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Effects of Anesthesia and Paralysis on Diaphragmatic Mechanics in Man

Alison B. Froese, M.D., and A. Charles Bryan, M.D., Ph.D., F.R.C.P.(C)†

Using a radiologic technique, the position and pattern of movement of the diaphragm have been evaluated in three adult volunteers, both awake and anesthetized, during spontaneous ventilation and with muscle paralysis and mechanical ventilation. Studies were made with the subjects in supine and left lateral decubitus positions with tidal and large-volume breaths. Positive end-expiratory pressure (PEEP) was added in studies of two subjects. During spontaneous ventilation awake or anesthetized, because of regional mechanical advantages, the dependent part of the diaphragm had the greatest displacement despite the higher intra-abdominal pressure in this region. Paralysis, awake or anesthetized, caused a cephalad shift of the end-expiratory position of the diaphragm that was disproportionately large in dependent regions. It also reversed the pattern of diaphragmatic displacement. The passive diaphragm was displaced preferentially in nondependent zones where abdominal pressure is least. Consequently, PEEP could not restore the diaphragm to its awake functional residual capacity position, and large breaths also could not duplicate the pattern of displacement achieved spontaneously. (Key words: Ventilation: diaphragm, mechanics; Ventilation: distribution; Ventilation: positive end-expiratory pressure.)

IMPAIRED GAS EXCHANGE is a frequent accompaniment of anesthesia.[1,2] The etiology of this is not clearly understood. Even the basic question of the effect of anesthesia on lung volume has resulted in conflicting answers. Although most investigators show a decrease of functional residual capacity (FRC) with anesthesia,[3–7] some have found no change.[8]

* Research Fellow in Anesthesia. Supported by M.R.C. Research Fellowship.

† Associate Professor, Department of Anesthesia.

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Most studies have concentrated on overall changes in respiratory values, with few attempts to describe the spatial relationships of these changes. The work of West,[9] Ball,[10] and many others since 1960 has emphasized the importance of gravitational gradients of both ventilation ($\dot{V}$) and perfusion ($\dot{Q}$) in determining regional $\dot{V}/\dot{Q}$ ratios and the resultant gas exchange.

Normal, spontaneous ventilation results in preferential distribution of volume to dependent areas in upright, lateral, supine or prone positions.[11,12] Milic-Emili[12] emphasized that this distribution of ventilation parallels regional lung compliance, which is determined by variations in alveolar volume created by the gravity-related gradient of pleural pressure. Agostoni subsequently showed that this pleural pressure gradient is not merely a reflection of pulmonary density. Approximately 40 per cent is contributed by the abdominal contents and diaphragm in dogs and rabbits.[13]

A striking reversal of the normal pattern of ventilation occurs with anesthesia and paralysis. Potgieter,[14] Nunn,[15] and Rehder[16] have all shown preferential ventilation of the nondependent lung in anesthetized, paralyzed subjects in either lateral decubitus position.

Possible explanations of this shift in ventilation away from dependent areas include: 1) some effect of anesthesia on regional lung compliance; 2) a marked increase in the regional variation of airway resistance, such that dependent zone resistance increases enough to affect distribution; 3) a change in the regional mechanics of the chest wall (*i.e.*, abdomen/diaphragm) due to the effects of anesthesia and paralysis.

However, important features of Potgieter's study are the rapidity with which changes occurred and their consistent relationship to

TABLE 1. Physical Characteristics of Subjects Studied

	Age (Years)	Height (cm)	Weight (kg)	Total Lung Capacity (l)	Functional Residual Capacity (l)	Residual Volume (l)
Subject 1	46	180	77.0	6.5	3.34	2.11
Subject 2	37	172	67.8	7.4	3.71	1.90
Subject 3	47	175.5	91.4	7.9	3.03	1.86

Volumes are in liters, BTPS.

alterations of posture.[14] These observations suggest that the primary mechanism is a gravity-related change in the mechanics of the chest wall.

With this rationale, we have investigated the regional effects of anesthesia and paralysis on the mechanics of the diaphragm in normal adult subjects. Our data suggest that the redistribution of ventilation can be explained by the effects of anesthetic agents and muscle relaxants on the diaphragm, which has a crucial role in separating two compartments of markedly different densities, the thorax and the abdomen.

Method

Subjects were three volunteer staff anesthetists of widely differing configuration who were fully informed of the nature of the proposed study. Their physical characteristics are listed in table 1.

The position and the pattern of movement of the diaphragm in both supine and left lateral decubitus positions were assessed. Only one decubitus position was studied, to minimize exposure to radiation. To separate the effects of general anesthesia from those of muscle paralysis, two subjects were studied awake, supine, paralyzed with succinylcholine, and mechanically ventilated. All subjects were also studied awake, breathing spontaneously; anesthetized, breathing spontaneously; and anesthetized, paralyzed, with positive-pressure ventilation.

Movement of the diaphragm was monitored fluoroscopically using a well-collimated Siemens 3-phase image intensification system, data being recorded on videotape for subsequent analysis. Since the x-ray beam was directed "cross-table," the patterns of diaphragmatic movement observed reflected the influence of gravitational forces.

The dosages of radiation to the skin using

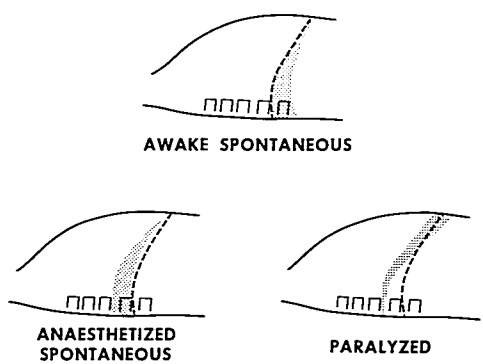

FIG. 1. Diaphragm position and displacement during tidal breathing in supine Subject 2. Dashed line = control functional residual capacity position of the diaphragm. Stippled area represents diaphragmatic excursion during tidal breathing.

TABLE 2. Regional Changes in the FRC Position of the Diaphragm with Onset of Anesthesia or Paralysis in Supine Position

	Change in Condition*	Displacement† (in cm)		
		Bottom‡	Middle	Top
Subject 1	A-S to AN-S	−0.8	−0.3	+1.2
	A-S to A-P	−1.1	−0.2	+1.0
Subject 2	A-S to AN-S	−3.1	−2.8	−1.4
	A-S to A-P	−3.3	−3.0	−1.5

* A-S = awake, spontaneous; AN-S = anesthetized, spontaneous; A-P = awake, paralyzed.

† + = caudad displacement; − = cephalad displacement.

‡ Throughout bottom, middle, and top levels through the diaphragm are selected from dependent to nondependent regions as in inset, figure 2.

this system averaged 200 m rad/min. Exposure times were 8–19 minutes. (For comparison, the surface exposure per anteroposterior film of the abdomen is approximately 700 m rad.[17])

Lead surface markers were used to identify an intervertebral space as a point of reference for position of the diaphragm. Magnification factors were calculated using a lead calibration strip in the field of view, so that displacements could be determined in cm. Volume changes were recorded at the airway using a Vertek VR4000 heated digital pneumotachograph and integrator. Flow and volume signals were charted on a strip recorder together with airway pressure, which was measured from a side port at the airway using a Statham PR23-2D-300 pressure transducer. Appropriate correction factors were used for the pneumotachograph according to the composition of the anesthetic mixture used.

Subjects were fasting and had no premedication. Control measurements were made in both positions. In the two subjects who were to be paralyzed awake, nasotracheal intubation was then performed under local cocaine analgesia using a cuffed tube (Portex 7.5 mm). Control measurements were repeated and results were found to be the same as those of the previous measurements made via mouthpiece.

An intravenous infusion of 5 per cent dextrose in water was established and maintained for the duration of the procedure. Muscle paralysis was induced with succinylcholine (1.5 mg/kg, iv) after pretreatment with *d*-tubocurarine (0.06 mg/kg) to minimize muscle fasciculations. The awake, paralyzed subjects were ventilated with 100 per cent O_2. Anesthesia was induced with thiopental, iv, and maintained with 1–1.5 per cent halothane–67 per cent N_2O–33 per cent O_2. In the third subject, a routine intravenous barbiturate–succinylcholine induction with

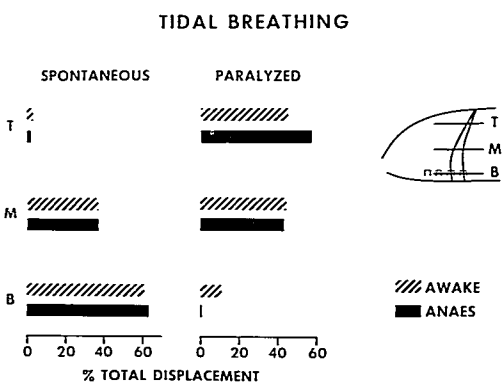

FIG. 2. Regional diaphragm displacements during breathing in supine Subject 2, while awake–breathing spontaneously, anesthetized–breathing spontaneously, awake–paralyzed, and anesthetized–paralyzed and mechanically ventilated. Inset indicates the three levels at which diaphragmatic displacement was analyzed. The linear displacements measured at T (top) + M(middle) + B(bottom) = total displacement. Regional displacements were expressed as percentages of this total value.

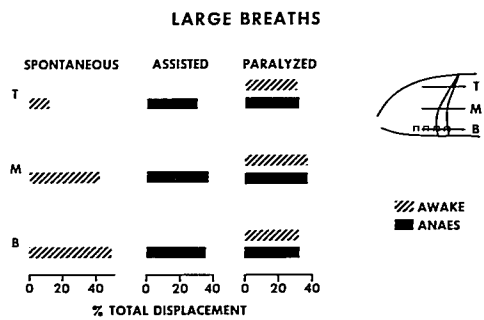

FIG. 3. Regional diaphragmatic displacements (as per cent of total) for supine Subject 2 during large breaths, awake–spontaneous ventilation, anesthetized–assisted ventilation, and anesthetized–paralyzed. Inset indicates the three levels analyzed.

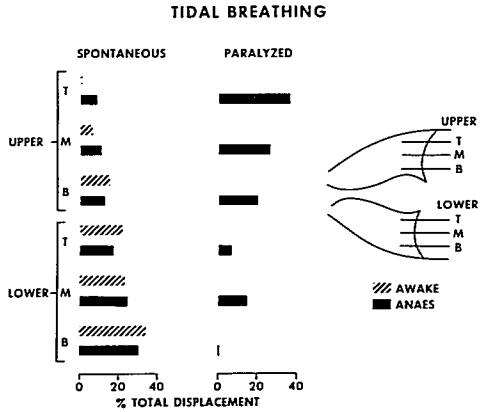

FIG. 4. Regional diaphragmatic displacements (as per cent of total) for Subject 2 in the left lateral decubitus position during tidal breathing, awake–spontaneous ventilation, anesthetized–spontaneous ventilation, and anesthetized–paralyzed. Inset indicates the six levels at which diaphragmatic displacement was measured. All six values were summed to give the total displacement.

oral intubation was carried out after control studies. Following measurements made during spontaneous breathing under anesthesia and then while paralyzed with positive-pressure ventilation, the subjects emerged from the anesthesia and were observed for several hours in the recovery area.

Two volume ranges were studied. Spontaneous tidal volume was measured awake and matched during positive-pressure ventilation (4.6–6.3 ml/kg). During anesthesia, spontaneous tidal volume was reduced, as expected, to approximately 60 per cent of control. Carbon dioxide was, therefore, added to the anesthetic mixture in two cases, with resulting increases in tidal volume to 69 and 100 per cent of control values. Large breaths (14.5–25.9 ml/kg) were delivered from a large calibrated syringe.

Three to four consecutive breaths were recorded under each condition. Prior to each series of measurements, three maximal inflations were performed, and the desired breathing pattern was then re-established and recorded. Positive end-expiratory pressure (PEEP) was added by connecting the ex-

piratory port of a one-way valve to a 10 cm H_2O threshold resistor (*i.e.*, tube under water) for several breaths.

After the study, the videorecordings were reviewed in slow motion so end-inspiratory and end-expiratory positions of the diaphragm could be determined accurately. The videotape was stopped at the correct frame and the diaphragmatic configuration and position relative to bony landmarks traced out. This technique surmounted the problem of trying to study dynamic breathing maneuvers with single-shot x-rays, since timing was critical. For purposes of analysis, three levels were selected on the tracings of diaphragmatic position, from nondependent to dependent regions. (see inset, figure 2, for diagrammatic representation). For a given breath the linear displacement of the diaphragm at each of these levels was measured and the values summed to give total displacement. Regional displacement at each level was expressed as a percentage of the total. In supine studies the bottom level was drawn through the vertebral bodies, with the other two levels equally spaced above.

Results

Figure 1 summarizes the patterns of diaphragmatic displacement. Spontaneous ventilation awake was characterized by predominant movement at the dependent level of the diaphragm. Anesthesia induced a cephalad shift in end-expiratory (FRC) diaphragmatic position that was greater in dependent areas. Despite this shift, diaphragmatic movement remained greatest in dependent areas when the subject breathed spontaneously.

Paralysis caused a cephalad shift in the diaphragmatic FRC position similar to the shift with anesthesia alone. The quantitative changes in position are given in table 2 for two subjects. In addition, there was a striking reversal of the pattern of diaphragmatic movement. Most displacement now occurred in nondependent areas, with minimal movement at the most dependent level. That is, two distinctly different patterns of diaphragmatic displacement were seen from the same new FRC position.

Figure 2 shows the quantitative relationship during tidal breathing between the level of the diaphragm and the percentage of total diaphragmatic displacement observed at that level. The change in pattern of movement correlates with the presence or absence of the muscle relaxant rather than the anesthetic agent itself. In this subject 61–64 per cent of the total displacement of the diaphragm was generated at the most dependent level during spontaneous ventilation, awake or anesthetized. In contrast, only 0–11 per cent of the total movement occurred there with paralysis and mechanical ventilation at the same tidal volume. The effects of muscle paralysis on the distribution of diaphragmatic displacement were identical, awake or anesthetized.

The patterns of diaphragmatic movement with large breaths (1,500 ml = 22.1 ml/kg in this subject) are depicted in figure 3. Movement remains predominantly dependent with spontaneous large breaths. During paralysis, diaphragmatic displacement is more evenly distributed with large-volume ventilation.

Large breaths during anesthesia without paralysis are depicted separately in figure 3. They result from an initial spontaneous inspiratory effort (displacement mainly dependent) which is assisted by positive pressure to the final 1,500 ml volume (displacement now in nondependent areas). The end result most closely resembles the paralyzed distribution.

Tidal volume measurements in the left lateral decubitus position are shown in figure 4. In this position, three levels of each diaphragm were assessed, and displacement expressed as a percentage of the total observed at all six. The same general patterns are seen. Although the lower diaphragm is pushed further into the thorax by the weight of abdominal contents, the most dependent level of the lower diaphragm still has the greatest displacement during spontaneous ventilation, awake or anesthetized. However, with paralysis and mechanical ventilation there is minimal movement of the dependent diaphragm, and the pattern of displacement is reversed.

TABLE 3A. Regional Diaphragmatic Displacements in Three Subjects with Varied Breathing Patterns, Supine

	Regional Displacements as Per Cent of Total					
	Tidal Breaths			Large Breaths		
	Top	Middle	Bottom	Top	Middle	Bottom
Awake, spontaneous ventilation	14.5 2.5 15	38.5 36.5 34	46.5 61 51	13 10.5 23	37 41.5 32	50 48 45
Anesthetized, spontaneous ventilation	21 0 0	42 36.5 36	37 63.5 64	27* 29 19	36 36 41	36 35 41
Awake, paralyzed	40 44	40 44	20 11	26 30	38 37	36 33
Anesthetized, paralyzed	45 57 40	40 43 46	15 0 14	33 35 31	43 36 39	24 29 30

* Anesthetized, assisted ventilation.

TABLE 3B. Regional Diaphragmatic Displacements in Three Subjects with Varied Breathing Patterns, Lateral Decubitus Position

	Regional Displacements as Per Cent of Total					
	Right			Left		
	Top	Middle	Bottom	Top	Middle	Bottom
Tidal breaths						
Awake, spontaneous ventilation	0 0 14	12.5 6.5 14	20 15.5 13	14 20.5 19	25 22.5 19.5	30 35 20
Anesthetized, spontaneous ventilation	9 8 17	12 10 11	12 12 7	22 17 12.5	21 24 22	24 29 31
Anesthetized, paralyzed	27 35 45	23 25 30	19 19 12	19 6 9	11.5 15 4.5	0 0 0
Large breaths						
Awake, spontaneous ventilation	− 7 −13 10	9 4 13	15 12 14	19 18 19	25 17 20	25 36 24
Anesthetized, assisted ventilation	11.5 28	14 19	14 15	22 11	19 12	19 15
Anesthetized, paralyzed	20 25 40	19 21.5 27	18 17 14	19 9.5 11	14 15.5 6	10 11 2

TABLE 4. Regional, Diaphragmatic Displacements for Spontaneous Breaths with Varied Tidal Volumes (Subject 3), Supine

Tidal Volume	Regional Displacements as Per Cent of Total		
	Top	Middle	Bottom
Spontaneous			
400 ml	0	36	64
580 ml	15	34	51
1,000 ml	9	32	59
Paralyzed			
420 ml	40	46	14

Two further observations were made during large breaths in the lateral decubitus position: 1) In the lateral decubitus position motion of the dependent chest wall is restricted. Nevertheless, most of the diaphragmatic movement is in these dependent regions. In the presence of a fixed lower chest wall, this displacement of the diaphragm is reflected in upward movement of the mediastinum, and an outward, cephalad swing of the nondependent rib cage. It can be seen from table 3 (*B*) that in two subjects the result was an actual cephalad diaphragmatic displacement at the top upon inspiration. These patterns were reversed by paralysis and mechanical ventilation. The greatest caudad displacement was now at the upper diaphragm levels, the mediastinum was pushed downwards with inspiration, and the movement of the dependent diaphragm was markedly decreased. 2) In the lateral position large-volume ventilations were not as evenly distributed as breaths of equal volume in the supine position. Instead, displacement of the upper diaphragm remained greater than that of dependent levels even with large-volume, slow ventilation (table 7).The figures above are based on data from Subject 2 alone. In table 3 (*A*) and (*B*) the percentage displacement at each level is shown for all three subjects in the supine and lateral decubitus positions under the different conditions. The consistency of the patterns is obvious despite interpersonal variations.

Table 4 shows data for the distribution of displacement in one subject during spontaneous breaths ranging from 400 to 1,000 ml in tidal volume. For comparison, the paralyzed distribution is also given. It is evident that the distribution during spontaneous ventilation is relatively volume-insensitive. It is the change in mode of ventilation, not the size of the breath, that alters the pattern.

The effects of adding 10 cm H_2O PEEP are given for one subject in tables 5 and 6. The greatest cephalad shift in the FRC position of the diaphragm was in nondependent regions. Regional diaphragmatic displacements measured with and without PEEP are also listed. No significant change in the pattern of displacement was seen with the addition of PEEP.

Discussion

These observations show that in horizontal postures the regional distribution of diaphragmatic displacement depends primarily on whether a volume change results from active contraction of the diaphragm or from application of an external force to a passive diaphragm.

Although the actual measurements made were of regional diaphragmatic position rather than regional volume change, two observations support our deduction that diaphragmatic displacement is a qualitative indicator of regional ventilation and volume: 1) The regional change in radiolucency of the lung next to the diaphragm paralleled regional diaphragmatic movement during a breath, *i.e.*, large diaphragmatic displacements were accompanied by marked increases in radiolucency (greater aeration) of the lung at that level. 2) Specific bronchovascular lung markings could be followed throughout a given breath. In the absence of diaphragmatic displacement there was no change in the position of a particular marking, whereas with increased diaphragmatic movement the regional lung markings lengthened and spread apart. In subsequent discussion we assume that regional diaphragmatic displacement is proportional to regional volume change in a qualitative but not strictly quantitative way.

TABLE 5. Change in FRC Position of the Diaphragm with Addition of 10 cm H_2O PEEP (Subject 3)

	Caudad Displacement in cm		
	Top	Middle	Bottom
Supine			
Awake, spontaneous ventilation to A-S with PEEP	2.2	1.9	1.7
Anesthetized, paralyzed, to AN-P with PEEP	1.7	1.2	0.8
Left lateral decubitus position			
Anesthetized, paralyzed, to AN-P with PEEP			
Right	3.3	2.3	0.9
Left	0.8	0.6	0

TABLE 6. Regional Diaphragmatic Displacement with and without PEEP

	Regional Displacement as Per Cent of Total		
	Top	Middle	Bottom
Supine			
Awake, spontaneous ventilation			
Tidal breaths			
ZEEP*	15	34	51
PEEP*	9	32	59
Large breaths			
ZEEP	23	32	45
PEEP	10	33	57
Anesthetized, paralyzed			
Tidal breaths			
ZEEP	40	46	14
PEEP	35	47	18
Large breaths			
ZEEP	31	39	30
PEEP	33	36	31

	Right			Left		
	Top	Middle	Bottom	Top	Middle	Bottom
Left lateral decubitus position						
Anesthetized, paralyzed						
Tidal breaths						
ZEEP	45	30	12	9	4.5	0
PEEP	32.5	32.5	21	14	0	0
Anesthetized, paralyzed						
Large breaths						
ZEEP	40	27	14	11	6	2
PEEP	30	18	17	17	11.5	5

* ZEEP = Zero end-expiratory airway pressure.
PEEP = 10 cm H_2O end-expiratory airway pressure.

Change in Diaphragm Position

In classic concepts, the FRC of the lung in the upright subject is established by the balance of elastic and hydraulic forces. There is no transdiaphragmatic pressure gradient: the position of the diaphragm is determined by the balance between lung elastic recoil pulling it cephalad and the weight of the abdominal contents pulling it caudad.[18]

The situation is more complex in the supine position. The diaphragm is a partition between two compartments of markedly different hydrostatic gradients. On the thoracic side, pressure increases approximately 0.2 cm H_2O/cm lung height[19]; on the abdominal side, 1.0 cm H_2O/cm.[18] This means that in horizontal postures, progressively higher transdiaphragmatic pressures must be generated towards dependent parts of the diaphragm to keep the abdominal contents out of the thorax. This tension could be developed by passive stretch and shape changes of the diaphragm, or by active tension. With acute muscle paralysis, we saw a shift of the diaphragm to a more cephalad position. The latter must express the true balance of elastic forces in the system, unmodified by any muscle activity. We conclude that the more caudad position of the diaphragm in the supine awake subject is the result of active muscle tension. This suggests that in horizontal postures, active diaphragmatic tension as well as passive stretch is required to maintain lung volume and to minimize the decrease in FRC known to occur in going from upright to supine postures.[18]

An alternative explanation of our observed changes is that, in normal supine man, the abdominal muscles have active tone that acts to decrease the abdominal pressure and shift the diaphragm caudad from its paralyzed position. However, abdominal muscles, more accessible to direct needle electromyogram (EMG) study than the diaphragm, have been shown to be electrically silent at supine FRC and even during quiet breathing, developing expiratory phasic activity only with $\dot{V}$ greater than 40 l/min.[20] On the other hand, present diaphragmatic EMG techniques have high noise-to-signal ratios, and it cannot be firmly concluded from data available in the literature that the diaphragm is, in fact, silent at FRC in the supine position.[21–23]

In our study the cephalad displacement of the diaphragm with paralysis was similar to that produced by anesthesia alone. This agrees with the results of Don,[3] who was unable to show a further decrease of FRC with the acute administration of succinylcholine after induction of anesthesia with halothane had caused an initial decrease in lung volume. The anesthetic-induced decrease of FRC may be the result of a decrease in neural output to the diaphragm, or an increase in expiratory tone of the abdominal muscles, raising intra-abdominal pressure and forcing the diaphragm cephalad. Freund *et al.*[24] demonstrated that onset of expiratory activity of the lateral abdominal muscles coincided with loss of consciousness during either N_2O–O_2 or halothane anesthesia, using needle electrode EMG's. Kallos *et al.*[25] present evidence that Innovar may increase tone in expiratory muscles to such an extent that the reduction of FRC with Innovar anesthesia alone is greater than that with Innovar plus muscle relaxation with succinylcholine.

The above data suggest that the FRC position of the diaphragm during anesthesia without muscle paralysis is the end result of factors acting on other respiratory muscle groups as well as the diaphragm. It is quite probable that the magnitude of this change in FRC is dependent on the anesthetic agents used, as well as body habitus.

Change in Pattern of Displacement

During spontaneous breathing in the awake subject, the dependent parts of the lung are the best ventilated, in both supine and lateral positions.[11] Our data show that there is greater displacement of the dependent part of the diaphragm, in both supine and lateral positions. This greater displacement is achieved despite the fact that the force opposing displacement—the hydrostatic pressure of the abdomen—is greatest in the dependent region.

Two mechanisms may enable the actively-contracting diaphragm to generate more force in the dependent region. The first

is that the dependent part of the diaphragm has a smaller radius of curvature, in both supine and lateral positions (for example, see fig. 5). Therefore, from the Laplace relationship, for the same diaphragmatic tension more pressure will be generated across dependent parts of the diaphragm because of their smaller radius of curvature. A second possible factor is that the isometric force developed by a muscle increases as its resting length increases. Marshall[26] has shown in cats and Pengelly[27] in man that, as lung volume decreases and the muscle fibers of the diaphragm lengthen, its effectiveness as a pressure generator increases. It is possible that the dependent part of the diaphragm is not only more curved, but also more stretched and, therefore, can develop greater force due to the length/tension relationship.

In our study we found that the pattern of diaphragmatic displacement was the same during spontaneous breathing during anesthesia as it was awake. This follows since the contracting diaphragm confers the same regional mechanical advantages on dependent areas during spontaneous breathing during anesthesia as it does awake, because the same shape/tension relationships apply.

When the diaphragm is paralyzed, its motion is no longer determined by active contraction, and these regional mechanical advantages are no longer in effect. During mechanical ventilation a relatively uniform pressure is applied to the thoracic side of the diaphragm through the airway. This is opposed by the hydrostatic pressure gradient of the abdominal contents. As a result, the applied positive pressure displaces the diaphragm preferentially where the abdominal pressure is least, namely in the nondependent areas. This distribution is similar to attempting to inflate an excised lung under water. Most of the volume would go to the top of the lung where the surrounding pressure is least, with little volume change at the bottom where the hydrostatic pressure opposing expansion is high.

Therefore, the distribution of diaphragmatic displacement depends on whether the force is being developed by active contraction of the diaphragm or by an external force

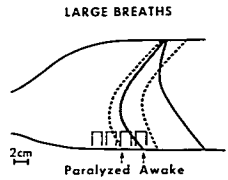

FIG. 5. Scale drawing of the displacement of the diaphragm during 1,800-ml breaths in supine Subject 2. Spontaneous ventilation = solid black outline; mechanical ventilation with paralysis = stippled area with dashed outline.

acting on a passive diaphragm. In the awake spontaneously-breathing subject the greatest displacement is in the dependent region, and this region is also the best ventilated. We also found spontaneous breathing during anesthesia to be characterized by greater displacement of the dependent region and concluded that these areas had the best ventilation under our conditions of study. This does not necessarily mean that dependent zones will receive more ventilation under all circumstances with general anesthesia and spontaneous ventilation. The distribution of spontaneous ventilation depends markedly on the FRC because, as Milic-Emili[12] has shown, there is a reversal of ventilation distribution at low lung volumes due to dependent airway closure. Reductions of FRC during anesthesia vary in magnitude; therefore, the precise distribution of ventilation will depend on how much the FRC decreases, the relationship of that volume to the subject's closing volume, and the size of the tidal volume. As these factors are all variable, the ventilation distribution during spontaneous breathing during anesthesia will be variable, and this may account for some of the confusion in the literature. However, when the diaphragm is paralyzed the distribution of diaphragmatic displacement is completely reversed. As a result, the distribution of ventilation must change, leading to preferential ventilation of nondependent regions even when lung volume is maintained. This must apply not only during anesthesia but also to many patients undergoing controlled ventilation for respiratory failure.

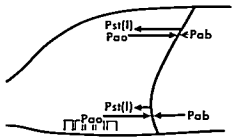

FIG. 6. Diagram showing the balance of forces across the diaphragm in a dependent and a nondependent region during maintained positive airway pressure with the subject supine. P_{ao} = airway pressure, $P_{st}(l)$ = elastic recoil pressure of lung, P_{ab} = abdominal pressure. The pressure relationships are shown for a lung volume large enough (*e.g.* > 60 per cent of vital capacity) for transdiaphragmatic pressure to be zero in the relaxed subject. $\therefore P_{st}(l) = P_{ao} - P_{ab}$ in each region.

CHANGES IN FRC AND AIRWAY CLOSURE

The relationship of FRC to the lung volume at which closure begins has been used to infer the presence of airway closure during tidal breathing.[28] Closure is recognized to be a regional dependent-zone phenomenon, whereas reductions in FRC have been considered to be uniformly distributed.

We observed a disproportionately large cephalad shift of the dependent part of the diaphragm with either anesthesia or paralysis (table 2). Consistently, the major displacement occurs where the abdominal pressure is highest. That is, the loss of lung volume is regionally distributed. This must increase the probability of closure in dependent zones and make the effect of a reduction of FRC on gas exchange more serious than predicted from the overall relationship of FRC and closing volume.

The disproportionate loss of dependent lung volume accentuates the importance of the ventilatory pattern during anesthesia. With spontaneous ventilation the greatest diaphragmatic displacement was in dependent areas and, therefore, more alveoli should open during at least part of the respiratory cycle, improving regional gas exchange and lessening chances of absorption atelectasis. With the redistribution of $\dot{V}$ away from these areas during paralysis and positive-pressure ventilation, however, dependent regions would remain areas of low $\dot{V}/\dot{Q}$ throughout the respiratory cycle, increasing the risk of dependent-zone atelectasis and postanesthetic complications.

Our results suggest that dependent-zone ventilation should be better maintained during anesthesia by spontaneous ventilation than by paralysis and mechanical ventilation. Wulff and Aulin demonstrated this using ^{133}Xe $\dot{V}$ and $\dot{Q}$ studies.[29] In the lateral decubitus positions, regional ventilation/unit volume in the dependent lung was significantly decreased (−17.4 per cent) by paralysis and controlled ventilation, but only slightly decreased (−5.5 per cent) in the spontaneously breathing, anesthetized subject. Since regional perfusion decreased slightly in both conditions, the resulting regional $\dot{V}/\dot{Q}$ was unchanged with spontaneous respiration but decreased significantly with paralysis.

EFFECTS OF PEEP ON REGIONAL LUNG VOLUME AND $\dot{V}$

Westbrook *et al.*[7] suggested that part of the abnormality of gas exchange during anesthesia was due to the reduction of FRC. They proposed that positive end-expiratory pressure (PEEP) could be expected to restore lung volume and also gas exchange in anesthetized subjects.

We found a predominantly dependent-zone cephalad displacement of the diaphragm with anesthesia or paralysis. In order to correct this volume loss and the coincident gas exchange problem, a maneuver that affects mainly dependent zones is needed. In contrast to this desired effect, we observed that 10 cm H_2O PEEP displaced mainly the superior part of the diaphragm, with little alteration in dependent diaphragmatic position (tables 5 and 6). This is what one would expect from the balance of hydrostatic forces across the paralyzed diaphragm. The result was that PEEP increased lung volume in areas already well expanded and failed to influence the distribution of diaphragmatic displacement. Recently, Rehder[30] confirmed the inability of 9 cm H_2O PEEP to restore the FRC lost from the dependent lung during anesthesia. Although the fraction of dependent lung volume decreased by 16 per cent going from the supine to the lateral position, it increased only 2 per cent with PEEP.

The observed clinical effectiveness of PEEP in respiratory failure must be the result of opening of lung units in some mid-regions of the lung. This effect must be less, however, than that which would result if the same volume change occurred specifically in dependent lung zones.

Distribution of Large Breaths

Throughout the volume range from tidal to inspiratory capacity, diaphragmatic displacement was predominantly in dependent zones in the awake, spontaneously-breathing subject. That is, so long as inspiration resulted from contraction of the diaphragm and other inspiratory muscles, the result was greater dependent-zone displacement.

In the paralyzed subject the pattern altered with volume. A 500-ml "tidal breath" at <.5 lps flow rate resulted in minimal displacement of the dependent diaphragm. With increasing inspiratory volumes during mechanical ventilation and, therefore, increasing airway pressures, the distribution became more even as the change in transpulmonary pressure became large relative to the magnitude of the abdominal hydrostatic gradient. At best, this resulted in approximate equalization of displacement in the most dependent and least dependent areas, in supine subjects. In figure 5, an 1,800-ml or 26.5 mg/kg breath delivered at <.5 lps over 4 seconds is compared with an 1,800-ml breath attained spontaneously, awake, at a similar flow rate. The difference in the caudad excursion of the diaphragm is obvious. This observation raises the question whether "true TLC" is attainable in the paralyzed subject. It may explain the striking apparent loss of lung volume in Westbrook's study, where during anesthesia lung volume could not be brought up to awake total lung capacity values even with sustained positive airway pressures of 35–40 cm H_2O.[7]

Using a static rather than dynamic situation, one can analyze the forces acting on the lower diaphragm when attempting to reach total lung capacity by positive-pressure ventilation. Assuming transdiaphragmatic pressure in the relaxed subject to be zero at high lung volumes,[31] the forces acting cephalad are abdominal pressure (P_{ab}) and the static recoil of the lung [P_{st} (1)]. The only caudally-directed force is airway pressure (P_{ao}). This balance is depicted in figure 6 for two levels of the diaphragm. At each point P_{st} (1) = P_{ao} – P_{ab}, and regional lung expansion will depend on this pressure difference plus the regional pressure–volume curve of the lung. In these terms, dependent lung expansion is a function of local P_{ab}—which has a hydrostatic component related to abdominal height, and will be modified as well by things such as external abdominal compression, and increased abdominal tension due to ascites, peritonitis, pregnancy, and other factors. In support of this, we observed that in the lateral decubitus position, the discrepancy between dependent-zone and superior diaphragmatic displacements remained even at large volumes. This is shown in table 7. The displacements of the most superior and most dependent levels became equal with large breaths in the supine position, but remained greater at upper levels in the lateral position (*e.g.*, 4.9 cm bottom, *vs.* 8.6 cm, top). This correlates with the greater effective height of the abdomen (and, therefore, greater dependent-zone P_{ab}) in the lateral decubitus position as compared with the supine.

TABLE 7. Actual Displacements (in cm) of Top and Bottom Levels of the Diaphragm in Supine and Lateral Positions: Left Panel, Tidal Breaths; Right Panel, Large Breaths*

	Displacement of Diaphragm (in cm)			
	Tidal Breaths		Large Breaths	
	Top	Bottom	Top	Bottom
Supine				
Subject 1	1.0	0.4	1.8	1.9
Subject 2	0.8	0.0	2.5	2.1
Subject 3	0.7	0.25	1.9	1.8
Lateral decubitus				
Subject 1	1.8	0.8	6.7	5.1
Subject 2	4.1	1.0	8.6	4.9
Subject 3	2.9	0.5	10.6	2.5

* *Note*: Greater displacement occurs at the top level in both positions with tidal breaths. During large breaths displacement evens out in the supine position, but remains greatest at the top level in the lateral position. For discussion, see text.

It is clear that neither PEEP nor large breaths can fully restore ventilation to dependent lung zones in the horizontal paralyzed subject. The only way to ventilate these regions is to modify the effect of abdominal mass by manipulating posture. In this respect the optimal position would be prone with abdomen unsupported.

A failure to attain "true" total lung capacity in dependent lung regions must affect measurements of compliance made during anesthesia. It is well established that the compliance of the lung is reduced when it is not expanded fully prior to measurements, due to hysteresis of surface forces as well as recruitment of lung units.[32] A marked reduction in total respiratory compliance (C_{rs}) has been documented by Margaria *et al.* (C_{rs} .084 l/cm H_2O awake *vs.* C_{rs} .054 l/cm H_2O anesthetized),[33] as well as Westbrook *et al.* (.121 l/cm H_2O awake *vs.* .099 l/cm H_2O anesthetized)[7] and others. The previous explanation of this change had centered on the reduction of FRC occurring with anesthesia. A decrease in FRC towards residual volume will increase the number of units closed in the tidal volume range and create a "stiffer" lung, because fewer units now participate in volume change. However, Westbrook *et al.* showed a decrease in C_{rs} even at volumes equivalent to the awake tidal volume range (*i.e.*, considerably above the anesthetized FRC). They attributed this to changes in surface forces due to low-lung-volume breathing, concluding that their observations were not affected by any difference in the patterns of chest expansion awake *vs.* anesthetized.

However, our data show that there is a further explanation. Regional restriction of movement of the dependent diaphragm will change the local volume history of the lung, and therefore would decrease compliance even in the absence of a change in FRC and amount of airway closure. We conclude that the reduction of respiratory compliance in paralyzed subjects is due in part to the changed patterns of pulmonary expansion, with regional failure to reach "true" total lung capacity.

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