIVE AREA OF THE LUNG LOCATED UNDER THE HEART

	Patient No.	Section 1 (subcarinal)			Section 2			Section 3			Section 4		
		Left	Right	Total	Left	Right	Total	Left	Right	Total	Left	Right	Total
Supine	1	0.04	0.09	0.06	0.10	0.09	0.09	0.27	0.14	0.20	0.35	0.15	0.24
	2	0.02	0.13	0.07	0.20	0.12	0.16	0.32	0.17	0.24	0.43	0.18	0.29
	3	0.08	0.09	0.08	0.32	0.13	0.22	0.46	0.15	0.28	0.56	0.12	0.30
	4	0.09	0.15	0.12	0.28	0.19	0.23	0.44	0.17	0.27	0.42	0.19	0.28
	5	0.05	0.06	0.05	0.12	0.07	0.09	0.30	0.12	0.19	0.33	0.09	0.18
	6	0.14	0.17	0.16	0.14	0.12	0.13	0.31	0.18	0.23	0.50	0.21	0.32
	7	0.10	0.11	0.11	0.10	0.09	0.09	0.13	0.11	0.12	0.37	0.15	0.24
Mean		0.07	0.11	0.09	0.18	0.12	0.14	0.32	0.15	0.22	0.42	0.16	0.26
SD		0.04	0.04	0.04	0.09	0.04	0.06	0.11	0.02	0.05	0.08	0.04	0.05
Prone	1	0.02	0.01	0.01	0.00	0.02	0.01	0.04	0.02	0.03	0.00	0.02	0.02
	2	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	3	0.03	0.09	0.06	0.03	0.04	0.03	0.02	0.03	0.02	0.00	0.01	0.01
	4	0.01	0.00	0.01	0.02	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00
	5	0.02	0.05	0.03	0.03	0.00	0.00	0.01	0.01	0.01	0.00	0.00	0.00
	6	0.02	0.07	0.05	0.02	0.02	0.02	0.02	0.02	0.02	0.00	0.01	0.01
	7	0.00	0.02	0.01	0.00	0.02	0.01	0.01	0.02	0.01	0.00	0.00	0.00
Mean		0.01	0.04	0.03	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.01	0.00
SD		0.01	0.03	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.01	0.01

that the weight of the heart might account for regional differences in pleural pressure (Ppl) (16–19), and these were confirmed by studies documenting this point 15 yr later (20–22). The supporting idea that lung distention could affect the heart was confirmed in 1977 (24), and subsequently in the 1980s (25, 26, as summarized in 27).

In 1965, Bosman and Lee (28) suggested that heart–lung interactions might affect ventilation, when they noted oscillations in expired gas flow (measured at the mouth with a body plethysmograph), observed that these were coincident to the heart beat, and attributed them, in part, to the direct local mechanical distortion of the lungs. Clarke and colleagues in 1969 (29), and Cortese and colleagues in 1976 (30) found marked postural differences in the single breath oxygen test, which they attributed in part to the weight of the heart. Wiener and colleagues (31) found that left lower lobe ventilation was impaired when patients with cardiomegaly were positioned supine, but not when they were prone.

Ball and colleagues (32) performed the first CT imaging study of the effects of posture on thoracic anatomy and noted that "the heart and great vessels moved ventrally and caudally [and a] much larger area of the heart came into contact with the anterior chest wall." Subsequently, Hoffman (33) used dynamic spatial reconstruction and observed marked supine-to-prone differences in the regional air content of dog and sloth lungs (i.e., more uniform end-expiratory lung volume when prone), found that this difference correlated with shifts in the position of the mediastinal contents, and concluded that "the heart is an important component of the lung's container."

Compressive Force of the Heart

How the weight of the heart transfers to a compressive force on the lungs is governed by how the heart and lungs fit into the thorax. Factors that affect the fit include the distensibility of the lung (which, in turn, depends on the lung volume and whether the lung is filled with air or fluid), the distensibility of the thorax (which depends on chest wall mass and compliance, abdominal mass, and compliance, heart mass, and perhaps on the anatomy of the diaphragm as it pertains to the curvatures relative to the ventral and dorsal thorax) and on body position (16–22, 34, 35).

The compressive force of the heart in normal subjects at FRC can be estimated from a study by Milic-Emili and colleagues (36) who found that esophageal pressure measured in the region of the heart averaged approximately 5 cm H_2O more in the supine compared with the prone position. This is consistent with the results of Liu and colleagues (22) who modeled regional pleural pressures of dog lungs from volumetric-computed tomograms and estimated that, at FRC, pressures beneath the heart would be approximately 3 cm H_2O greater than the mean pleural pressure when the animals were supine. These studies also indicated that the weight of the heart was supported by the sternum when the animals were prone. The data presented in Table 1 indicate that a similar situation exists in prone humans.

The compressive force of the heart would predictably be greater in patients with cardiomegaly, as has been demonstrated by Wiener and colleagues (31), and would probably be reduced in those with the smaller hearts associated with emphysema. The isolated effects of heart weight would be difficult to estimate in patients with ARDS in that there are marked regional variations in lung volume, the lung is fluid-filled to a variable extent, soft tissue edema could alter thoracic and abdominal masses and compliances, bowel wall edema, ascites and increased abdominal pressures are variably present, and patients may or may not have cardiomegaly. These variations could contribute to the variability in gas exchange improvement that is seen in patients with ARDS when they are turned from supine to prone.

Gattinoni and colleagues (11, 12) have proposed that the pressure causing air-space collapse in patients with ARDS should be called the "lung superimposed pressure," which they suggest would equal sum of the height of the lung times its density. If this were true, the lung collapsing pressure would remain constant on turning from supine to prone, but be directed to ventral rather than to dorsal lung regions. This implication is inconsistent with a number of studies reporting that (*1*) there are postural differences in small airways closure (30); (*2*) the measured gravitational pleural pressure gradient is *greater* than the hydrostatic gradient for a fluid with the density of lung when measured in the supine position; and (*3*) there is no substantive gravitational pleural pressure gradient, and no gravitational differences in regional lung expansion when these variables are measured in the prone position (summarized in 37). Accordingly, forces *in addition to gravity* must be acting on the lung (e.g., the weights of the heart and the abdominal contents, among others, as summarized above). These observations, along with the recent finding that regional perfusion is preferentially directed to dorsal lung regions *regardless* of whether the dorsal lung is in the dependent or non-dependent position (9, 38), demonstrate that turning from supine to prone will not have equal but anatomically opposite effects on lung volume, on regional ventilation, or on regional ventilation-perfusion relationships.

Clinical Implications

As shown in Figures 1 and 2, turning prone eliminates the compressive force of the heart on dorsal lung regions and redirects it to only a small portion of the ventral lung regions. This change will (*1*) lower the inspiratory pressure required to obtain maximal air-space recruitment (i.e., alveolar recruitment and/or airway opening); (*2*) lower the end-expiratory pressure required to maintain maximal air-space recruitment; and (*3*) reduce cyclical air-space opening and closing. If ventilator-induced lung injury is related to any or all of these factors, routine use of the prone position could translate into reductions in morbidity and mortality for patients with ARDS. The data presented further suggest that the variations in the degree of gas exchange improvement seen on turning patients with ARDS from supine to prone could, in part, relate to differences in the volume and/or position of the heart.

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