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Lung Function under High Thoracic Segmental Epidural Anesthesia with Ropivacaine or Bupivacaine in Patients with Severe Obstructive Pulmonary Disease Undergoing Breast Surgery

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Background: **Because general anesthesia with tracheal intubation can elicit life-threatening bronchospasm in patients with bronchial hyperreactivity, epidural anesthesia is often preferred. However, segmental high thoracic epidural anesthesia (sTEA) causes pulmonary sympathetic and respiratory motor blockade. Whether it can be safely used for chest wall surgery as a primary anesthetic technique in patients with chronic obstructive pulmonary disease or asthma is unclear. Furthermore, ropivacaine supposedly evokes less motor blockade than bupivacaine and might minimize side effects. To test the feasibility of the technique and the hypotheses that (1) sTEA with ropivacaine or bupivacaine does not change lung function and (2) there is no difference between sTEA with ropivacaine or bupivacaine, the authors studied 20 patients with severe chronic obstructive pulmonary disease (forced expiratory volume in 1 s [FEV_1] = 52.1 ± 17.3% of predicted [mean ± SD]) or asthma who were undergoing breast surgery.**

Methods: **In a double-blind, randomized fashion, sTEA was performed with 6.6 ± 0.5 ml of either ropivacaine, 0.75% (n = 10), or bupivacaine, 0.75% (n = 10). FEV_1, vital capacity, FEV_1 over vital capacity, spread of analgesia (pin prick), hand and foot skin temperatures, mean arterial pressure, heart rate, and local anesthetic plasma concentrations were measured with patients in the sitting and supine positions before and during sTEA.**

Results: **Segmental high thoracic epidural anesthesia (segmental spread C4–T8 [bupivacaine] and C5–T9 [ropivacaine]) significantly decreased FEV_1 from 1.22 ± 0.54 l (supine) to 1.09 ± 0.56 l (ropivacaine) and from 1.23 ± 0.49 l to 1.12 ± 0.46 l (bupivacaine). In contrast, FEV_1 over vital capacity increased from 64.6 ± 13.5 to 68.2 ± 14.5% (ropivacaine) and from 62.8 ± 12.4 to 66.5 ± 13.6% (bupivacaine). There was no difference between ropivacaine and bupivacaine. Skin temperatures increased significantly, whereas arterial pressure and heart rate significantly decreased indicating widespread sympathetic blockade. All 20 patients tolerated surgery well.**

Conclusions: **Despite sympathetic blockade, sTEA does not increase airway obstruction and evokes only a small decrease in FEV_1 as a sign of mild respiratory motor blockade with no difference between ropivacaine and bupivacaine. Therefore, sTEA can be used in patients with severe chronic obstructive pulmonary disease and asthma undergoing chest wall surgery as an alternative technique to general anesthesia.**

PATIENTS with asthma or chronic obstructive pulmonary disease (COPD) share a high incidence of bronchial hyperreactivity.[1,2] General anesthesia with tracheal intubation can elicit bronchospasm, which, in some cases, can be life-threatening in these patients.[3,4]

Accordingly, regional anesthesia techniques are commonly used to avoid the strong stimulus of tracheal intubation. However, high thoracic epidural anesthesia bears the inevitable side effects of both pulmonary sympathetic and respiratory motor blockade, possibly precluding its use in these patients.[5]

However, potential increases in bronchial tone and bronchial reactivity by pulmonary sympathetic blockade may be outweighed by the systemic effect of bupivacaine, which, like other amide local anesthetics, decreases bronchial reactivity.[6] Moreover, forced expiratory volume in 1 s (FEV_1) and vital capacity (VC) have been reported to decrease by less than 10% in patients with obstructive pulmonary disease.[6]

However, whether high thoracic epidural anesthesia can be used in patients with severe COPD or asthma as a sole anesthesia technique is unclear. Furthermore, it is unknown whether epidural anesthesia with ropivacaine, which supposedly causes less motor blockade than bupivacaine,[7–9] offers advantages when used for high segmental thoracic epidural anesthesia (sTEA).

Accordingly, in patients with severe COPD or asthma scheduled to undergo breast surgery with or without axillary lymph node dissection, we evaluated the feasibility of sTEA as a sole anesthesia technique and its effect on lung function. Finally, we tested the hypothesis that there is no difference in the effect on lung function of ropivacaine and bupivacaine.

Materials and Methods

Patients

After institutional approval and the patients' informed written consent, 20 unpremedicated women (American Society of Anesthesiologists physical status class II or III) scheduled to undergo elective breast surgery were consecutively enrolled in the study and randomly assigned to receive epidural anesthesia with either ropivacaine or bupivacaine. Surgery was indicated to treat breast carcinoma by partial or total mastectomy (n = 16) with (n = 4) or without (n = 12) axillary lymph node dissection or for plastic surgery (n = 4).

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Table 1. Disease, Current Medication, and Baseline Lung Function of 20 Patients Undergoing High Segmental Thoracic Epidural Anesthesia with Either Ropivacaine or Bupivacaine

	Patient	Age (yr)	Weight (kg)	Height (cm)	R_{tot} (mbar · l^{-1} · s)	FEV_1 (l)	FEV_{1pred} (l)	VC (l)	VC_{pred} (l)	FRC (l)	FRC_{pred} (l)	Disease	Medication
Ropivacaine	1	74	80	160	0.635	1.42	1.87	2.43	2.40	3.39	2.66	Asthma	2,5
	2	36	54	164	0.274	3.27	3.00	3.94	3.52	2.93	2.71	Asthma*	1,4,5
	3	67	49	163	1.000	0.66	2.19	1.66	2.73	5.03	2.72	COPD	5
	4	74	88	167	0.649	1.20	2.19	1.71	2.77	5.14	2.84	COPD	2,5,6
	5	63	78	170	0.528	1.82	2.54	2.74	3.13	3.53	2.87	Asthma	1,2,3
	6	37	62	158	0.303	2.02	2.72	3.43	3.19	2.89	2.58	Asthma	1,5,7
	7	69	57	168	0.777	0.82	2.34	1.93	2.92	3.43	2.83	COPD	4,5
	8	58	54	163	1.510	0.69	2.39	1.43	2.92	4.31	2.71	COPD	5
	9	77	64	156	0.873	1.28	1.61	2.05	2.12	2.70	2.57	COPD	5,6,7
	10	75	79	172	1.091	1.10	2.36	2.40	2.98	4.02	2.95	Asthma	2,4,5,6
	Mean	63	67	164	0.764	1.43	2.32	2.37	2.87	3.74	2.74		
	SD	15	14	5	0.375	0.79	0.40	0.81	0.40	0.87	0.13		
Bupivacaine	11	53	73	162	0.948	1.42	2.50	2.52	3.02	3.32	2.68	COPD	3,5,7
	12	57	84	167	0.932	1.65	2.57	3.03	3.13	3.37	2.80	COPD	5,7
	13	35	80	158	0.802	1.77	2.79	2.61	3.27	3.10	2.57	Asthma	2,4,5
	14	45	58	155	0.856	1.04	2.40	2.01	2.86	3.63	2.52	Asthma	2,4,5,7
	15	50	73	163	0.282	1.81	2.59	3.90	3.12	3.98	2.70	Asthma*	2,5
	16	38	84	161	0.512	1.96	2.81	3.08	3.31	4.38	2.64	COPD	
	17	65	64	162	1.110	0.91	2.15	1.41	2.69	3.94	2.69	COPD	3
	18	45	90	159	0.588	1.75	2.56	3.17	3.05	2.68	2.61	COPD	3
	19	77	97	167	0.753	0.88	2.05	1.60	2.63	5.52	2.82	Asthma	1,2,3,4,5,7
	20	58	56	168	1.380	0.72	2.64	1.90	3.20	3.57	2.82	Asthma	1,5,7
	Mean	52	76	162	0.816	1.39	2.51	2.52	3.03	3.75	2.69		
	SD	13	14	4	0.311	0.46	0.25	0.79	0.23	0.79	0.11		
	P value	0.11	0.14	0.38	0.74	0.90	0.23	0.68	0.29	0.98	0.27		

$P < 0.05$ indicating significant difference between ropivacaine and bupivacaine groups.

Medication: 1 = oral corticosteroids; 2 = corticosteroid, metered dose inhaler; 3 = oral theophylline; 4 = long-acting β_2-adrenergic agonist, metered dose inhaler; 5 = short-acting β_2-adrenergic agonist, metered dose inhaler; 6 = oral β_2-adrenergic agonist; 7 = anticholinergic, metered dose inhaler.

* Patients 5 and 12 had a history of severe bronchospasm after endotracheal intubation during general anesthesia.

R_{tot} = airway resistance (body plethysmography); FEV_1 = forced expiratory volume in 1 s; VC = vital capacity; FRC = functional residual capacity; pred = predicted value; COPD = chronic obstructive pulmonary disease.

All patients had a history of severe COPD or asthma with a history of severe bronchial hyperreactivity.[10] Two patients had a history of severe bronchospasm complicating general anesthesia with tracheal intubation. COPD was diagnosed by a history of productive coughing, dyspnea attacks, wheezing lasting longer than 2 yr, and abnormal lung function tests (airway resistance 0.89 ± 0.29 mbar · l^{-1} · s and forced expiratory volume in 1 s (FEV_1) of $52.1 \pm 17.3\%$ of predicted; mean ± SD). Patients' underlying disease, current medication, anthropometric data, and preoperative lung function data are presented in table 1. There were no significant differences between the ropivacaine and bupivacaine groups.

Measurements

One to 7 days before surgery, all patients underwent lung function measurements in sitting position to evaluate baseline lung function by body plethysmography (Masterlab; Jaeger, Würzburg, Germany). On the day of surgery, FEV_1 and VC were obtained using the same pneumotachograph (Jaeger). FEV_1 and VC were always measured three times with the best result chosen for further analysis. All measurements on the day of surgery were performed with the patient in supine position.

Arterial oxygen saturation (pulse oximetry, Nellcor; Nellcor Inc., Hayward CA), heart rate (electrocardiographic lead II), mean arterial pressure (oscillometry), skin temperatures on hand and foot (infrared telethermometry, Bio-Therm C-600 M; Linear Laboratories, Los Altos, CA), and room temperature (mercury thermometer) were also measured.[11] To measure ropivacaine and bupivacaine plasma concentrations, blood was drawn from an antecubital vein. Ropivacaine and bupivacaine were measured by high-pressure liquid chromatography (Waters 2690; Waters, Eschborn, Germany; photo diode array detector, spectrophotometric election at 200 nm; lower level of detection 0.01 μg/ml; coefficient of variation less than 0.5%).

Study Protocol

On the morning of surgery, a peripheral venous cannula was placed on the forearm of the nonsurgical site for blood sampling and infusion of Ringer's' lactate. A pulse oximeter, an electrocardiograph, and a blood pressure cuff were applied. Epidural catheterization was performed in the T2–T4 interspace with the patient in sitting position using the "loss of resistance" technique with air and the catheter advanced 4–6 cm into the epidural space.

After values, including FEV_1 and VC, had been recorded for 15 min with the patient in the supine position, venous blood samples were drawn for measurement of ropivacaine or bupivacaine plasma concentrations.

Thereafter, 10 patients each received epidurally either ropivacaine or bupivacaine, 0.75%, the dose adjusted to each patient's height (mean doses: 6.6 ± 0.5 ml for bupivacaine and 6.6 ± 0.5 ml for ropivacaine). Sensory blockade was assessed by pin prick every 5 min for up to 35 min after epidural injection. Borders were defined as the most cranial and the most caudal dermatomes unresponsive to the stimulus.

Heart rate, mean arterial pressure, and skin temperatures on the thumb and fifth toe were measured before and every 5 min after epidural injection for up to 35 min.

Thirty-five minutes after epidural injection, *i.e.*, after full spread of epidural anesthesia, lung function tests were repeated, and venous blood was drawn for measurements of ropivacaine or bupivacaine plasma concentrations.

Subsequently, surgery was started, and the patients received mild sedation with continuous infusion of propofol or 1-mg boluses of midazolam, if desired. The need for additional analgesic medication, development of dyspnea, and tolerance of regional anesthesia were recorded. Epidural anesthesia was maintained with injections of ropivacaine or bupivacaine (0.375%). The epidural catheter was removed at the end of the day of surgery.

Statistical Analysis

Data are presented as mean ± SD. Power analysis was based on repeated measurements, with an α error of 5% and a β error of 20%. Two *a priori* null hypotheses were tested: (1) there is no difference in FEV_1, VC, heart rate, blood pressure, and temperatures before and 35 min after epidural injection of bupivacaine and ropivacaine; and (2) there is no difference between the effect of ropivacaine and bupivacaine on FEV_1 and VC. Hypotheses were tested with one- and two-way analysis of variance followed by *post hoc t* test with Bonferroni correction for multiple comparisons. Null hypotheses were rejected, and significance was assumed with $P < 0.05$.

Results

Segmental high thoracic epidural anesthesia led to significant decrease in both FEV_1 and VC ($P < 0.0001$) compared with the supine position, with a small but significant increase ($P = 0.0005$) in the ratio of FEV_1 over VC. There was no difference between the effects of ropivacaine and bupivacaine.

Effect of Change in Posture

Changing posture from sitting to supine decreased FEV_1 from 1.43 ± 0.8 l to 1.22 ± 0.5 l in the patients later receiving ropivacaine and from 1.39 ± 0.5 l to 1.23 ± 0.5 l in those of the bupivacaine group. A similar effect was detected for VC, *i.e.*, a decrease from 2.37 ± 0.8 l to 1.87 ± 0.7 l (ropivacaine group; $P = 0.0005$) and from 2.52 ± 0.8 l to 1.92 ± 0.6 l (bupivacaine group; $P = 0.0005$), respectively (fig. 1). The ratio of FEV_1 over VC significantly improved from 57.6 ± 13.7% (sitting) to 64.6 ± 13.5% (supine before sTEA) in the ropivacaine group and from 55.3 ± 8.9% to 62.8 ± 12.3% in the bupivacaine group, respectively). There was no difference between the ropivacaine and bupivacaine groups ($P = 0.6918$).

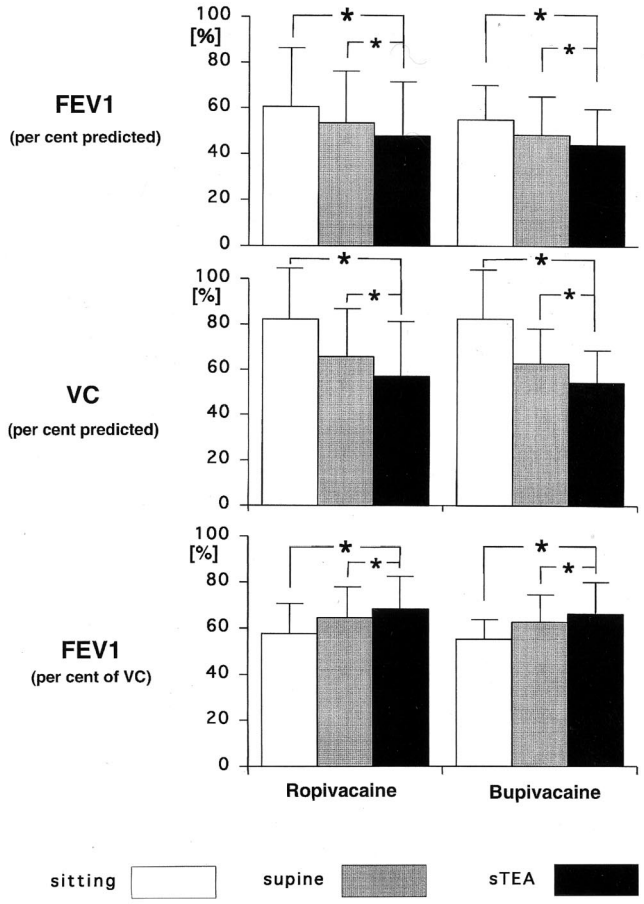

Fig. 1. Forced expiratory volume in 1 s (FEV_1), vital capacity (VC) (percent predicted), and ratio of FEV_1 over VC (percent) of 20 patients with chronic obstructive pulmonary disease or asthma in sitting (open bars) and in supine position before (gray bars) and during (black bars) epidural anesthesia with either ropivacaine (*left*, n = 10) or bupivacaine (*right*, n = 10). Mean ± SD; *$P < 0.05$. VC and FEV_1 decreased significantly both when attaining supine position and during high thoracic segmental epidural anesthesia. As a measure of airway obstruction, FEV_1 as a percentage of VC increased significantly when attaining supine position and during segmental high thoracic epidural anesthesia (sTEA).

Effect of Segmental Thoracic Epidural Anesthesia on Lung Function

Segmental high thoracic epidural anesthesia decreased FEV_1 significantly from 1.22 ± 0.5 l to 1.09 ± 0.2 l for ropivacaine ($P = 0.0001$) and from 1.23 ± 0.5 l to

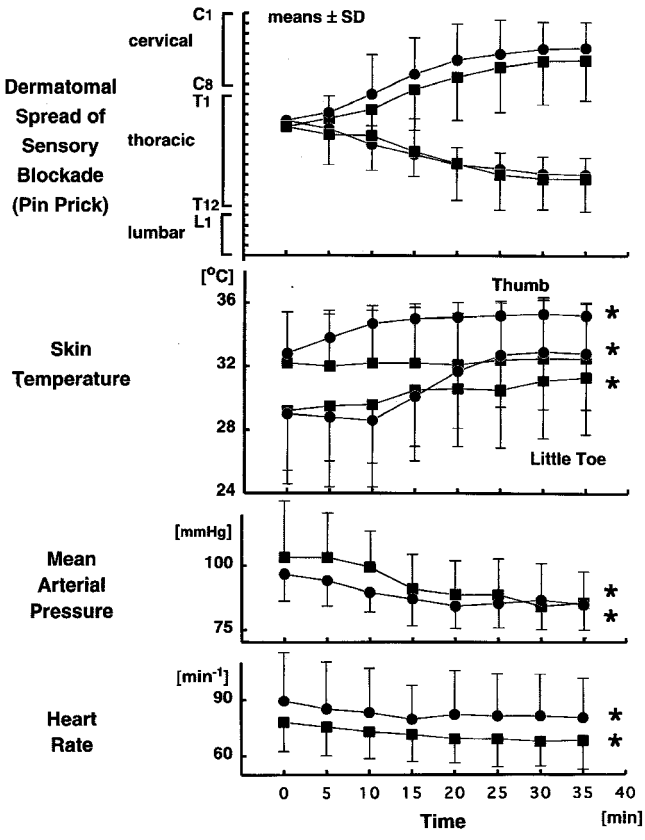

Fig. 2. Time course of dermatomal spread of sensory blockade (pin prick) along the spinal axis, skin temperatures on thumb and little toe, mean arterial blood pressure, and heart rate before and during high segmental thoracic epidural anesthesia. Patients were randomized to receive either epidural ropivacaine (squares, n = 10) or bupivacaine (circles, n = 10) injection. As an indicator of widespread sympathetic blockade, skin temperature increased significantly. Epidural anesthesia with both local anesthetics led to significant decrease in mean arterial blood pressure and heart rate. Mean ± SD; *$P < 0.05$.

1.12 ± 0.5 l for bupivacaine ($P = 0.0019$) in supine position (fig. 1). VC decreased during sTEA from 1.87 ± 0.7 l to 1.63 ± 0.7 l ($P = 0.0022$) for ropivacaine and from 1.92 ± 0.6 l to 1.66 ± 0.5 l ($P = 0.0005$) for bupivacaine, respectively (fig. 1).

Whereas absolute numbers of FEV_1 and VC both decreased with sTEA, the ratio of FEV_1 over VC significantly improved with sTEA, from 64.6 ± 13.5% (supine) to 68.5 ± 14.2% for ropivacaine ($P = 0.0003$) and from 62.8 ± 12.3% (supine) to 66.5 ± 13.8% for bupivacaine ($P = 0.0003$), respectively. There was no difference between the effects of ropivacaine and bupivacaine ($P = 0.9419$).

Spread of Segmental Thoracic Epidural Anesthesia

The time course of sensory blockade after epidural administration of ropivacaine or bupivacaine, along with skin temperatures, mean arterial pressure, and heart rate, is shown in figure 2. Thirty-five minutes after epidural injection of ropivacaine, sensory blockade averaged 12 ± 3 segments, with a cranial border of C5 ± 3 and a caudal border of T9 ± 3. Epidural injection of bupivacaine evoked a sensory blockade of 13 ± 1 segments, with a cranial border of C4 ± 1 and a caudal border of T8 ± 1.

As an indicator of sympathetic blockade, skin temperatures increased ($P < 0.0001$) after epidural bupivacaine on the thumb (spinal segment C6) and on the little toe (S1) ($P < 0.0001$). Ropivacaine increased skin temperature on the little toe ($P = 0.0046$) but not on the thumb ($P = 0.5924$). Room temperature averaged 22.7 ± 1.2°C.

Mean arterial pressure decreased significantly after ropivacaine ($P < 0.0001$) and bupivacaine ($P < 0.0001$), whereas heart rate significantly diminished (ropivacaine, $P = 0.002$; bupivacaine, $P < 0.0001$). Oxygen saturation remained unchanged.

Plasma Concentrations of Local Anesthetics

Thirty-five minutes after epidural injection of ropivacaine or bupivacaine, mean plasma concentrations were 0.75 ± 0.71 μg/ml and 0.50 ± 0.49 μg/ml, respectively.

Surgery

Surgery lasted 85 ± 38 min in the ropivacaine group and 97 ± 50 min in the bupivacaine group. Including the initial bolus, the patients received a total dose of 114 ± 32 mg ropivacaine or 107 ± 26 mg bupivacaine for epidural anesthesia. During breast surgery, patients were slightly sedated with either propofol (58 ± 36 mg/h in the ropivacaine group and 86 ± 16 mg/h in the bupivacaine group) or with midazolam (2 ± 1 mg/h in the ropivacaine group and 2 ± 2 mg/h in the bupivacaine group, respectively). Two patients declined any sedation. None of the patients required supplemental analgesics or reported dyspnea, and high thoracic epidural anesthesia did not have to be converted to general anesthesia in any patient.

Discussion

High thoracic segmental epidural anesthesia with either ropivacaine or bupivacaine slightly decreased FEV_1 and VC compared with baseline in supine position. Although ropivacaine is believed to cause lesser motor blockade than bupivacaine, no difference between the effects of the two local anesthetics was found. Furthermore, despite severe COPD or asthma, surgery was well-tolerated by all patients during high thoracic segmental epidural anesthesia.

Half of the enrolled patients had asthma with, despite intense antiobstructive treatment, persisting diminution of FEV_1, increased airway resistance, and evidence of increased bronchial reactivity. Two of the asthma patients also had a recent history of life-threatening bronchospasm after tracheal intubation for a surgical procedure and requiring prolonged mechanical ventilation

and intensive care treatment. The other half of the patients had severe COPD with a mean FEV_1 of 52.1 ± 17.3% of predicted, including four patients with an FEV_1 of less than 1 l.

In patients free of obstructive pulmonary disease, thoracic or cervical epidural anesthesia show evidence of only slight respiratory motor blockade, *i.e.*, approximately a 10% decrease of FEV_1 and VC and no increase in airway resistance.[12,13] Therefore, sTEA has been used for chest wall surgery in patients without obstructive pulmonary disease.[14,15] In patients with obstructive pulmonary disease, however, the effects of sTEA for chest wall surgery have never been evaluated, and there is no proof that sTEA can be used safely as the sole anesthetic technique for chest wall surgery in patients with severe obstructive pulmonary disease. Furthermore, the effects on pulmonary function of ropivacaine during sTEA, with its potential advantage of less motor blockade compared with bupivacaine,[7–9] have not been addressed. Accordingly, we evaluated the effect on lung function of sTEA with either ropivacaine or bupivacaine in patients with obstructive pulmonary disease.

Attaining the resting supine position alone decreased FEV_1 and VC by up to 23%. During sTEA, FEV_1 and VC decreased further by an additional 12 and 14% for ropivacaine and by 9 and 16% for bupivacaine, respectively. Thus, the effect of sTEA on pulmonary function is even smaller than the effect of the change in position. Furthermore, it is similar to the effect described in patients free of pulmonary disease as well as in patients with bronchial hyperreactivity or COPD.[6] The fact that the ratio of FEV_1 over VC significantly improved during sTEA indicates that the decrease of FEV_1 and VC is due to mild motor blockade of respiratory musculature rather than to increased airway obstruction.

Several studies have addressed the relation of the degree of motor blockade to the concentrations of epidural ropivacaine and bupivacaine. In some of the studies, a significant lesser degree and duration of motor blockade of the lower extremities was found when equal concentrations of ropivacaine and bupivacaine were injected epidurally.[7–9] Accordingly, epidural injection of 0.75% ropivacaine *versus* 0.75% bupivacaine might also evoke differences in pulmonary function during sTEA. However, no such difference was found, although this might be explained by the fact that differences between ropivacaine and bupivacaine effects are small and that the number of patients was to small to detect a difference, if present. However, even studies comparing effects on motor blockade under lumbar epidural anesthesia in a larger sample have not detected any differences.[16] Furthermore, FEV_1 and VC are indirect estimates of muscle strength and might not be sensitive enough to detect small differences, if present. However, even if there were undetected differences in muscle strength, they obviously can be considered clinically negligible for chest wall surgery. Overall, epidural injection of both local anesthetics led to a mild decrease in FEV_1 and VC and was well-tolerated by all patients, with none of the patients reporting dyspnea during sTEA and surgery.

These results were determined when sensory blockade extended from C5 to T9 after epidural ropivacaine and from C4 to T8 after bupivacaine, with no differences between groups. Moreover, the increase in skin temperature and the decrease in mean arterial blood pressure and heart rate, as indicators of sympathetic blockade, suggest for both local anesthetics a widespread sympathetic blockade. Considering that pulmonary sympathetic efferents emerging from the spinal cord between T2 and T7, pulmonary sympathetic blockade can be assumed.[17] Accordingly, the balance between parasympathetic bronchoconstrictor tone and sympathetic bronchodilation might be shifted toward a higher bronchomotor tone, an increased bronchial reactivity, or both, as suggested by a study of patients with high paraplegia and several case reports.[18–21] However, direct sympathetic innervation of the bronchial musculature in humans is only rudimental, and the bronchial tone is preferentially determined by β_2-adrenergic receptors stimulated by circulating catecholamines.[22–24]

Therefore, sTEA alone increases neither airway resistance at rest nor bronchial reactivity to acetylcholine.[3,25] In fact, bronchial reactivity to acetylcholine is attenuated by amid local anesthetics absorbed from the epidural space.[3,26] In volunteers with bronchial hyperreactivity, both bupivacaine and lidocaine dose dependently attenuate bronchial hyperreactivity.[26] Therefore, with a bupivacaine plasma concentration of 0.50 ± 0.49 μg/ml, significant attenuation of bronchial reactivity can be expected.[26] A possible bronchodilatory effect of sTEA is indicated by a significant, albeit slight, increase of the ratio of FEV_1 over VC and supports the hypothesis that the overall decrease in absolute FEV_1 and VC is due to mild motor blockade rather than increased bronchial tone.[27]

In summary, in patients with severe COPD or asthma, sTEA with ropivacaine or bupivacaine, mildly decreasing FEV_1 and VC, can be used as the primary anesthesia technique for chest wall surgery and is well-tolerated. Furthermore, we did not find a difference in lung function between the two local anesthetics when yielding a similar spread of sensory blockade.

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