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Human Chest Wall Function during Epidural Anesthesia

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Background: Although epidural anesthesia (EA) can significantly disrupt the function of the respiratory system, data concerning its effects on respiratory muscle activity and the resulting motion of the chest wall are scarce. This study aimed to determine the effects of lumbar EA on human chest wall function during quiet breathing.

Methods: Six persons were studied while awake and during mid-thoracic (approximately a T6 sensory level) and high (approximately a T1 sensory level) lumbar EA produced by either 2% lidocaine (two persons) or 1.5% etidocaine (four persons) with 1:200,000 epinephrine. Respiratory muscle activity was measured using fine-wire electromyography electrodes. Chest wall configuration during high EA was determined using images of the thorax obtained by three-dimensional, fast computed tomography. The functional residual capacity was measured using a nitrogen dilution technique.

Results: High EA abolished activity in the parasternal intercostal muscles of every participant but one, whereas the mean phasic activity of the scalene muscles was unchanged. High EA significantly decreased the inspiratory volume displacement of the rib cage compared with intact breathing but did not have a significant effect on diaphragm displacement. Therefore, high EA decreased the percentage contribution of rib cage expansion to inspiratory increases in thoracic volume (ΔV_{th}) (from 27 ± 2 [MSE] to $10 \pm 11\%$ of ΔV_{th}). Paradoxical rib cage motion during inspiration (*i.e.*, a net inward motion during inspiration) developed in only one participant. High EA substantially increased the functional residual capacity (by 295 ± 89 ml), with a significant net caudad motion of the end-expiratory position of the diaphragm. In addition, high EA significantly decreased the volume of liquid in the thorax at end expiration in five of the six participants, a factor that also

contributed to the increase in functional residual capacity in these persons.

Conclusions: Rib cage expansion continues to contribute to tidal volume during high EA in most subjects, even when most of the muscles of the rib cage are paralyzed; the mean phasic electrical activity of unblocked respiratory muscles such as scalenes does not increase in response to rib cage muscle paralysis produced by EA; and high EA increases the functional residual capacity, an increase produced in most participants by a caudad motion of the diaphragm and a decrease in intrathoracic blood volume. (Key words: Measurement technique: dynamic spatial reconstructor; fast computed tomography; respiratory impedance plethysmography; electromyogram. Anesthetic technique: lumbar epidural anesthesia. Anesthetic drugs, local: etidocaine; lidocaine. Lung: functional residual capacity; breathing pattern; intrathoracic blood volume; diaphragm; rib cage. Muscle: respiratory; diaphragm; parasternal intercostal; transversus abdominis; scalene.)

LUMBAR or thoracic epidural anesthesia (EA) could affect the respiratory system in several ways. Because the coordinated activity of several rib cage muscles is necessary for normal inspiratory expansion of the rib cage in humans,¹⁻³ motor blockade of these muscles should hinder this expansion. Tonic activity in these muscles may also be important for maintaining end-expiratory rib cage position, so that elimination of any such activity may decrease the functional residual capacity (FRC).⁴⁻⁶ The respiratory muscles also provide important afferent information from muscle spindles and tendon organs, and interruption of this information may affect respiratory control.⁷ Apart from these effects on muscle function, EA also significantly affects cardiovascular function.⁸ As a result, it may change the volume of blood in the thorax,^{9,10} which may in turn affect the amount of gas in the lungs. Finally, systemic absorption of local anesthetic may also affect respiratory control.^{11,12}

It is therefore perhaps surprising that many previous studies found that epidural (or subarachnoid) block has little effect on inspiratory respiratory function in healthy humans. The distribution of inspired gas and pulmonary gas exchange are changed only minimally.¹³⁻¹⁸ Although total lung capacity and inspiratory

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Table 1. Subject Characteristics

Subject No.	Age (yr)	Gender	Weight (kg)	Height (cm)
1	36	M	76	181
2	26	F	59	168
3	42	M	80	176
4	28	M	76	183
5	40	M	71	183
6	26	M	70	170

capacity are reduced slightly, only small, inconsistent changes in FRC have been found.^{13-16,19-21} Ventilatory responses to hypercapnia are preserved or enhanced in most studies.^{18,22-26} Although information concerning chest wall motion during EA in adult human subjects is limited, inspiratory rib cage expansion appears to be maintained.^{17,22}

The apparent conflict between the importance of rib cage muscles to chest wall function and the lack of effect of EA may arise from (1) low dermatomal levels of anesthesia that involve little of the rib cage, (2) lack of motor blockade of the respiratory muscles,^{20,27} (3) compensation for paralyzed muscles by intact muscles such as the scalenes that can expand the rib cage,^{28,29} (4) changes in intrathoracic blood volume that may affect lung gas volumes,^{9,10} or (5) inadequacies of techniques used to measure chest wall motion. Most previous studies of EA have not measured respiratory muscle activation, so that the factors responsible for the apparent preservation of chest wall activity during EA remain unclear.

The overall objective of this study was to determine the effects of lumbar EA on chest wall function in humans, combining measurements of respiratory muscle electromyograms (EMG) with imaging of the thorax using three-dimensional, fast computed tomography. The study addressed three hypotheses: (1) Recruitment of scalene muscle activity will maintain inspiratory rib cage expansion during high EA; (2) high EA will reduce end-expiratory thoracic volume by eliminating chest wall muscle tone and thus decreasing rib cage dimensions; and (3) high EA will also decrease intrathoracic blood volume, so that the volume of gas in the thorax at end expiration (*i.e.*, the FRC) will be unchanged.

Materials and Methods

This study was approved by our institution's review board. Six healthy persons who did not smoke were studied after they gave informed consent (table 1). Re-

sults of physical examination, chest roentgenogram, and pulmonary function tests were normal for each person. A pregnancy test was performed on the one female participant. Participants were brought to the laboratory the day before the actual experiment to become familiar with the experimental procedures. They were not allowed to consume oral fluids after midnight the day before the experiment.

Instrumentation

A lumbar epidural catheter was placed at the L3-L4 interspace, and a test dose of 3 ml 2% lidocaine with 1:200,000 epinephrine was administered. Each participant was placed in the supine position. Respiratory impedance plethysmography (RIP) belts (Respirace, Ardsley, NY) were placed around the upper rib cage and mid-abdomen. An intravenous catheter was inserted, and the radial artery was cannulated to obtain samples for blood gas analysis (IL 1302; Instrumentation Laboratories, Lexington, MA) and to monitor blood pressure. Ringer's lactate was infused at approximately 30 ml/hr *via* the intravenous catheter throughout the experiment.

Bipolar EMG electrodes were inserted into several respiratory muscles. The electrodes were fashioned by removing 1 mm of insulation from the end of 0.002-inch teflon-insulated wires. Two wires were passed through an insulated 30-gauge needle and then bent 1 mm from the end to form hooks. The electrodes were inserted under ultrasonic guidance³⁰ while electrical activity was monitored in the desired location, the needle was removed, leaving the wires in the desired muscle. Electrodes were placed in the transversus abdominis muscle in anterior axillary line approximately 4 cm inferior to the costal margin (at approximately the T10 dermatome), in the parasternal intercostal muscle (PS) at the third right interspace, approximately 3 cm lateral to the midline, and in the right anterior scalene muscle (SCA) near the base of the neck. An electrode was also placed in the diaphragms of three participants.³¹ To insert the diaphragm electrode, ultrasound was first used to define the diaphragm position at full inspiration. The needle was then inserted one interspace inferior to this point in the mid-axillary line and advanced until phasic inspiratory electrical activity was detected.³² This procedure minimized the possibility of causing pneumothorax as the electrode was placed through the intercostal muscles into the diaphragm close to its insertion on the rib cage. Electromyogram signals were amplified (Grass P511, Quincy, MA), band-

pass filtered between 30 and 3000 Hz, and recorded on digital audio tape (TEAC RT100, Montebello, CA) for later processing.

The RIP bands were calibrated using the method of Mankikian and colleagues.³³ Changes in dimensions of the rib cage and abdomen measured with the RIP bands were related to the volumes displaced by the rib cage and abdominal compartments during tidal breathing; these relationships were expressed as volume-motion coefficients of the rib cage and abdomen. These coefficients were calculated from data obtained by asking the participants to alternate predominately abdominal and thoracic breathing. Calibrations were performed at the beginning and end of each experimental condition (intact or EA), and average values were calculated for each condition. Inspiratory and expiratory gas flows were measured using a pneumotachograph (Fleisch 3, Richmond, VA) connected to a differential pressure transducer (Validyne MP-45, Northridge, CA). Gas flows were integrated to obtain changes in lung volume, which were corrected to BTPS conditions. Subjects breathed through a mouthpiece and nose clip during all measurements. Similar to our previous study,³¹ we found that this apparatus did not change the pattern of breathing (data not shown).

Heart muscle activity, blood pressure, and arterial oxygen saturation were monitored throughout the study.

Procedures

Participants were placed in the dynamic spatial reconstructor (DSR), a high-speed X-ray scanner that uses the computed tomography principle to provide three-dimensional images of the thorax. This technique has been described in detail elsewhere.^{31,34-37} The DSR has sufficient temporal resolution to visualize thoracic structures during quiet breathing and sufficient volume resolution to determine a known volume to within 2%.

The participants were scanned while breathing quietly through the mouthpiece and nose clip, with their arms brought above their heads with the humeri vertical to allow for thoracic imaging. Scans lasting 300 ms were triggered manually at end expiration and end inspiration. Scans at the same point in the breathing cycle were recorded during three consecutive breaths and gated together during later analysis to provide end-expiratory and end-inspiratory images. Because cephalocaudal height of the imaging field was not sufficient to include the entire thorax, these initial scans included only the superior half of the thorax. The participants

were then shifted cephalad, and a similar sequence of scans was obtained to visualize the inferior portion of the thorax.³⁶ Respiratory impedance plethysmography measurements, tidal volume, and EMGs were recorded simultaneously to ensure stability of the breathing pattern. During later analysis, the superior and inferior images were joined to produce end-inspiratory and end-expiratory images of the entire thorax.

Immediately after scanning, the FRC was measured in duplicate using a nitrogen-dilution technique.^{31,38,39} The participants each performed six vital capacity maneuvers into a 4-liter bag initially filled with 100% oxygen after the bag was connected to the mouthpiece at end expiration. Nitrogen concentrations in the bag were determined before and after this maneuver using a mass spectrometer (Perkin-Elmer MGA 1100, Buffalo Grove, IL).

After these measurements were taken during quiet breathing (referred to hereafter as "intact" breathing), local anesthetic solution was injected incrementally through the epidural catheter to produce sensory block to approximately the T6 dermatome. In participants 1 and 2, 2% lidocaine with 1:200,000 epinephrine was used; in the other four participants, 1.5% etidocaine with 1:200,000 epinephrine was used to maximize motor block. At this level of block, subsequently called "mid-thoracic EA," activity in the transversus abdominis muscle was eliminated (both during quiet breathing, if present, and during voluntary activation), but activity in the PS was intact. After a stable level was achieved, measurements were made during quiet breathing; DSR scans were not performed because the cumulative amount of radiation exposure had to be limited in these volunteers. More local anesthetic was injected through the catheter to produce motor block of the PS at the third interspace, a condition called "high EA." After a stable level was confirmed, DSR scans and FRC measurements were repeated.

Data Analysis

Details of image processing to define chest wall boundaries and to validate the DSR in measuring chest wall motion have been previously described.^{31,34-37} Briefly, each scan produced a three-dimensional volume image of the thorax composed of cubic volume elements (voxels) with edge lengths of 1.3 mm. Images were processed to define each voxel in the image as being in the thoracic cavity, the abdominal cavity, or the background. Thoracic volume (V_{th}) was determined by counting the number of voxels in the tho-

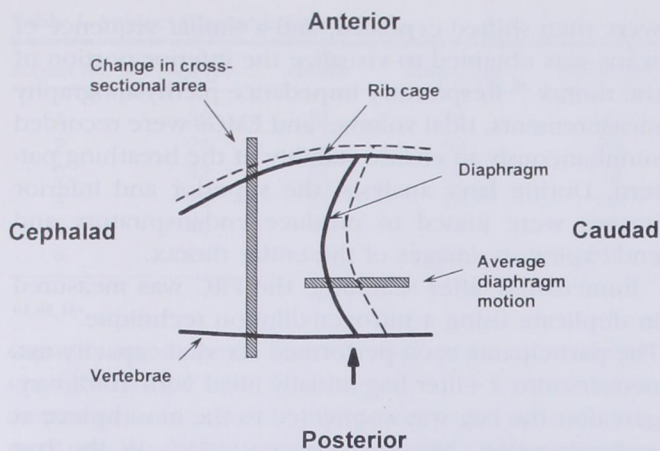


Fig. 1. Diagrammatic representation of scheme used to quantify changes in the pattern of chest wall motion depicted in figures 4 to 6. A mid-sagittal section of the thorax is shown, with end-expiratory (solid lines) and end-inspiratory (dashed lines) positions of the chest wall depicted. Stippled areas indicate edge views of planes used to quantify changes in transverse rib cage area and average diaphragm motion. The arrow indicates the cephalad extent of the area of apposition at end expiration.

racic cavity. Changes in thoracic liquid volume during inspiration (ΔV_{liq}), presumably representing changes in thoracic blood volume,³⁶ were calculated as the difference between changes in V_{th} from the beginning to the end of inspiration (ΔV_{th}) and tidal volume (V_T) measured by the integration of gas flow ($\Delta V_{liq} = \Delta V_{th} - V_T$). Changes in thoracic volumes between any two scans were partitioned into volumes swept by the diaphragmatic and rib cage surfaces.³⁴ Changes in the pattern of chest wall motion were quantified as previously described³⁴ (fig. 1).

Changes in the volume of the heart and major vessels were also estimated directly from these images.^{31,38} The heart and other mediastinal structures were isolated in each image using a combination of computer thresholding and operator interaction. The pulmonary vessels were truncated at approximately the same location in all scans from each participant. The total volume of these mediastinal structures (V_{ms}) was measured by counting the number of voxels within these structures and multiplying by the volume of one voxel. Because the images were obtained by gating several scans together at random times in the cardiac cycle, this volume represents an average during this cycle.

Electromyogram signals recorded on tape were processed with a third-order Paynter filter to provide a 100-msec moving time average (MTA).⁴⁰ To quantify

this activity, the MTA tracings were digitized and the mean MTA activity per breath was calculated as the area under the MTA signal divided by its duration.⁴¹ If EMG activity persisted into expiration, only the portion of the signal before the onset of expiratory flow was used to calculate this mean.⁴²

Paired statistical comparisons were made by paired *t* tests. Multiple comparisons were made using repeated measures analysis of variance, with Dunnett's test used for *post hoc* comparisons. $P < 0.05$ was taken as significant. Values are expressed as $M \pm SE$.

Results

Mid-thoracic EA required a total dose of 16 ± 2 ml of anesthetic and produced sensory levels ranging from T4 to T8 dermatomes (table 2). High EA required a total dose of 34 ± 2 ml anesthetic and produced sensory levels ranging from C6 to T2 in 5 participants. In participant 4, the sensory level did not exceed T4.

Compared with values before block, high EA did not significantly change arterial P_{O_2} (from 93 ± 3 to 97 ± 5 mmHg) or arterial P_{CO_2} (from 39 ± 1 to 36 ± 2 mmHg). High EA significantly decreased mean arterial pressure (from 101 ± 5 to 83 ± 6 mmHg) without changing the heart rate (from 66 ± 3 to 69 ± 4 min^{-1}). Participant 6 required a 500-ml bolus of intravenous fluid for transient hypotension when establishing high EA.

Electromyogram Activity

During intact breathing, consistent phasic EMG activation, predominantly during inspiration, was observed in the PS and the diaphragms in the three participants in whom this activity was measured (fig. 2, table 2). In these three persons, phasic expiratory activity was also detected by the diaphragm electrode (Fig. 2). Phasic inspiratory activity was present in the SCA of every participant except 6. In participant 5, tonic activity was present in the PS and SCA in addition to phasic activity. Tonic activity was noted in the transversus abdominis muscle in two participants.

Mid-thoracic EA abolished any activity in the transversus abdominis muscle and produced tonic activity in the SCA of participant 6. This level of EA did not significantly change phasic inspiratory EMG activity in the SCA or the PS (mean MTA of 1.57 ± 0.69 and 0.93 ± 0.18 , respectively, normalized to the activity present before block).

High EA abolished activity in the PS of every partici-

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Table 2. Dermatomal and Myotomal Levels of Block

Subject No.	Sensory				Motor		
	Sharp		Paresthesia		TA (T10)	PS (T3)	SCA (C4-C7)
	Left	Right	Left	Right			
Intact							
1	—	—	—	—	—	P	P
2	—	—	—	—	T	P	P
3	—	—	—	—	T	P	P
4	—	—	—	—	—	P	P
5	—	—	—	—	—	P,T	P,T
6	—	—	—	—	—	P	—
Midthoracic epidural anesthesia							
1	T ₄	T ₄	—	—	—	P	P
2	T ₄	T ₆	—	—	—	P	P
3	T ₄	T ₄	—	—	—	P	P
4	T ₈	T ₆	—	—	—	P	P
5	T ₆	T ₇	—	—	—	P,T	P,T
6	T ₅	T ₄	—	—	—	P	T
High epidural anesthesia							
1	T ₂	T ₁	—	C ₈	—	—	P
2	T ₂	T ₂	C ₈	C ₈	—	—	P,T
3	C ₈	C ₇	C ₈	C ₇	—	—	P
4	T ₄	T ₄	—	—	—	P	P,T
5	C ₆	C ₆	C ₆	C ₆	—	—	P,T
6	C ₇	C ₇	C ₇	C ₇	—	—	P,T

TA = transversus abdominis; PS = parasternal intercostal; SCA = scalene; P = phasic activity predominantly during inspiration; T = tonic activity; — = no activity or not present.

pant except in the person designated number 4. Additional tonic activity was noted in the SCA of some persons (fig. 3, table 2). High EA did not significantly change phasic inspiratory EMG activity in the SCA (mean MTA of 0.85 ± 0.20 , normalized to the MTA activity before block).

Inspiratory Chest Wall Motion

High EA did not significantly change tidal volume or breathing frequency (table 3). The change in intrathoracic volume during inspiration, as measured by the DSR, was significantly greater than the tidal volume measured by integrating gas flow, indicating that V_{liq} consistently increased during inspiration (table 3). This increase in V_{liq} comprised a substantial ($20 \pm 4\%$) fraction of ΔV_{th} during intact breathing and corresponded closely to the change in V_{mg} during inspiration, measured directly from DSR images of the mediastinal structures. These findings are consistent with our previous observations during quiet breathing.³¹ During high EA, V_{liq} and V_{mg} increased with inspiration in five of the six participants (table 3).

High EA significantly decreased inspiratory rib cage volume displacement, compared with intact breathing, but did not change inspiratory diaphragm displacement (table 3). Therefore, high EA decreased the relative contribution of rib cage expansion to inspiratory increases in thoracic volume (from 27 ± 2 to $10 \pm 11\%$ of ΔV_{th}).

The pattern of inspiratory rib cage expansion before block varied among participants (fig. 4). The effect of high EA also varied. Paradoxical rib cage motion (*i.e.*, a net inward inspiratory rib cage motion) was noted during inspiration in participant 5. In participants 2, 4, and 6, the expansion of the most cephalad portion of the rib cage was unchanged or augmented by high EA. Consistent with earlier reports, the greatest motion of the diaphragm in the cephalocaudal axis was always in the most dependent regions (fig. 5).

The effects of mid-thoracic EA were assessed by analyzing RIP measurements (table 4). This level of EA did not significantly affect compartmental volume displacements estimated by RIP. Consistent with the DSR measurements, high EA significantly reduced the inspiratory rib cage volume expansion estimated by RIP (table

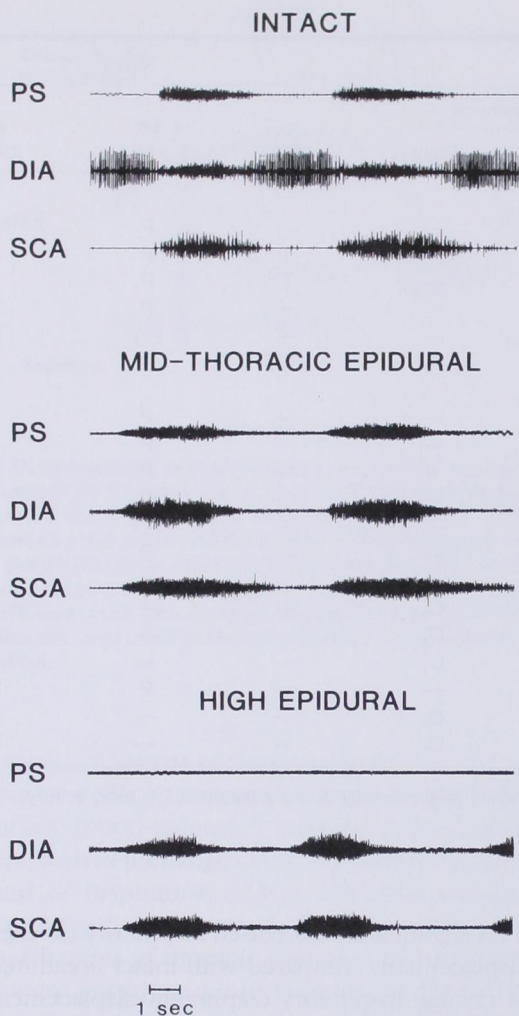


Fig. 2. Electromyograms of the parasternal intercostal muscle (PS) at the third right interspace, the diaphragm (DIA), and the anterior scalene muscle (SCA) during breathing while intact (before epidural anesthesia), during mid-thoracic epidural anesthesia, and during high epidural anesthesia in one participant. Activity occurs predominantly during inspiration in most muscles. Phasic expiratory activity is present in the diaphragm electrode during intact breathing, probably representing activity in the overlying internal intercostal muscle (see text for further discussion).

4). Neither level of EA changed any measure of ventilatory timing (table 4).

Functional Residual Capacity

High EA significantly increased the FRC, measured by nitrogen dilution (table 5). In five participants, the change in FRC was greater than the change in V_{th} , showing that V_{liq} at end expiration decreased in these

participants. Consistent with this observation, V_{mg} measured directly from the thoracic images also decreased in these five participants (table 5). These results suggest that in these participants, high EA decreased the intrathoracic blood volume at end expiration and thus contributed to the increase in FRC. In contrast, intrathoracic blood volume at end expiration increased in participant 6, who was unique in requiring an additional 500-ml fluid bolus during the establishment of high EA.

In four participants, an expansion of the rib cage contributed to an increase in end-expiratory V_{th} (table 5). There was also a net caudad motion of the end-expiratory position of the diaphragm in five of the six participants. In every participant, the anterior portion of the diaphragm moved caudad (fig. 6). In five participants, the posterior portion of the diaphragm also moved caudad. This posterior portion moved cephalad in one person; this was the same participant (#4) in whom a sensory block above T4 could not be achieved.

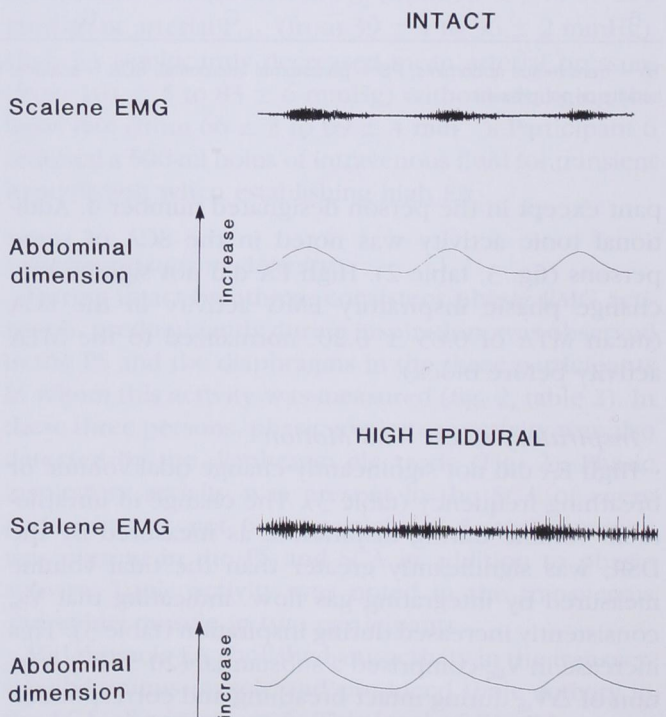


Fig. 3. Example of tonic electromyogram (EMG) activity developing in the scalene EMG during high epidural anesthesia in one participant. Abdominal dimensions measured by respiratory impedance plethysmography show the phases of breathing, with an increase in dimension during inspiration. Both phasic and tonic activities are present during high epidural anesthesia.

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Table 3. Volume Displacements

Subject No.	f(min ⁻¹)	V _T (ml)	ΔV _{th} (ml)	ΔV _{liq} (ml)	ΔV _{ms} (ml)	Volume Displaced by Rib Cage		Volume Displaced by Diaphragm		
						ml	%ΔV _{th}	ml	%ΔV _{th}	
Intact										
1	9.1	802	975	173	116	198	20	777	80	
2	11.1	509	622	113	121	174	28	448	72	
3	9.9	548	610	62	79	180	30	429	70	
4	12.8	522	707	185	179	224	32	483	68	
5	10.4	777	983	206	186	192	20	790	80	
6	11.1	629	774	145	60	236	30	538	70	
Mean	10.7	631	778	147	19	201	27	578	73	
SE	0.5	53	68	22	2	10	2	67	2	
High epidural anesthesia										
1	8.6	690	778	182	21	81	9	791	91	
2	14.5	425	541	116	22	62	11	480	89	
3	11.5	1,084	1,019	-65	-6	42	4	977	96	
4	11.5	667	681	14	2	164	24	517	76	
5	11.9	722	861	139	16	-55	-6	916	106	
6	9.8	899	962	63	7	180	19	782	81	
Mean	11.3	748	823	75	10	79*	10*	744	90*	
SE	0.8	91	73	37	5	11	11	204	11	

f = breathing frequency; V_T = tidal volume, measured by integrating gas flows; ΔV_{th} = change in thoracic volume, measured from DSR images; ΔV_{liq} = change in thoracic liquid volume during inspiration; ΔV_{ms} = change in volume of heart and mediastinal structures during inspiration.

* Significant difference from intact, *P* < 0.05, paired *t* test.

Discussion

Major findings of this study include the following: (1) Rib cage expansion contributes to tidal volume during high EA in most participants, even when most of the muscles of the rib cage are paralyzed; (2) the mean phasic electrical activity of intact respiratory muscles such as scalenes does not significantly increase in response to rib cage muscle paralysis produced by EA; and (3) high EA increases the FRC, an increase produced in most participants by a caudad motion of the diaphragm and a decrease in intrathoracic blood volume.

Chest Wall Function during Inspiration

Inspiratory rib cage expansion requires the coordinated activity of several respiratory muscles, including phasic inspiratory activation of PS and scalene muscles, and phasic expiratory activation of internal intercostal muscles in the lower lateral interspaces.^{1,2,32} We confirmed the presence of phasic activity in these muscles in most participants during quiet breathing. As in a previous study,³¹ we again noted phasic expiratory activity in the diaphragm electrode. The present study confirms our previous speculation that its source was

the overlying internal intercostal muscles, because paralysis of this myotome by EA consistently eliminated this activity.

Studies in animals and in quadriplegic humans have demonstrated that isolated diaphragm contraction constricts the upper rib cage and expands the lower rib cage.^{1,29,35,43} Constriction of the upper rib cage is caused by an inspiratory decrease in intrathoracic pressure that pulls this portion of the rib cage inward. Expansion of the lower rib cage results from the action of the diaphragm through its insertion on the rib cage, which elevates and expands the ribs (an "insertional" component), and inspiratory increases in abdominal pressure that act *via* the portion of the diaphragm that is apposed to the inner rib cage surface (an "appositional" component). Paralysis of rib cage muscles by regional anesthetic techniques might be expected to produce this pattern of chest wall motion.

Data regarding chest wall motion during epidural or spinal anesthesia from previous human studies are limited. Eisele and coworkers⁴⁴ mentioned passive retraction of the upper rib cage during continuous spinal anesthesia to the T1 level in three persons, but they did not quantify chest wall motion. Pascucci and associ-

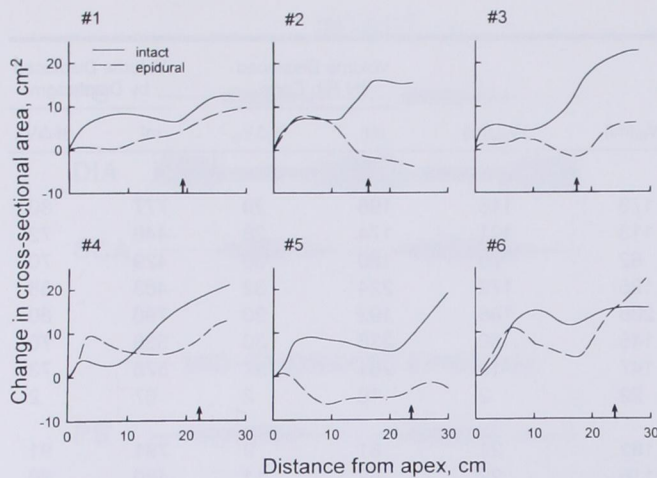


Fig. 4. Inspiratory change in the internal transverse cross-sectional area of the thorax as a function of distance from the lung apex in six participants during intact breathing and high epidural anesthesia. Arrows denote the cephalad extent of the area of apposition.

ates⁴⁵ noted paradoxical inward motion of the rib cage during inspiration in four of six infants receiving spinal anesthesia. The deformability of the infant rib cage makes it difficult to extrapolate these results to adults. Kochi and coworkers²² reported that thoracic EA with 2% lidocaine (with resulting sensory levels from approximately T1 to T6) caused a small but significant

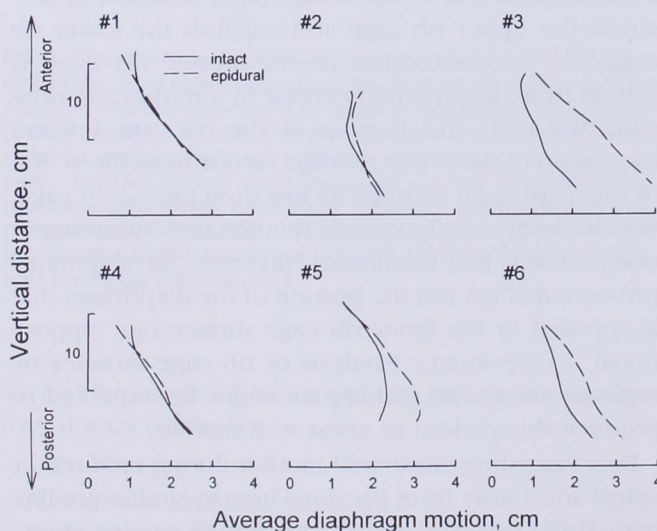


Fig. 5. Average inspiratory cephalocaudal motion of the diaphragm as a function of vertical distance in supine participants breathing while intact and during high epidural anesthesia.

Table 4. Effects of Midthoracic Epidural Anesthesia

	Intact	Midthoracic EA	High EA
Breathing frequency (min ⁻¹)	11.0 ± 1.0	10.4 ± 0.6	11.4 ± 1.4
Inspiratory time (s)	2.44 ± 0.33	2.23 ± 0.27	2.15 ± 0.34
Tidal volume (ml, pneumotachograph)	634 ± 77	714 ± 78	645 ± 98
Compartmental volume displacement by RIP			
Rib cage			
ml	187 ± 35	157 ± 42	103 ± 19*
%V _T	30 ± 3	22 ± 3	18 ± 4*
Abdomen			
ml	427 ± 46	517 ± 62	545 ± 96
%V _T	70 ± 3	78 ± 3	82 ± 4*

EA = epidural anesthesia; RIP = respiratory impedance plethysmography; V_T = tidal volume measured by RIP.

* Significant difference from intact, Dunnett's test.

decrease in the rib cage contribution to tidal volume during quiet breathing. Yamakage and associates¹⁷ found that spinal anesthesia to a T6 sensory level actually increased the rib cage contribution to tidal volume. This increase disappeared when the patients later fell asleep, suggesting that it represented a voluntary response to the sensations engendered by spinal anesthesia.

We found that although high EA diminished the contribution of the rib cage to tidal volume, it did not cause inspiratory constriction of the upper rib cage in most participants, suggesting that other muscles still active during EA must have expanded the rib cage. The scalene muscles, formerly thought to be "accessory" muscles of respiration, are in fact regularly active during quiet breathing in humans in supine and upright postures.^{28,46} There is evidence that SCA activity can significantly affect the motion of the upper rib cage, especially when the intercostal muscles are paralyzed.⁴⁷ Estenne and De Troyer²⁹ found that the upper rib cage moved inward during inspiration in quadriplegic participants when SCA activity was absent. In most participants with phasic inspiratory SCA activity, the upper rib cage expanded during inspiration. De Troyer and Estenne⁴⁶ found similar results in healthy participants performing voluntary respiratory maneuvers. These results suggest that the continued SCA activity observed during high block could account for rib cage expansion despite paralysis of most intercostal muscles. We previously found that high EA caused par-

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Table 5. Effects of High Epidural Anesthesia on End-expiratory Configuration of the Chest Wall

Subject No.	FRC			ΔV_{th}				
	Intact	Epidural	ΔFRC	Total ΔV_{th}	Volume Displaced by Rib Cage (ml)	Volume Displaced by Diaphragm (ml)	ΔV_{liq} (ml)	ΔV_{ms} (ml)
1	3,150	3,270	120	-12	-168	156	-132	-153
2	1,360	2,760	400	180	30	151	-220	-56
3	2,880	3,380	500	474	200	274	-26	-36
4	2,600	2,980	380	199	235	-36	-181	-83
5	3,580	3,520	-60	-103	-278	175	-43	-50
6	2,450	2,880	430	531	466	65	101	95
Mean	2,670	2,965	295*	212	81	131*	-83	-47
SE	309	260	89	104	113	43	48	33

FRC = functional residual capacity, measured by nitrogen dilution; ΔV_{th} = change in end-expiratory thoracic volume measured from thoracic images; ΔV_{liq} = change in end-expiratory thoracic volume; ΔV_{ms} = change in end-expiratory volume of the heart and mediastinal structures. All changes are calculated as the difference between high EA and intact values at end-expiration; thus, positive values indicate that high EA produced an increase in volume.

adoxical inspiratory rib cage motion in pentobarbital-anesthetized dogs.³⁵ In these dogs, SCA activity was never present in implanted EMG electrodes. Although there are many species differences between the chest wall of dogs and humans, this may represent further evidence for the importance of SCA in preventing paradoxical rib cage motion during high EA in humans.

It is also possible that intercostal muscles cephalad to the T3 myotome (at which parasternal intercostal activity was monitored) were active and contributed to rib cage expansion during high block. We could not eliminate PS activity at this level in participant 4; continued rib cage expansion can be attributed to this activity in this person. However, the complete block

at the T3 level achieved in the other participants makes it likely that muscles at more cephalad interspaces were at least partially paralyzed. We used etidocaine in the last four participants we studied to maximize intercostal muscle paralysis, yet we noted paradoxical rib cage motion in participant 5 only.

We are uncertain why this participant could not maintain inspiratory rib cage expansion. Estenne and De Troyer²⁹ also found that some quadriplegic patients exhibited paradoxical rib cage motion despite phasic inspiratory SCA activity. This participant also experienced the highest sensory level of block and was unique in demonstrating tonic activity in both the PS and SCA before block.

Acute paralysis of selected respiratory muscles can increase activity in other respiratory muscles.^{48,49} Possible factors responsible for such recruitment include reflexes, with afferents arising from receptors in the lung or the chest wall, or increases in P_{aCO_2} , if ventilation is sufficiently depressed by paralysis. Brichant and colleagues⁴⁸ found that high EA in pentobarbital-anesthetized dogs increased the neural activation of the diaphragm, probably in response to increases in P_{aCO_2} . We had hypothesized that SCA EMG activity might increase to compensate for paralysis of the PS during EA. That we could not show a change in this activity during high EA suggests that perturbations of chest wall motion, lung volume, or P_{aCO_2} were insufficient to trigger compensatory responses, that blockade of chest wall afferents interfered with any reflex responses, or that other factors such as effects of absorbed local anesthetic on respiratory control affected

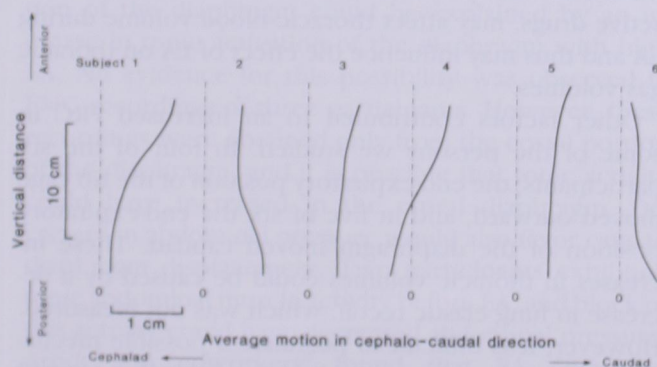


Fig. 6. Change in the average end-expiratory position of the diaphragm with the onset of high epidural anesthesia as a function of vertical distance in supine participants. A value of "0" would indicate that the position of the diaphragm did not change. The anterior portion of the diaphragm moved caudad in each participant, and the posterior portion also moved caudad in five of the participants.

any response. Similarly, mid-thoracic EA elicited no compensatory increase in PS or SCA activation; neither did it significantly affect the pattern of chest wall motion. Measurements of ventilatory timing also provided no evidence for a reflex response to EA, because they were not affected. The development of hypercarbia during high EA in pentobarbital-anesthetized dogs studied by Brichant and colleagues may also suggest a greater dependence of the dog on the actions of extradiaphragmatic respiratory muscles during quiet breathing compared with humans.^{34,35} Alternatively, pentobarbital anesthesia may have interfered with compensatory responses in the dogs.

Although we did not measure gas exchange in detail, arterial blood gases were consistent with previous reports that epidural or spinal anesthesia has little effect on Pa_{O_2} .^{13-17,30} Apparently the alterations in chest wall motion are insufficient to significantly affect regional ventilation and gas exchange, or sufficient compensatory mechanisms such as hypoxic pulmonary vasoconstriction prevent significant gas exchange abnormalities. Participant 5, who demonstrated the greatest change in rib cage function, showing paradoxical inspiratory rib cage motion, actually experienced an increase in Pa_{O_2} (from 94 to 108 mmHg), accompanied by a decrease in Pa_{CO_2} (from 37 to 31 mmHg). This trend toward a decrease in Pa_{CO_2} , although not statistically significant, is consistent with previous reports and may be related to effects of absorbed local anesthetic on ventilatory control.^{11,12} Maintenance of gas exchange may be related to the increase in FRC produced by EA, as discussed in the following section.

Functional Residual Capacity

Previous studies found that thoracic or lumbar EA has no effect¹⁴⁻¹⁶ or decreases¹³ the FRC. Paralysis of rib cage muscles by intercostal nerve blocks decreases the FRC.^{51,52}

In contrast to these reports, we found that high EA significantly increased the FRC (by approximately 15%). The mechanism responsible for this increase varied among participants. In five of the six, V_{liq} decreased, presumably caused by a shift of blood out of the thorax. This decrease, calculated on the basis of differences between changes in gas volume (measured by nitrogen dilution) and total thoracic volume (measured by the DSR), was confirmed by direct measurements of the volume of images of the heart and other mediastinal structures (V_{mg}). In these five participants, this change represented a $6.4 \pm 2.1\%$ decrease in the volume of

tissue in the thorax. Arndt and coworkers⁹ found that EA to a T4 sensory level decreased thoracic blood volume by $8.1 \pm 1.2\%$ as measured by radiolabeled erythrocytes, with this blood redistributed to the capacitance vessels of the legs in most persons studied.^{9,10} Although direct comparison of our values with those of Arndt and coworkers requires knowledge of the proportion of thoracic tissue composed of blood, they appear to be consistent. These shifts in blood volume presumably reflect the cardiovascular effects of the sympathectomy produced by EA. As found in previous work, most of these healthy volunteers could maintain an adequate heart rate and mean arterial pressure without requiring substantial amounts of intravenous fluids.⁸

In contrast, high EA increased end-expiratory thoracic liquid volume in participant 6. This person was unique in requiring additional intravenous fluid for transient hypotension during local anesthetic injection. We speculate that this increase in V_{liq} in participant 6 reflected the administration of this additional intravenous fluid. Consistent with this concept, Stanton-Hicks and associates¹⁰ found that decreases in thoracic blood volume produced by EA could be reversed by increasing cardiac filling by peripheral venoconstriction. In addition, we found that high EA increased end-expiratory thoracic liquid volume in a previous study of pentobarbital-anesthetized animals.³⁵ Although many other factors may have been responsible, these animals received a similar amount of intravenous fluid per hour as did participant 6 when scaled for weight (approximately $2 \text{ ml} \cdot \text{kg} \cdot \text{hr}^{-1}$). Measures directed toward the cardiovascular system, such as volume infusion or vasoactive drugs, may affect thoracic blood volume during EA and thus may influence the effect of EA on thoracic gas volumes.

Other factors contributed to an increased FRC in some of the persons we studied. In four of the six participants, the end-expiratory position of the rib cage moved outward, and in five of six the end-expiratory position of the diaphragm moved caudad. These increases in thoracic volumes could be caused by a decrease in lung elastic recoil, which was not measured. However, it is difficult to postulate a possible mechanism for such a decrease, directing attention toward direct effects of EA on the chest wall.

The rib cage could be expanded by the tonic activity in the SCA observed in some participants during high EA; however, there was no direct correspondence between the presence of such activity and rib cage expan-

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sion (see tables 2 and 5). Other muscles inserting on the rib cage, such as the sternocleidomastoids, could have also contributed. In a previous study, we found that halothane anesthesia changed the curvature of the thoracic spine, which may have affected rib cage dimensions³¹; however, we saw no such changes in the present study (data not shown). Here, as in previous studies,^{31,32} we found evidence for phasic expiratory activity in the lateral intercostal muscles; expiratory activity may also be present in the transversus thoracis muscle in supine persons when breathing quietly.⁵³ Because this activity constricts the rib cage during expiration,⁵⁴ its abolition should increase end-expiratory rib cage dimensions. This mechanism is partly responsible for the increase in FRC noted with high EA in dogs.³⁸

If tonic activity is normally present in muscles that expand the rib cage,⁴⁻⁶ such as the PS muscles, then paralysis of these muscles should decrease rib cage dimensions. A similar mechanism has been advanced to explain decreases in rib cage dimensions produced by general anesthesia.⁴ However, many studies have failed to find evidence for significant tonic activity in rib cage muscles during quiet breathing in humans lying supine.^{2,31,55,56} We also could find no evidence of tonic activity in the PS or SCA during intact breathing, with the exception of participant 5. The decrease in rib cage dimensions produced by high EA in this person could be explained by the abolition of tonic PS activity. However, the increase in rib cage dimensions produced in our study by high EA in four other participants is not consistent with the presence of such tonic activity.

The caudad displacement of the end-expiratory position of the diaphragm could be explained by an increase in tonic activation of the diaphragm with high EA. No evidence for this possibility was observed in EMG recordings of three participants. However, these recordings were obtained only from the costal portion of the diaphragm, and it is possible that tonic activity could have increased in the crural diaphragm. Decreases in abdominal pressure would also favor caudad diaphragm displacement. Two participants exhibited tonic abdominal muscle activity before EA, and block of this activity could have decreased abdominal pressure. Arndt and coworkers⁹ found that EA decreased splanchnic blood volume in most participants, a factor that would also decrease abdominal pressure.

Clinical Application

These measurements of chest wall function were obtained using dermatomal levels of block that are at the

limit of those desired in clinical practice, using agents that provide substantial motor blockade. Thus they can be viewed as representing the maximal effect that might be expected during the conduct of lumbar EA in the clinical setting. Despite considerable block of the extradiaphragmatic muscles under these maximal conditions, most of these healthy persons maintained some degree of rib cage expansion when breathing quietly, with no significant effects on gas exchange as measured by arterial blood gases. This maintenance of gas exchange may have been aided by a concomitant increase in FRC. Unlike common clinical practice, most of these participants did not receive intravenous fluid boluses during the establishment of block. Data from the one participant who did receive such a bolus suggest that this practice may significantly affect intrathoracic blood volume, and hence the FRC.

Individual patients undergoing surgery may respond in different ways, as shown by the variability within our group of participants. One healthy participant in our study could not maintain inspiratory rib cage expansion, for reasons that are unknown. Patients with abnormal chest wall mechanics, such as the obese or those with chronic obstructive pulmonary disease, may rely more on rib cage expansion to generate tidal volume compared with normal participants,⁵⁷ and they may be more susceptible to rib cage muscle paralysis produced by EA. Additional effects of surgical stimulation must also be considered. For example, intraabdominal manipulation produces diaphragm dysfunction, probably due to reflex inhibition of the diaphragm.^{58,59} During EA, the ability of the rib cage muscles to compensate for this inhibition may be impaired.

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