EFFECT OF AIRWAY OCCLUSION ON RESPIRATORY TIMING DURING ANAESTHESIA WITH ENFLURANE OR HALOTHANE

G. B. DRUMMOND

SUMMARY

Eighteen patients were studied, during operations under subarachnoid or extradural anaesthesia combined with general anaesthesia, to assess the influence of occlusion of inspiration on the duration of inspiration, and the likelihood of a relationship between lung volume and duration of inspiration. Ten patients breathed 2% enflurane and eight breathed 1% halothane, using 67% nitrous oxide in oxygen as carrier gases. The durations of inspiration and of expiration were significantly longer during enflurane administration than during halothane. Occlusion of inspiration reduced significantly the duration of the inspiratory attempt in both groups of patients, but the duration of a complete respiratory cycle was not changed. In patients receiving enflurane, but not in those given halothane, the relative duration of occluded inspiration was positively correlated with the duration of the unoccluded inspiration. There was no evidence with either agent of a reflex relationship between lung volume and inspiratory duration which would have suggested an active Hering–Breuer reflex.

During enflurane anaesthesia, in contrast to anaesthesia with other agents, the duration of inspiration is influenced by tidal volume, both in dogs (Marsh, Rehder and Hyatt, 1981) and, more surprisingly, in man (Polacheck et al., 1980). The latter workers showed that, when the usual increase in lung volume during inspiration was decreased, or prevented, the duration of inspiration was increased. This finding suggests that a Hering–Breuer reflex was present. When this reflex is present, increasing lung stretch receptor activity, associated with the increase in lung volume during inspiration, contributes to factors that end inspiration. If the increase in lung volume is prevented as, for example, by occlusion of the airway, inspiration is prolonged. The findings of Polacheck and co-workers were surprising, since this reflex is considered to be weak in eupnoic man (Guz et al., 1964) and does not appear to be responsible for the tachypnoea caused by inhalation or i.v. anaesthetic agents (Paskin, Skovsted and Smith, 1968; Gautier, Bonora and Gaudy, 1981).

This study was undertaken to assess the relationship of inspiratory duration and tidal volume in patients receiving enflurane, and to compare this relationship with that found in similar patients given halothane. Both agents were vaporized in 67% nitrous oxide in oxygen, and regional analgesia was used to prevent the effects of surgical stimulation (during light anaesthesia) influencing the results.

PATIENTS AND METHODS

Eighteen patients were studied during combined general and regional anaesthesia administered for varicose vein or inguinal hernia surgery. Consent was obtained and the study was approved by the Ethics Committee. Details of the patients are given in table I. None had overt respiratory disease. All received temazepam 20 mg by mouth 1 h before the induction of anaesthesia.

Subarachnoid or extradural analgesia, to about

Table I. Patient details. SA = subarachnoid, ED = extradural spinal analgesia.

<table>
<thead>
<tr>
<th>Group</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Regional analgesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>H 1</td>
<td>M</td>
<td>33</td>
<td>170</td>
<td>77</td>
<td>SA</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>66</td>
<td>161</td>
<td>61</td>
<td>SA</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>26</td>
<td>178</td>
<td>65.5</td>
<td>SA</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>64</td>
<td>160</td>
<td>59.5</td>
<td>SA</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>39</td>
<td>183</td>
<td>89</td>
<td>SA</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>64</td>
<td>167</td>
<td>68</td>
<td>ED</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>61</td>
<td>173</td>
<td>52</td>
<td>ED</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>48</td>
<td>182</td>
<td>74</td>
<td>ED</td>
</tr>
<tr>
<td>E 1</td>
<td>M</td>
<td>62</td>
<td>185</td>
<td>101</td>
<td>ED</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>52</td>
<td>173</td>
<td>80</td>
<td>SA</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>64</td>
<td>167</td>
<td>78</td>
<td>SA</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>59</td>
<td>178</td>
<td>69</td>
<td>SA</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>22</td>
<td>183</td>
<td>71</td>
<td>SA</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>38</td>
<td>188</td>
<td>83</td>
<td>SA</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>40</td>
<td>178</td>
<td>116</td>
<td>SA</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>45</td>
<td>163</td>
<td>60</td>
<td>ED</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>54</td>
<td>178</td>
<td>57</td>
<td>SA</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>42</td>
<td>160</td>
<td>61</td>
<td>ED</td>
</tr>
</tbody>
</table>
the 10th thoracic dermatome was achieved, and then
general anaesthesia was induced with either prop-
anidid 500 mg or etomidate 20 mg. After suxa-
methonium 1 mg kg\(^{-1}\) i.v., the trachea was intu-
tubated using a plastic tube (Portex) with a low volume
cuff, 8.0 mm for females and 9.0 mm for males. The
traceal tube was connected by its 15-mm connector
directly to the breathing system, without an angle
piece, and connectors were made or adapted so that
there were no abrupt changes in internal diameter.

After spontaneous respiration had returned, the
patients breathed either 2% enflurane or 1%
halothane vaporized in 67% nitrous oxide in oxygen,
from a T-piece system through a large-bore tap, a
low resistance non-rebreathing valve (Ambu Hesse)
and a screen pneumotachograph (Mercury Elec-
tronics F100 L) (fig. 1). Airway pressure at the
valve, and the pneumotachograph pressure, were
measured with transducers (Furness MDC) and
recorded on an oscillograph (Bell and Howell 5-
127). The volume of the pneumotachograph was
15 ml.

Measurements were not made until stable values
of inspiratory flow had been present for at least
20 min. Airway occlusion for the duration of a single
inspiratory attempt was imposed at 1-min intervals
by closing the inspiratory tubing with the large-bore
tap during expiration. Airway pressure and flow
were recorded during the preceding breath and
during the occluded inspiratory effort. At least five
occlusions were performed in each patient. In eight
of the 10 patients given enflurane, additional meas-
urements were made of single inspiratory attempts
from a large air container at ambient pressure with
an elastance of about 4.9 kPa litre\(^{-1}\). This decreased
the tidal volume by about 50–60%.

The traces were digitized (Summagraphics Bit
Pad One) and analysed (Commodore PET 3032) to
give tidal volume, airway pressure during occlusion,
and the durations of the normal, loaded and oc-
cluded inspirations and expirations. The duration of
the normal inspiration (\(T_i\)) was measured from the
duration of inspiratory flow. The durations of the
loaded and occluded breaths (\(T_i^P\)) were measured as
the time from the start of the decrease of airway
pressure to the minimum airway pressure during the
inspiratory attempt. There was very little variation
in inspiratory duration, tidal volume or occlusion
pressure from breath to breath. As an index of the
uniformity of the breath duration within individu-
als, the mean of the standard errors of the durations
of inspiration in each subject was 0.025 s. Student’s
\(t\) test for unpaired and paired data, and least squares
linear regression analysis were used, as appropriate,
to assess statistical significance.

RESULTS
The administration of enflurane was associated with
significantly longer inspiratory and expiratory times
than was halothane (table II). Tidal volumes were
greater in the patients given enflurane and minute
volumes were less, but these differences were not
significant. Mean inspiratory flow rate, calculated as
tidal volume/inspiratory duration (\(Vt/Ti\)) was simi-
lar in the two groups (table III). However, the mean
rate of decrease of airway pressure during occlusion,
calculated as minimum airway pressure/duration of
occluded inspiration (\(Pt/Ti^P\)) was less in the pa-
tients breathing enflurane, and this difference just
reached statistical significance (\(P< 0.05\)).

In both groups, the mean duration of inspiration
was decreased significantly by occlusion. The mean
decrease was similar, being 0.09 ± 0.06 s in group H
\((P< 0.01)\) and 0.09 ± 0.11 s in group E \((P< 0.05)\).
However, the duration of the total respiratory cycle
was not increased by the occlusion of inspiration in
either group. Mean \(T_{\text{ox}}\) in the halothane group
decreased from 2.26 to 2.17 s, and in the enflurane
group an increase from 3.11 to 3.12 s occurred.

The durations of inspiration from the enclosed
container, in the patients given enflurane, are given
in table II and illustrated in figure 2. In general,
these breaths had a duration intermediate between
normal and occluded breaths.

In the patients breathing enflurane, the change in
duration of inspiration during occlusion was related
to the duration of the unimpeded inspiration, so that
those patients with a longer inspiratory duration
showed a smaller decrease, or even an increase, in
inspiratory duration with occlusion. In contrast, the

![Fig. 1. Diagram of the breathing and measurement system.](https://academic.oup.com/bja/article-abstract/56/3/215/503217)
TABLE II. Tidal volume ($V_r$) and duration of inspiration ($T_i$) and expiration ($T_e$) in patients breathing halothane (H) and enflurane (E).

**P < 0.01 compared with group H,** ***P < 0.001 compared with group H.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Control</th>
<th>Occlusion</th>
<th>Control</th>
<th>Occlusion</th>
<th>Load</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$V_r$ (ml)</td>
<td>$T_i$ (s)</td>
<td>$T_e$ (s)</td>
<td>$T_o$ (s)</td>
<td>$T_e$ (s)</td>
</tr>
<tr>
<td>1</td>
<td>207</td>
<td>0.96</td>
<td>1.42</td>
<td>0.93</td>
<td>1.24</td>
</tr>
<tr>
<td>2</td>
<td>168</td>
<td>0.93</td>
<td>1.07</td>
<td>0.86</td>
<td>1.14</td>
</tr>
<tr>
<td>3</td>
<td>187</td>
<td>1.06</td>
<td>1.59</td>
<td>0.96</td>
<td>1.53</td>
</tr>
<tr>
<td>4</td>
<td>165</td>
<td>1.39</td>
<td>1.59</td>
<td>1.33</td>
<td>1.63</td>
</tr>
<tr>
<td>5</td>
<td>207</td>
<td>1.00</td>
<td>1.12</td>
<td>0.92</td>
<td>1.07</td>
</tr>
<tr>
<td>6</td>
<td>240</td>
<td>1.08</td>
<td>1.26</td>
<td>0.87</td>
<td>1.32</td>
</tr>
<tr>
<td>7</td>
<td>177</td>
<td>0.89</td>
<td>0.98</td>
<td>0.87</td>
<td>1.19</td>
</tr>
<tr>
<td>8</td>
<td>179</td>
<td>0.74</td>
<td>0.99</td>
<td>0.60</td>
<td>0.94</td>
</tr>
<tr>
<td>Mean</td>
<td>191</td>
<td>1.01</td>
<td>1.25</td>
<td>0.92</td>
<td>1.26</td>
</tr>
<tr>
<td>SD</td>
<td>25</td>
<td>0.19</td>
<td>0.25</td>
<td>0.20</td>
<td>0.23</td>
</tr>
</tbody>
</table>

The decrease in inspiration during occlusion was relatively constant in the group anaesthetized with halothane.

These observations were confirmed by a positive correlation between the relative duration of occluded inspiration ($T_o/T_i$) and $T_i$ in the enflurane group ($r = 0.69, P < 0.05$), whereas there was no correlation in the patients given halothane (fig. 3). A similar correlation was observed for the total respiratory cycle for the patients given enflurane ($r = 0.62, P < 0.05$), but not in those breathing halothane.

TABLE III. Mean rate of inspiratory flow ($V_r/T_i$) and development of occlusion pressure ($P_T/T_o$) in patients given halothane (H) and enflurane (E). *P < 0.05 compared with group H.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Group H</th>
<th>Group E</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$V_r/T_i$ (ml s$^{-1}$)</td>
<td>$P_T/T_o$ (kPa s$^{-1}$)</td>
</tr>
<tr>
<td>1</td>
<td>216</td>
<td>0.96</td>
</tr>
<tr>
<td>2</td>
<td>216</td>
<td>0.93</td>
</tr>
<tr>
<td>3</td>
<td>176</td>
<td>0.88</td>
</tr>
<tr>
<td>4</td>
<td>119</td>
<td>0.67</td>
</tr>
<tr>
<td>5</td>
<td>207</td>
<td>1.07</td>
</tr>
<tr>
<td>6</td>
<td>222</td>
<td>0.76</td>
</tr>
<tr>
<td>7</td>
<td>200</td>
<td>1.14</td>
</tr>
<tr>
<td>8</td>
<td>242</td>
<td>1.30</td>
</tr>
<tr>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>195</td>
<td>0.96</td>
</tr>
<tr>
<td>SD</td>
<td>37</td>
<td>0.20</td>
</tr>
</tbody>
</table>

DISCUSSION

Methods

Although end-tidal concentrations were not controlled, 1% halothane and 2% enflurane are likely to...
have provided comparable depths of anaesthesia (Eger, 1981).

Ideally, the timing of the events of the respiratory cycle should be assessed from neural signals, since errors may be introduced if other methods are used. In this study, the duration of inspiration was assessed from the flow signal for control breaths and from the pressure signal for the occluded and loaded inspiratory attempts. These mechanical effects can persist for a time after neural activity in the inspiratory muscles ceases (Clark and von Euler, 1972; Miserochi and Milic-Evili, 1976; Polacheck et al., 1980) (fig. 4). The discrepancy between neural and mechanical duration is decreased by decreasing the mechanical time constant of the respiratory system, by adding an external load to decrease the compliance. Airway occlusion represents an infinite load. In anaesthetized cats the duration of the inspiratory attempt, assessed by the occlusion pressure, was identical to the duration of electrical activity of the diaphragm (Miserochi and Milic-Evili, 1976). Consequently, the duration of an occluded inspiration, measured from airway pressure, would be less than the duration of a normal breath measured from airway flow, if the duration of the neural activation were the same in both circumstances. The mean time difference between peak electrical activity of the diaphragm and the end of inspiratory flow,

Fig. 3. Relationship of the duration of occluded inspiration ($T_{oc}$) and respiratory cycle ($T_{tot}$) as a fraction of the control duration, with the duration of control inspiration and respiratory cycle. H = patients given halothane; E = patients given enflurane. Linear regression relationships for group E were significant: for $T_1$, $r = 0.69$; for $T_{tot}$, $r = 0.62$.

Fig. 4. Diagram of the relationship between neural activation (integrated neural activity) and mechanical events in a respiratory cycle. Left, flow during a normal breath starts shortly after neural activity and persists for some time after it. $T_1$ measured from flow is thus longer than neural $T_1$. Right, airway pressure during airway occlusion is almost in phase with neural activity and $T_1$ from airway pressure has the same duration as neural $T_1$. $T_{tot}$ measured from either flow or pressure has the same duration as $T_{tot}$ from neural activity.
measured in three patients anaesthetized with enflurane, was 290 ms, whereas the mean difference between peak electrical activity and minimum airway pressure during occlusion was only 90 ms (Polacheck et al., 1980). However, the actual values of these differences will depend upon the time constants associated with normal and occluded breaths, in the individual subjects, which themselves depend on factors such as lung volume, and respiratory resistance and compliance.

There may also be a small time difference between the onset of neural action and inspiratory flow, but Miserocchi and Milic Emili (1976) were unable to detect such a difference in cats. However, there should be no difference between the duration of the total respiratory cycle assessed from neural or from mechanical events. Neural timing was not measured in this study, which was planned as a comparison between agents, assuming that similar influences would be present in both groups.

Results

Halothane anaesthesia. $T^o$ was less than $T_1$, probably because of the mechanical influence mentioned above. The lack of change in $T_{ox}$, which should not be susceptible to this effect, supports this possibility. However, the mean decrease in inspiratory duration was about 90 ms, which was less than the 190 ms expected from the observations of Polacheck and co-workers (1980).

Nevertheless, $T^o$ did not exceed $T_1$, suggesting that, during anaesthesia with halothane in nitrous oxide, the duration of inspiration was not influenced markedly by preventing an increase in lung volume during inspiration (other than the small change caused by decompression of lung gas). It is possible that a slight prolongation of $T_1$ (8%) occurred, if the difference between $T^o$ and $T_1$ for the same neural $T_1$ is the same as that found by Polacheck and colleagues (1980). In animals, airway occlusion markedly inhibits volume-related vagal activity (Richardson et al., 1973) and experiments with airway occlusion before and after section or cooling of the vagal nerves show that the prolongation of inspiration by occlusion is vagally mediated (Grunstein, Younes and Milic-Emili, 1973; Younes, Iscoe and Milic-Emili, 1975). Although halothane can increase the phasic activity of lung receptors (Coleridge et al., 1968), studies in animals suggest species differences in the inhibition of inspiration caused by this activity. Inspiration is prolonged by airway occlusion in cats anaesthetized with halothane or halothane and pentobarbitone. Vagal section or cooling increases the duration of inspiration and abolishes the effect of occlusion (Younes and Youssef, 1978; Mazzarelli et al., 1979). However, in dogs anaesthetized with halothane, airway occlusion does not prolong inspiration (Marsh, Rehder and Hyatt, 1981). Guz and colleagues (1964) showed that vagal blockade in patients anaesthetized with halothane did not influence respiratory timing, and the occlusion technique used in the present study substantiates the conclusion that the Hering–Breuer reflex is not present during halothane anaesthesia in man.

Enflurane anaesthesia. Respiratory frequency was less during the administration of enflurane than during the administration of halothane. Strict comparisons cannot be made, since factors such as temperature, $P_{ACO_2}$ and alveolar anaesthetic concentrations were not measured or controlled to ensure comparability. The aim of the study was merely to compare the response to occlusion with the two agents. However, ventilation and $V_{T}/T_1$ were similar in the two groups, although $P_{T}/T^o$ was greater in the patients given halothane, suggesting less depression of central ventilatory drive in these patients.

Other workers have reported similar differences in respiratory frequency. At equal MAC values, the respiratory frequency of dogs given halothane was greater than during enflurane anaesthesia (Rich et al., 1979) and the difference in frequency persisted after section of the vagi (Marsh, Rehder and Hyatt, 1981), suggesting that the breathing patterns associated with these agents are not caused solely by vagal reflex action.

During the administration of enflurane, $T^o$ was significantly less than $T_1$, and $T_{ox}^o$ did not differ from $T_{ox}$, suggesting that volume-related feed-back does not reflexly control inspiratory duration. This conclusion is not consistent with observations in dogs (Marsh, Rehder and Hyatt, 1981) or in man (Polacheck et al., 1980). It is possible that effects were not found in the present study because tidal volume was smaller than in the study by Polacheck and colleagues (1980). Their data suggested, but did not prove, a relationship between tidal volume and the prolongation of inspiration. Vagal control of inspiratory duration is present in conscious dogs (Phillipson, 1974). This control is abolished in REM sleep (Phillipson, Murphy and Kozar, 1976), but could possibly persist during enflurane anaesthesia. The reflex may not be detected during halothane anaesthesia because rapid shallow breathing de-
increased the size of the volume feed-back signal.

In man there is little evidence for vagal control, either during quiet breathing while awake, or during i.v. anaesthesia (Clark and von Euler, 1972; Gautier, Bonora and Gaudy, 1981). Polacheck and co-workers (1980) suggested that this reflex control was present during enfurane anaesthesia in man, having found that occlusion increased $T_1$ by 12.5% (range -10 to +50%). In the present study, $T_{10}$ was decreased to 91% of $T_1$, and this was similar to the decrease seen with halothane.

However, the present study corroborates one finding of the previous workers (Polacheck et al., 1980), namely a relationship between the change in inspiratory duration caused by occlusion and the duration of the control inspiration (fig. 3). This relationship could be affected by the differences in duration of neural and mechanical events, which would result in measured $T_{10}$ being less than $T_1$ despite an identical duration of neural events. If this caused a constant difference between $T_{10}$ and $T_1$, then the relative effect of this constant difference would depend on the duration of $T_1$. A positive relationship between $T_{10}/T_1$ and $T_1$ would result, and the slope would be related to the difference. However, the difference would have to be of the order of 500 ms to account for the slope seen in figure 3, which is most unlikely.

Comparison of the present study with that of Polacheck and co-workers (1980) is difficult. Polacheck’s group administered different inhaled concentrations to the patients they studied (from 1.5% to 3% enfurane) so as to obtain no visible response to painful stimuli. Diazepam was used for premedication, thiopentone for the induction of anaesthesia, and it was not stated if nitrous oxide was used.

In this study, occlusion was associated with prolongation of inspiration in two subjects. These subjects might possibly have had particularly strong vagal reflexes, and thus demonstrated this effect, despite other influences that would tend to make $T_{10}$ less than $T_1$, such as the mechanical effect described by Miserocchi and Milic-Emili (1976), or possibly another reflex such as the inspiratory inhibitory intercostal reflex. This is a polysynaptic reflex mediated by intercostal muscle spindles that has been observed in animals (Remmers, 1970; Remmers and Martilla, 1975), newborn sleeping infants (Knill and Bryan, 1976) and anaesthetized man (Read, Freeman and Kafer, 1974). It shortens the duration of occluded inspiration and may have been augmented in the present study by the presence of nitrous oxide, which selectively depresses spinal monosynaptic reflexes (Sugai, Maruyama and Goto, 1982). However, subjects with strong vagal reflexes would be expected to have shorter $T_1$ values, and this was not observed.

ACKNOWLEDGEMENTS

I should like to thank Mr I. B. Macleod for agreeing to studies of patients in his care, and Abbott Laboratories for purchase of some of the apparatus.

REFERENCES


RESPIRATORY TIMING WITH ENFLURANE OR HALOTHANE


**EFFET DE L'OCCLUSION DES VOIES AERIENNES SUR LE CYCLE RESPIRATOIRE AU COURS DE L'ANESTHESIE A L'ENFLURANE ET A L'HALOTANE**

Dix huit patients ont été étudiés au cours d'interventions chirurgicales sous rachianesthésie ou anesthésie peridurale associées à des anesthésies générales, pour contrôler l'influence de l'occlusion inspiratoire sur la durée de l'inspiration, et l'éventualité d'une relation entre le volume pulmonaire et la durée de l'inspiration. Dix patients respiraient de l'enflurane (2%) et huit de l'halothane (1%), avec comme gaz vecteur un mélange de 67% de protoxyde d'azote dans l'oxygène. Les durées de l’inspiration et de l'expiration étaient significativement plus augmentées par l'administration d'enflurane que par l'administration d'halothane. L'occlusion inspiratoire diminuait, de façon significative, la durée des tentatives d'inspiration, et l'éventualité d'une relation entre le volume pulmonaire et la durée de l'inspiration. Dix patients respiraient de l'enflurane (2%) et huit de l'halothane (1%), avec comme gaz vecteur un mélange de 67% de protoxyde d'azote dans l'oxygène. Les durées de l’inspiration et de l’expiration étaient significativement plus augmentées par l’administration d’enflurane que par l’administration d’halothane. L’occlusion inspiratoire diminuait, de façon significative, la durée des tentatives d’inspiration dans les deux groupes de patients, mais la durée d’un cycle respiratoire complet n'était pas modifiée. Chez les patients qui recevaient de l’enflurane, mais pas chez ceux qui recevaient de l’halothane, la durée relative de l’inspiration occlue était positivement corrélée à celle de l’inspiration non occlue. Aucune preuve n’a pu être apportée avec l’un ou l’autre agent d’une relation réfléxe entre le volume pulmonaire et la durée de l’inspiration, relation qui aurait fait supposer l’existence d’un réflexe de Hering – Breuer actif.

**SUMARIO**

Se llevaron a cabo estudios sobre 18 pacientes sometidos a operaciones quirúrgicas bajo anestesia subaracnoidea o extradural, combinada con anestesia general con el objeto de evaluar la influencia de la oclusión de la inspiración sobre la duración de la inspiración y la probabilidad de una relación entre el volumen pulmonar y la duración de la inspiración. Diez pacientes respiraron enflurano al 2% y ocho respiraron halotano al 1%, usando óxido nitruso al 67% en oxígeno como gas portador. La duración de la inspiración y de la expiración fue bastante mayor durante la administración de enflurano que durante la de halotano. La oclusión de la inspiración redujo de manera significativa la duración de la tentativa inspiratoria en ambos grupos de pacientes, pero la duración de un ciclo respiratorio completo no se modificó. En pacientes con enflurano, pero no así en los con halotano, la duración relativa de la inspiración occluida tenía una correlación positiva con la duración de la inspiración no-obstruida. No hubo ninguna prueba con cualquiera de los agentes de una relación de reflejo entre el volumen pulmonar y la duración inspiratoria, lo que hubiera sugerido un reflejo Hering – Breuer activo.