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Effects of Thoracic Extradural Block on Diaphragmatic Electrical Activity and Contractility after Upper Abdominal Surgery

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Background: **Upper abdominal surgery (UAS) induces diaphragmatic dysfunction. Thoracic extradural block (TEB) using 0.5% bupivacaine improves some pressure and motion indices of diaphragmatic function. However, no direct information on diaphragmatic activity is available after UAS. The aim of this study was to assess diaphragmatic electrical activity (Edi) after UAS before and after TEB.**

Methods: **A postoperative electromyogram was obtained, using intramuscular electrodes inserted by the surgeon in the costal and crural parts of the diaphragm, in 14 patients undergoing abdominal aortic surgery. Tidal changes in abdominal (V_{AB}) and rib-cage (V_{RC}) volumes, and gastric ($\Delta Pgas$), esophageal (ΔPes), and transdiaphragmatic (ΔPdi) pressures were used to measure tidal volume (V_T) and respiratory rate and to provide indirect indices of diaphragmatic activity from the two ratios V_{AB}/V_T and $\Delta Pgas/\Delta Pdi$. These respiratory variables were obtained preoperatively. Postoperatively, measurements including Edi were obtained before and after a segmental epidural block, reaching a T4 level was achieved with 0.5% plain bupivacaine.**

Results: **Upper abdominal surgery induced an increase in respiratory rate (+28 ± 15%; $P < .01$), associated with a decrease in V_{AB}/V_T (from 0.75 ± 0.11 to 0.07 ± 0.08; $P < .01$), $\Delta Pgas/\Delta Pdi$ (from 0.3 ± 0.08 to 0.01 ± 0.19; $P < .05$), and V_T (−30 ± 14%; $P < .01$). After surgery, all patients exhibited electrical diaphragmatic activity that increased with TEB by 48 ± 28% ($P < .01$) and 60 ± 22% ($P < .001$) for the cural and costal segments, respectively. The ratio $\Delta Pdi/Edi$, used to evaluate diaphragmatic contractility, was not modified by TEB. Tidal volume, respiratory rate, and $\Delta Pgas/\Delta Pdi$ returned to preoperative levels, whereas V_{AB}/V_T increased but remained different from preoperative values.**

Conclusions: **This study demonstrates that TEB produces an increase in diaphragmatic activity, identical for the two segments of the muscle. Interruption of afferents that produce an inhibitory effect on diaphragmatic activity appears the most attractive hypothesis to explain the consequences of TEB after UAS. (Key words: Anesthetics, local: bupivacaine. Lungs: postoperative pulmonary function. Measurement technique, diaphragm: electromyogram. Muscle: diaphragm. Surgery: abdominal.)**

PATIENTS undergoing upper abdominal surgery (UAS) generally develop a severe restrictive pattern in the postoperative period.[1] The decrease in the diaphragm's contribution to tidal volume during both quiet breathing and forced maneuvers[2,3] appears a major determinant in this restrictive pattern. Since diaphragmatic contractility is not impaired after UAS,[4] reflex inhibition of the phrenic nerve output appears as the most attractive hypothesis to explain this reduction in diaphragmatic activity.[5]

The diaphragmatic contribution to ventilation is currently assessed from changes in transdiaphragmatic pressure (ΔPdi), the ratio between changes in gastric and transdiaphragmatic pressure ($\Delta Pgas/\Delta Pdi$),[6] and the relative changes in dimensions of the abdomen (X_{AB}) and of the rib cage (X_{RC}), *i.e.*, $X_{AB}/(X_{AB} + X_{RC})$.[7] However, these variables also depend on abdominal muscle activity.[8] Therefore, their ability to reflect diaphragmatic activity may be questioned, especially in the postoperative period, since abdominal surgery has been demonstrated to produce an increase in the activity of abdominal muscles.[9] It has been demonstrated that a thoracic extradural block (TEB) using 0.5% bupivacaine increases pressure and motion indices of diaphragmatic function after UAS, suggesting a partial reversal of the diaphragmatic dysfunction.[10] But variations in pressure and motion indices induced by TEB could reflect changes in abdominal muscle activity rather than changes in diaphragmatic activity. To resolve this controversy, direct measurement of diaphragmatic activity is required.

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This study was designed to assess the effects of TEB on diaphragmatic electrical activity after UAS. Because the ability to accurately quantify diaphragmatic electrical activity using either percutaneous or esophageal electrodes has been questioned,[11,12] the electromyogram (EMG) was obtained using intramuscular electrodes, inserted at the end of the surgical procedure into the costal and crural parts of the diaphragm.

Methods

Fourteen patients (11 men, 3 women) undergoing elective abdominal aortic surgery through a xyphopubic incision were studied. All subjects included in the study were free of cardiac disease, and their preoperative pulmonary function tests were within the normal range. Their mean age was 55 ± 10 yr (mean ± SD), mean height 171 ± 10 cm, and mean weight 67 ± 12 kg. All subjects gave their informed consent, and the study was approved by the Ethical Committee of the authors' institution.

Prior to general anesthesia, an epidural catheter was inserted at a T8–9 level, and correct positioning was confirmed by an injection of 4 ml 2% lidocaine with 1:200,000 epinephrine. Flunitrazepam, fentanyl, and pancuronium were used for anesthesia induction. Anesthesia maintenance was obtained with N_2O, isoflurane, fentanyl, and pancuronium, as required. The patients' lungs were mechanically ventilated during and after surgery, until the patients were completely rewarmed and awake. Between tracheal extubation and the time this study was started, postoperative analgesia was provided using intravenous paracetamol, as required.

All respiratory measurements were carried out with the subjects lying supine.

Thoracic and Abdominal Dimensions

Tidal changes in rib cage and abdominal circumferences were simultaneously measured using two differential linear transformers (DLTs, Shaewitz Orgeval, France)[13] connected to belts positioned at the nipple level and 2 cm above the umbilicus. Technical description and physical properties of DLT and belts have been previously published. Briefly, it consists of a ferromagnetic core that moves freely inside a hollow cylindrical support containing coils. The electrical output signal induced by the displacement of the core inside the transformer during respiratory movements is linearly related to chest wall circumference changes. Assuming that the volume measured at the mouth by spirometry is equal to the sum of changes in rib cage and abdominal volumes,[8] DLT signals could be calibrated in terms of volume, by measuring the spirometer volume and the rib cage and abdominal signals simultaneously while breathing in such a way as to vary the contribution of the two compartments as much as possible. This procedure enabled volume motion coefficients to be calculated for rib cage and abdomen. Details concerning the calibration procedure, volume motion coefficients calculation, and the precision of this method have been published previously.[14] Upper abdominal surgery and/or TEB may change chest wall geometry and consequently the volume-motion coefficients. Therefore the calibration of DLT in terms of volume was repeated before each set of measurements. After calibration, DLTs were used to determine abdominal and rib cage tidal volume changes (V_{AB}, V_{RC}), tidal volume ($V_T = V_{AB} + V_{RC}$) and abdominal contribution to tidal volume (V_{AB}/V_T), which is assumed to represent the diaphragm contribution to the breathing process. These variables were calculated as the means of all cycles during a 2-min period of quiet breathing.

Gastric and Esophageal Pressures

Gastric and esophageal pressures (Pgas, Pes) were measured using two thin-walled 10-cm long balloons, each connected to a differential pressure transducer (Validyne DP 15, Northridge, CA). The esophageal balloon, filled with 0.5 ml of air, was positioned in the mid esophagus and used as a pleural pressure index, after correct positioning was ascertained with the occlusion method.[15] The gastric balloon was filled with 1 ml of air. Its tip was positioned 65 cm from the nares. Gastric pressure was used as an index of abdominal pressure according to the method of Agostini and Rahn.[16] Changes in Pgas (ΔPgas) and Pes (ΔPes) were calculated as the difference between peak inspiratory minus peak expiratory pressure. Transdiaphragmatic pressure variation for each breath (ΔPdi) was obtained by electronically subtracting Pes from Pgas simultaneously obtained. The ratio ΔPgas/ΔPdi was taken as an index of the relative contribution of the diaphragm to the breathing process.

The dynamic abdominal compliance was calculated cycle by cycle as the ratio V_{AB}/ΔPgas.

Respiratory Timing

The inspiratory (Ti), expiratory (Te), and total times (Ttot) were measured at each period of the study. Since flow was not recorded, the beginning and end of each

inspiration were determined by both pressure and motion signals. When a phase lag was noted between these variables, respiratory cycles were not used for analysis. The ratios V_T/Ti and Ti/Ttot were calculated cycle by cycle.

Electromyography

In the postoperative period, electrical activity of the diaphragm (Edi) was recorded, using two pairs of electrodes inserted by the surgeon before abdominal wall closure, respectively in the costal and crural parts of the right hemidiaphragm.

The electrodes were steel wires, similar to those used for epicardial pacing. They were insulated except for their last 3 cm, which was inserted parallel to the muscle fibers of each segment, 1.5 cm apart. They were implanted consistently in approximately the same region of the respective segment in each patient. The crural was located in the posterior paravertebral region of the segment and the costal was placed close to the central tendon. The electrodes were exteriorized through the abdominal wall. Crural and costal Edi signals were amplified (band width 100–1,000 Hz), using a DISA 15 G 01 electromyograph (Dantec, Denmark). The EMG signals were integrated with a time constant of 100 ms (DISA integrator), and recorded on a Gould ES 1000 strip chart recorder (Cleveland, OH) simultaneously with circumference and pressure signals. Phasic electrical activity of crural (Edi cru) and costal (Edi cost) diaphragmatic segments was assessed by measuring the amplitude of the inspiratory peak recorded from the integrated signal. The results of each set of measurements were the means of all cycles during a 2-min period of quiet breathing. The ratio ΔPdi/Edi calculated from simultaneously recorded pressures and electromyographic signals was used to evaluate diaphragmatic contractility.[17,18] This index was expressed in arbitrary units.

The electrodes were easily withdrawn at the end of the study, and no complication was observed.

Lung Function Tests

Functional residual capacity (FRC) was measured at each time of the study by the helium dilution method (Pulmonet III, Gould).

Experimental Procedure

Respiratory variables including FRC, pressure, and motion measurements were obtained the day prior to surgery. After surgery, the same variables were obtained. In addition, the second set of measurements included EMG recording. The mean delay between the end of surgery and the beginning of measurements was 19 h. Then, 0.5% plain bupivacaine was injected through the epidural catheter to achieve a sensory block reaching a T4 level. The mean dose of bupivacaine was 50 ± 5 mg. The last set of measurements was performed 1 h later.

Arterial and capillary wedge pressures were monitored *via* radial and pulmonary artery catheters inserted preoperatively for the standard anesthetic and postoperative management. To avoid a decrease in mean arterial pressure greater than 20% of control values, modified gelatin solutions were infused if necessary. No adrenergic agent was used.

Statistical Analysis

Data are expressed as mean ± SD. Statistical analysis was achieved using a two-way analysis of variance for changes in pressure, circumference, and FRC. A paired Student's *t* test was used for the analysis of EMG signals and diaphragmatic efficiency. Differences were considered significant when $P < .05$.

Results

Sequential changes in respiratory variables are represented in table 1. After UAS, FRC was reduced by 23 ± 8% ($P < .01$) when compared to preoperative values. $\dot{V}E$ remained unchanged, while marked changes in the breathing pattern occurred, characterized by a 30 ± 14% ($P < .01$) decrease in tidal volume and a 28 ± 15% ($P < .01$) increase in respiratory rate. Ti decreased by 26 ± 20% ($P < .05$) while Ti/Ttot and V_T/Ti remained unchanged.

Four patients presented an expiratory increase in Pgas related to a phasic activity of abdominal muscles during expiration. To overcome the influence of abdominal muscle activity on inspiratory gastric pressure measurement ΔPgas was measured in these patients as the difference between inspiratory peak and the value obtained just after abdominal muscle relaxation (fig. 1). Upper abdominal surgery was associated with a decrease in ΔPdi (22 ± 16%; $P < .01$). ΔPgas/ΔPdi, and V_{AB}/V_T decreased respectively from 0.3 ± 0.08 to 0.01 ± 0.19 ($P < .05$) and from 0.75 ± 0.11 to 0.07 ± 0.08 ($P < .01$). Postoperatively, phasic electrical activity was still observed in both the costal and crural parts of the diaphragm (fig. 2). No asynchrony was observed between the two diaphragmatic segments, since the onset of activity and the peak of electrical activation

Table 1. Sequential Changes in Respiratory Variables

		Postoperative	
	Preoperative	Pre-block	Post-block
V_T (ml)	500 ± 100	351 ± 72*	457 ± 97†
RR (c/min)	13.9 ± 2.2	17.8 ± 2.4*	14.8 ± 3.4†
$\dot{V}E$ (L/min)	6.8 ± 0.4	6.5 ± 0.5	6.7 ± 0.5
V_{AB} (ml)	370 ± 70	33 ± 32*	180 ± 69†‡
V_{RC} (ml)	133 ± 73	315 ± 65*	280 ± 49*
V_{AB}/V_T	0.75 ± 0.11	0.07 ± 0.08*	0.41 ± 0.09*†
ΔPgas (cmH_2O)	2.5 ± 0.7	0.1 ± 1.3*	2.3 ± 0.8†
ΔPes (cmH_2O)	6.3 ± 2.5	6.8 ± 2.8	7.0 ± 3.0
ΔPdi (cmH_2O)	8.82 ± 1.85	6.92 ± 2.72*	9.33 ± 2.89†
ΔPgas/ΔPdi	0.30 ± 0.08	0.01 ± 0.19‡	0.27 ± 0.09†
FRC (l)	2.6 ± 0.5	2.0 ± 0.5*	2.1 ± 0.5*
Ti (s)	1.47 ± 0.34	1.08 ± 0.28‡	1.43 ± 0.29§
Ti/Ttot	0.34 ± 0.04	0.33 ± 0.04	0.35 ± 0.02
V_T/Ti (l/s)	0.34 ± 0.09	0.33 ± 0.09	0.32 ± 0.09
V_{AB}/ΔPgas (ml/cmH_2O)	146 ± 48	57 ± 36*	89 ± 66‡§
End-exp Pgas (cmH_2O)	4.1 ± 1.2	4.8 ± 2.3	4.6 ± 1.8

Data are expressed as mean ± SD.

V_T = tidal volume; RR = respiratory rate; $\dot{V}E$ = minute ventilation; V_{AB} = inspiratory changes in abdominal volume; V_{RC} = inspiratory changes in rib cage volume; V_{AB}/V_T = abdominal contribution to tidal volume; ΔPgas = inspiratory changes in gastric pressure; ΔPes = inspiratory changes in esophageal pressure; ΔPdi = inspiratory changes in transdiaphragmatic pressure; FRC = functional residual capacity; Ti = inspiratory time; Ttot = total respiratory time; V_{AB}/ΔPgas = dynamic abdominal compliance; End-exp Pgas = end-expiratory gastric pressure.

* $P < .01$ *versus* preoperative.

† $P < .01$ *versus* pre-block.

‡ $P < .05$ *versus* preoperative.

§ $P < .05$ *versus* preblock.

appeared simultaneously for costal and crural diaphragm. Three subjects demonstrated paradoxical abdominal breathing, characterized by a negative inspiratory change in both gastric pressure and abdominal circumference. In these subjects, the two parts of the diaphragm still exhibited inspiratory phasic activity. Abdominal dynamic compliance was not calculated in these three patients. For the other 11 subjects, the compliance decreased from 146.8 ± 48 to 57.5 ± 36 ml/cmH_2O ($P < .01$).

One hour after TEB there was a 17 ± 11% ($P < .01$) decrease in respiratory rate associated with a 34 ± 18% ($P < .01$) increase in tidal volume. $\dot{V}I$ and FRC remained unchanged. Ti increased (32 ± 18%; $P < .05$), while Ti/Ttot and V_T/Ti were not altered.

Thoracic extradural block induced an increase in ΔPdi (35 ± 20%; $P < .01$). ΔPgas/ΔPdi increased (from 0.01 ± 0.19 to 0.27 ± 0.09; $P < .01$) and returned to preoperative values, whereas V_{AB}/V_T increased (from 0.07 ± 0.08 to 0.41 ± 0.09; $P < .01$) and remained different from baseline values. Abdominal compliance increased by 56 ± 32% ($P < .01$).

Both Edi cru and Edi cost increased after TEB (table 2) by 48 ± 28% ($P < .01$) and 60 ± 22% ($P < .001$), respectively. The increase in electrical activity was greater for the costal segment, but the difference between the two parts of the muscle was not significant. An increase in Edi was observed for all of the 14 patients (fig. 3). The patients who presented a postoperative paradoxical pattern of breathing recovered a positive inspiratory change in both gastric pressure and abdominal circumference after TEB that was coincident with the observed increase in Edi.

The ratio ΔPdi/Edi assessed for the two segments of the muscle (ΔPdi/Edi cru and ΔPdi/Edi cost) remained unchanged after TEB (table 2).

Discussion

The current study demonstrates that, after UAS, TEB using 0.5% bupivacaine increases diaphragmatic activity. Both pressure and motion indices of diaphragmatic contribution to ventilation, markedly reduced by surgery, and phasic electrical activity of costal and crural

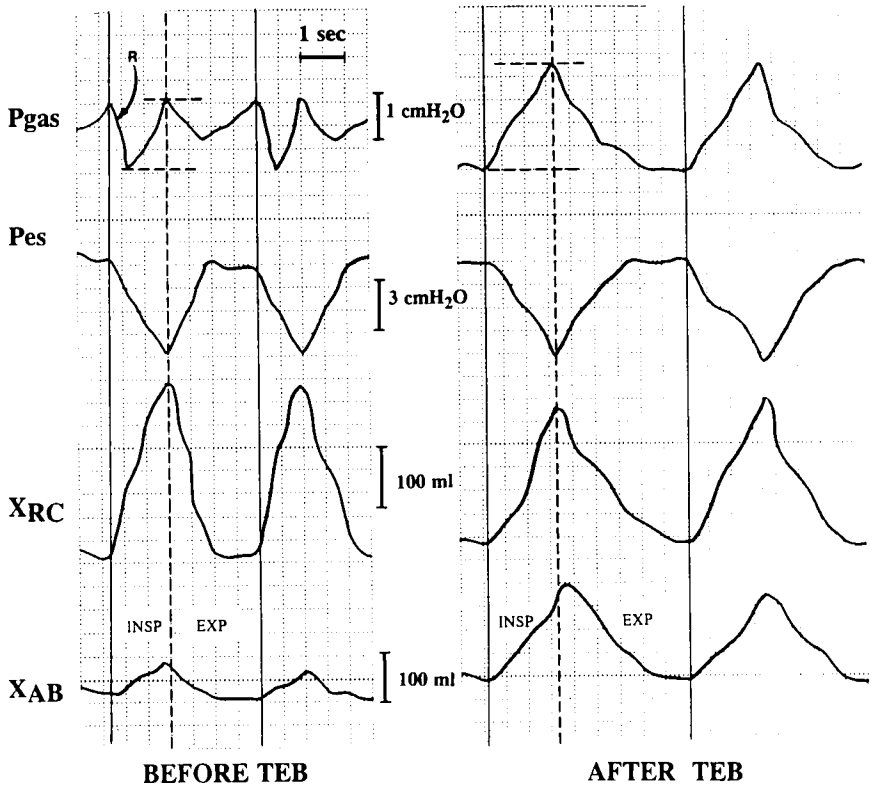

Fig. 1. Simultaneous changes in chest wall circumferences (XAB, XRC), gastric (Pgas), and esophageal (Pes) pressure in one patient who exhibited phasic expiratory activity of abdominal muscles. At the beginning of inspiration, the initial decrease in Pgas is related to abdominal muscles relaxation (R). ΔPgas was measured as the difference between inspiratory peak and the value obtained just after abdominal muscle relaxation. This pattern of breathing disappeared after thoracic extradural block.

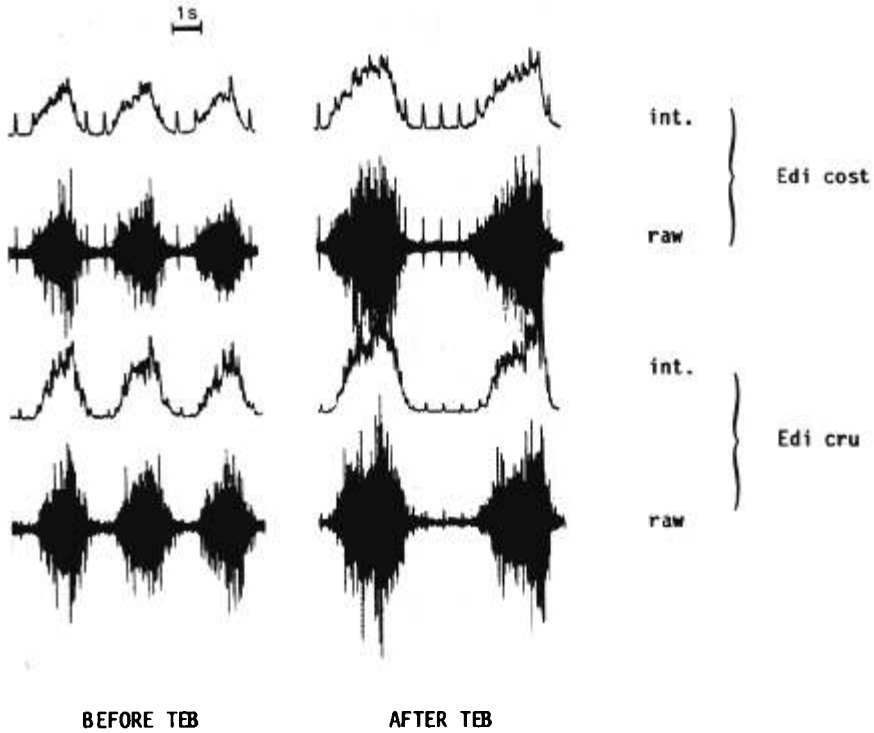

Fig. 2. Effects of thoracic extradural block (TEB) on raw (raw) and integrated (int) electromyogram of costal (Edi cost) and crural (Edi cru) parts of the diaphragm after upper abdominal surgery, before and after TEB. The increase in diaphragmatic electromyogram is associated with a decrease in respiratory rate.

Table 2. Effects of TEB on Diaphragmatic Electrical Activity and Contractility

	Pre Block	Post Block
Edi cost	12.86 ± 2.95	20.03 ± 5.21*
Edi cru	13.02 ± 4.80	19.02 ± 6.70†
ΔPdi/Edi cost	0.54 ± 0.27	0.52 ± 0.25‡
ΔPdi/Edi cru	0.60 ± 0.28	0.57 ± 0.24‡

Edi cost = integrated electromyogram of costal diaphragm (arb. units); Edi cru = integrated electromyogram of crural diaphragm (arb. units); ΔPdi = inspiratory changes in transdiaphragmatic pressure (cmH_2O).

* $P < .001$ *versus* pre-block.

† $P < .01$ *versus* pre-block.

‡ Not significant.

parts of the diaphragm increase after TEB following UAS.

Intramuscular electrodes should give short-term reliable measurements of diaphragm electrical activity. The wires were placed close enough to the central tendon that they could not have been subject to changing conductivity of their surroundings due to movement of the lungs nor to contamination by EMGs from other muscles sometimes observed when esophageal or percutaneous electrodes are used.[11] While EMG amplitude can be affected slightly by changes in length or velocity of the muscle, the differences observed are only in the range of 5–10%,[11] not enough to substantially alter the conclusions. Moreover, important variations in diaphragm length were unlikely since FRC did not change after TEB was performed. Although the trauma to the diaphragm was trivial, other experiments[19] have shown that fine-wire EMG electrodes may precipitate a significant local edema that would be expected to decrease the tissue impedance between the electrode poles, changing the potential difference sensed in the volume conductor, and artifactually increasing the recorded signal. However, the short delay between pre- and post-TEB recordings makes the change in degree of peri-wire edema an unlikely hypothesis to explain the differences induced by block.

Costal and crural parts of the diaphragm sometimes are presented as two different muscles as to their embryologic origin, pattern of neural and muscular activity, or functional characteristics.[20–23] However, some authors do not mention any difference between the two diaphragmatic segments as regards their pattern of activation.[24,25] In cats after laparotomy, Oyer *et al.*[26] observed that the onset and termination of activity were similar for both costal and crural parts of the diaphragm. Little is known about the electrical activity of costal and crural parts of the diaphragm in humans. In our study, we were unable to identify any difference between the two parts of this muscle, for either the onset or the pattern of electrical activation. This suggests that UAS does not induce asynchrony of the two diaphragmatic segments. Moreover the increase in electrical activity obtained after TEB was similar for the two parts of the diaphragm.

Several studies, using indirect motion and pressure indices of diaphragmatic function, previously reported that UAS induced a reduction in the diaphragmatic contribution to ventilation. However, in a recent study, Duggan and Drummond[27] demonstrated that abdominal muscles presented phasic expiratory activity after UAS and questioned the reality of diaphragmatic dysfunction. Since both pressure and motion indices are influenced by abdominal muscle contraction, these authors suggested that these indices were unlikely to be of value in characterizing diaphragmatic activity after UAS. In-

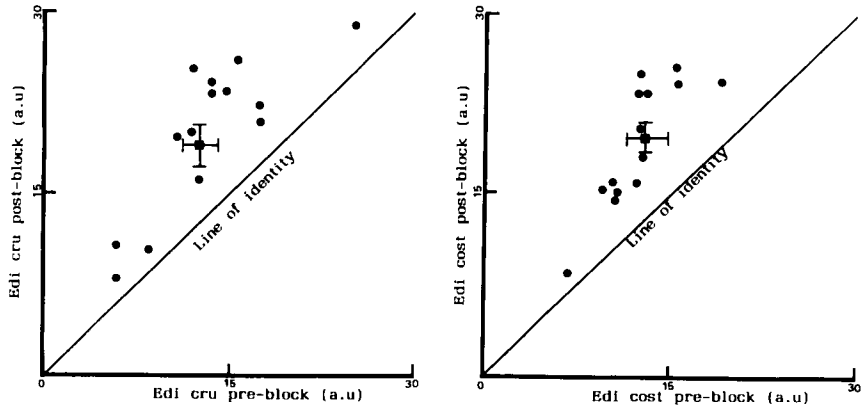

Fig. 3. Individual and mean changes in diaphragmatic integrated electromyogram induced by thoracic extradural block. Edi cru = crural diaphragm; Edi cost = costal diaphragm. Bars ± SD.

deed, phasic abdominal muscle contraction, by increasing end-expiratory Pgas, could artificially decrease ΔPgas if this variable had been obtained as end-inspiratory minus end-expiratory Pgas. That was taken into account in this study and the method used to overcome, at least partially, abdominal muscle activity in the measurement of inspiratory ΔPgas is illustrated in figure 1: ΔPgas was measured as the difference between inspiratory peak and the value obtained just after abdominal muscle relaxation. Therefore the decrease in ΔPgas measured after surgery was probably related to a decrease in diaphragmatic activity but not to an increased abdominal muscle activity. Besides this phasic expiratory contraction of abdominal muscles, tonic activity could be involved as well in the postoperative decrease of abdominal compliance. This phenomenon could be responsible for the decreased inspiratory abdominal expansion observed after surgery. However, a decreased abdominal compliance associated with an unchanged diaphragmatic activation would produce an increase in ΔPgas in concert with an increase in rib-cage expansion (rise in fulcrum effect) with no major change in tidal volume. Such an event is observed in normal subjects when posture is changed from supine to standing.[18] Rib cage tidal volume increased, but ΔPgas and V_T decreased after UAS, suggesting that diaphragm activity probably decreased. Supporting the hypothesis of a postoperative diaphragmatic dysfunction, Easton *et al.*,[19] using chronically implanted sonomicrometers, recently demonstrated in dogs that tidal shortening of costal and crural segments of the diaphragm increased from the 2nd to the 21st day after laparotomy, suggesting that diaphragmatic function, impaired by laparotomy, progressively returned to the preoperative condition.

Even in the three subjects who presented a paradoxical abdominal breathing characterized by a real inspiratory decrease in gastric pressure and abdominal motion, both crural and costal parts of the diaphragm still exhibited electrical activity. This apparent paradox could be related to several mechanisms. First, after UAS, the inspiratory variations in abdominal pressure resulting from diaphragmatic activation could coincide with abdominal muscles relaxation.[18,28] The gastric balloon method, used to assess changes in abdominal pressure from changes in gastric pressure, may be inaccurate in detecting this rapid sequence of events. The abdominal cavity is considered a liquid-filled system that transmits pressure changes induced by diaphragm and/or abdominal muscles to the gastric cavity. This concept is probably imperfect, and abdominal pressure changes possibly differ from gastric pressure variations, especially when rapid events occur.[29] Second, in normal supine subjects, diaphragmatic contraction prevents abdominal viscera from moving cephalad with the transmission of an inspiratory decrease in pleural pressure.[18,30,31] After UAS, even if the diaphragm still functions, its contraction could be insufficient in some patients to prevent the transmission of inspiratory pleural depression to the abdominal compartment, producing a paradoxical abdominal pattern of breathing.

Several hypotheses could explain the increase in diaphragmatic activity induced by TEB. An increase in phrenic drive could occur to compensate for a reduction in parasternal muscle inspiratory activity produced by motor blockade. Such a phenomenon seems unlikely since rib cage motion did not change after TEB, whereas a decreased activity of parasternal muscles should have induced a decrease in rib-cage excursion[32] that would have been detected by DLT. Indeed, compartmental and overall volume changes derived from the DLT depend on principles similar to those used for magnetometers and inductance plethysmographs, validated in the same way in normal subjects. That is, there is a one-to-one correspondence between deflection of the DLT compartmental signals and volume of rib cage or abdomen, as well as between the sum signal and tidal volume. Once volume-motion coefficients are obtained by a calibration procedure, the device will give the correct volume as long as the assumption holds that the system behaves with only one degree of freedom, whatever the dimension measured (area, diameter, or perimeter). The recalibration of the device before each run ensures that, within the limitations of the device, the volumes measured remain valid despite slippages of the belts or changes in chest wall configuration due to the block. In particular, if the epidural were to cause a reduction in upper rib cage expansion compared to lower rib cage expansion, as observed in quadriplegic persons,[33] then after the block, the ratio Δperimeter/ΔV_{RC} will be smaller than before the block because relatively small movements of the upper rib cage will correspond to relatively bigger volume change of the whole rib cage. Nevertheless, using the corrected coefficient, the V_{RC} will be correct. A problem arises if the system develops more than one degree of freedom. Were this to occur, than the way the rib cage changes shape as it expands would be different during the measured breaths than during the calibration. This happens,

for example, if DLTs are calibrated during relaxation maneuvers but then used to assess breaths during which large tension in respiratory muscles are generated, because large forces can distort the chest wall in such a way as to change the relation between perimeter and volume. This phenomenon is possible but not likely to have a large effect since the calibration was performed on normal breaths up to breaths of about two times normal tidal volume of natural breaths.

The influence of TEB on lower inspiratory intercostal muscle activity also should be considered. In normal supine subjects, these muscles exhibit no phasic inspiratory activity during quiet breathing but contract during hyperventilation.[34,35] Their activity has never been evaluated after UAS, but an increase in their contribution to ventilation is questionable since minute ventilation remained unchanged. It thus seems unlikely that diaphragmatic activity increased after TEB to compensate for either complete or incomplete paralysis of these muscles.

A direct effect of bupivacaine on respiratory centers is probably not responsible for the effects of TEB on diaphragmatic activity. Indeed, minute ventilation remained unchanged. Furthermore, the increase in diaphragmatic function does not appear immediately after bupivacaine injection and seems more likely to be associated with the development of the epidural block than with the changes in bupivacaine plasma levels.[36] Moreover, the stability of diaphragmatic contractility associated with the increase in the ΔPgas/ΔPdi index rules out a direct stimulation to the respiratory centers. Indeed, as mentioned by Macklem,[18] an increase in central drive would only increase ΔPgas/ΔPdi if the action were selectively on the diaphragm drive, without a proportional increase in the other respiratory muscles drive. Although the influence of bupivacaine on the central drive of accessory respiratory muscles is unknown, an increase in central drive probably would concern all the muscles and not only the diaphragm. Such an effect on all respiratory muscles would induce no change in the ratio ΔPgas/ΔPdi. Because ΔPgas/ΔPdi increases with TEB, the hypothesis of direct central stimulation seems unlikely. The stability of VT/Ti and Ti/Ttot provides another argument against a central stimulation.

As suggested by the stability of the ΔPdi/Edi ratio, the consequences of TEB on diaphragmatic activity probably do not result from a direct effect on the muscle. This variable is not a perfect index of diaphragmatic contractility. It should be altered by changes in length-tension parameters or variations in diaphragm geometry. However, integrated Edi is usually considered as a reliable indicator of diaphragmatic activation and ΔPdi as an acceptable index of mechanical output of the diaphragm. If these points are agreed on, then the ratio ΔPdi/Edi should be a valid index of the efficiency of the muscle.

The most likely explanation for the increase in diaphragmatic activity is the interruption of an inhibitory reflex. Reflexes arising from viscera, muscles, or chest wall have been shown to interact with respiratory centers and increase or decrease phrenic output.[37–39] Recently Ford *et al.*[5] demonstrated that mechanical stimulation of the gallbladder produced a marked decrease in diaphragmatic contraction in spontaneously breathing dogs. The afferents involved are unknown, but some inhibitory reflexes mediated by phrenic,[40] vagal, or sympathetic[41] pathways have been shown. After UAS, the inhibition of phrenic output is not solely related to pain, since analgesia provided with extradural fentanyl does not improve diaphragmatic contribution to ventilation.[3] Improvement of diaphragmatic function induced by TEB could be related to a direct interruption in such inhibitory reflexes but also to indirect effects. Relaxation of abdominal muscle tone (either by direct motor block or by deafferentation of abdominal proprioceptors) is suggested by the increased abdominal compliance observed after TEB. Then the load placed on the diaphragm may decrease, leading to a reduction in phrenophrenic inhibitory afferents.

In conclusion, this study demonstrates that, although diaphragmatic function was reduced, some phasic activity of the diaphragm was still present after UAS. Diaphragmatic electrical activity was markedly increased after a 0.5% bupivacaine TEB. However, the clinical benefit and/or potential risk (diaphragmatic fatigue) of an increase in diaphragm activation remains to be determined.

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