Circulatory Responses to Airway Stimulation and Cervical Epidural Blockade

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In order to investigate the effects of acute cardiac sympathectomy, the circulatory responses due to tracheal intubation and broncho-carinal stimulation were compared in lightly anesthetized, paralyzed patients who had either cervical (n = 18) or lumbar epidural blockade (n = 18), or neither (n = 12). The mean analgesic levels obtained using 2% mepivacaine with epinephrine were C4–T8 in the cervical group, and T7–S1 in the lumbar group.

Increases in arterial blood pressure (AP) and heart rate (HR) due to laryngoscopy were significant (P < 0.01) but similar in the three groups of patients. Broncho-carinal stimulation by a suction catheter did not produce noticeable changes in AP and HR in the cervical group; significant increases were observed in the other two groups (P < 0.01). It was concluded from this study that acute sympathectomy induced in part by cervical or lumbar epidural block does not attenuate circulatory responses due to laryngoscopy and tracheal intubation. The cervical blockade, however, can prevent such responses due to broncho-carinal stimulation without predominate vagotonic reflexes; this may be attributed to the interruption of sympathetic afferents to the spinal cord at the epidural level. (Key words: Anesthetic techniques: epidural, cervical. Heart: heart rate; blood pressure. Sympathetic nervous system: acute sympathectomy. Complications: tracheal intubation; broncho-carinal stimulation.)

It is well-known that stimulation of the airway, such as by direct laryngoscopy, tracheal intubation and carinal irritation, affects the cardiovascular system, although the mechanism(s) involved is not clear. Experimentally, mechanical stimulation of the airways has also been reported to excite various cardiovascular and respiratory reflexes. In a study by Tomori and Widdicombe of paralyzed, mechanically ventilated cats, stimulation of the laryngeal and tracheal regions caused a reflex increase in systemic blood pressure associated with reflex tracheo-bronchial constriction. This has also been confirmed in a recent clinical study by Dohi and Gold in anesthetized humans. These observations suggest that stimulation of the airways can cause a sympathomimetic and a parasympathomimetic reflex simultaneously.

Upper thoracic epidural blockade will interrupt cardiac afferent and efferent neural impulses at the level of the spinal cord, but the parasympathetic reflexes that do not involve the spinal cord remain intact. In addition, afferent input from the lung and airway travels along both the vagus and the sympathetic nerves to the upper four or five thoracic segments; the latter can also be blocked by upper thoracic epidural blockade with minimum cardiovascular changes.

Therefore, acute sympathectomy produced by cervical epidural blockade could diminish the deleterious circulatory effects of airway manipulation, or may elicit a profound parasympathetic response. To examine these questions, we compared the circulatory responses to tracheal intubation and carinal stimulation in anesthetized patients who had either cervical or lumbar epidural blockade, or neither.

Materials and Methods

Forty-eight patients, who were scheduled to have general anesthesia plus cervical or lumbar epidural anesthesia or general anesthesia alone for their surgical procedures, were selected for this study. The protocol was approved by the Institutional Human Studies Committee and informed consent was obtained from each patient. All patients selected did not have cardiopulmonary or neurological disorders. Premedication with 1.2–1.4 mg/kg hydroxyzine and 0.4–0.5 mg atropine was administered by intramuscular injection one hour before arrival in the operating room. An intravenous cannula was placed for infusion of lactated Ringer’s solution (8 ml · kg⁻¹ · 30 min⁻¹) during the study. A radial artery catheter was inserted to permit continuous recording of arterial blood pressure and electronic calculation of heart rate. Thirty-six of these patients had either cervical (cervical group, n = 18) or lumbar epidural anesthesia (lumbar group, n = 18); the rest had no epidural block and served as the control (control, n = 12).

Following sterile preparation and draping of each patient in the lateral decubitus position, a local infiltration of lidocaine (1%, 2–3 ml) was injected intradermally and subcutaneously at C7–T1 intervertebral space for the cervical epidural group or at L1–2 for the lumbar group. A 17-gauge Touhy needle was then inserted into the epidural space using the loss-of-resistance technique. Following identification of the epidural space, 8–10 ml of 2% mepivacaine with freshly added epinephrine (1/200,000) was injected into the space.
Table 1. Mean Values of Age, Body Weight, Area of Sensory Block, and Arterial Blood-gas Analyses during Control Ventilation in the Three Groups of Patients

<table>
<thead>
<tr>
<th></th>
<th>Age (yr)</th>
<th>Body Weight (kg)</th>
<th>Segmental Area of Sensory Block</th>
<th>P_ACO2 (mmHg)</th>
<th>P_Ao2 (mmHg)</th>
<th>pH</th>
<th>BE (mEq/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical epidural (n = 18)</td>
<td>44 ± 9</td>
<td>53 ± 10</td>
<td>C3.8 ± 0.8–7.7 ± 1.5</td>
<td>181 ± 34</td>
<td>34 ± 3</td>
<td>7.46 ± 0.05</td>
<td>0.4 ± 2.4</td>
</tr>
<tr>
<td>Lumbar epidural (n = 18)</td>
<td>41 ± 8</td>
<td>53 ± 8</td>
<td>T7.0 ± 1.6–S1.1 ± 1.8</td>
<td>181 ± 24</td>
<td>33 ± 4</td>
<td>7.45 ± 0.04</td>
<td>0.3 ± 2.1</td>
</tr>
<tr>
<td>Control (n = 12)</td>
<td>44 ± 12</td>
<td>58 ± 5</td>
<td>–</td>
<td>180 ± 24</td>
<td>34 ± 3</td>
<td>7.47 ± 0.04</td>
<td>1.0 ± 2.8</td>
</tr>
</tbody>
</table>

Values are means ± SD.

over 45 s and was followed by the placement of an epidural catheter for continuous blockade. The patient then was placed in the supine position. The level of analgesia was measured using a pin-prick method 15 min after the injection.

After the establishment of either cervical or lumbar epidural anesthesia, general anesthesia was induced intravenously with 4 mg/kg thiamylal following 1 mg pancuronium. Succinylcholine chloride, 1.5 mg/kg, was administered to facilitate the insertion of an endotracheal tube. Manual ventilation with oxygen was instituted after loss of the eyelid reflex. Direct laryngoscopy with a curved blade and the placement of a cuffed tracheal tube were performed by one of the authors (S.D.) and was intended to require approximately 30 s in all patients studied. No aqueous lidocaine was sprayed into the larynx and trachea. Following proper placement, the cuff was inflated with air until no gas leak around the tube was audible. Control patients also were anesthetized with thiopental and managed in an identical manner. Each patient then was given 2 L/min oxygen and 4 L/min nitrous oxide via a semiclosed circle with a carbon dioxide absorption system. This was supplemented by a total dose of 100 μg fentanyl after tracheal intubation. Muscle paralysis was maintained with 0.08 mg/kg intravenous pancuronium chloride, and the lungs were ventilated mechanically. The end-tidal carbon dioxide concentration was maintained at approximately 4.5% with the use of an infrared analyzer (Beckman® LB 2).

After stabilization of blood pressure and heart rate following intubation (approximately 10 min), a suction catheter was inserted into the tracheal tube until the tip was about 40 cm from the orifice of the tube. The broncho-carinal regions were stimulated by advancing and retracting the catheter five times within seven seconds; the catheter was then withdrawn.

Data were analysed using Student's t test and by covariance analysis. Results are presented as means ± 1 SD with a statistically significant change being considered to have occurred when P values were 0.05 or less.

Results

There were no differences in age distribution, body weight, control blood pressure, and heart rate among the three groups of patients (tables 1 and 2). The mean analgesic levels obtained by epidural anesthesia were between C4 and T8 in the cervical epidural group, and between T7–S1 in the lumbar epidural group.

Laryngoscopy and tracheal intubation produced significant increases in blood pressure and heart rate in all patients studied. The magnitude of blood pressure increase with laryngoscopy and tracheal intubation was not significantly different among the three groups of patients (table 2). The maximum per cent changes in systolic blood pressure were 55 ± 15% in the cervical group, 56 ± 13% in the lumbar group, and 69 ± 14% in the control group. The changes in heart rate were also significant compared with values before laryngoscopy in the three groups of patients (P < 0.01) (table 2).

The responses to broncho-carinal stimulation were significantly different among the three groups. In the cervical group, broncho-carinal stimulation produced no change at all in blood pressure and heart rate in 12 patients, a slight decrease in two, and a slight increase in four patients, resulting in no statistically significant differences in either value from the pre-stimulation values (table 2). On the other hand, broncho-carinal stimulation caused significant increases in blood pressure and heart rate in all patients in the control and lumbar epidural groups. The magnitude of increases in blood pressure ranged from 10 to 45 mmHg in the control and from 5 to 32 mmHg in the lumbar group. In these groups, the increases in heart rate and blood pressure usually occurred simultaneously and returned to control values within 5–6 min. Figure 1 illustrates changes in blood pressure and heart rate with broncho-carinal stimulation in a patient with cervical epidural blockade (upper half of figure) and in a patient without blockade (lower half of figure). In the lumbar epidural and control groups, the responses in blood pressure and heart
TABLE 2. The Changes in Heart Rate (beats/min) and Systolic (SAP) and Diastolic (DAP) Arterial Blood Pressure (mmHg) following Tracheal Intubation and Carinal Stimulation

<table>
<thead>
<tr>
<th></th>
<th>Awake Control</th>
<th>Intubation</th>
<th>Carinal Stimulation</th>
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<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>Cervical (n = 18)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>85 ± 13</td>
<td>82 ± 13</td>
<td>105 ± 19*</td>
</tr>
<tr>
<td>SAP</td>
<td>127 ± 17</td>
<td>106 ± 14</td>
<td>167 ± 31*</td>
</tr>
<tr>
<td>DAP</td>
<td>65 ± 14</td>
<td>58 ± 9</td>
<td>95 ± 19*</td>
</tr>
<tr>
<td>Lumbar (n = 18)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>87 ± 14</td>
<td>85 ± 17</td>
<td>116 ± 19*</td>
</tr>
<tr>
<td>SAP</td>
<td>130 ± 20</td>
<td>111 ± 21</td>
<td>189 ± 29*</td>
</tr>
<tr>
<td>DAP</td>
<td>68 ± 11</td>
<td>64 ± 15</td>
<td>100 ± 21*</td>
</tr>
<tr>
<td>Control (n = 12)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>82 ± 14</td>
<td>80 ± 11</td>
<td>116 ± 11*</td>
</tr>
<tr>
<td>SAP</td>
<td>129 ± 18</td>
<td>109 ± 15</td>
<td>185 ± 34*</td>
</tr>
<tr>
<td>DAP</td>
<td>70 ± 18</td>
<td>72 ± 19</td>
<td>111 ± 17*</td>
</tr>
</tbody>
</table>

Values are means ± SD.

* P < 0.01 vs. the values before intubation or carinal stimulation.

rate due to carinal stimulation were significantly less than those following laryngoscopy and tracheal intubation in any of the three groups (P < 0.001). Between these two groups, there was a statistically significant difference in the per cent increase in blood pressure from the pre-stimulation value due to carinal stimulation (P < 0.05), but no significant difference between the two groups in the increase in heart rate.

None of the patients received a pressor agent to maintain arterial pressure following the epidural blockade during the study.

Discussion

The purpose of this investigation was to determine the effects of lower cervical and upper thoracic sym-

![Figure 1](image-url)

**Fig. 1.** Changes in heart rate (HR) and arterial pressure (AP) following bronco-carinal stimulation in a patient with cervical epidural anesthesia (upper) and in a patient without block (lower). In both cases, the stimulation was repeated 5 min later.
pathectomy by epidural anesthesia on the cardiovascular responses to airway stimulation. We used a potent local anesthetic with epinephrine in order to ensure a complete blockade of all neural modalities. All patients in the cervical group should have had complete preganglionic sympathetic denervation of the heart, because the mean analgesic levels obtained were between C4–T8. In addition, the preganglionic B-fiber group is blocked more readily than any other fiber group, being at least three times more sensitive than nonmyelinated C fibers. Epinephrine containing local anesthetic solution should also have provided a more intense sympathetic blockade. On the other hand, the local anesthetic per se can affect the cardiovascular responses to airway stimulation such as from laryngoscopy and intubation, or cough reflexes. Recently, Stoelting reported several studies showing that lidocaine given both topically or intravenously only partially suppressed blood pressure and heart rate changes following laryngoscopy and intubation. In an effort to maximize the opportunity to observe circulatory changes we did not apply local anesthetic to the trachea.

Bromage stated that epidurally administered local anesthetics may affect cardiovascular dynamics mainly in two ways, neurally and pharmacologically; the latter effect relating to the plasma concentration of local anesthetic achieved due to vascular absorption from the epidural space. Mepivacaine, when absorbed from the epidural space, may suppress the cough reflex, or suppress dorsal-horn neurons in the spinal cord as lidocaine does. In order to distinguish these possible pharmacologic systemic effects from neural effects, we compared circulatory reflexes in the cervical group with those in the lumbar group in which the same dose and volume of mepivacaine were administered into the epidural space. Since we have found no differences in the time course and the peak values of the plasma concentration of a local anesthetic injected into the cervical, thoracic, and lumbar epidural space with the same amount of the drug, systemic effects, if any, should have been the same in both the cervical and lumbar groups in the present study. In addition, we used light general anesthesia and muscle relaxant to avoid some of the hemodynamic effects of responses such as "bucking." It is possible that the difference in blood pressure response due to carinal stimulation between the lumbar and control groups was related to a systemic effect of the mepivacaine; however, intravenously administered lidocaine, even in a dose of 1.5 mg/kg, does not seem to suppress the increase in blood pressure following endotracheal suctioning.

The lower cervical and upper thoracic epidural blockade did not suppress the increases in blood pressure and heart rate following laryngoscopy and tracheal intubation. Tomori and Widdicombe observed in the cat that mechanical stimulation of four different areas of the upper respiratory tract, the nose, the epipharynx, and laryngopharynx, and the tracheobronchial tree, induced reflex cardiovascular responses. These cardiovascular responses, such as tachycardia and hypertension, and the enhanced neural activity were most pronounced during stimulation of the epipharynx, while those arising from stimulation of the tracheobronchial tree were least marked. These differences in the responses related to sites of airway stimulation also seem to exist in humans In addition, even with the same stimulation, arterial blood pressure increases progressively with the duration of laryngoscopy. Accordingly, the results that the circulatory responses due to laryngoscopy and tracheal intubation were more intense than those due to carinal stimulation are conceivable. In addition, it is possible that cervical block of itself may cause more circulatory depression than does lumbar block. In the present study, however, little difference was noticed between the two groups. In 10 volunteers who had thoracic epidural analgesia (mean analgesic levels, C7.2–T6.3), Oettesen reported that the changes in circulatory variables such as cardiac output, heart rate, blood pressure, and stroke work index, with physical exercise were not remarkably different from those in control subjects. In this regard, acute cardiac sympathetic blockade induced by epidural blockade seems to preserve circulatory responses to intense stimulation such as laryngoscopy and intubation.

At the same time, the circulatory responses due to broncho-carinal stimulation were suppressed by cervical epidural blockade. There may be anatomic differences in the neural impulses arising from laryngoscopy and tracheal intubation and those from broncho-carinal stimulation. Although the effects of neural stimulation on the airways are extremely complex and only partially understood, it seems clear that acute sympathetic block of the heart and lung was instituted in our patients with the cervical epidural blockade. Afferent input from the pharyngo-laryngeal regions traverses through the cranial nerves, IX and X, to the vasomotor center. This cannot be blocked by the cervical block, whereas the sympathetic efferent pathways, at least in part, can be blocked by cervical or lumbar epidural blockade. However, in neither group was the response to laryngoscopy suppressed. Therefore, the differences in the responses due to carinal stimulation between the cervical and lumbar groups are mainly attributed to the neural effect of the epidural anesthesia. Tomori and Widdicombe also suggest that responses to mechanical stimulation of the

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airways could be partly due to release of catecholamines from the adrenal medulla.

In the lungs and airways, there are several receptors, including mechanoreceptors, pulmonary stretch receptors, and irritant receptors. The afferent pathways for the pulmonary reflexes elicited by these receptors have been described in the vagus nerve and its branches. However, it has been suggested recently that pulmonary receptors with afferents in sympathetic nerves exist. This may be one explanation why stimulation of the airway can elicit sympathomimetic circulatory responses and parasympathomimetic responses in the respiratory tracts simultaneously. Cervical epidural blockade should interrupt the sympathetic afferents from receptors in the broncho-carinal regions, resulting in no sympathetic circulatory responses to mechanical stimulation in this area.

There have been many studies on reflex bronchoconstriction elicited by stimulation of the respiratory tracts. This reflex bronchoconstriction is interpreted as increased activity in different parasympathetic nerves to the trachea and bronchi. In addition, the vagus normally plays a potentially dominant role in which sympathetic activity appears to function as an inhibitor of the vagus rather than as an active bronchodilator. In this regard, one would speculate that reflex bradycardia and hypotension should occur following broncho-carinal stimulation in patients with cervical epidural blockade. This did not occur in our study.

We, therefore, conclude that cervical epidural anesthesia can diminish some deleterious circulatory effects due to broncho-carinal stimulation or tracheal suction without predominant vagotonic reflexes in lightly anesthetized, intubated, and paralyzed patients. The mechanisms involved remain to be elucidated; however, we suggest that the interruption of sympathetic afferents from the broncho-carinal region to the spinal cord at the epidural level could contribute to this effect.

References

7. Stoehling RK: Circulatory response to laryngoscopy and tracheal intubation with or without prior oropharyngeal viscous lidocaine. Anesth Analg (Clev) 56:618–621, 1977