Anesthesiology 2002; 97:298–305

Motion of the Diaphragm in Patients with Chronic Obstructive Pulmonary Disease while Spontaneously Breathing versus *during Positive Pressure Breathing after Anesthesia and Neuromuscular Blockade*

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Background: **Diaphragmatic excursion during spontaneous ventilation (SV) in normal supine volunteers is greatest in the dependent regions (bottom). During positive pressure ventilation (PPV) after anesthesia and neuromuscular blockade and depending on tidal volume, the nondependent region (top) undergoes the greatest excursion, or the diaphragm moves uniformly. The purpose of this study was to compare diaphragmatic excursion (during SV and PPV) in patients with chronic obstructive pulmonary disease (COPD) with patients having normal pulmonary function.**

Methods: **Twelve COPD patients and 12 normal control subjects were compared. Cross-table diaphragmatic fluoroscopy was performed while patients breathed spontaneously. After anesthetic induction and pharmacologic paralysis and during PPV, diaphragmatic fluoroscopy was repeated. For analytic purposes, the diaphragm was divided into three segments: top, middle, and bottom. Percentage of excursion of each segment during SV and PPV in normal subjects was compared with the percentage of excursion of each segment in patients with COPD.**

Results: **There was no significant difference in the pattern of regional diaphragmatic excursion (as a percentage of total excursion)—top, middle, bottom—when comparing COPD patients with control subjects during SV and PPV. In the control subjects, regional diaphragmatic excursion was 16 ± (5), 33 ± (5), 51 ± (4) during SV and 49 ± (13), 32 ± (6), 19 ± (9) during PPV. In COPD patients, regional diaphragmatic excursion was 18 ± (7), 34 ± (5), 49 ± (7) during SV and 47 ± (10), 32 ± (6), 21 ± (9) during PPV.**

Conclusion: **Regional diaphragmatic excursion in patients with COPD during SV and PPV is similar to that in persons with normal pulmonary function.**

This article is accompanied by an Editorial View. Please see Warner DO: Diaphragm function during anesthesia: Still crazy after all these years. ANESTHESIOLOGY 2002; 97:295–7.

THE classic study by Froese and Bryan[1] showed that diaphragmatic motion in spontaneously breathing normal volunteers is markedly altered when the volunteers are paralyzed with neuromuscular-blocking drugs. Specifically, Froese and Bryan concluded that in the supine position, during spontaneous ventilation (SV), the dependent part (bottom) of the diaphragm had the greatest displacement. However, after neuromuscular blockade and positive pressure ventilation (PPV), exactly the opposite was seen: the nondependent part (top) had the greatest displacement. Sixteen years later, Krayer *et al.*, using more sophisticated technology (high-speed, three-dimensional x-ray computed tomography [CT]) showed similar results as those obtained by Froese and Bryan with some striking differences.[2] Among those differences were that Krayer *et al.* showed that during anesthesia and paralysis and during PPV, diaphragmatic motion became piston-like, *i.e.*, excursion being equal at all levels. Froese and Bryan, on the other hand, observed piston-like behavior of the diaphragm at large tidal volumes and a reversal of the normal spontaneous ventilation pattern at smaller tidal volumes—tidal volumes that were considerably less than those used by Krayer *et al.* The studies of Froese and Bryan and Krayer *et al.* have contributed to our understanding of how diaphragmatic motion is altered during PPV and neuromuscular blockade. However, the aforementioned investigations studied healthy volunteers.

Patients with chronic obstructive pulmonary disease (COPD) have profound chest wall and diaphragmatic abnormalities.[3–6] Such abnormalities could theoretically lead to altered pulmonary function during anesthesia and neuromuscular blockade. Gas exchange in patients with COPD during anesthesia and neuromuscular blockade is different when compared with healthy subjects.[7] Therefore, the purpose of this study is to test the following hypothesis: Diaphragmatic motion in supine patients with COPD during spontaneous breathing and during PPV after anesthesia and neuromuscular blockade is different than diaphragmatic motion in supine persons

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Received from Anesthesiology and Diagnostic Radiology Services and Section of Cardiothoracic Surgery at the Edward Hines Jr. Veterans Affairs Hospital, Hines, Illinois. Submitted for publication October 3, 2001. Accepted for publication March 6, 2002. Supported by the office of Research and Development of the Edward Hines Jr. Veterans Affairs Hospital, and the Department of Anesthesiology, Loyola University Stritch School of Medicine, Maywood, Illinois. Presented in part at the Annual Meeting of the American Society of Anesthesiologists, October 20, 1998, Orlando, Florida.

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Table 1. Characteristics of Normal Patients

	Age (yr)	Sex	Weight (kg)	Operation	FEV_1 Liters (% of Predicted)	FEV_1% (FEV_1/FVC)	Tidal Volume (ml) Baseline/Large SV	Tidal Volume (ml) Baseline/Large PPV
1.	51	M	93	CABG	4.20 (118)	76	600/1190	600/1200
2.	50	M	118	CABG	5.70 (120)	83	700/1200	670/1180
3.	55	M	66	MVR	2.86 (82)	83	420/950	400/920
4.	45	M	110	Lap choly	4.52 (110)	79	620/1210	600/1200
5.	46	M	88	Lap choly	4.67 (120)	78	480/1070	500/1100
6.	65	M	100	Um Hernia	2.56 (85)	79	450/950	450/970
7.	57	M	110	Sh arthroscopy	3.00 (84)	73	520/1090	500/1100
8.	66	M	116	CABG	2.92 (80)	76	580/1080	600/1100
9.	77	M	70	CABG	2.98 (123)	77	410/990	400/1000
10.	64	M	113	Mastectomy	2.72 (94)	82	550/1060	570/1090
11.	61	M	89	AVR	3.03 (105)	76	640/1180	650/1200
12.	49	M	90	Lap choly	4.39 (121)	87	490/1190	500/1200

Tidal volumes to nearest 10 ml.

SV = spontaneous ventilation; PPV = positive pressure ventilation; CABG = coronary artery bypass graft; MVR = mitral valve replacement; AVR = aortic valve replacement; Lap choly = laparoscopic cholycyctectomy; Sh = shoulder; Um = umbilical; FEV_1 = forced expiratory volume in 1s; FVC = forced vital capacity.

with normal pulmonary function during spontaneous breathing and during PPV after anesthesia and neuromuscular blockade.

Materials and Methods

Subjects

This project was approved by the Hines Hospital Institutional Review Board for the Protection of Human Subjects. Informed consent was obtained from 24 patients. Twelve patients presenting for a variety of surgeries and with normal pulmonary spirograms served as the control group (table 1). Their diaphragmatic radius of curvature was 16 cm (SD ± 8). Spirometry and flow-volume loops were performed using a portable pulmonary function device (Satellite/Base Station Spirometry System, Jones Medical, Oak Brook, IL) in the preanesthesia evaluation clinic.

Twelve patients (undergoing thoracic procedures) had pulmonary function tests consistent with moderate or severe COPD (table 2). COPD was defined by history and pulmonary function testing. Smoking history and symptoms of dyspnea in the absence of obvious heart failure or a diagnosis of asthma was diagnostic. To qualify as a candidate for study, the pulmonary function spirogram had to show a forced expiratory volume percent (FEV_1%) of 60% or less, in the absence of extra thoracic airway obstruction (which was ruled out by the flow-volume loop). Pulmonary function tests were performed in the pulmonary function laboratory. The mean residual volume (RV)—an index of hyperinflation—was 198% (SD ± 66%) of predicted. These patients had a diaphragmatic radius of curvature of 28 cm (SD ± 12).

Experimental Protocol

Cross-table lateral fluoroscopy of the diaphragm was performed before induction of general anesthesia. This necessitated the subjects have their arms above their heads. Initially, patients were asked to breath as they

Table 2. Characteristics of Patients with Chronic Obstructive Pulmonary Disease

	Age (yr)	Sex	Weight (kg)	Operation	FEV_1 Liters (% of Predicted)	FEV_1% (FEV_1/FVC)	Tidal Volume (ml) Baseline/Large SV	Tidal Volume (ml) Baseline/Large PPV
1.	78	M	85	VATS	1.28 (44)	56	450/990	470/1000
2.	54	M	77	Bronchoscopy	1.55 (43)	53	220/630	230/650
3.	72	M	73	Lobectomy (R)	1.10 (38)	52	390/1180	400/1120
4.	77	M	60	Lobectomy (L)	1.47 (56)	48	580/1200	590/1180
5.	67	M	64	Lobectomy (R)	1.60 (50)	53	520/910	510/900
6.	71	M	91	Lung reduction	0.96 (32)	41	480/1160	490/1080
7.	60	M	85	Lung reduction	0.88 (25)	48	380/1150	390/1120
8.	64	M	61	Lung reduction	1.10 (42)	33	550/1090	550/1100
9.	69	M	63	Lobectomy (R)	1.80 (52)	49	460/1480	480/1450
10.	74	M	77	Lobectomy (R)	1.00 (45)	51	480/970	470/990
11.	59	M	91	Lung reduction	1.04 (26)	34	390/1150	400/1120
12.	78	M	55	Lobectomy (R)	1.38 (51)	37	460/1150	450/1200

Tidal volumes to nearest 10 ml.

TURP = prostatic resection; R = right; L = left; VATS = video assisted thoroscopic surgery; SV = spontaneous ventilation; PPV = positive pressure ventilation; FEV_1 = forced expiratory volume in 1 s; FVC = forced vital capacity.

normally would (baseline measure), during which fluoroscopy was performed. Fluoroscopy was then performed again while the patients took a series of deep breaths (2 or 3 times their baseline tidal volumes). After the patients were anesthetized and pharmacologically paralyzed and their lungs ventilated by positive pressure, the fluoroscopic studies were repeated in the same sequence (baseline tidal volumes followed by large tidal volumes). Also, the position of their arms remained as they were during the awake phase of the study.

Tidal volumes were measured with a respirometer (Haloscale Standard Wright Respirometer, Ferraris Medical, London, UK). The use of the respirometer was demonstrated to patients before entry into the operating room. They were instructed how to breath through a tubular metallic mouthpiece that was connected to the respirometer. In the operating room, patients were told to breathe normally and then take a series of deep breaths. After induction of anesthesia, tidal volumes were measured by the anesthesia machine's digital spirometer positioned on the expiratory limb of the breathing circuit. Tidal volumes were duplicated during positive pressure breathing to match the ones during spontaneous breathing.

Management of Positive Pressure Breathing

After the patients were anesthetized and pharmacologically paralyzed, they were ventilated by the anesthesia machine ventilator (Narkomed 2B, North American Drager, Telford, PA). The fraction of inspired oxygen (F_{IO_2}) was 40%. Total fresh gas flow was approximately 2 l/min. The rate was set at 6 breaths/min. The ventilator settings were adjusted to permit as long an expiratory time as reasonably possible. This prevented any expiratory flow persisting into the onset of the next tidal volume. Therefore, the potential for the development of intrinsic positive end-expiratory pressure (PEEP) was minimized. Tidal volumes were adjusted to approximate as closely as possible the tidal volumes during the spontaneous phase of the study. When the tidal volumes were deemed close enough approximations of baseline tidal volumes, the positive pressure breaths were repeated during fluoroscopic examination. This resulted in the recording of at least one positive pressure breath whose tidal volume closely approximated the tidal volume of the baseline spontaneous breaths.

Anesthetic Management

Anesthesia was induced with sodium thiopental, 4 mg/kg. Succinylcholine, 1 mg/kg, was used to facilitate tracheal intubation. Immediately after tracheal intubation, either pancuronium, 0.1 mg/kg, or atracurium, 0.5 mg/kg, was administered for maintenance of neuromuscular blockade. Isoflurane was used for maintenance of anesthesia. Diaphragmatic excursion was examined by fluoroscopy after a blockade monitor confirmed the absence of twitches.

Fluoroscopy

Fluoroscopy was performed using a collimated digital mobile C-arm x-ray 9-inch image intensifier system (Siremobil 2000, Siemens, Iselin, NJ). The field of view spanned from the vertebral bodies to 1 or 2 cm from the ventral surface of the lung. To limit radiation exposure, total fluoroscopy time was restricted to 60 s per subject, which resulted in a calculated radiation dose of 0.04 Gy.

Diaphragmatic Measurements

The motion of the diaphragm was recorded on standard videotape. A lead surface marker on the rib cage and the vertebral bodies were used as points of reference to assist in comparing diaphragmatic positions at functional residual capacity (FRC). The video recordings were reviewed in a stop action mode—frame by frame—such that end-inspiratory and end-expiratory positions of the diaphragm could be accurately determined. The videotape was stopped at the appropriate frame, and the outline of the diaphragm was traced. Its relative position to the lead surface markers and the bony landmarks was noted. The excursion (from FRC to maximum excursion) was then traced on transparencies. The method previously described by Froese and Bryan was used for analysis.[1] The diaphragm was divided into three parts from nondependent (top) to dependent regions (middle to bottom). For a given tidal volume, the linear displacement, defined by motion of the midpoint of each of these three segments of the diaphragm, was measured (in cm), and the values were summed to give total displacement. The regional displacement at each level was expressed in centimeters and as a percentage of the total displacement. The bottom level (B) was drawn through the midpoint of the vertebral bodies. The other two levels—middle (M) and top (T)—were equally spaced above the bottom level. Total excursion (in cm) was equal to T + M + B. Percent of excursion of each segment (in cm) was equal to the excursion of that segment divided by T + M + B. A lead calibration strip (in cm) was inserted into the field of view to correct for magnification effect, such that diaphragmatic displacement could be determined in centimeters. Means of the T, M, and B excursions (as a percentage of the total: T + M + B, and absolute displacement in cm) during SV (baseline tidal volumes and large tidal volumes) and PPV (normal tidal volumes and large tidal volumes after anesthesia and neuromuscular blockade) were compared. Lead markers and vertebral landmarks enabled us to ensure constancy of patient position during anesthetic induction.

The position of the diaphragm at FRC before the induction of anesthesia and pharmacologic paralysis was traced on a transparency. After paralysis, its position was similarly traced. Changes in position of top, middle, and bottom segments from the corresponding positions at FRC were measured in centimeters. For purposes of

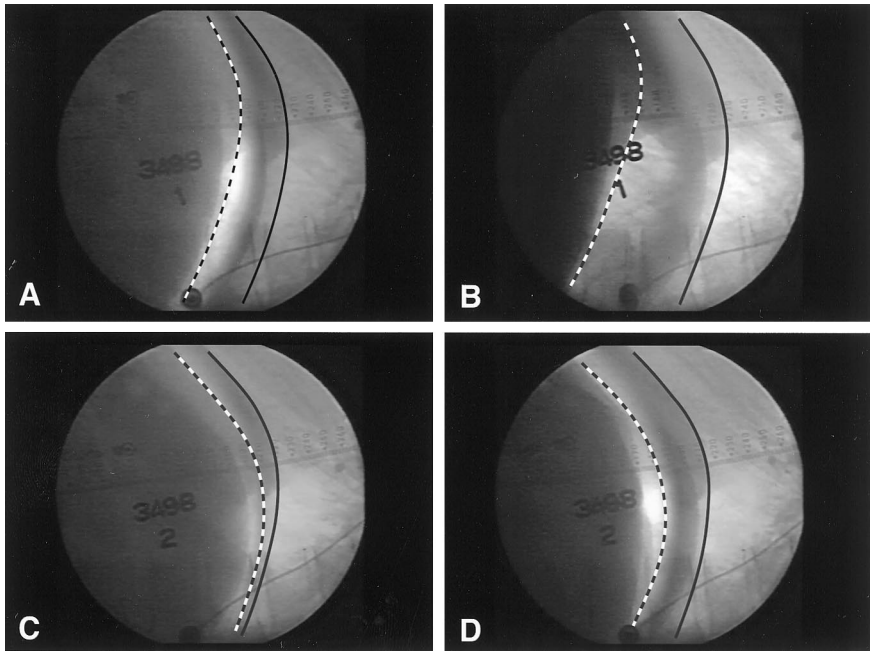

Fig. 1. Diaphragmatic excursion from control patient no. 5 (from table 1). End-inspiratory video frame has been digitally pasted on video frame of diaphragm at functional residual capacity (FRC) position. Diaphragmatic borders are graphically enhanced. Stippled outline represents end inspiration; thick black line is diaphragm at FRC position. Area between stippled outline and thick black line represents diaphragmatic displacement. *A*, Spontaneous breathing, baseline tidal volume. *B*, Spontaneous breathing, large tidal volume. *C*, Positive pressure ventilation, baseline tidal volume. *D*, Positive pressure ventilation, large tidal volume. Note greater excursion in nondependent segments as contrasted with spontaneous breaths; *A versus C*.

analysis, cephalad (cranial) movement was designated as positive (+), and caudal movement was designated as negative (−). Total excursion in centimeters for each segment from FRC position during paralysis was measured. In addition, qualitative assessment was made as to whether segments of the diaphragm moved either cephalad (cranial) or caudad after the onset of pharmacologic paralysis.

Radius of curvature of the diaphragms was measured by constructing a circle of "best fit" (using a compass) incorporating the middle segment of the diaphragmatic silhouette (at FRC during normal SV) into the circle of best fit. The radius of curvature of the diaphragm was therefore the radius (in cm) of the circle of best fit.

Analysis

One investigator (M.V.D.) blinded to clinical conditions reviewed the videotapes. One investigator (B.K.), who was not blinded to clinical conditions, reviewed tapes on several different occasions. Several months after completing subject acquisition, tapes were re-reviewed by two investigators (B.K. and M.V.D.). There was substantial agreement between the investigators with a κ of 0.76 (interobserver agreement). Agreement between B.K.'s first readings and those subsequently was also high, with a κ of 0.84 (intraobserver agreement). The final arbiter of any questions related to diaphragmatic excursion or shift of diaphragmatic position at FRC was M.V.D. Spontaneous breaths and positive pressure breaths whose tidal volumes approximated each other were analyzed. This resulted in the analysis of one measure per subject and condition, *i.e.*, one normal spontaneous breath *versus* one normal positive pressure breath and one large spontaneous breath *versus* one large positive pressure breath.

Statistical Analysis

Means (relative in % and absolute in centimeters) and standard deviations were calculated. Two-tailed paired *t* tests were used to determine statistical significance between segmental (top, middle, bottom) diaphragmatic excursions during SV *versus* PPV within each group (control subjects and patients with COPD). Two-tailed *t* tests were then used to compare statistical significance between segmental diaphragmatic excursions in control subjects *versus* those with COPD. A two-tailed paired *t* test was also used to compare segmental diaphragmatic excursion from FRC (in cm) after paralysis in control subjects *versus* excursion in those with COPD. Statistical significance was set a $P \leq 0.05$.

Results

During spontaneous breaths, diaphragmatic displacement was substantially greater in dependent regions compared with that in nondependent regions in patients with normal pulmonary function and in those with COPD. However, during PPV, two patterns were observed in both groups, depending on the size of the tidal volumes. First, with normal breaths, there was preferential motion of nondependent zones, *i.e.*, with normal tidal volumes there was a significant reversal of the pattern seen during SV (figs. 1, and 2; tables 3 and 4). However, two patients in the control group and three patients in the COPD group deviated from this overall pattern by exhibiting equal excursion of all diaphragmatic segments (so called piston-like motion). In addition, one patient's diaphragm in the control group showed greatest excursion in the middle segment. Second, with large tidal breaths during PPV, diaphragmatic

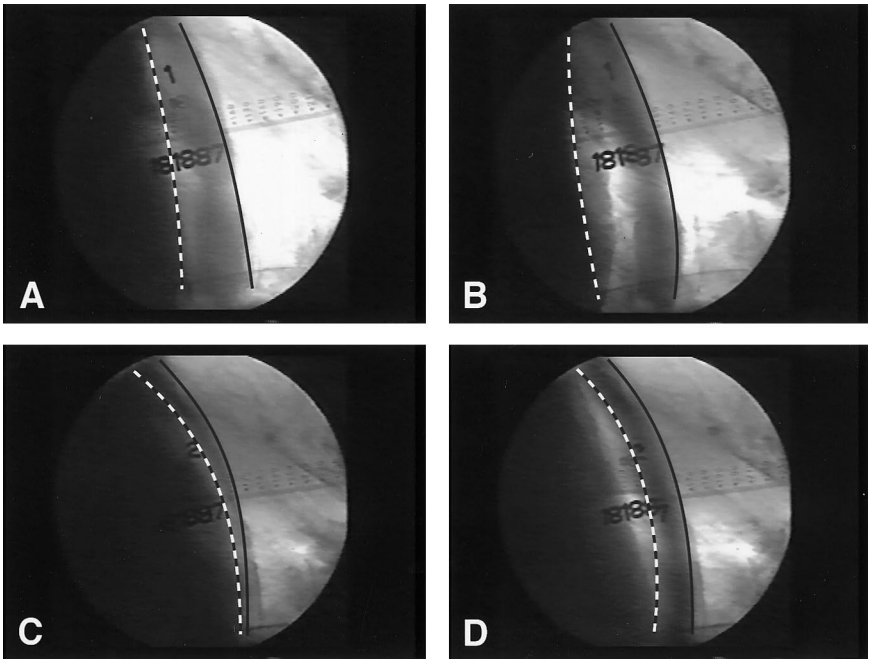

Fig. 2. Diaphragmatic excursion from chronic obstructive pulmonary disease (COPD) patient no. 4 (from table 2). Sequence the same as in figure 1. Note similar pattern of diaphragmatic excursion to control subjects, particularly comparing *A versus C*.

motion became piston-like, being approximately equal at all heights (figs. 1 and 2; tables 3 and 4). One patient with COPD deviated markedly from this pattern. His diaphragm had the greatest excursion in the middle. Overall, there was no significant difference observed in the pattern of diaphragmatic excursion during SV and PPV between the group with normal pulmonary function and the group with COPD (table 3 and 4).

During normal spontaneous breaths, total absolute diaphragmatic displacement (excursion of top + middle + bottom in cm) was significantly greater than total displacement during corresponding positive pressure breaths in both groups: 7.6 cm ± (2.4 cm) *versus* 3.2 cm ± (1.2 cm) in the COPD group; 6.6 cm ± (1.4 cm) *versus* 4.0 cm (± 0.8 cm) in the control group. In terms of total absolute diaphragmatic directional shift after pharmacologic paralysis (in cm), there was a small net cephalad (cranial) shift of all segments in the COPD group but was not statistically significant (table 5). In the control group, there was a small net caudal shift of the top and middle segments and a small net cranial shift of the bottom segment, which also was not statistically significant (table 5). Differences between groups were not significant. On a qualitative basis in the COPD group, six diaphragms shifted cranially, three shifted caudally, and three did not shift in either direction. For each patient within the COPD group, there was no heterogeneity of diaphragmatic directional shift. In other words, within patients in the COPD group, there was no difference in the direction of change (cranial *vs.* caudal) for the three segments. Results were a little different in the control group. Five diaphragms moved cranially and four moved caudally. However, three were heterogeneous: some segments moved cranially, whereas others moved caudally.

Discussion

It was once believed that the greater displacement of the dependent part of the diaphragm during SV was achieved despite the fact that opposing forces—the hy-

Table 3. Distribution of Diaphragmatic Excursion (%) Normals *versus* Chronic Obstructive Pulmonary Disease

	Top		Middle		Bottom	
	NL	COPD	NL	COPD	NL	COPD
SV						
Baseline	16 ± (5)	18 ± (7)	33 ± (5)	34 ± (5)	51 ± (4)	49 ± (7)
Large	21 ± (5)	18 ± (7)	33 ± (4)	35 ± (4)	46 ± (7)	46 ± (5)
PPV						
Baseline	49 ± (13)*	47 ± (10)*	32 ± (6)	32 ± (6)	19 ± (9)*	21 ± (9)*
Large	36 ± (7)*	33 ± (3)*	33 ± (4)	35 ± (4)	31 ± (5)*	32 ± (5)*

Values are mean % (± SD), to the nearest percent.* = significantly different from corresponding SV.

SV = spontaneous ventilation; PPV = positive pressure ventilation. Baseline tidal volumes; Large, tidal volumes; NL = normal pulmonary function; COPD = chronic obstructive pulmonary disease.

Table 4. Distribution of Diaphragmatic Excursion (cm) Normals versus Chronic Obsructive Pulmonary Disease

	Top		Middle		Bottom	
	NL	COPD	NL	COPD	NL	COPD
SV						
Baseline	1.1 ± (0.6)	1.6 ± (0.3)	2.1 ± (0.7)	2.5 ± (1.1)	3.3 ± (0.8)	3.5 ± (1.3)
Large	2.8 ± (1.2)	2.7 ± (1.6)	4.3 ± (1.3)	5.0 ± (1.6)	5.8 ± (1.7)	6.5 ± (1.6)
PPV						
Baseline	2.0 ± (0.7)	1.5 ± (0.5)*	1.3 ± (0.4)*	1.0 ± (0.2)*	0.8 ± (0.4)*	0.7 ± (0.3)*
Large	3.1 ± (0.9)	3.1 ± (1.6)	2.9 ± (1.1)*	3.4 ± (1.7)*	2.7 ± (1.2)*	3.3 ± (1.8)*

Values are mean in cm (± SD), to the nearest 0.1 cm. *= significantly different from corresponding SV.

SV = spontaneous ventilation; PPV = positive pressure ventilation. Baseline tidal volumes; Large, tidal volumes; NL = normal pulmonary function. COPD = chronic obstructive pulmonary disease.

drostatic pressures in the abdomen—were greatest in the dependent regions.[1] Mechanisms proposed to explain how the actively contracting diaphragm was able to generate more force in the dependent region included the Laplace relationship and second, if the dependent part of the diaphragm was not only more curved but more stretched, then like any other muscle, the diaphragm could develop more force.[1] Subsequent evidence suggests that the Laplace relationship is an oversimplification and plays little, if any, role in diaphragmatic mechanics.[8,9] Further, the diaphragm is unique. It consists of crural and costal segments that have different force–length relationships. These segments may even function as two distinct muscles, muscles that in fact may have different orientations to the chest wall.[10–12]

Our findings in patients with COPD who are pharmacologically paralyzed, as well as in control subjects, show there is a reversal in regional diaphragmatic motion from what occurs during spontaneous ventilation. These findings duplicate to a large extent what has previously been described in healthy subjects in previous literature[1,2] Froese and Bryan proposed that with neuromuscular blockade, diaphragmatic excursion was no longer determined by active contraction.[1] Rather, excursion was determined by the net effects of two opposing forces: a uniform force applied to the airway that was transmitted to the thoracic side of the diaphragm *versus* an opposition force provided by a presumed nonuniform hydrostatic pressure gradient of the abdominal contents ("water column effect").[1] Therefore, maximal diaphragmatic excursion would occur where the transdiaphragmatic pressure gradient was greatest, namely, the nondependent regions of the diaphragm. On the other hand, if diaphragmatic motion is determined by a vertical hydrostatic gradient, then diaphragmatic motion should be independent of body position during SV and PPV. However, Krayer *et al.* found that the motion of the diaphragm was different in the prone *versus* supine position.[2] Our findings in three of our control subjects and three of our patients with COPD lend support to Krayer *et al.*, in that diaphragmatic excursion was uniform during PPV with normal breaths in five of six subjects, and in one (with COPD), the greatest excursion was in the middle segment. In fact, canine studies have confirmed that the abdomen does not behave as a liquid-filled container or water column.[13] Therefore, there must be other factors besides an abdominal hydrostatic gradient to account for regional diaphragmatic motion in healthy persons and in patients with COPD. However, the primary objective of our investigation was to first characterize regional diaphragmatic motion in patients with COPD, and then to see if it was the same or different from a relatively large group of patients with normal pulmonary function. Further studies will be needed to define mechanisms not only in healthy persons but also in those with COPD.

Table 5. Position of the Diaphragm after Paralysis at FRC Relative to the Position at FRC before Paralysis (cm)

	COPD	Normal
Top	0.3 ± (1.2)	−1.0 ± (3.1)
Middle	0.3 ± (1.3)	−0.4 ± (1.7)
Bottom	0.4 ± (1.0)	0.7 ± (1.4)

+ = cephalad direction; − = caudal direction; mean in cm ± SD.

COPD = chronic obstructise pulmonary disease.

Profound respiratory muscle mechanical changes have previously been reported in patients with COPD.[3–6] Chief among these mechanical changes is anatomic flattening of the diaphragm. Such flattening would appear to put the diaphragm at a mechanical disadvantage when compared with the diaphragm in healthy persons.[3,4] However, more recent investigations have shown that diaphragmatic contractile function is surprisingly well preserved in patients with COPD, even in those patients with chronic hyperinflation.[14,15] The mechanism for this preservation of function is not clear. Animal data suggest it may be related to a process called *length adaptation*.[16] Length adaptation is a process whereby a muscle is passively shortened to less than its optimal resting length, loses its force-generating capacity, but with time adapts and is able to generate almost normal force at shorter lengths. Anatomically, chronically shortened muscles, such as diaphragms in patients with COPD, lose

excess sarcomeres such that remaining sarcomeres are restored to the proper optimal operating length, thereby restoring virtual normal contractile function.[15,17] If motion and function are related, then the above may partially explain our unexpected results showing similar regional diaphragmatic motion between patients with COPD and hyperinflation (residual volume of 198% of predicted and a significantly greater radius of curvature than in control subjects) and patients with normal pulmonary function. Therefore, our findings disprove our initial hypothesis that diaphragmatic motion in patients with COPD would be different than diaphragmatic motion in persons with normal pulmonary function.

The diaphragm did not significantly change from its resting FRC position after anesthesia and paralysis in either control subjects or patients with COPD. This was also true when comparing diaphragmatic shift, after anesthesia and paralysis, between the two groups. Overall, in both groups, some patients' diaphragms showed no movement from FRC after paralysis. Some subjects' diaphragms showed caudal, others' cephalad, and three in the control group displayed heterogeneous diaphragmatic segmental displacement. These findings are at odds with results of Hedenstierna *et al.*,[18] who reported cephalad (cranial) movement in six patients after anesthesia and paralysis; the dome of the diaphragm being displaced by as much as a mean of 1.9 cm. Kryer *et al.*, Warner *et al.*, and Drummond *et al.*, however, reported findings similar to ours.[2,19–21] Warner *et al.*, in two separate studies (comparing awake, anesthetized, and anesthetized–paralyzed subjects), found that dependent segments of the diaphragm were displaced cranially, whereas the nondependent segments were often displaced caudally.[19,20] Kryer *et al.* found that in three anesthetized–paralyzed subjects, the diaphragms of two subjects were displaced cranially, and one was displaced caudally.[2] Finally, Drummond *et al.* found that in anesthetized, nonparalyzed subjects, a cranial shift of the diaphragm occurred in only 10 of 20 subjects studied.[21] Thus, previously reported decreases in FRC[18,20] after anesthesia and anesthesia and paralysis cannot be solely attributed to changes in diaphragmatic position.

Gunnarsson *et al.* used CT scanning to characterize diaphragmatic position after pharmacologic paralysis in patients with COPD.[7] He found that only 2 of 10 patients showed significant cephalad, diaphragmatic displacement after pharmacologic paralysis. In the other eight patients, there was either no or minor displacement. Gunnarsson *et al.* believed that these findings helped account for the maintenance of FRC and preserved gas exchange in patients with COPD when compared with healthy persons. Because our data show there is no difference in FRC position of the diaphragm between control subjects and those with COPD after anesthesia and paralysis, previously reported maintenance of FRC and preservation of gas exchange in patients with COPD, therefore, cannot be solely attributed to difference in the behavior of the diaphragm between persons with normal pulmonary function and those with COPD.

Our finding that in both groups total diaphragmatic excursion after anesthesia and pharmacologic blockade was less than total excursion during spontaneous breaths confirms earlier work in healthy persons.[2] This finding is probably observed because after muscle paralysis, the distribution of a breath within the chest depends on the relative compliances of the rib cage and the diaphragm such that more volume is displaced by motion of the rib cage.[19,22]

The main limitation of our study is that we analyzed a three-dimensional structure (the diaphragm) using one x-ray silhouette. Using a lateral silhouette to make generalizations about a three-dimensional structure is akin to viewing the heart with the limited "ice-pick" view of M-mode echocardiography and trying to make generalizations about a three-dimensional cardiac structure. Clearly, areas of regional wall dyskinesis are easily missed. This, likewise, may be true of areas of regional diaphragmatic dyskinesis; they, too, may be missed.

In conclusion, we found that diaphragmatic motion in supine patients with COPD and hyperinflation, during spontaneous breathing and during PPV after anesthesia and paralysis, is similar to diaphragmatic motion in persons with normal pulmonary function during spontaneous breathing and PPV after anesthesia and paralysis. This surprising finding is perhaps understandable, when viewed in the context of previous studies showing preservation of diaphragmatic function in patients with COPD. In addition, we demonstrated that previously reported decreases in FRC in healthy persons, after anesthesia and paralysis, cannot be solely attributed to changes in diaphragmatic position. Likewise, previously reported preservation of FRC, after anesthesia and paralysis, in patients with COPD also cannot be solely attributed to changes (or lack there of) in diaphragmatic position.

The authors thank Mr. Bernard Robinson, A.S., A.R.R.T., (Chief Technician, Diagnostic Radiology-114A, Edward Hines Jr. Veterans Affairs Hospital, Hines, Illinois) for his efforts and expertise in the acquisition of the diaphragmatic fluoroscopic images.

References

1. Froese AB, Bryan A: Effects of anesthesia and paralysis on diaphragmatic mechanics in man. ANESTHESIOLOGY 1974; 41:242–55

2. Krayer S, Rehder K, Vetterman J, Didier EF, Ritman EL: Position and motion of the human diaphragm during anesthesia-paralysis. ANESTHESIOLOGY 1989; 70: 891–8

3. Butler C: Diaphragmatic changes in emphysema. Am Rev Resp Dis 1976; 114:115–21

4. Gibson GJ, Pride NB, Clark E: Function of the diaphragm in patient with severe hyperinflation. Am Rev Resp Dis 1979; 119(suppl):175–7

5. Rochester DF, Braun NMT, Arora NS: Respiratory muscle strength in chronic obstructive pulmonary disease. Am Rev Resp Dis 1979; 119(suppl): 151–4

6. Polkey MI, Kyroussis D, Hamnegard C-H, Mills GH, Green M, Moxham J: Diaphragm strength in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1996; 154:1310–7

7. Gunnarsson L, Tokics L, Lundquist H, Brismar B, Strandberg B, Berg B, Hedenstierna G: Chronic obstructive pulmonary disease and anesthesia: Formation of atelectasis and gas exchange impairment. Eur Respir J 1991; 4:1106–16
8. Decramer M: Hyperinflation and respiratory muscle interaction. Eur Respir J 1997; 10:934–41
9. Kim MJ, Druz WS, Danon J, Machnach W, Sharp JT: Mechanisms of the canine diaphragm. J Appl Physiol 1976; 41(3):369–82
10. Decramer M, De Troyer A, Kelly S, Macklem PT: Mechanical arrangement of costal and crural diaphragms in dogs. J Appl Physiol 1984; 56:1484–90
11. De Troyer A, Sampson, Sigrist S, Macklem PT: The diaphragm: Two muscles. Science 1981; 213:237–8
12. Road J, Newman J, Derenne P, Grassino A: In vivo length-force relationship of canine diaphragm. J Appl Physiol 1986; 60:63–70
13. Decramer M, De Troyer A, Kelly S, Zocchi L, Macklem P: Regional differences in abdominal pressure swings in dogs. J Appl Physiol 1984; 57:1682–7
14. Similowski T, Sheng Y, Gauthier AP, Macklem PT, Bellemare F: Contractile properties of the human diaphragm during chronic hyperinflation. N Engl J Med 1991; 325:917–23
15. De Troyer A: Effect of hyperinflation on the diaphragm. Eur Respir J 1997; 10:708–13
16. Farkas GA, Roussos C: Diaphragm in emphysematous hamsters: Sarcomere adaptability. J Appl Physiol 1983; 54:1635–40
17. Rochester DF: The diaphragm in COPD (editorial). N Engl J Med 1991; 325:961–2
18. Hedenstierna G, Stranberg A, Brismar B, Lundquist H, Svensson L, Tokics L: Functional residual capacity, thoracoabdominal dimensions, and central blood volume during general anesthesia with muscle paralysis and mechanical ventilation. ANESTHESIOLOGY 1985; 62:247–54
19. Warner DO, Warner M, Ritman E: Mechanical significance of respiratory muscle activity in humans during halothane anesthesia. ANESTHESIOLOGY 1996; 84:309–21
20. Warner DO, Warner M, Ritman E: Human chest wall function while awake and during halothane anesthesia. ANESTHESIOLOGY 1995; 82:6–19
21. Drummond GB, Allan PL, Logan MR: Changes in diaphragmatic position in association with the induction of anesthesia. Br J Anaesth 1986; 58:1246–51
22. Van Lith P, Johnson FN, Sharp JT: Respiratory elastances in relaxed and paralyzed states in normal and abnormal man. J Appl Physiol 1967; 23:475–86