Effects of Interpleural Bupivacaine on Respiratory Muscle Strength and Pulmonary Function

Lluís Gallart, M.D., Joaquim Gea, M.D., Ph.D., † M. Carmen Aguar, M.D., ‡
Joan M. Broquetas, M.D., Ph.D., § Margarita M. Puig, M.D., Ph.D. ¶

Background: Several reports suggest that interpleural local anesthetics may have deleterious effects on respiratory function. The current study investigated the effects of interpleural bupivacaine on human respiratory muscles and lung function in 18 healthy male subjects (<65 ± 5 yr old) with normal respiratory function and scheduled for cholecystectomy entered the study before surgery. Respiratory parameters were compared before and after the interpleural administration of 20 ml 0.5% bupivacaine plus 1.200,000 epinephrine while patients were supine; we evaluated breathing pattern, dynamic and static lung volumes, airway conductance, maximal inspiratory pressures (at the mouth; at the esophagus [Pes;]; at the abdomen [Pga;]; and transdiaphragmatic [Pdi;]) functional reserve (tension-time index) of the diaphragm, and maximal expiratory pressures (at the mouth; at the esophagus [Pem]; and at the abdomen [Pga;]). Hemoglobin oxygen saturation by pulse oximetry, heart rate, and mean arterial pressure were continuously monitored.

Results: Respiratory rate (15 ± 1 to 19 ± 1 breaths/min; P < 0.01) and heart rate (78 ± 3 to 85 ± 3 beats/min; P < 0.01) were slightly increased. Dynamic and static lung volumes, airway conductance, hemoglobin saturation, and the remaining breathing pattern parameters were unchanged. Regarding respiratory muscles, maximal inspiratory pressure at the mouth, Pes, and tension-time index of the diaphragm did not change. Pdi decreased slightly (102 ± 10 to 92 ± 10 cmH2O; P < 0.05) because of a change in Pga (24.2 ± 7.4 to 18.4 ± 6.8 cmH2O; P < 0.05). Maximal expiratory pressure at the mouth remained unaltered, but Pga showed a trend to decrease (92 ± 15 to 78 ± 10 cmH2O; P < 0.05).

Conclusions: In our experimental conditions, interpleural bupivacaine did not significantly change lung function or inspiratory muscle strength but induced a slight decrease in abdominal muscle strength. Although this effect was minimal, its clinical relevance needs to be evaluated further in patients with impaired respiratory function. (Key words: Anesthesiology, local: bupivacaine. Anesthetic techniques: interpleural. Muscles, respiratory: physiology. Respiration: function tests.)

INTERPLEURAL local anesthetics produce sensory blockade of the hemithorax and superior hemiabdomen. However, the extent and characteristics of the motor blockade and the effects on respiratory function have not been clearly established. The block may affect muscles innervated by thoracic nerves, including the external intercostal muscles, used during inspiration, and the internal intercostal and abdominal muscles, which are the main respiratory muscles. On the other hand, the diaphragm, which is the main inspiratory muscle, is less likely to be blocked because the phrenic nerve travels in the mediastinum, remote from the posterior rib cage, where local anesthetics are located when administered with the patient supine. However, a large part of the surface of the diaphragm is in opposition with the lower rib cage; in this area, the muscle or the terminal branches of the phrenic nerve may be blocked by local anesthetics. Therefore, both inspiratory and expiratory muscles may be affected by interpleural anesthetics.

Studies in animals have shown that interpleural anesthetics induce blockade of the intercostal nerves and dramatically decrease the electromyographic activity of the diaphragm. It has also been reported that in humans, interpleural anesthetics can occasionally result in unilateral bronchospasm or phrenic nerve paralysis. However, no studies have been specifically designed or performed to investigate the effects of interpleural anesthetics on respiratory muscle strength and pul-

Materials and Methods

Patients

Thirteen Institutional approval and healthy adults for whom the resus-
citation was normal and who had no history of obstructive airway disease, neurologic disease, neuromuscular disease, or severe cardiovascular disease were included in the study. Subjects excluded were those with chronic obstructive airway disease, neurologic disease, or severe cardiovascular disease.

Catheter Placement

Patients received 5000 KU of heparin intravenously and were premedicated with 5 mg atropine intravenously, 20 mg ketamine intramuscularly, and 10 mg diazepam intravenously 30 min before surgery. Anesthesia was induced with 1.5 mg/kg thiopental, 0.6 mg/kg propofol, and 0.6 mg/kg fentanyl intravenously. Tracheal intubation was facilitated with 10 mg/kg succinylcholine. Anesthesia was maintained with 50% nitrous oxide in oxygen, 4% isoflurane, and 100 ng to 200 ng of fentanyl intravenously.

Address reprint requests to Dr. Gallart: Servicio de Anestesiología, Hospital Universitari del Mar, Passeig Marítim 25, 08003 Barcelona, Spain.

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Fig. 1. Sequence of the study. BP = breathing pattern; FS = forced spirometry (dynamic lung volumes); DLco = carbon monoxide diffusion; SLV = static lung volumes; SGaw = airway conductance; RMF = respiratory muscle function; SpO2 = hemoglobin oxygen saturation by pulse oximetry; HR = heart rate; MAP = mean arterial blood pressure.

A trend to impaired sensory and sensory-motor functions of the hemiabdominals of the respiratory muscle may affect breathing, including the diaphragm and intercostal muscles. However, the other abdominal and respiratory muscles, including the phrenic nerve, were not affected by bupivacaine.

Interpleural anesthesia of the phrenic nerves and respiratory activity has been reported that in turn can result in nerve paralysis.

Catheter Placement

Patients received no preanesthetic medication. Before surgery and with the patient sitting, an interpleural catheter (Perifix, Braun, Melsungen, Germany) was introduced through an 18-G Hustead-type needle (Monoject, Sherwood Medical, West Sussex, United Kingdom). The needle was inserted in the eighth intercostal space below the right scapular vertex, using the technique described by Scott. The catheter was gently inserted and was then withdrawn so that the 10-cm mark could be seen on the skin surface. A test dose (3 ml 0.5% bupivacaine plus 1:200,000 epinephrine) was administered to rule out intravascular injection, and a radiographic control was performed to rule out the presence of pneumothorax. Interpleural radiologic contrast was not used, for two reasons: to avoid diluting or altering the distribution of bupivacaine and to prevent any possible effect of the dye on respiratory muscle function.

Experimental Protocol

Figure 1 describes the protocol used in this study. With the pleural catheter inserted and the patient sitting, breathing pattern, dynamic lung volumes (measured by forced spirometry), carbon monoxide diffusion, static lung volumes, and airway conductance were assessed.

The subjects were then placed supine, and 15 min later, breathing pattern, dynamic lung volumes, and respiratory muscle function were assessed. These parameters were again evaluated in the same position 30 min after administration of 20 ml 0.5% bupivacaine plus 1:200,000 epinephrine. To verify the extension and effectiveness of the analgesia, the pinprick test was performed, with the left hemithorax and superior hemiabdomen used as controls. The limits of cutaneous analgesia to be checked were as follows: cranially, a dermatome line between the clavicle and the nipple, related to the upper thoracic nerves; caudally, the T10 dermatome related to the umbilical line; and medially, the midline.

Static lung volumes and airway conductance were assessed after the interpleural blockade with the patients seated and were compared with the previous data obtained in the same position. They were not obtained in the supine position because plethysmography needed to be performed while the patient was sitting.

Hemoglobin saturation by pulse oximetry (Biox 3740, Ohmeda, Louisville, CO), heart rate, and noninvasive mean arterial blood pressure (Superion 7210, Kontron Instruments, Milano, Italy) were monitored throughout the study. Patients breathed room air during the entire procedure.

Materials and Methods

Patients

After Institutional approval and informed consent, 13 healthy adults for whom the results of respiratory function tests were normal and who were scheduled for subcostal cholecystectomy were consecutively included in the study. Subjects excluded were those with abnormal chest anatomy; neurologic, muscular, pulmonary or cardiac disease; morbid obesity (body mass index > 35 kg · m⁻²); known drug allergy; diabetes mellitus; coagulation disorders; or acute or chronic pain.

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When the study had been concluded, patients entered the operating room. The interpleural catheter was used for the administration of bupivacaine during surgery and for postoperative analgesia.

**Functional Evaluation Techniques**

**Respiratory Function Tests.** These included dynamic lung volumes measured by forced spirometry (Spirometer Datospir 92, Sibel, Barcelona, Spain) and determinations of static lung volumes and airway conductance (body plethysmography, Masterlab, Jaeger, Würzburg, Germany) and carbon monoxide diffusion (single-breath method, Masterlab). Reference values were those for a Mediterranean population.10,11

**Breathing Pattern.** Patients breathed through a mouthpiece and a two-way low-resistance valve (Hans-Rudolph, Kansas City, MO). Breathing pattern was obtained with a pneumotachometer (Screenmate, Jaeger) placed in the external inspiratory circuit. The flow signal was converted into a volume signal and registered with a multichannel recorder (R-611, Sensormedics, Anaheim, CA). Tidal volume, respiratory rate, minute ventilation, and inspiratory and total respiratory times were obtained from the recording. The system was calibrated at the beginning of each study. To ensure steady state, variables were evaluated after 5 min of quiet breathing.

**Respiratory Muscle Function.** Respiratory muscle function12 was evaluated by determining maximal inspiratory and expiratory pressures measured at the mouth (PImax and PEmax, respectively), at the esophagus (Pesniff and Pescough, respectively), and at the abdomen (Pginiff and Pgcough, respectively); transdiaphragmatic pressure (Pdi) was computed as Pga − Pes. The PImax was measured from the residual volume, and the PEmax was determined from total lung capacity. Both efforts were performed against a closed mouthpiece, by using the same manometer (Sibelmed 63, Sibel). The Pes and Pga were obtained with the classic two-balloon-catheter technique. The balloons (Jaeger) were the standard ones used to determine lung compliance. Each balloon’s unstressed volume was 6 ml, and they were filled with the predetermined minimum air volume necessary to obtain the best recording. Thus, one balloon was placed in the esophagus and filled with 0.75 ml air, and the other was positioned in the stomach and filled with 1 ml. Each was attached to a pressure transducer (Transpac II, Abbot, Chicago, IL) that was connected to the above mentioned recorder. A pop test13 previously performed confirmed that the system was critically damped. The system was calibrated at the beginning of each study, and balloon volumes were checked at the end of the procedure to rule out air leakage. Mean values of Pes, Pga, and Pdi were measured at tidal volume (Pes, Pga, and Pdi) and during maximal respiratory efforts. The sniff maneuver (a short, sharp inspiratory noise effort from functional residual capacity) was chosen to evaluate the maximal inspiratory effort, and a voluntary cough from total lung capacity was used to evaluate the maximal expiratory effort. Thus Pesniff, Pginiff, Pdcough, and Pgcough were obtained (figs. 2 and 3). All measurements, except sniff and cough maneuvers, were performed using nose clips.

Maximal respiratory measurements (PImax, PEmax, forced spirometry, and sniff and cough measurements) were always conducted by the same physician and were randomly performed (with a standard random number table) to avoid interference from train-
Statistical Analysis

Data are presented as means ± SEM. Normal distribution for each variable was tested with the Kolmogorov-Smirnov test. Student’s paired t test was used to compare variables from the same patient (before and after bupivacaine). Pearson’s coefficient was used to assess correlation, and linear regression analysis was applied where appropriate. A P value < 0.05 was considered significant.

Results

Demographic data of the subjects are listed in table 1. As previously mentioned, respiratory function was normal in all subjects at the beginning of the study (table 2). Unilateral skin analgesia of the thorax and superior abdomen within the limits previously mentioned was obtained in all the patients, without evidence of analgesia on the left side.

After the administration of bupivacaine, dynamic and static lung volumes, and airway conductance were unaltered. An increase in respiratory rate without changes in the other parameters of the breathing pattern was observed; this change caused an increase in minute ventilation (table 3).

In the comparison of variables that express inspiratory muscle strength, no changes were detected in Pmax and Pes,cough. However, Pdi,sniff exhibited a slight decrease, which was entirely attributable to a decrease in Pga,sniff; a positive correlation between changes in these two variables was obtained (r = 0.84; P < 0.001). Pes and Pga during quiet breathing (Pes,sniff and Pga) as well as the functional reserve of the diaphragm against fatigue (Pdi/Pdi,sniff, tension–time index of the diaphragm) remained unaltered (table 4).

Regarding expiratory muscle strength, no changes were observed in PEmax. In contrast, Pga,cough significantly decreased, and a similar pattern was observed in Pes,cough (table 4).

Heart rate significantly increased (78 ± 3 to 93 ± 3 beats/min; P < 0.01) whereas mean arterial blood

<table>
<thead>
<tr>
<th>Table 1. Demographic Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>55 ± 4</td>
</tr>
</tbody>
</table>

Values are mean ± SEM, where applicable.
pressure decreased (98 ± 4 to 90 ± 3 mmHg; P < 0.01) after interpleural bupivacaine. The hemoglobin saturation remained unchanged (97 ± 0.4 vs. 97 ± 0.5%) throughout the study.

Discussion

The current study demonstrates that the administration of interpleural bupivacaine to healthy patients in the supine position has no deleterious effects on pulmonary function or inspiratory muscle strength. The possibility of respiratory impairment induced by interpleural anesthetics has been suggested by several groups of investigators.4,6,8,14-19 In this situation, maneuvers such as coughing and sighing would be altered and could result in a greater rate of pulmonary complications in the postoperative period.

Although some studies have evaluated respiratory function after the administration of interpleural anesthetics,4,6,10-22 they are not useful enough to address this issue. All of these studies used only forced spirometry, which is not an appropriate method to diagnose muscle weakness caused by nerve blockade.12 In addition, they were performed in the immediate postoperative period, and their results may have been influenced by pain, residual anesthetics or the surgery itself. Moreover, results differ among these studies, maintaining the controversy.

In a study in dogs1 assessing the effects of interpleural bupivacaine on respiratory muscle function, diaphragmatic electromyographic activity was markedly diminished. However, this report had several limitations. First, the validity of the model may be questioned: upper abdominal surgery, which can induce diaphragm dysfunction,23 was performed. In addition, there are important differences between dogs and humans in anatomic position and thorax shape.2 Second, because only the electromyographic activity from the costal diaphragm was measured and because the crural and costal diaphragm are considered two different muscles,24 the results may have been biased. Moreover, the electromyogram reflects electric activity; it does not measure muscle strength.12,25 Third, the strength of inspiratory muscles was evaluated only partially, because maximal inspiratory pressures were not recorded. Finally, expiratory muscles were not considered at all. Thus, we believe it important to clarify the effect of interpleural blockade on humans with normal respiratory function.

In the current study, several points concerning method should be considered.

Because interpleural blockade is a relatively invasive technique, the ethical aspects of this study were considered. Therefore, in all instances, the catheter was used for intra- and postoperative analgesia, and there was no placebo group. Thus, the patients acted as their own control. In addition, the study was designed to be performed entirely before surgery to avoid any confounding effects from other factors (such as pain, surgery, and anesthetics).

We evaluated the effects of interpleural bupivacaine on respiratory function in conditions similar to those during its use for postoperative analgesia. Respiratory parameters were recorded, when possible, with the patient supine, because the anesthetic is usually administered to supine patients, and patients remain supine in the postoperative period. Moreover, physiologic respiratory maneuvers (i.e., cough) were used together with classic maneuvers. Finally, the volume and doses of local anesthetics were those most commonly used in clinical practice.

Analysis of Results

Parameters from forced spirometry and static lung volumes did not change after interpleural bupivacaine. This finding is consistent with the hypothesis that there was no serious impairment in respiratory muscle function, although to support these results more specific indicators of respiratory muscle strength, such as maximal respiratory pressures,12 were used.
Table 3. Effects of Interpleural Bupivacaine on Respiratory Function (Supine)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before Bupivacaine</th>
<th>After Bupivacaine</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (L)</td>
<td>2.6 ± 0.2 (87 ± 3%)</td>
<td>2.5 ± 0.2 (84 ± 3%)</td>
<td>NS</td>
</tr>
<tr>
<td>FEV₁/FVC (%)</td>
<td>80 ± 1</td>
<td>78 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>SGaw (1/kPa · s)</td>
<td>0.8 ± 0.06</td>
<td>0.7 ± 0.08</td>
<td>NS</td>
</tr>
<tr>
<td>TLC (L)*</td>
<td>4.4 ± 0.2 (103 ± 3%)</td>
<td>4.5 ± 0.3 (103 ± 2%)</td>
<td>NS</td>
</tr>
<tr>
<td>RV (L)*</td>
<td>1.5 ± 0.2 (93 ± 8%)</td>
<td>1.4 ± 0.2 (94 ± 9%)</td>
<td>NS</td>
</tr>
<tr>
<td>RR (min⁻¹)</td>
<td>15 ± 1</td>
<td>19 ± 1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Vt (L)</td>
<td>0.5 ± 0.05</td>
<td>0.48 ± 0.05</td>
<td>NS</td>
</tr>
<tr>
<td>VE (L)</td>
<td>7.6 ± 0.84</td>
<td>8.3 ± 1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Ti (s)</td>
<td>1.38 ± 0.08</td>
<td>1.24 ± 0.07</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ttot (s)</td>
<td>4 ± 0.3</td>
<td>3.3 ± 0.2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ti/Ttot</td>
<td>0.35 ± 0.01</td>
<td>0.38 ± 0.01</td>
<td>NS</td>
</tr>
<tr>
<td>Vt/Ti (L/s)</td>
<td>0.37 ± 0.04</td>
<td>0.41 ± 0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Pes (cmH₂O)</td>
<td>-5.62 ± 0.69</td>
<td>-5.23 ± 0.43</td>
<td>NS</td>
</tr>
<tr>
<td>Pga (cmH₂O)</td>
<td>1.7 ± 0.3</td>
<td>1.44 ± 0.22</td>
<td>NS</td>
</tr>
<tr>
<td>Pdi (cmH₂O)</td>
<td>7.32 ± 0.77</td>
<td>6.67 ± 0.51</td>
<td>NS</td>
</tr>
</tbody>
</table>

FVC = forced vital capacity; FEV₁/FVC = forced expiratory volume in 1 s/FVC ratio; TLC = total lung capacity; RV = residual volume; SGaw = airway conductance; RR = respiratory rate; Vt = tidal volume; VE = expired minute volume; Ti = inspiratory time; Ttot = total respiratory time; Pes = mean esophageal pressure at Vt; Pga = mean gastric pressure at Vt; Pdi = mean transdiaphragmatic pressure at Vt; %p = % of the predicted value; NS = not significant.

Values are mean ± SEM. Student's t tests for paired data are used to compare the parameters.

* Compared with sitting position.

Airway conductance also remained unchanged in our study. This finding and the absence of changes in dynamic lung volumes disagree with the hypothesis that interpleural bupivacaine may cause bronchospasm through sympathetic blockade in healthy persons. However, to rule out this possibility, further studies are needed, especially in patients with a predisposition to airway reactivity.

With reference to inspiratory muscles, Pimax and Pesₘₘₙ were unchanged after bupivacaine. However, Pdiₘₘₙ, which specifically expresses the strength of the diaphragm, slightly decreased. This finding would indicate an impairment in the strength of this muscle. Nevertheless, the decrease in Pdiₘₘₙ was caused completely by a decrease in Pgaₘₘₙ, and this parameter reflects the abdominal pressure changes attributable to

Table 4. Effects of Interpleural Bupivacaine on Respiratory Muscle Strength (Supine)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before Bupivacaine</th>
<th>After Bupivacaine</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspiratory muscles</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pimax (cmH₂O)</td>
<td>-71 ± 7 (107 ± 10%)</td>
<td>-67 ± 8 (99 ± 10%)</td>
<td>NS</td>
</tr>
<tr>
<td>Pesₘₘₙ (cmH₂O)</td>
<td>-77.6 ± 5.2</td>
<td>-73.6 ± 5.1</td>
<td>NS</td>
</tr>
<tr>
<td>Pgaₘₘₙ (cmH₂O)</td>
<td>24.2 ± 7.4</td>
<td>18.4 ± 6.8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Pdiₘₘₙ (cmH₂O)</td>
<td>102 ± 10</td>
<td>92 ± 10</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TTDi</td>
<td>0.073 ± 0.005</td>
<td>0.079 ± 0.009</td>
<td>NS</td>
</tr>
<tr>
<td>Expiratory muscles</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PEmₘₘₙ (cmH₂O)</td>
<td>104 ± 12 (74 ± 9%)</td>
<td>100 ± 13 (72 ± 10%)</td>
<td>NS</td>
</tr>
<tr>
<td>Pesₘₘₙ (cmH₂O)</td>
<td>92 ± 13</td>
<td>78 ± 10</td>
<td>0.074</td>
</tr>
<tr>
<td>Pgaₘₘₙ (cmH₂O)</td>
<td>108 ± 10</td>
<td>92 ± 8</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Pimax = maximal inspiratory pressure (at mouth); Pesₘₘₙ = maximal inspiratory esophageal pressure; Pgaₘₘₙ = maximal inspiratory gastric pressure; Pdiₘₘₙ = maximal transdiaphragmatic pressure; TTDi = tension-time index of the diaphragm; PEmₘₘₙ = maximal expiratory pressure (at mouth); Pesₘₘₙ = maximal expiratory esophageal pressure; Pgaₘₘₙ = maximal expiratory gastric pressure; %p = % of the predicted value; NS = not significant.

Values are mean ± SEM. Student's t tests for paired data are used to compare the parameters.
the caudal displacement of the diaphragm. This decrease in Pgasalv may be related to increased abdominal compliance caused by the decreased motor tone. Because Pimax and Pesup remained unaltered and both parameters evaluate the global inspiratory muscle strength, changes in Pdi may be considered irrelevant regarding inspiratory function. The tension-time index of the diaphragm also remained unchanged. Thus, the risk of diaphragmatic fatigue is not increased in healthy adults receiving interpleural bupivacaine. However, these results cannot be extrapolated to cases of patients at increased risk of diaphragmatic fatigue, such as patients with severe chronic obstructive pulmonary disease.

Maximal inspiratory maneuvers also suggested that there was a degree of motor blockade of the abdominal wall muscles, manifested as a decreased Pgasalv. Because abdominal expiratory effort is transmitted to the thorax, Pgasalv showed a trend to decrease. This finding appears to be clinically unimportant in healthy subjects because the magnitude of the changes was small and because PEmax, which closely indicates the effective expulsive efforts performed with all the expiratory muscles, remained unmodified. However, these effects may be more important in patients with obstructive airways diseases, who frequently need the recruitment of abdominal muscles.

The increase in respiratory rate and minute ventilation after interpleural bupivacaine, without changes in the remaining parameters of the breathing pattern and hemoglobin saturation, was an unexpected result. It may be attributable to central ventilatory effects of the absorbed local anesthetic, or to the absorbed epinephrine. On the other hand, mean arterial blood pressure slightly decreased, perhaps because of the sympathetic blockade induced by bupivacaine and the α-agonist effect of epinephrine, which also may explain the increase in heart rate. None of these mechanisms could be confirmed in this study.

Our results demonstrate that the effects of interpleural bupivacaine on the respiratory system are minimal if given with the patient supine. However, when local anesthetics are given to patients in the lateral decubitus, a similar degree of analgesia is obtained, but the safety of the technique in that position has not been clearly defined. In the lateral decubitus, the anesthetic spreads to the mediastinal pleural space, where it may induce a blockade of the phrenic nerve, which is in contact with the mediastinal pleura.

Finally, these results cannot be extrapolated to larger concentrations or volumes of bupivacaine or to continuous infusions.

In conclusion, interpleural bupivacaine, when administered preoperatively to healthy supine subjects, does not significantly impair lung or inspiratory muscle function. Bupivacaine produces a slight decrease in the strength of abdominal muscles, probably because of the motor block it induces. Although this impairment is small and does not reflect the effective expulsive pressures, its clinical relevance in the postoperative period is unknown, especially in patients with respiratory or neuromuscular diseases.

The authors thank Dr. O. Pol and Dr. J. Valls for their help with the statistical evaluation of the results.

References


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