EFFECT OF HIGH THORACIC EXTRADURAL ANAESTHESIA ON VENTILATORY RESPONSE TO HYPERCAPNIA IN NORMAL VOLUNTEERS

T. KOCHI, S. SAKO, T. NISHINO AND T. MIZUGUCHI

It is generally accepted that the parasternal intercostal muscles (interchondral part of the internal intercostal muscles) are inspiratory and serve to raise the ribs [1, 2]. Thus reduced power in these muscles could impair outward movement of the upper rib cage and hence reduce ventilation. Furthermore, afferent intercostal nerve activity has been shown to affect ventilatory control [3–5]. Thoracic extradural anaesthesia might affect ventilation by both of these mechanisms. The present study was designed to determine the degree of ventilatory impairment following high thoracic extradural anaesthesia, by measurement of the hypercapnic ventilatory response (HVR).

SUBJECTS AND METHODS

We studied six healthy, male volunteers aged 18–29 yr, who had experienced HVR previously. None had any significant medical history, took regular medication or had recently ingested alcohol. The study was reviewed by our Institutional Ethics Committee and was conducted according to the Declaration of Helsinki. The nature and extent of the investigation had been explained to the subjects and their informed consent obtained.

A 20-gauge cannula was inserted into a forearm vein to permit administration of fluids. Ringer-lactate solution 300–700 ml was administered during the course of the study.

Each subject was placed in the lateral position and an 18-gauge Tuohy needle inserted in the 3rd or 4th thoracic extradural space by the loss of resistance technique. A polyethylene catheter was inserted cranially to a distance of 4 cm into the extradural space. Following removal of the needle, the subject was moved into the supine position and two transducer bands were placed around the trunk, one at the level of the nipples and another at the level of the umbilicus.

Systemic arterial pressure and heart rate were monitored continuously with an automatic sphygmomanometer (Nippon Colin BX-5) and

SUMMARY

We have investigated, in six healthy male volunteers, the effect of high thoracic extradural anaesthesia on the ventilatory pattern and hypercapnic ventilatory response. Ventilatory variables were determined using a respiratory inductive plethysmograph. Duration of inspiration, rib cage excursion and its contribution to tidal volume decreased significantly following extradural anaesthesia, while mean inspiratory flow rate and minute ventilation increased. End-tidal $\text{PCO}_2$ and the tidal excursion of the abdomen were unchanged. Hypercapnic ventilatory response decreased significantly following extradural anaesthesia, principally because of the rib cage component. The slope of the abdominal component did not change significantly. The results indicate that mechanical impairment of rib cage movement can produce decreased ventilatory response to carbon dioxide. The ventilatory impairment and the changes in breathing pattern induced by the high thoracic extradural anaesthesia probably reflect blockade of the efferent or afferent pathway (or both) of the intercostal nerve roots.

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VENTILATORY EFFECT OF THORACIC EXTRADURAL ANAESTHESIA

363

The respiratory inductive plethysmograph (RIP) (Non-Invasive Monitoring Systems Inc.) was calibrated against spirometry by the least-squares method [6] and checked during tidal breathing in the supine position before and after the study. Data were considered unacceptable if RIP measurements differed from simultaneous spirometry by > 10%.

Duration of inspiration (T I), expiration (T E) and V T were determined from the sum signal of RIP. Breath duration (T T), duty ratio (T I/T T), mean inspiratory flow rate (V T/T I) and breath-to-breath minute ventilation (V I) were calculated. V I was separated into the contributions of the rib cage (V I,rc) and abdomen (V I,ab).

The ventilatory response to carbon dioxide was measured by a modified Read rebreathing technique [7]. Subjects rebreathed through a mouthpiece into a 7-litre reservoir charged with 7% carbon dioxide in oxygen until PE' CO 2 reached approximately 9 kPa. Expired gas was sampled at 250 ml min⁻¹ and returned to the reservoir system.

Variables were recorded continuously during 15 min of quiet breathing and during rebreathing. The subjects were allowed to recover for 30 min following rebreathing and then received 2% lignocaine 9-12 ml through the extradural catheter. Fifteen minutes later, recording was restarted and the second carbon dioxide rebreathing was performed 25 min after the administration of lignocaine. Dermatome levels of reduced sensibility to pinprick were determined before and on completion of rebreathing.

Data analysis

Resting ventilatory variables for pre- and post-extradural anaesthesia were determined from the data measured during the final 1 min of quiet breathing and averaged for each subject. Each fifth breath was analysed during the steady state period of carbon dioxide rebreathing.

Data were analysed using least squares regression for HVR and Wilcoxon's Rank test for comparison of variables between pre- and post-extradural anaesthesia. P < 0.05 was regarded as significant.

RESULTS

Anthropomorphic data and extent of extradural block for each subject are shown in table I. The dose of lignocaine varied between subjects, depending on the physical characteristics: subjects 1 and 5 received 2% lignocaine 9 ml and the others received 12 ml. The mean (SD) number of segments blocked was 6.7 (2.5). Mean arterial pressure during quiet breathing did not change significantly from 77.7 (5.2) mm Hg to 74.3 (7.4) mm Hg, whereas heart rate decreased from 77 (6) to 67 (3) beat min⁻¹ (P < 0.01). Homer's sign was observed bilaterally in all subjects.

Resting breathing patterns

Following thoracic extradural anaesthesia, T I decreased significantly, while T E remained unchanged (table II). As a result, T T decreased significantly (P < 0.05). V rc decreased significantly (P < 0.025), whereas V ab was unchanged following extradural anaesthesia (fig. 1). Consequently, the contribution of the rib cage to tidal

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Upper and lower levels of analgesia</th>
<th>No. of segments</th>
<th>Dose of lignocaine (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29</td>
<td>168</td>
<td>54</td>
<td>C5-T7</td>
<td>11</td>
<td>180</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>180</td>
<td>82</td>
<td>T1-T5</td>
<td>5</td>
<td>240</td>
</tr>
<tr>
<td>3</td>
<td>29</td>
<td>172</td>
<td>60</td>
<td>T2-T5</td>
<td>4</td>
<td>240</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td>182</td>
<td>70</td>
<td>T2-T7</td>
<td>6</td>
<td>240</td>
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<tr>
<td>5</td>
<td>18</td>
<td>167</td>
<td>61</td>
<td>C7-T6</td>
<td>8</td>
<td>180</td>
</tr>
<tr>
<td>6</td>
<td>19</td>
<td>170</td>
<td>59</td>
<td>C8-T5</td>
<td>6</td>
<td>240</td>
</tr>
<tr>
<td>Mean</td>
<td>23.5</td>
<td>173.2</td>
<td>64.3</td>
<td></td>
<td>6.7</td>
<td>240</td>
</tr>
<tr>
<td>SD</td>
<td>5.1</td>
<td>6.3</td>
<td>10.1</td>
<td></td>
<td>2.5</td>
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</table>
TABLE II. Changes of respiratory timing variables and \( P_{\text{CO}_2} \) before and after thoracic extradural anaesthesia (mean (SEM)). *P < 0.05; **P < 0.025 compared with control

<table>
<thead>
<tr>
<th></th>
<th>( T_i ) (s)</th>
<th>( T_e ) (s)</th>
<th>( T_T ) (s)</th>
<th>( T_i/T_T )</th>
<th>( P_{\text{CO}_2} ) (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>2.05 (0.61)</td>
<td>2.56 (0.57)</td>
<td>4.54 (1.12)</td>
<td>0.403 (0.026)</td>
<td>5.16 (0.20)</td>
</tr>
<tr>
<td>Extradural anaesthesia</td>
<td>1.47 (0.23)**</td>
<td>2.10 (0.34)</td>
<td>3.49 (0.50)*</td>
<td>0.420 (0.024)</td>
<td>4.97 (0.28)</td>
</tr>
</tbody>
</table>

Fig. 1. Changes in tidal excursion of the rib cage (\( V_{rc} \)), abdomen (\( V_{ab} \)), overall sum (\( V_T \)), compartmental contribution of the rib cage to tidal volume (\( V_{rc}/V_T \)%), mean inspiratory flow rate (\( V_T/T_i \)) and minute ventilation (\( V_t \)), before (left) and following (right) thoracic extradural anaesthesia. O△□●△■ = Individual data; --- = mean value. *P < 0.05; **P < 0.025 compared with control.

breathing (\( V_{rc}/V_T \)% ) decreased significantly (\( P < 0.025 \)) and, as a result, tidal volume was decreased slightly and not significantly. Mean inspiratory flow rate (\( V_T/T_i \)) and minute ventilation (\( V_t \)) increased significantly (\( P < 0.05 \)), mainly because of the shortened \( T_i \) (fig. 1).

Ventilatory response to carbon dioxide

Figure 2 shows the slopes of the ventilatory response to carbon dioxide for subject 3. The overall response decreased by approximately 25%, because of a reduction in the rib cage
Fig. 2. Ventilatory response to carbon dioxide and its partitioning into rib cage and abdominal components determined in subject 3. ● = Control measurements; ○ = following extradural anaesthesia. Values for Slope are litre min⁻¹ kPa⁻¹.

TABLE III. Slope of the linear relationships of $\dot{V}_t$, $\dot{V}_{1,rc}$ and $\dot{V}_{1,ab}$ with $P_{E'CO_2}$ before and after thoracic extradural anaesthesia. Individual data and mean (SD) of six subjects. *P < 0.05; **P < 0.025 compared with control.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>$\dot{V}<em>t/P</em>{E'CO_2}$ (litre min⁻¹ kPa⁻¹)</th>
<th>$\dot{V}<em>{1,rc}/P</em>{E'CO_2}$ (litre min⁻¹ kPa⁻¹)</th>
<th>$\dot{V}<em>{1,ab}/P</em>{E'CO_2}$ (litre min⁻¹ kPa⁻¹)</th>
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<td>Anaesthesia</td>
<td>Control</td>
</tr>
<tr>
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<td>5.00</td>
</tr>
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<td>20.95</td>
<td>18.44</td>
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<td>3</td>
<td>13.22</td>
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<td>4</td>
<td>16.25</td>
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<td>11.68</td>
</tr>
<tr>
<td>5</td>
<td>13.03</td>
<td>8.18</td>
<td>2.01</td>
</tr>
<tr>
<td>6</td>
<td>19.25</td>
<td>19.43</td>
<td>5.67</td>
</tr>
<tr>
<td>Mean</td>
<td>16.16</td>
<td>12.94*</td>
<td>6.66</td>
</tr>
<tr>
<td>SD</td>
<td>3.31</td>
<td>4.79</td>
<td>3.30</td>
</tr>
</tbody>
</table>
component from 8.63 to 6.15 litre min\(^{-1}\) kPa\(^{-1}\), while the compartmental slope of the abdomen was unchanged. The slope of the mean ventilatory response to carbon dioxide for the group decreased following high thoracic extradural anaesthesia, as a result of a decrease of the rib cage contribution. The abdominal contribution was unchanged (table III).

**DISCUSSION**

**Resting breathing pattern**

Carbon dioxide rebreathing increases rib cage movement by increasing activity of the rib cage inspiratory muscles, particularly the parasternal intercostal muscles [8]. Although lumbar extradural anaesthesia with lignocaine increases the hypercapnic ventilatory response (HVR) by direct action on the respiratory centre [9], high thoracic extradural anaesthesia might impair ventilation if parasternal intercostal muscles are blocked.

We observed a significant change in breathing pattern, namely decreases in \(Ti\) and \(Tt\) and an increase in \(VT/Ti\), following thoracic extradural anaesthesia. \(TE\) and \(VT\) also decreased, but not significantly. Consequently \(VT\) increased significantly, although \(P\epsilon'_{CO2}\) was unchanged, suggesting an increase in deadspace ventilation. It has been shown that \(VT/Ti\) is related closely to central inspiratory activity, while \(Ti/Tt\) represents respiratory timing in normal humans in whom the inspiratory volume-time relationship is linear [10]. Because \(P\epsilon'_{CO2}\) remains unchanged, the observed increase in \(VT/Ti\) following extradural anaesthesia indicates an increased central neural drive which may be related to a number of factors. Emotional stress caused by the extradural block could have changed the breathing pattern through cortical influences. The central nervous system (CNS) stimulating effect of lignocaine could also contribute [11]. The excitatory action of lignocaine on the CNS has been reported by several authors. Lignocaine selectively blocks inhibitory neurones in the midbrain reticular formation of the rat, thus facilitating spontaneous neuronal discharge in those neurones under strong inhibitory control [12]. de Jong and colleagues reported enhanced spinal monosynaptic transmission following i.v. lignocaine [13]. The relevance of these findings to our results is not entirely certain, but a facilitatory effect of lignocaine on CNS activity could be one of the causes of the observed increase in neural drive.

**Hypercapnic ventilatory response**

HVR decreased in our study following extradural anaesthesia, in contrast with observations of an increase following lumbar extradural anaesthesia [9], which was suggested to result from a central effect of the lignocaine. However, high thoracic extradural anaesthesia decreased rib cage motion during tidal breathing at rest and during rebreathing to an extent that may have overcome the central stimulant effect of lignocaine. This was confirmed by the observation that \(\dot{V}i/rc/P\epsilon'_{CO2}\) decreased, while \(\dot{V}i,ab/P\epsilon'_{CO2}\) was unchanged.
The largest reductions in $\dot{V}_i/rc/\dot{P}e_{CO_2}$ were found in subjects 1 and 5, who had the most extensive extradural block, which also supports this theory. It is of interest that the ventilatory depressant effect of halothane is also explained partially by its action on the rib cage. Tusiewicz and colleagues examined the effect of halothane on HVR and its partitioning into rib cage and abdominal components in human subjects [17] and found a significant decrease of HVR, mainly because of a decrease in the rib cage contribution, during halothane anaesthesia. These findings, together with those of the present study, suggest that mechanical impairment of the rib cage muscles, whatever the cause, can significantly influence the breathing pattern and decrease HVR in healthy human volunteers. However, the central chemoreceptor drive produced by a change in carbon dioxide concentration is not developed fully in the short time required to perform the Read rebreathing technique. Thus if the diaphragm is capable of functioning normally, adequate ventilation should be maintained even with a reduction of the thoracic component of ventilation, after the full hypercapnic drive has developed. On the other hand, if diaphragmatic function is severely impaired, high thoracic extradural anaesthesia could produce ventilatory failure.

We conclude that thoracic extradural anaesthesia may be applied relatively safely to patients with normal diaphragmatic function, but may augment the likelihood of hypoventilation in patients with ventilatory muscle fatigue such as those with severe chronic obstructive pulmonary disease.

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REFERENCES