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Improvement of Diaphragmatic Function by a Thoracic Extradural Block After Upper Abdominal Surgery

B. Mankikian, J. P. Cantineau,† M. Bertrand,* E. Kieffer,‡ R. Sartene,§ P. Viars¶*

The effects on diaphragmatic function of a thoracic epidural block were assessed in 13 patients after upper abdominal surgery (UAS). Lung volumes and tidal changes in chest wall circumferences and gastric (ΔPgas) and esophageal (ΔPes) pressures were measured pre- and postoperatively. Volume displacement of the abdomen divided by tidal volume ($\Delta V_{AB}/V_T$) and ΔPgas/ΔPes were taken as indices of the diaphragmatic contribution to tidal breathing. These respiratory variables were obtained in the postoperative period, before and after epidural injection of 0.5% plain bupivacaine to achieve a block up to the T4 segment. UAS was constantly associated with a decrease in V_T, $\Delta V_{AB}/V_T$, ΔPgas/ΔPes, and forced vital capacity (FVC). Epidural block was associated with an increase in V_T, $\Delta V_{AB}/V_T$, and FVC. ΔPgas and ΔPgas/ΔPes returned to their preoperative values. It is concluded that: 1) diaphragmatic dysfunction observed after UAS is partialy reversed by thoracic epidural block; and 2) that inhibitory reflexes of phrenic activity arising from the abdominal compartment (abdominal wall and/or viscera) could be involved in this diaphragmatic dysfunction. (Key words: Anesthetic technique: bupivacaine 0.5%; epidural. Muscle, diaphragm: postoperative function. Postoperative period: breathing patterns. Surgery: upper abdominal. Ventilation: abdominal; rib cage.)

FOLLOWING UPPER ABDOMINAL surgery (UAS), a long-lasting decrease in lung volume associated with hypoxemia and atelectasis is frequently seen. Most studies[1–4] have focused on changes in lung function and on the therapeutic maneuvers used to treat these changes and thus reduce the incidence of respiratory complications.[5–9] The original hypothesis[10] that this acute and reversible pulmonary failure could be secondary to ventilatory pump failure has recently regained interest. Using chest wall external motion and transdiaphragmatic pressure changes to determine the relative contribution to breathing of the respiratory muscles, several studies[11–14] have demonstrated diaphragmatic dysfunction after UAS. Decrease in diaphragmatic contractility seems unlikely, since transdiaphragmatic pressure excursions during bilateral phrenic nerve stimulation have been reported unchanged in humans[14] after UAS. Consequently, the diaphragmatic dysfunction can be secondary to an impairment in diaphragmatic mechanics related to an increase in abdominal wall tone (increased abdominal stiffness) and/or a reflex decrease of phrenic activity by inhibitory afferents arising from the abdominal compartment. Considering these two possibly interrelated hypotheses, extradural anesthesia could reduce the diaphragmatic dysfunction by interrupting inhibitory afferents conducted by medullary pathways and/or decreasing the abdominal wall tone. To test these hypotheses, the effects of a thoracic epidural block on the breathing pattern, the diaphragmatic contribution to breathing, and lung volumes were assessed after upper abdominal surgery.

Materials and Methods

Thirteen ASA physical status II patients, undergoing elective abdominal aortic surgery *via* a midline xiphopubic incision, were studied pre- and post-operatively.

Individual characteristics, and lung function tests performed while the subjects were in the sitting position obtained 1 week before surgery, are presented in table 1. None of the patients were obese. All, except two, were smokers with a clinical history of mild chronic bronchitis. Forced vital capacity and forced expiratory volume in 1 s (FEV_1) were normal. Individual informed consent and institutional ethical committee approval were obtained. Prior to general anesthesia, a catheter was inserted in the thoracic extradural space through the T_7–T_8 interspace. Four milliliters of 2% lidocaine with 1:200000 epinephrine were injected to confirm placement of the catheter. Thiopental (5 $mg \cdot kg^{-1}$ iv) was used for induction of anesthesia and was followed by pancuronium bromide (0.1 $mg \cdot kg^{-1}$) and fentanyl (6 $\mu g \cdot kg^{-1}$) for tracheal intubation. Anesthesia was maintained by 60% nitrous oxide in O_2 and halothane (0.5–1.5%) with controlled ventilation. Fentanyl and pancuronium were given as required. The mean (±SEM) duration of surgery was 199 min (±20). At the end of surgery, in the recovery room, tracheal extubation was carried out when clinical conditions were judged satisfactory. To provide postoperative analgesia, SC morphine (10 mg) was administered when necessary.

* Assistant Professor of Anesthesiology.
† Resident of Anesthesiology.
‡ Professor of Vascular Surgery.
§ Professor of Physics.
¶ Professor of Anesthesiology.

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Address reprint requests to Dr. Mankikian: Department of Anesthesiology, G.H. Pitie-Salpetriere, 83 boulevard de l'hôpital, 75013 Paris, France.

TABLE 1. Individual Characteristics and Static Lung Volumes (Sitting Position)

Patient No.	Sex	Age	Weight (kg)	Height (cm)	FVC (liters)	FEV_1 (liters)	FRC (liters)	RV (liters)
1	M	65	86	175	3.99	2.70	3.85	2.00
2	M	59	74	183	5.50	3.46	4.92	3.00
3	M	53	60	170	4.24	3.14	3.16	1.38
4	M	72	58	162	3.99	2.57	3.96	2.67
5	M	57	86	182	5.68	3.97	3.93	3.17
6	M	53	78	170	4.38	3.14	3.00	2.47
7	M	75	58	163	3.93	2.44	4.68	2.94
8	M	74	70	170	3.58	2.51	4.20	2.95
9	M	55	75	165	3.89	2.49	3.22	2.30
10	M	72	75	170	3.49	2.80	2.80	2.19
11	M	57	78	179	4.58	3.60	4.36	2.58
12	F	37	53	155	3.60	2.64	2.68	1.69
13	M	49	68	177	4.06	3.14	4.85	2.50
Mean		60	71	171	4.22	2.97	3.75	2.45
±SEM		3	0.8	2.3	0.19	0.13	0.20	0.15

FVC = forced vital capacity; FEV_1 = forced expiratory volume in 1 s; FRC = functional residual capacity; RV = residual volume.

THORACIC AND ABDOMINAL DIMENSIONS

Tidal changes in rib cage (RC) and abdominal (AB) circumferences were simultaneously measured using two differential linear transformers (DLT, Shaewitz, Orgeval, France) connected to belts positioned at the nipple level and 2 cm above the umbilicus. The positions of the belts were carefully marked on the skin to keep the same measurement levels in the pre- and postoperative periods. Technical description and physical properties of DLT and belts have been previously published.[15,16]

Before each measurement period, a calibration procedure was performed to calculate volume-motion coefficients (VMC) for RC and AB. Patients were asked to alternate between predominantly abdominal and thoracic breathing while circumference changes (ΔX_{AB}, ΔX_{RC}) were matched to simultaneously measured spirometric volumes (ΔV, Mijnhardt 9-liter water-filled spirometer). From experimental recording $\Delta X_{AB}/\Delta V$ was then plotted on the x axis and $\Delta X_{RC}/\Delta V$ on the y axis, and a regression line was found by the least square technique. The intercepts of the regression line with the x and y axis produced, respectively, $1/VMC_{AB}$ and $1/VMC_{RC}$. Details concerning the calibration procedure and the calculation of VMC have been previously published.[15]

At each measurement period, circumference changes of each compartment were calculated as the means of all cycles during a 5-min period of quiet breathing. Circumference changes and VMC permitted calculation of AB and RC tidal volume changes ($\Delta V_{AB} = \Delta X_{AB} \cdot VMC_{AB}$, $\Delta V_{RC} = \Delta X_{RC} \cdot VMC_{RC}$), tidal volume ($V_T = \Delta V_{AB} + \Delta V_{RC}$), and abdominal contribution to tidal volume ($\Delta V_{AB}/V_T$). Minute ventilation ($\dot{V}_E$) was calculated as the product of V_T and respiratory rate (RR).

It can be anticipated that UAS may change chest wall geometry and, consequently, the VMCs. Since accuracy of volume measured by a non-invasive method depends mainly upon the accuracy of VMC, a calibration procedure was obtained before each period. Results concerning calibration are given in table 2. Calibrations performed were considered satisfactory because: 1) ranges of V_T and RC/AB partitioning used for calculation of VMCs were broad; 2) the correlation coefficient between $\Delta X_{AB}/\Delta V$ and $\Delta X_{RC}/\Delta V$ was sufficiently high to validate a linear regression analysis; and 3) the correlation coefficient between DLTs (V_{DLT}) and spirometric volumes (V_S) was always greater than .960.

GASTRIC AND ESOPHAGEAL PRESSURES

Two latex thin-walled 10-cm long balloons connected to polyethylene tubing (120 cm in length and 1.7 mm ID) were positioned in the middle third of the esophagus and in the stomach, and filled, respectively, with 0.5 ml and 1 ml of air. Each balloon catheter system was connected to a pressure transducer (Validyne® DP 15). Changes in esophageal (ΔPes) and gastric pressures (ΔPgas) were measured as the difference between peak inspiratory minus peak expiratory pressures and used as indices of pleural and abdominal pressure swings. $\Delta Pgas/\Delta Pes$ ratio was calculated. Pressures were calculated as the mean of all cycles recorded during 2 min of quiet breathing. In addition, end-expiratory gastric pressure (End-exp Pgas) was measured. Abdominal dynamic compliance was calculated as the $\Delta V_{AB}/\Delta Pgas$ ratio. Since catheters were removed between pre- and

TABLE 2. Calibrations and Volume-motion Coefficients (Values are Expressed as Mean ± SEM)

	Preoperatively (Control)	Postoperatively			
		Prior to Epidural Injection	Hours after Epidural Injection		
			1 h	2 h	3 h
Number of points	26 ± 3	28 ± 3	29 ± 1	29 ± 3	30 ± 2
ΔV Ranges (ml)	360 ± 33 1450 ± 195	330 ± 30 1440 ± 100	350 ± 40 1100 ± 100	370 ± 30 1140 ± 130	400 ± 40 1020 ± 100
$\Delta V_{AB}/V_T$ Ranges (%)	84 ± 3 28 ± 3	60 ± 6 13 ± 3	80 ± 5 30 ± 4	81 ± 5 24 ± 4	73 ± 7 20 ± 5
Correlation coefficient ($\Delta X_{AB}/V$, $\Delta X_{RC}/V$)	.896 ± .018	.802 ± .004	.851 ± .003	.871 ± .026	.820 ± .045
Correlation coefficient (V_{DLT}, V_S)	.979 ± .003	.962 ± .001	.970 ± .003	.960 ± .013	.967 ± .014
VMC_{AB} (ml/mm)	97 ± 10	193 ± 44	173 ± 23	172 ± 24	156 ± 24
VMC_{RC} (ml/mm)	83 ± 11	117 ± 20	123 ± 14	114 ± 10	111 ± 13

VMC_{AB} and VMC_{RC} volume-motion coefficients for the abdominal (AB) and ribcage (RC) compartments.

postoperative measurements, the distance from the nostril for each catheter was noted at the end of the control period. Catheters could thus be repositioned in the postoperative period at the same levels. In addition, after surgery, the stomach was emptied before each set of measurements using a gastric suction tube inserted during anesthesia.

Tidal changes in chest wall circumferences and pressures were recorded on a six channel strip-chart recorder (Sefram-Enertec, Velizy, France). ΔPgas/ΔPes and $\Delta V_{AB}/V_T$ ratios were used to assess the diaphragmatic contribution to tidal breathing.[17-19]

Lung Function Tests

Functional residual capacity (FRC) and forced vital capacity (FVC) were measured using a 9-liter water-filled spirometer (Mijnhardt, Odjik, Holland) with CO_2 absorber and helium analyzer (Mijnhardt UG 45). The helium analyzer was calibrated before each determination. Patients were connected to the circuit at the end of expiration as determined by chest wall circumference monitoring. Oxygen was continuously added to obtain a constant end-expiratory circuit volume. A 2-min plateau in helium concentration was obtained before measurement. Values were corrected to BTPS. A single measurement of FRC was made for each patient at each period. FVC was performed in triplicate and the best value retained.

Experimental Procedure

At each measurement period, respiratory measurements (including calibration procedure) were performed with patients in the supine 30° head-up position. Respiratory variables were obtained 24 h before surgery (preoperative baseline) and after the end of surgery (postoperative, pre-injection), as patients had fully recovered from general anesthesia. The mean time (±SEM) from the end of surgery to the beginning of postoperative measurements was 17 h (±2). Then, 0.5% plain bupivacaine was slowly (1 min) injected through the epidural catheter to achieve loss of pinprick sensation up to T4 segment. This level of block was achieved in all patients. The volume injected ranged between 8 and 14 ml. Arterial blood pressure was continuously monitored during the study by a radial artery catheter inserted preoperatively for the standard anesthetic management. If necessary, modified gelatine solution was infused (Plasmion®, Roger Bellon, France) to avoid decrease in mean arterial pressure greater than 20% of control values. No adrenergic agents were used. Measurements were repeated 1, 2, and 3 h after bupivacaine administration. At each period, after the calibration procedure between direct and indirect spirometry, patients were left in a quiet environment without nose clip and mouth piece to obtain a close to steady state breathing pattern. FVC, FRC, and arterial blood gases were measured 24 h preoperatively and postoperatively, before and 1 h after the epidural block. Arterial blood gases were obtained after patients had been breathing room air for 20 min. In this study, pain was not assessed. However, no patient complained of sufficient pain to receive SC morphine in the 9.3 ± 0.7 h (mean ± SEM) preceeding the first set of postoperative measurements.

Statistical Analysis

Comparison between the different periods was performed using two-way analysis of variance and a Student's *t* test corrected for multiple comparison. All values are given as the mean ± SEM. *P* value less than 0.05 was taken to be significant.

TABLE 3. Sequential Changes in Respiratory Variables

	Preoperatively (Control)	Postoperatively			
		Prior to Epidural Injection	Hours after Epidural Injection		
			1 h	2 h	3 h
RR ($c \cdot min^{-1}$)	12.9 ± 0.9	18.7‡ ± 0.9	15.9†¶ ± 0.5	18.4‡ ± 0.8	19.3‡ ± 1.4
V_T (ml)	531 ± 36	337‡ ± 22	464¶ ± 24	419†§ ± 30	342‡ ± 27
V_E ($l \cdot min^{-1}$)	6.66 ± 0.47	6.24 ± 0.45	7.33 ± 0.37	7.70 ± 0.62	6.52 ± 0.51
ΔV_{RC} (ml)	157 ± 27	263‡ ± 23	219* ± 22	226* ± 26	248† ± 28
$\Delta V_{AB}/V_T$ (%)	69.2 ± 3.4	15.2‡ ± 3.7	46.2‡** ± 5.3	34.9‡** ± 4.7	16.9‡ ± 5.5
ΔPes (cm H_2O)	5.63 ± 0.45	4.93 ± 0.48	5.27 ± 0.35	5.03 ± 0.39	5.14 ± 0.45
ΔPgas (cm H_2O)	2.77 ± 0.43	0.39‡ ± 0.30	2.58** ± 0.33	1.58†¶ ± 0.31	0.77‡ ± 0.14
ΔPgas/ΔPes (%)	50.5 ± 6.7	7.4‡ ± 9.3	51.7** ± 9.3	33.9*¶ ± 8.7	14.0‡ ± 4.5
End-exp Pgas (cm H_2O)	5.67 ± 1.08	5.90 ± 0.84	4.40 ± 0.84	4.59 ± 0.86	6.20 ± 1.02
$\Delta V_{AB}/\Delta Pgas$ ($ml \cdot cm\ H_2O^{-1}$)§	146 ± 20	64‡ ± 14	113§ ± 24	94* ± 20	73‡ ± 18

Values are expressed as mean ± SEM. RR = respiratory rate; V_T = tidal volume; V_E = minute ventilation; ΔV_{RC} = inspiratory change in rib cage volume; $\Delta V_{AB}/V_T$ = abdominal contribution to tidal volume; ΔPes = inspiratory change in esophageal pressure; ΔPgas = inspiratory change in gastric pressure; End-exp Pgas = end-expiratory gastric pressure; $\Delta V_{AB}/\Delta Pgas$ = change in abdominal volume over change in gastric pressure obtained in 11 patients.

* $P < 0.05$; † $P < 0.01$; ‡ $P < 0.001$ *vs.* control.

§ $P < 0.05$; ¶ $P < 0.01$; ** $P < 0.001$ *vs.* prior to epidural injection.

Results

Sequential changes in respiratory parameters measured during the study are given in tables 3 and 4 and figures 1–3.

Effects of Upper Abdominal Surgery

When compared to preoperative baseline values, the breathing pattern observed after UAS was characterized by a significant decrease in VT and a significant increase in RR. $\dot{V}_E$ remained unchanged. In all patients, external chest wall movements were modified with a shift from predominantly abdominal to predominantly rib cage breathing. Both pressure ($\Delta Pgas/\Delta Pes$) and motion ($\Delta V_{AB}/V_T$) indices of diaphragmatic contribution to tidal breathing were significantly decreased. Abdominal dynamic compliance ($\Delta V_{AB}/\Delta P_{gas}$) was significantly reduced. End-expiratory gastric pressure remained unchanged. FVC, FRC, and Pa_{O_2} were significantly reduced, while Pa_{CO_2} was unchanged. The mechanical behavior of the abdominal compartment was abnormal in all patients when compared to the preoperative condition. In ten patients, inspiratory swings in gastric pressure and abdominal circumference, though markedly decreased, remained slightly positive. In only one patient was a paradoxical abdominal breathing, defined as simultaneous inspiratory negative changes in both gastric pressure and abdominal circumference, observed. No phasic expiratory activity in abdominal muscles could be clinically detected in this patient. In these 11 patients, no phase lag was observed between the circumference and pressure signals. In the remaining two patients, a strong expiratory contraction of abdominal muscles was clinically observed, showing a different pattern (fig. 4). During expiration, gastric pressure increased progressively, while abdominal circumference decreased slightly. The following inspira-

TABLE 4. Arterial Blood Gases and Static Lung Volumes Before and After Surgery (Supine Position)

	Preoperatively (Control)	Postoperatively	
		Prior to Epidural Injection	1 h after Epidural Injection
FVC (ml)	3300 ± 208	1380† ± 115	1930† ± 144‡
FRC (ml)	2500 ± 104	2208† ± 127	2220† ± 130
*p*H	7.43 ± 0.1	7.41 ± 0.1	7.42 ± 0.1
Pa_{O_2} (mmHg)	74 ± 3	64* ± 3	67 ± 2
Pa_{CO_2} (mmHg)	39 ± 1	39 ± 1	38 ± 2

* $P < 0.05$; † $P < 0.01$ *vs.* control.

‡ $P < 0.01$ *vs.* prior to epidural injection.

Values are expressed as mean ± SEM. FVC = forced vital capacity; FRC = functional residual capacity; Pa_{O_2} = partial pressure of arterial oxygen; Pa_{CO_2} = Partial pressure of arterial carbon dioxyde.

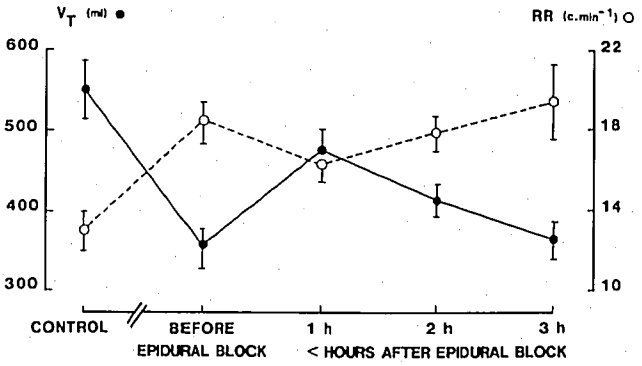

FIG. 1. Mean changes (±SEM) in tidal volume (V_T), represented by closed circles, and respiratory rate (RR), represented by open circles, for each of the designated time periods. Statistical significances are given in table 2.

tion was characterized by two successive phases: 1) at the very beginning of inspiration, gastric pressure fell abruptly, and then 2) increased until the end of inspiration with a simultaneous increase in abdominal circumference. The first phase was interpreted as secondary to the abrupt relaxation of the abdominal muscles, while the second phase was interpreted as corresponding to the inspiratory diaphragmatic motion. Therefore, phasic expiratory activity of abdominal muscles was responsible for a false aspect of abdominal paradoxical breathing, and a phase lag between inspiratory changes in rib cage and abdominal signals was observed. In these two patients, abdominal compliance could not be measured, since the abdominal compartment behavior was not uniform during the inspiratory phase.

Effects of the Thoracic Epidural Block with 0.5% Plain Bupivacaine

One hour after epidural bupivacaine, all patients had a block up to the T_4 level, and none complained of abdominal pain or any other discomfort. When compared to postoperative pre-injection values, there was a significant increase in V_T and a significant decrease in RR. $\dot{V}_E$, Pa_{O_2}, and Pa_{CO_2} remained unchanged. $\Delta Pgas/\Delta Pes$ and $\Delta V_{AB}/V_T$ ratios were significantly increased. During this period, V_T and pressure indices did not change from preoperative values, while RR and $\Delta V_{AB}/V_T$ remained different. This pattern was observed in all patients.

Each of the three abnormalities in abdominal compartment behavior induced by UAS was reversed by the block. In addition, abdominal compliance increased significantly, while end-expiratory gastric pressure was not modified. FVC was significantly increased, and FRC, Pa_{O_2}, and Pa_{CO_2} remained unchanged.

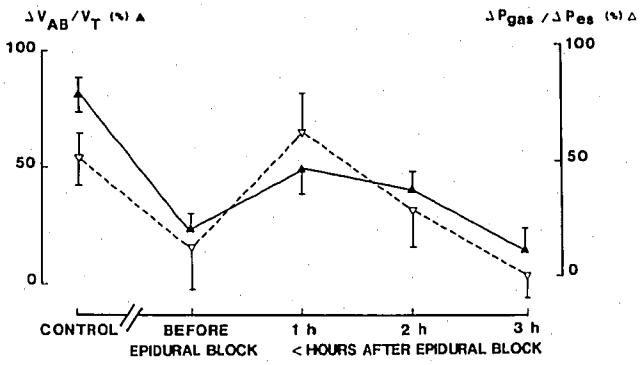

FIG. 2. Mean changes (±SEM) in abdominal contribution to tidal breathing ($\Delta V_{AB}/V_T$), represented by closed triangles, and ratio of inspiratory swings in gastric pressure over inspiratory swings in esophageal pressure ($\Delta Pgas/\Delta Pes$), represented by open triangles, for each of the designated time periods. Statistical significances are given in table 2.

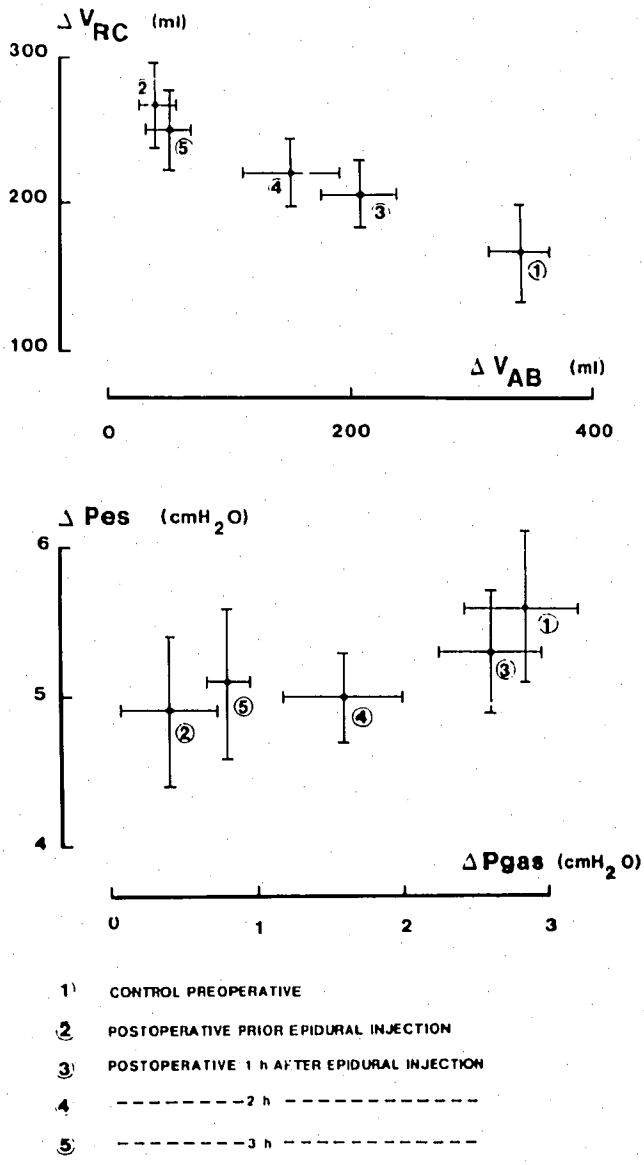

FIG. 3. Mean tidal changes in rib cage volume (ΔV_{RC}) plotted against mean tidal changes in abdominal volume (ΔV_{AB}) (upper panel) and mean inspiratory swings in esophageal pressure (ΔPes) plotted against mean inspiratory swings in gastric pressure ($\Delta Pgas$) (lower panel) for each of the designated time period. Statistical significances are given in table 2.

Time Course of the Respiratory Effects Induced by the Thoracic Epidural Block

As shown in table 3 and figures 1–3, all respiratory variables returned progressively to postoperative pre-injection values. Two hours after bupivacaine injection, V_T, $\Delta V_{AB}/V_T$, $\Delta Pgas$, and $\Delta Pgas/\Delta Pes$ remained significantly different from postoperative pre-injection values. Three hours after bupivacaine, as there was a complete clinical resolution of the block, all respiratory

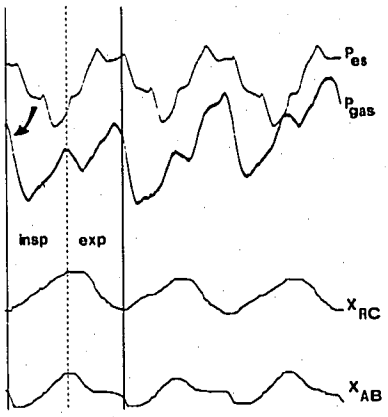

FIG. 4. Recording of simultaneous changes in chest wall circumferences (X_{AB}, X_{RC}), gastric (Pgas), and esophageal pressure (Pes) in one patient with a phasic expiratory activity of abdominal muscles after upper abdominal surgery. The initial decrease in Pgas at the very beginning of inspiration (insp) is related to relaxation of abdominal muscles (indicated by arrow), giving a false aspect of abdominal paradoxical breathing (see text).

variables returned to their postoperative pre-injection values.

Discussion

This study, similar to several previous studies,[11-14] demonstrates that UAS is responsible for respiratory dysfunction characterized by rapid and shallow breathing, a shift from predominantly abdominal to predominantly rib cage breathing, and a decrease in FVC and FRC. The new finding in this study is that the respiratory dysfunction is partially reversed by a segmental epidural block reaching the T_4 level. One hour after the block, tidal volume and abdominal contribution to breathing are both increased (49% and 50%, respectively), and the respiratory rate is decreased as the vital capacity increased.

The relative contribution to breathing of the respiratory muscles has been indirectly estimated from the simultaneous measurements of tidal changes in transdiaphragmatic pressures (ΔPgas, ΔPes), chest wall external dimensions (ΔX_{AB}, ΔX_{RC}), and lung volumes. Changes in $\Delta V_{AB}/V_T$ and in ΔPgas/ΔPes are commonly used as indices of changes in the diaphragmatic contribution to breathing, while changes in $\Delta V_{RC}/V_T$ could reflect alterations in the contribution of intercostal/accessory muscles.[17-19] There are several methodological limitations with this approach. The most important comes from the mechanical interdependence between rib cage and abdominal compartments.[20,21] In normal conditions, diaphragmatic shortening is associated with a lowering of the diaphragmatic dome into the abdominal cavity (piston-like action) causing an increase in abdominal pressure and abdominal external dimensions. However, the diaphragm also acts by increasing the rib cage external dimensions; directly, by its costal insertions, and, indirectly, by the increase in abdominal pressure through the zone of apposition.[20,22-24] Thus, for a given diaphragmatic shortening, the extent of increase in transdiaphragmatic pressure and chest wall external dimensions will depend on the relative compliance and geometry of the two compartments. Finally, changes in muscular activity can be grossly inferred from chest wall motions only if mechanical conditions (including compartment's geometry) remain constant. Alterations of chest wall mechanics secondary to changes in posture illustrate this mechanical interdependence.[25] When the posture is changed from supine to standing, abdominal contribution to breathing ($\Delta V_{AB}/V_T$) is decreased, while the diaphragmatic electromyographic phasic activity increases. The shift from predominantly abdominal to mostly rib cage breathing is related to a stiffened abdominal compartment. ΔPgas and V_T remain unchanged. In our study, a decrease in dynamic abdominal compliance ($\Delta V_{AB}/\Delta$Pgas) was also observed after UAS and associated with a decrease in $\Delta V_{AB}/V_T$. However, change in abdominal compliance in this condition cannot explain the shift from abdominal to rib cage breathing, since ΔPgas and tidal volume are consistently decreased (−85% and −36%, respectively). These results suggest that the decrease in abdominal contribution to tidal breathing observed after UAS is mainly related to a decrease in diaphragmatic activity. In addition, the increase in the absolute value of ΔV_{RC} suggests an augmentation of the intercostal/accessory muscles contribution. In the same way, the increase in V_T and the return to normal of ΔPgas observed after thoracic epidural block suggest that the greater $\Delta V_{AB}/V_T$ is related to an improvement of the diaphragmatic function. Normalization of ΔPgas/ΔPoes after epidural anesthesia, while $\Delta V_{AB}/V_T$ remains different from control values, does not imply that diaphragmatic function is normalized. As previously discussed, ΔPgas/ΔPes and $\Delta V_{AB}/V_T$ are dependent upon chest wall geometry and relative compliance of the two compartments (RC and AB). Since chest wall geometry is modified (lower FRC), and abdominal compliance decreased postoperatively, pressure and motion indexes should be considered only in the magnitude and direction of their changes, and not in their absolute values.

It should be emphasized that the changes observed in forced vital capacity after UAS and epidural block are parallel to the changes in diaphragmatic function. The discrepancy between the improvement of diaphragmatic function and the only moderate increase in FVC may be related, in part, to the lower intercostal and abdominal blockade during bupivacaine epidural anesthesia.

Two main mechanisms can be responsible for the diaphragmatic dysfunction after UAS: 1) a decrease in con-

tractility, and 2) a decrease in phrenic nerve activity. A decrease in diaphragmatic contractility cannot be viewed as a predominant factor, since transdiaphragmatic pressure swings during bilateral phrenic nerve stimulation have been reported unchanged after UAS in humans.[14] In contrast, animal studies have demonstrated inhibitory reflexes of phrenic motor output during or after stimulation of visceral or somatic afferents. In anesthetized cats, distension of the lower esophagus reflexly decreases diaphragmatic activity while, in the same time, intercostal activity is increased. This reflex is abolished after bilateral cervical vagotomy.[26] In cats, inhibitory somatic afferents arising from chest wall mechanoreceptors have been demonstrated. Stimulation of intercostal and abdominal muscle proprioceptor afferents reflexly decreases phrenic motor activity.[27] In this model, inhibitory afferents are conducted through medullary pathways. Phreno-phrenic inhibitory loops have also been demonstrated.[28] Inhibition of phrenic motor output can be triggered by direct mechanical stimulation of diaphragmatic golgi tendon organs.[29]

After UAS, inhibitory reflexes may be present in humans originating from visceral stimulation and/or increase in abdominal wall tone. Partial reversal of the diaphragmatic dysfunction after thoracic epidural block may be related to their direct or indirect interruption. Inhibitory afferents, either visceral or somatic, can be blocked by the local anesthetic at the level of spinal dorsal roots. An indirect mechanical effect can also be hypothetized. Relaxation of abdominal muscle tone, either by direct motor block (spinal ventral roots) or by deafferentiation of abdominal proprioceptors, may decrease the tension exercised on the diaphragm, and, thus, decrease the phreno-phrenic inhibitory afferents.

On the other hand, nociceptive afferents might not be involved. It has been shown that the respiratory dysfunction observed after UAS was not modified by selective spinal analgesia achieved by extradural or subarachnoid injection of opiates. Using the same methodology as ours already described in this study, Simmoneau *et al.*[13] demonstrated that the dysfunction was not modified after epidural opiates. The shallow and rapid breathing, the decrease in the diaphragmatic contribution to tidal breathing, and the decrease in transdiaphragmatic pressure generated during a Muller manoeuver, were not modified after a 150-μg fentanyl epidural injection. Potent analgesia was obtained, since the pain intensity was scored 2.6 before and 0.25 after fentanyl (mean values). In the same way, Clergue *et al.*[30] have shown that the shallow and rapid breathing observed after UAS were not modified by intrathecal injection of morphine (2 and 5 mg), while complete pain relief was obtained. However, Bromage *et al.*,[6] comparing the effect on forced expiratory volume in 1 s (FEV_1) of epidural morphine and 2% lidocaine, have drawn different conclusions. Since the same improvement in FEV_1 was reported with either morphine or 2% lidocaine, the authors conclude that the respiratory dysfunction was equally reversed. However, despite the similarity on FEV_1 improvement between the two analgesic regimens, the mechanism is implicitely different. FEV_1 mainly depends on three determinants: initial lung volume (inspiratory capacity), airway diameter, and force generated by expiratory muscle contraction (mainly abdominal muscles). Two percent lidocaine gives a potent motor block, while opiates have no motor effect. Therefore, if we assume a constant airway diameter, improved FEV_1 in the lidocaine group could reflect an increased initial lung volume, while the greater FEV_1 in the opiate group could be secondary to the analgesia along with the preservation of abdominal muscle function. It appears from clinical studies that pain, which is a major clinical problem after UAS, is not the key mechanism underlying respiratory dysfunction.

It is unlikely that direct stimulation of respiratory centers by the local anesthetic after its vascular resorption was involved in the partial reversal of diaphragmatic dysfunction. In a complementary study, the ventilatory response to CO_2 rebreathing was measured before and after epidural injection of 20 ml .5% plain bupivacaine at lumbar level.[31] The bupivacaine plasma levels achieved were low (1.10 ± 0.15 $\mu g \cdot ml^{-1}$), and no ventilatory stimulation was observed during both quiet and CO_2 stimulated breathing. In the present study, doses of bupivacaine used were low (ranging from 40 to 70 mg), respiratory rate was decreased after the block, and no signs of toxicity were observed.

To avoid changes in diaphragmatic contractility, bupivacaine without epinephrine was administered and arterial pressure was maintained by volume expansion. Therefore, no adrenergic agent known to increase diaphragmatic contractility[32] was administered.

Thus, the effect of the epidural block on diaphragmatic function provides indirect evidence that inhibitory reflex(es) of phrenic motor activity is (are) probably responsible for the diaphragmatic dysfunction after UAS. We cannot discriminate among the several inhibitory afferents potentially involved; in particular, between a pure sensory mechanism (intraabdominal afferents) and/or a mechanical antagonism between the abdominal muscles and the diaphragm. The effects of lower concentrations of bupivacaine (0.25%) could be interesting in the differentiation of the sensory from the motor block.

In conclusion, this study confirms that diaphragmatic dysfunction is a major determinant of the decrease in lung volumes observed after UAS. This diaphragmatic dysfunction is partially reversed by a thoracic epidural

block using .5% bupivacaine. Inhibitory reflexes of phrenic activity are probably involved. The clinical importance of this study is that, among the several analgesic methods used after UAS, epidural block is the only one to achieve *per se* a decrease of the respiratory dysfunction. However, the beneficial effect of an acute epidural administration of .5% bupivacaine is transitory. Additional studies must be performed to assess the effect of long-term administration and of a lower concentration of bupivacaine on respiratory function and on the incidence of pulmonary complications.

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