Effects of airway anaesthesia on the ability to detect added inspiratory resistive loads

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Summary

1. The effects of airway anaesthesia on the ability to detect added inspiratory resistive loads were studied in normal subjects. A 4% solution of lignocaine hydrochloride was used for anaesthesia of the airways.

2. After anaesthesia of the mouth and upper airways to the level of the vocal cords there was a significant deterioration in the detection ability expressed in terms of the absolute added resistance ($\Delta R$), with a concomitant increase in pulmonary resistance ($R_{\text{int}}$). However, there was no significant change in the detection ability expressed in terms of the ratio of $\Delta R$ to the sum of $R_{\text{int}}$ and the minimal resistance of the apparatus ($\Delta R/R_0$).

3. After combined anaesthesia of the upper and lower airways there was no significant change in pulmonary resistance or in the detection ability expressed either as $\Delta R$ or as $\Delta R/R_0$.

4. We conclude that, in normal subjects, the main site of detection of added inspiratory resistive loads does not lie in the upper or lower airways. Our results and those of previous studies suggest that the diaphragm is the most likely site of detection of added resistive loads.

Key words: airway anaesthesia, lignocaine, respiratory sensation.

Abbreviations: FEV$_{1\text{-sp}}$, forced expiratory volume in 1 s; FRC, functional residual capacity; FVC, forced vital capacity; $R_{\text{int}}$, pulmonary resistance; $R_0$, sum of $R_{\text{int}}$ and initial resistance of apparatus.

Introduction

The ability to detect added pulmonary resistive loads has been studied in normal human subjects (Bennet, Jayson, Rubenstein & Campbell, 1962; Wiley & Zechman, 1966), in patients with increased airways resistance (Burki, Mitchell, Chaudhary & Zechman, 1976) and also in patients with various neurological abnormalities (Davis, 1967; Noble, Frankel, Else & Guz, 1972). However, the site at which the detection of added resistance occurs is disputed. Some workers (Noble, Eisele, Trenchard & Guz, 1970) have suggested that the major site of detection is in the upper respiratory tract, and others (Bennet et al., 1962; Davis, 1967) have thought that receptors in the chest wall are more likely to be the major site of detection. The present study was undertaken to define the role of the airways in the detection of added inspiratory resistive loads by means of selective anaesthesia of the upper airways and lower airways in human subjects.

Materials and methods

Six healthy men and one woman, aged 20–35 years, were studied, none having any evidence of pulmonary disease and all being non-smokers. Informed consent was obtained from each subject. In five subjects (D.B., N.B., B.C., B.H. and N.M.) the effects of upper airway anaesthesia were studied and in three of these subjects (D.B., N.B. and B.C.) and a further two subjects (J.B. and
For upper airways anaesthesia the subject gargled with a mixture of 2% lignocaine hydrochloride jelly and 4% lignocaine hydrochloride solution. After this a pledget of gauze soaked with 4% lignocaine was applied to the soft palate, pharynx and vocal cords until tactile sensation and taste were lost and the gag reflex disappeared. The maximum amount of lignocaine required to achieve anaesthesia was 300 mg.

For anaesthesia of both upper and lower airways, the upper airways were first anaesthetized as described above; the subject then inhaled a 4% solution of lignocaine hydrochloride from a fine-particle nebulizer (Hudson Model Co. 1700). The size of 75% of the particles generated by this nebulizer is between 0.5 and 3.5 μm. Each subject breathed the lignocaine aerosol for 5 min at a respiratory rate of 3 breaths/min with a breathing pattern consisting of 5 s for inhalation to total lung capacity, breath holding for 10 s and slow exhalation for 5 s, followed by 5 min of eupnoeic breathing of the aerosol. In two subjects (N.B. and B.C.) a fibre-optic bronchoscope was passed into the trachea after the lignocaine aerosol inhalation; there was no cough reflex or discomfort with this procedure.

Spirometry was performed by standard methods (American College of Chest Physicians, 1975) with a 13.5 litre bell spirometer (Collins Inc.). The forced vital capacity (FVC), forced expiratory volume in the first second (FEV₁,ₐ) and the flow during the middle 25–75% of FVC (FEF₂₅–₇₅%) were obtained. Functional residual capacity was measured by plethysmography (Dubois, Botelho, Bedell, Marshall & Comroe, 1956).

The pattern of ventilation and pulmonary resistance was measured with the subject seated comfortably in an adjustable dental chair, breathing through a Collins J valve. The flow signal from a pneumotachograph (Fleisch no. 2) attached to the inspiratory side of the valve and the integrated volume signal were recorded on a direct-writing pen recorder (Beckman R411). When it was judged that the subject was comfortably relaxed and adjusted to the mouthpiece, the breathing pattern was recorded and peak inspiratory flow rate, respiratory frequency, tidal volume and minute ventilation were measured. Pulmonary resistance ($R_{\text{int.}}$), was measured by the interrupter technique (Jackson, Milhorn & Norman, 1974) at an inspiratory flow rate of 0.5 litre/s with an electronically operated shutter.

The ability to detect added inspiratory resistive loads was measured by a variable resistance manifold (Zechman & Burki, 1976) connected to the inspiratory side of the valve and hidden from the subject’s view. The manifold consisted of 11 approximately equal levels of resistance provided by sintered-bronze discs arranged in series; the level of resistance presented to the subject could be altered by means of rubber stoppers in the manifold. The dead space of the system was 60 ml. The approximate detection threshold for added inspiratory resistive loads was determined by a tracking procedure described previously (Zechman & Burki, 1976). After this, five levels of resistance chosen to bracket the detection threshold were presented randomly, each load being presented five times. The load was presented after every three to five breaths and allowed to remain for two breaths unless it was detected on the first breath. The subject signalled detection of a load by pressing a button. Extraneous stimuli were kept to a minimum. A scoring system was used in which detection of a load on the first breath was assigned 2 points, and 1 point was assigned for detection on the second breath. The percentage detection score for each level of added resistance was calculated by dividing the scores obtained by the maximum possible score and multiplying the fraction by 100.

The threshold of detection was defined as the load detected 50% of the time (50% $\Delta R$). The detection ability was also calculated in terms of the fractional added resistive load ($\Delta R/R_{\text{a}}$), expressed as the ratio of the added resistance ($\Delta R$) to the sum of the subject’s measured pulmonary resistance and the initial resistance of the apparatus ($R_{\text{a}}$).

In each subject the study sequence in the control state consisted of spirometry and body plethysmography, followed by measurement of the pattern of ventilation and interrupter resistance; finally, the ability to detect added inspiratory resistive loads was measured. The sequence was repeated immediately after airway anaesthesia. In subjects D.B., N.B. and B.C., studies were repeated on a minimum of two occasions to a maximum of six occasions, each study being performed on a different day. It took approximately 15–20 min for the completion of each study after airway anaesthesia.

**Results**

There was a significant increase in $R_{\text{int.}}$ in all subjects after upper airway anaesthesia, but there were no significant differences in any of the other measurements of pulmonary function after either
Airway anaesthesia and load detection

TABLE 1. Mean values for pulmonary function tests before (a) and after (b) anaesthesia

<table>
<thead>
<tr>
<th>Test</th>
<th>Upper airways anaesthesia</th>
<th>Combined upper and lower airways anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (l)</td>
<td>a = 4.79, b = 4.78</td>
<td>a = 5.33, b = 5.31</td>
</tr>
<tr>
<td>FEV\textsubscript{1.0} (l)</td>
<td>a = 3.80, b = 3.94</td>
<td>a = 4.07, b = 4.00</td>
</tr>
<tr>
<td>FEF\textsubscript{25-75%} (l s\textsuperscript{-1})</td>
<td>a = 4.72, b = 4.84</td>
<td>a = 4.27, b = 4.30</td>
</tr>
<tr>
<td>FRC (l)</td>
<td>a = 2.94, b = 3.03</td>
<td>a = 2.56, b = 2.77</td>
</tr>
<tr>
<td>$R_{\text{int}}$ (kPa l\textsuperscript{-1} s)</td>
<td>a = 0.245, b = 0.284</td>
<td>a = 0.280, b = 0.292</td>
</tr>
</tbody>
</table>

Fig. 1. Percentage detection of added inspiratory loads ($\Delta R$), expressed as ratio of $\Delta R$ to the sum of the subject's pulmonary resistance and the minimal resistance of the apparatus ($R_0$), before and after upper airway anaesthesia, in five subjects. Each point represents the percentage detection score of five presentations of the load before (O, control) and after (●) anaesthesia. The line of linear regression does not include the values at 0% and 100% detection.

Upper airway anaesthesia or combined upper and lower airway anaesthesia, as compared with the control state (Table 1). The pattern of ventilation did not change significantly from the control measurements either during upper or combined upper and lower airways anaesthesia.

Fig. 1 shows the percentage detection plotted against fractional changes in added resistive load ($\Delta R/R_0$) before and after upper airway anaesthesia. There was no significant change in the percentage detection of any given level of $\Delta R/R_0$ in any subject. The mean values of absolute load
detected 50% of the time (50% ΔR) and the 50% detection level of the fractional added resistive load (50% ΔR/ΔR₀) before and after upper airway anaesthesia in each subject show that, after upper airway anaesthesia, there was a significantly higher 50% ΔR in all subjects; however, there was no significant change in 50% ΔR/ΔR₀ (Table 2). The effects of combined upper and lower airway anaesthesia on the percentage detection plotted against ΔR/ΔR₀ showed no significant change in any subject (Fig. 2). There were no significant changes in either 50% ΔR or 50% ΔR/ΔR₀ after combined upper and lower airway anaesthesia (Table 3).

Discussion

Our results show that there is no significant change in the ability to detect added inspiratory resistive

![Graph](image)

**Table 2. Detection of inspiratory resistive loads before (a) and after (b) upper airway anaesthesia**

<table>
<thead>
<tr>
<th>Subject</th>
<th>50% R (kPa l⁻¹s⁻¹)</th>
<th>50% ΔR/ΔR₀</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>a</td>
<td>b</td>
</tr>
<tr>
<td>D.B.</td>
<td>0.070</td>
<td>0.102</td>
</tr>
<tr>
<td>N.B.</td>
<td>0.070</td>
<td>0.080</td>
</tr>
<tr>
<td>B.C.</td>
<td>0.055</td>
<td>0.089</td>
</tr>
<tr>
<td>B.H.</td>
<td>0.057</td>
<td>0.065</td>
</tr>
<tr>
<td>M.M.</td>
<td>0.055</td>
<td>0.078</td>
</tr>
<tr>
<td>Mean</td>
<td>0.061</td>
<td>0.083</td>
</tr>
</tbody>
</table>

Significance of difference (paired t-test): 1' < 0.02  P > 0.05

![Graph](image)

**Fig. 2.** Percentage detection of added inspiratory loads (ΔR), expressed as ratio of ΔR to the sum of the subject's pulmonary resistance and the minimal resistance of the apparatus (ΔR₀), before and after combined upper and lower airway anaesthesia, in five subjects. Each point represents the percentage detection score of five presentations of the load before (○, control) and after (●) anaesthesia. The line of linear regression does not include the values at 0% and 100% detection.
Airway anaesthesia and load detection

TABLE 3. Detection of inspiratory resistive loads before (a) and after (b) combined upper and lower airway anaesthesia

<table>
<thead>
<tr>
<th>Subject</th>
<th>50% $\Delta R$ (kPa l^{-1}s)</th>
<th>50% $\Delta R/R_0$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>a</td>
<td>b</td>
</tr>
<tr>
<td>D.B.</td>
<td>0.085</td>
<td>0.087</td>
</tr>
<tr>
<td>N.B.</td>
<td>0.064</td>
<td>0.085</td>
</tr>
<tr>
<td>B.C.</td>
<td>0.045</td>
<td>0.048</td>
</tr>
<tr>
<td>R.M.</td>
<td>0.076</td>
<td>0.075</td>
</tr>
<tr>
<td>J.B.</td>
<td>0.076</td>
<td>0.058</td>
</tr>
<tr>
<td>Mean</td>
<td>0.069</td>
<td>0.071</td>
</tr>
</tbody>
</table>

Significance of difference (paired t-test) $P > 0.5$ $P > 0.5$

loads after either upper airway anaesthesia alone or combined upper and lower airway anaesthesia.

In the present study, the upper airways were probably adequately anaesthetized as judged by loss of sensation in the oropharyngeal cavity and the gag reflex. The degree and extent of lower airway anaesthesia achieved is more difficult to determine. The lack of cough or irritation with passage of the fibre-optic bronchoscope after aerosol anaesthesia in two subjects suggests that the trachea and the main bronchi were adequately anaesthetized. Previous studies on the characteristics of aerosol deposition (Hatch, 1961) would suggest that, with the particle size produced by our nebulizer, and the slow deep breathing pattern used for aerosol inhalation, the anaesthetic aerosol probably penetrated to the alveoli.

The threshold of detection of added inspiratory loads, i.e. 50% $\Delta R$, was significantly higher after upper airway anaesthesia; however, there was a concomitant increase in pulmonary resistance so that the ratio $\Delta R/R_0$ did not change significantly. On the other hand, with combined anaesthesia of both upper and lower airways, we found no change in pulmonary resistance and there was no change in the threshold of detection, expressed either as 50% $\Delta R$ or 50% $\Delta R/R_0$.

The effect of upper airway anaesthesia on the ability to detect added respiratory loads has been studied previously (Davis, 1967), but there are no previous studies on the effects of combined upper and lower airway anaesthesia on load-detection ability.

Our results after upper airway anaesthesia are similar to the results of Davis (1967), who did not find any change in the ability to detect inspiratory resistive loads in normal subjects after oral anaesthesia; however, in patients with upper spinal cord (C4-C7) lesions he found an impaired detection ability, which was further reduced after oral anaesthesia. He concluded that receptors for load detection were mainly somatic receptors in the chest wall, as had previously been suggested by Bennet *et al.* (1962). However, in spinal cord transection, when such information cannot be received from the chest wall, Davis (1967) concluded that the mouth may become a major site of load detection. However, Noble *et al.* (1970, 1972), from studies in tracheostomized patients, concluded that in patients with minimally increased airways resistance the difference in detection ability between upper and lower airways was small. This could explain the lack of any significant change in detection ability after anaesthesia of the upper airways alone in the present study.

The results of studies of the chest wall as a site for detection of resistive loads are conflicting; the studies of Davis (1967) in patients with partial spinal cord transection would suggest that the chest wall is an important site of load detection. In contrast, patients with spinal cord lesions (Noble *et al.*, 1972) and spinal anaesthesia (Eisele, Trenchard, Burki & Guz, 1968) had no impairment in elastic or resistive load-detection ability. The apparent discrepancy in these results may be due to the fact that the airways resistance was not measured in these studies; if the airways resistance was elevated in the patients studied by Davis (1967) then previous studies (Wiley & Zechman, 1966; Burki *et al.*, 1976) would suggest that the load-detection ability expressed only in terms of $\Delta R$ would be impaired. A more recent study by Zechman & Wiley (1977) in six healthy normal subjects found that chest-cage restriction had no effect on load-detection ability expressed as $\Delta R/R_0$, and they concluded that, in normal subjects, the ability to detect loads is not dependent on afferent information coming from the chest wall.

The diaphragm as a site for load detection has not been adequately investigated. Noble *et al.* (1970) studied the ability to detect resistive loads after incomplete bilateral phrenic nerve block and in one patient with a spinal cord transection at C3 (Noble, Frankel, Else & Guz, 1971); the ability to detect resistive loads was not impaired in their experiments.

The increase in pulmonary resistance after upper airway anaesthesia in the present study may have been due to drooping of the soft palate, which was seen on inspection after anaesthesia. During com-
bined upper and lower airways anaesthesia an increase in airflow resistance of the upper respiratory tract may have been masked by a simultaneous decrease in resistance of the lower respiratory tract. The lack of any change in pulmonary function tests after combined upper and lower airway anaesthesia is consistent with previous studies of airway anaesthesia in normal subjects (Cross, Guz, Jain, Archer, Stevens & Reynolds, 1976). However, in subjects with hyper-reactive bronchi, Miller & Awe (1975) found a significant increase in airway resistance after aerosol anaesthesia of the airways with 1% lidocaine.

We conclude from this study that in normal subjects the major site of inspiratory resistive load detection does not lie in the upper or lower airways, although it is possible that the upper airways may become an important site of resistive load detection in patients with spinal cord transection. Other studies (Eisele et al., 1968; Zechman & Wiley, 1977) indicate that the chest wall does not play a major role in resistive load-detection ability. The most likely site in normal subjects appears to be the diaphragm but further definitive studies are required to investigate this possibility.

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References


