PATTERN OF RESPIRATION AND RESPONSES TO CARBON DIOXIDE DURING TRICHLOROETHYLENE ANAESTHESIA IN THE CAT

S. GREGORETTI, G. B. DRUMMOND AND J. MILIC-EMILI

SUMMARY

Tidal volume \( (V_T) \), minute ventilation \( (V_E) \), the duration of inspiration and expiration, and \( P_{aCO_2} \) were measured via a tracheostomy in adult cats anaesthetized with 0.7%, 1% and 1.5% (inspired) trichloroethylene (TCE). The tracheal cannula was occluded at intervals at the start of inspiration and the tracheal pressure was measured to assess the force of contraction of the respiratory muscles. Anaesthesia with TCE 0.7% was associated with an increase in \( VE \), a reduction in \( VT \), and a marked increase in respiratory frequency and mean inspiratory flow rate, but \( P_{aCO_2} \) values did not differ significantly from those in conscious animals. Ventilation was also greater than in conscious animals during anaesthesia with TCE 1%. TCE 1.5% caused a significantly greater \( P_{aCO_2} \) than in conscious animals. All concentrations of TCE caused a reduction in the ventilatory response to carbon dioxide, measured by the steady-state method. Cervical vagal section did not abolish the tachypnoea caused by TCE.

The characteristic pattern of breathing during anaesthesia with trichloroethylene (TCE) is a reduction in tidal volume \( (VT) \) and an increase in respiratory frequency \( (f) \). Although this pattern has been known for many years (Hewer and Hadfield, 1941; Dundee, 1953), information about the effect of TCE on the pattern of respiration and the ventilatory response to carbon dioxide is sparse and often conflicting.

Whitteridge and Bülbring (1944) suggested that TCE caused a persistent sensitization of the inflation and deflation receptors in the cat lung and this could account for the reduction in tidal volume and tachypnoea seen during TCE anaesthesia. However, section of the vagi did not prevent the onset of tachypnoea after inhalation of TCE in decerebrate cats (Ngai, Katz and Farhie, 1965), suggesting that TCE causes this effect by a central action. Talcott, Larson and Buechel (1965) reported that TCE did not influence the ventilatory response to carbon dioxide in man, but the response was severely depressed in cats (Ngai, Katz and Farhie, 1965).

We have studied the effects of TCE anaesthesia on...
mechanical properties of the respiratory system. For example, a reduction in $V_{0.1}$ may be caused by an increase in flow resistance or elastance of the respiratory system, in addition to a reduction in the rate of increase of activity of respiratory motoneurones or by neuromuscular impairment. An increase in the ratio $P_{0.1}/V_{0.1}$ would suggest that impedance had increased.

The ratio $T_{i}/T_{tot}$ indicates the fraction of time in which the inspiratory muscles are active. It has been called the "duty cycle" of the inspiratory muscles.

Ventilation, mean inspiratory flow and $T_{i}/T_{tot}$ can be related:

$$\dot{V}_E = \frac{V_T}{T_{i}} \times \frac{T_{i}}{T_{tot}}$$

(1)

The advantage of expressing ventilation in this way is that the first component ($V_T/T_{i}$) is related only to the increasing activity of the inspiratory muscles and the second only to the proportional timing of the events in the respiratory cycle. This is not so for the conventional relationship:

$$\dot{V}_E = \frac{V_T}{T_{tot}}$$

(2)

because $V_T$ depends upon both the rate of inspiratory flow and the duration of inspiration. Euler, Herrero and Wexler (1970) proposed that changes in ventilation could be analysed in terms of the rate of increase in lung volume and mechanisms responsible for the timing of events of the respiratory cycle, since these appeared to be controlled separately.

METHODS

Adult cats (six non-pregnant females and three males) were premedicated with atropine 0.6 mg s.c. Previous experiments had shown that atropine 0.6 mg i.v. did not modify the respiratory pattern of cats anaesthetized with either halothane or pentobarbital. Anaesthesia was induced with TCE 2% in 50% oxygen using a mask connected to a T-piece system with a gas flow rate of 8 litre min$^{-1}$. TCE was vaporized using a recently calibrated Tritec Mark 3 vaporizer (Cyprane Ltd). After 15–20 min a tracheal cannula was ligated firmly in the caudal part of the trachea and a femoral artery was cannulated. In five cats both vagi were dissected free in the neck and placed in loose ligatures to facilitate subsequent section.

The cat lay supine in a volume displacement body plethysmograph (Emerson) which had a linear response at frequencies less than 7 Hz. Tracheal pressure (from a sidearm in the tracheal cannula) and systemic arterial pressure were measured with Hewlett-Packard 267B and Statham P23AC transducers respectively and recorded with the volume signal on a Honeywell oscillograph. TCE was given by a T-piece connected to the tracheal cannula and the gas flow kept at three times the minute volume of the cat. The deadspace volume of the system from the tracheal cannula to the T-piece was 4 ml.

Arterial $P_{CO_2}$ was measured by taking two 1-ml samples from the arterial cannula, and analysing each in duplicate with a Corning 165 blood-gas analyser. $P_{aCO_2}$ was calculated as the mean of the four values (the mean absolute difference between the paired values was 0.18 kPa).

After preparation of the cat, which took about 1 h, any chosen TCE concentration was given for 60 min before measurements were made. Nine cats were studied with TCE 0.7%, five with 1% and two with TCE 1.5%. In the six cats in which more than one

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**FIG. 1.** A typical record of volume (upper trace) and tracheal pressure (lower trace) showing controls and an occluded breath, indicating the variables measured. The period of tracheal occlusion is indicated.
concentration was used, the concentration was increased in three and decreased in the other three.

At each TCE concentration, $P_{a\text{CO}_2}$ was measured and the airway was occluded on five occasions over a period of 2 min. Occlusion was performed at end-expiration, for the duration of one cycle of respiratory attempts, with at least 15–20 normal breaths between occlusions. Previous experiments had shown that the occlusions did not cause any change in the subsequent respiratory pattern or in $P_{a\text{CO}_2}$. Tidal volume, the duration of inspiration and expiration, $V_{0.1}$ and $P_{0.1}$, were measured as appropriate from the three breaths preceding each occlusion and the first occluded respiratory effort (fig. 1). $V_E$ and $V_T/T_I$ were calculated for each breath and the mean of all values calculated for the whole period.

These five occlusions were repeated after two concentrations of carbon dioxide (first 2–3% and then 4–6%) had been added to the carrier gas for 5 min to allow the response to carbon dioxide to be measured at each TCE concentration.

Cervical section of the vagi was made in three cats breathing TCE 0.7% and in two cats breathing TCE 1%. Twenty minutes later the carbon dioxide response

**Table 1.** Mean and SD of respiratory variables in awake cats (Gautier, 1976) (A); cats anaesthetized with TCE 0.7% (B); and cats anaesthetized with TCE 1% (C). $P$ values are given for comparison of groups A with B and B with C.

<table>
<thead>
<tr>
<th></th>
<th>Wt (g)</th>
<th>$V_E$ (ml min$^{-1}$)</th>
<th>$V_T$ (ml)</th>
<th>$T_I$ (s)</th>
<th>$T_{tot}$ (s)</th>
<th>Freq. (b.p.m.)</th>
<th>$V_T/T_I$ (ml s$^{-1}$)</th>
<th>$T_I/T_{tot}$</th>
<th>$V_{0.1}$ (ml)</th>
<th>$P_{0.1}$ (cm H$_2$O)</th>
<th>$P_{max}$ (cm H$_2$O)</th>
<th>$P_{a\text{CO}_2}$ (kPa)</th>
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<tr>
<td>A ($n = 11$)</td>
<td></td>
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<td></td>
<td></td>
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<td></td>
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<tr>
<td>Mean</td>
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<td>781</td>
<td>27.2</td>
<td>0.91</td>
<td>2.11</td>
<td>28.7</td>
<td>30.0</td>
<td>0.43</td>
<td>—</td>
<td>—</td>
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<tr>
<td>SD</td>
<td>505</td>
<td>167</td>
<td>5.0</td>
<td>0.05</td>
<td>0.20</td>
<td>3.3</td>
<td>6.2</td>
<td>0.05</td>
<td>—</td>
<td></td>
<td></td>
<td>0.23</td>
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<td>B ($n = 9$)</td>
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<tr>
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<td>2596</td>
<td>16.9</td>
<td>0.20</td>
<td>0.40</td>
<td>156.7</td>
<td>90.0</td>
<td>0.49</td>
<td>11.3</td>
<td>5.8</td>
<td>8.4</td>
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<tr>
<td>SD</td>
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<td>453</td>
<td>2.6</td>
<td>0.05</td>
<td>0.09</td>
<td>37.3</td>
<td>20.2</td>
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<td>1.6</td>
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<td>2.3</td>
<td>0.70</td>
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<td>$P$</td>
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<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.025</td>
<td>n.s.</td>
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<td></td>
<td>n.s.</td>
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<td>C ($n = 5$)</td>
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<tr>
<td>Mean</td>
<td>2592</td>
<td>1732</td>
<td>15.9</td>
<td>0.28</td>
<td>0.57</td>
<td>110.2</td>
<td>57.3</td>
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<td>337</td>
<td>1.2</td>
<td>0.06</td>
<td>0.13</td>
<td>26.1</td>
<td>11.4</td>
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<td>0.8</td>
<td>0.62</td>
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<tr>
<td>$P$</td>
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<td>n.s.</td>
<td>&lt;0.025</td>
<td>&lt;0.025</td>
<td>&lt;0.05</td>
<td>&lt;0.01</td>
<td>n.s.</td>
<td>&lt;0.001</td>
<td>&lt;0.005</td>
<td>n.s.</td>
<td>&lt;0.025</td>
</tr>
</tbody>
</table>

**Fig. 2.** Schematic spirograms using mean values ($\pm$ SD) from table I for cats awake (closed circles) and during anaesthesia with TCE 0.7% (open circles), and TCE 1% (open squares).
Fig. 3. Relationship of ventilation with arterial $P_{\text{aCO}_2}$ in individual cats anaesthetized with TCE 0.7% and 1%. Each cat is indicated by a reference number. The broken line represents the mean response of awake cats (Gautier, 1976).

was measured again. An aqueous solution of sodium chloride 0.9% was infused into the artery at a rate of 5 ml h$^{-1}$ for the duration of the experiment (4–6 h) and sampled blood was replaced with twice the volume of the saline solution.

Statistical analysis was made with Student’s $t$ test for paired and unpaired data as appropriate.

RESULTS

The mean values of the measured and derived variables for cats breathing TCE 0.7% and 1% in 50% oxygen are shown in table I, with similar data for conscious adult cats breathing air, studied by H. Gautier (personal communication). The weights of the groups were comparable. When compared with cats breathing air, the cats breathing TCE 0.7% had a greater $V_{\text{E}}$, a smaller $T_{\text{r}}$ and a markedly greater frequency. The mean rate of inspiration ($V_T/T_I$) was greater in proportion to the greater ventilation and $T_I/T_{\text{tot}}$ was slightly but statistically significantly greater. $P_{\text{aCO}_2}$ was less but this was not statistically significant. The cats breathing TCE 1% had $V_T$ similar to those breathing 0.7%, but $T_I$ and $T_{\text{tot}}$ were greater. Consequently $V_{\text{E}}$ and $V_T/T_I$ were less, $T_I/T_{\text{tot}}$ was not significantly different, and $P_{\text{aCO}_2}$ was greater than in cats breathing TCE 0.7%. These differences are shown in figure 2, in which spiograms have been drawn using the mean values from table I.

In the two cats studied during anaesthesia with TCE 1.5%, ventilation was less than in the 1% group but $V_T$ was about the same. Mean $V_{\text{E}}$ was 1546 ml
min⁻¹, \( V_T \) was 15.5 ml and \( P_{aCO_2} \) was 15.5 kPa.

The ventilatory responses to carbon dioxide for the individual cats in the TCE 0.7% and 1% groups are shown in figure 3, where the mean response of conscious cats is given for comparison (Gautier, 1976). In the cats breathing TCE 0.7%, the increase in \( V_E \) as \( P_{aCO_2} \) was increased was much less than in awake animals. Two animals (Nos 4 and 10) showed an increase and then a reduction in \( V_E \) as the inhaled concentration of carbon dioxide was increased. The response to \( P_{aCO_2} \) was even less in the TCE 1% group. No response to carbon dioxide was seen in the two animals breathing TCE 1.5%.

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The changes in \( V_E \) and frequency were reduced. However, only the change in \( V_T \) in the TCE 0.7% group was significant using the paired t test (\( P<0.05 \)). \( V_T/T_I \) and \( T_I/T_{tot} \) did not change. The changes in the spirogram are illustrated in figure 5 for the TCE 0.7% group. \( P_{aCO_2} \) decreased in all animals, except one breathing TCE 0.7%. The ventilatory response to carbon dioxide was characterized by an increase in \( V_T \) as \( P_{aCO_2} \) was increased. However, \( V_E \) increased in only three animals when \( P_{aCO_2} \) was increased (two breathing TCE 0.7%, and one breathing TCE 1%) and was associated with an increase in \( V_T/T_I \). The changes in timing of respiration that occurred with increasing \( P_{aCO_2} \) did not show a consistent pattern.

Vagal section did not influence mean arterial pressure which remained greater than 100 mm Hg throughout the experiment.

**DISCUSSION**

**Choice of species.** We studied cats because there is a large amount of previous experimental work on their

![Figure 5](image-url)
pattern of respiration in the awake and anaesthetized states. In addition previous authors have reported effects of TCE in this species (Whitteridge and Bülbir, 1944; Ngai, Katz and Farhie, 1965; Coleridge et al., 1968). The slow onset and prolonged recovery from stable TCE anaesthesia in man would have made this study difficult to perform in clinical circumstances.

Widdicombe (1961) suggested that the reflex inhibition of inspiration by lung inflation (Hering–Breuer reflex) was weak in man and relatively weak in the cat, in comparison with other animal species. However, this method of assessment of the strength of the Hering–Breuer reflex has been shown to be misleading (Younes, Vaillancourt and Milic-Emili, 1974). Clark and Euler (1972) showed that the responses of cat and man were similar, although the reflex is not present in man in the eupnoeic range of breathing (Guz et al., 1964).

Methods. The cats were allowed to breathe the appropriate concentration of TCE for 60 min before measurements were made. We consider that this was sufficient to achieve a stable brain concentration of anaesthetic. Measurement of end-expiratory concentrations of trichloroethylene was not possible because of tachypnoea and small tidal volumes. In trial experiments, the inhaled concentration was reduced to 0.5% after at least 1 h of anaesthesia with TCE 0.7%. Coughing and other reflex activity occurred within 10 min of this reduction, and attempts to study cats using this concentration were abandoned. These observations suggested that the change of brain concentration was rapid, possibly because of hyperventilation, and that 1 h would be an appropriate time to allow stabilization. The brain concentration of TCE could have been increased during carbon dioxide administration by an increase in ventilation and possibly in cerebral blood flow. This could account for the increase followed by a decrease in ventilation seen in two cats after inhalation of carbon dioxide. Alternative explanations would be a direct depressant effect of carbon dioxide or fatigue of the respiratory muscles.

Gabbard and others (1952) observed a similar reduction in ventilation at greater $P_{\text{a}CO_2}$ values in human subjects anaesthetized with ether.

Ventilation. Our results show that ventilation was much greater during inhalation of TCE 0.7% than in unanaesthetized animals. Although the respiratory frequency was markedly greater the relative duration of inspiration in the total respiratory cycle was not greatly increased, because $T_i/T_{tot}$ was 0.49 during TCE 0.7% anaesthesia, and 0.43 in awake cats. Consequently the rate of inspiration also had to be greater, and in fact $V_i/T_i$ was 200% greater during TCE 0.7% anaesthesia than in the conscious animals. It is most unlikely that this could have been caused by altered mechanics of the respiratory system, so that the greater flow rate must have been the result of a more rapid contraction of the respiratory muscles, caused by an increase in the rate of activation of respiratory motoneurones. Therefore TCE causes increased activity of these motoneurones. This conclusion is contrary to the opinion that TCE stimulates respiration by an increase in frequency alone (Severinghaus and Larson, 1965) and illustrates an advantage of considering ventilation in terms of the mean flow and relative duration of inspiration. In the cat, other respiratory stimulants such as caffeine (Mazzarelli, unpublished observations) and doxapram (Bopp, Drummond and Milic-Emili, unpublished observations) have been shown to act by increasing $V_i/T_i$, and respiratory depressants such as enflurane (Mazzarelli, Chiolero and Milic-Emili, 1979) by decreasing $V_i/T_i$. In the present study, greater inhaled concentrations of TCE were associated with smaller values of $V_i/T_i$. Greater inhaled concentrations were not associated with an increase in tachypnoea as has been suggested for man (Dundee, 1953). On the contrary, frequency was less, although $T_i/T_{tot}$ was not different.

The efficiency of ventilation was reduced markedly, presumably by tachypnoea. Ventilation during inhalation of TCE 0.7% was more than three times that in conscious animals, whereas $P_{\text{a}CO_2}$ values were not significantly different in the two states. $P_{\text{a}CO_2}$ values agree with values reported in clinical studies (Talcott, Larson and Buechel, 1965; Unni, McArdle and Dundee, 1972; Buchan and Baud, 1973). Marked hypercapnia was present only during anaesthesia with TCE 1.5%, presumably because of an inefficient pattern of respiration.

Diethyl ether is the only other agent that causes an increase in ventilation and a marked increase in frequency (Larson et al., 1969), but no information is available on the respiratory pattern during ether anaesthesia, except in newborn rabbits. Tachypnoea occurs also, but the slight increase in ventilation is caused entirely by an increase in $T_i/T_{tot}$ (Thatch, Wyszogrodski and Milic-Emili, 1976).

Vagotomy. Statistically significant results were not obtained after vagotomy because only a limited number of cats was studied. The most important finding was that tachypnoea was not abolished. Ventilation
was essentially unchanged. \( V_t \) increased significantly and frequency decreased, but this change was not significant statistically. The more efficient pattern of ventilation resulted in a decrease in \( P_{a\text{CO}_2} \) in four of the five animals studied. Vagotomy did not seem to influence \( V_t/T_t \) and \( T_i/T_{tot} \). The ratio of \( P_{0.1} \) to \( V_{0.1} \) (effective impedance) did not change markedly, so the mechanical properties of the respiratory system did not appear to be altered.

Whitteridge and Bulbring (1944), using cats after spinal section, showed that the discharge of pulmonary stretch receptors was increased by 30–50% during brief ventilation with TCE 0.5–1%. This finding was confirmed in intact cats anaesthetized with either chloralose or pentobarbitone by Coleridge and others (1968) who also showed that TCE 1% did not influence the activity of "high threshold receptors" whose impulses were conducted at mean velocities of 1.5 m s\(^{-1}\). Sensitization of pulmonary stretch receptors has been suggested to account for tachypnoea during anaesthesia with TCE and other agents, in animals and man. However, Ngai, Katz and Farhie (1965) showed that TCE caused tachypnoea in cats after vagotomy. Lung inflation does not cause increased tachypnoea in patients anaesthetized with halothane, methoxyflurane or fluroxene as would be expected if the tachypnoea were caused by an active expiration promoting Hering–Breuer reflex (Paskin, Skovsted and Smith, 1968). Vagal section in dogs anaesthetized with ether (Muallem, Larson and Eger, 1969) who also showed that TCE 1% did not influence the activity of "high threshold receptors" whose impulses were conducted at mean velocities of 1.5 m s\(^{-1}\). Sensitization of pulmonary stretch receptors has been suggested to account for tachypnoea during anaesthesia with TCE and other agents, in animals and man. However, Ngai, Katz and Farhie (1965) showed that TCE caused tachypnoea in cats after vagotomy. Lung inflation does not cause increased tachypnoea in patients anaesthetized with halothane, methoxyflurane or fluroxene as would be expected if the tachypnoea were caused by an active expiration promoting Hering–Breuer reflex (Paskin, Skovsted and Smith, 1968). Vagal section in dogs anaesthetized with ether (Muallem, Larson and Eger, 1969) and in cats during halothane administration (Younes and Yousef, 1978) did not prevent tachypnoea.

The ratio of total cycle duration before and after vagotomy in the present study was about the same as would be predicted on the basis of the relationship of this ratio to tidal volume, as described by Grunstein, Younes and Milic-Emili (1973) in a study of cats anaesthetized with pentobarbitone.

The cats in the present study had respiratory rates between 100 and 160 b.p.m. In decerebrate cats given TCE 1% the respiratory rate was increased to only 50 b.p.m. and vagal section and carotid denervation did not influence the ability of TCE to cause tachypnoea (Ngai, Katz and Farhie, 1965). The greater respiratory rates in the present study suggest that TCE may cause tachypnoea by action at a site proximal to the brain-stem colliculi, possibly in the way that hyperthermia causes tachypnoea (Pitts, Magoun and Ranson, 1939), or even proximal to the diencephalon (Cohen, 1964).

These findings support the analogy drawn by Ngai (1968), who pointed out that although baroreceptors are sensitized by anaesthetics, this is not responsible for hypotension during anaesthesia. An increase in vagal afferent activity may occur during TCE administration, but the response of the central mechanisms seems to be reduced so that the end result of vagal activity seems similar to that seen in other forms of anaesthesia. Afferent impulses from the chest wall, such as muscle spindles, have been shown to influence the duration of inspiration (Remmers, 1970) and TCE may cause tachypnoea by augmenting such a reflex.

**Ventilatory response to carbon dioxide.** The study of Ngai, Katz and Farhie (1965) in decerebrate cats showed that the ventilatory response to carbon dioxide was reduced by TCE. On the other hand, Talcott, Larson and Buechel (1965) suggested that the mean ventilatory response of five human subjects was not influenced by anaesthesia using TCE 1 and 2%. However, the mean ventilatory response of the conscious subjects was small, and the range of increase in carbon dioxide values (0.4–0.8 kPa) was also small (Severinghaus and Larson, 1965). Our results show that the actions of TCE resemble those of ether, consisting of an increase in ventilation in the absence of stimulation with inhaled carbon dioxide but a reduced carbon dioxide response (Muallem, Larson and Eger, 1969). Many other volatile anaesthetic agents cause a decrease in ventilation and a reduced carbon dioxide response (Munson et al., 1966).

**Acknowledgements**

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**References**


MODELE DE RESPIRATION ET REACTIONS AU GAZ CARBONIQUE PENDANT UNE ANESTHESIE AU TRICHLOROETHYLENE CHEZ LE CHAT

RESUME
On a mesuré le volume courant ($V_T$), la ventilation par minute ($V_E$), la durée de l'inhalation et de l'expiration, de même que la $P_{ACO_2}$ grâce à une trachéotomie effectuée sur des chats adultes anesthésiés au trichloroéthylène (TCE) inspiré à raison de 0,7%, 1% et 1,5%. La canule trachéale a été obturée à certains intervalles au début de l'inspiration et on a mesuré la pression trachéale pour estimer la force de contraction des muscles respiratoires. L'anesthésie à l'halothane a en général une pression trachéale à 1% de $P_{ACO_2}$ et à 1,5% de $P_{ACO_2}$ sensiblement plus grande que sur les animaux conscients. Toutes les concentrations de TCE ont causé une réduction de la tension respiratoire du gaz carbonique, mesurée à l'aide de la méthode de l'équilibre cinématique. La section cervicale vagale n'a pas empêché la tachypnée causée par le TCE.

ATMUNGSRHYTMUS UND REAKTIONEN AUF KOHLENSTOFFDIOXYD WÄHRENDE EINER TRICHLORÄTHYLEN-ANÄSTHESIE BEI KATZEN

ZUSAMMENFASSUNG
Atemvolumen, Minutenventilation, die Dauer von Ein- und Ausatmung, sowie $P_{ACO_2}$ wurden durch Tracheostomie bei Katzen gemessen, die durch die Einatmung von 0,7%, 1% und 1,5% Trichloräthylen (TCE) narkotisiert waren. Die Trachealkanüle wurde gelegentlich zu Beginn der Einatmung okkludiert, und der Tracheaumfang wurde gemessen, um die Kontraktilität der Atemmuskeln zu bewerten. Eine Narkose mit 0,7% TCE ergab einen Anstieg in $V_T$.
eine Reduktion in VT und einen deutlichen Ansteig von
Atmungsfrequenz und mittlerer Einatmungsstromrate, doch
unterschieden sich die Werte von $P_{\text{aco}}$ nicht wesentlich
von denen nichtnarkotisierter Tiere. Die Belüftung war
auch bei Narkose mit 1% TCE grösser als bei nichtnarko-
tisierten Tieren. 1,5% TCE bewirkte einen deutlich höh-
eren Wert von $P_{\text{aco}}$ als bei nichtnarkotisierter Tieren. Alle
Konzentrationen von TCE bewirkten eine Reduktion der
Belüftungsreaktion auf Kohlendioxyd, gemessen durch die
Gleichmassigkeitsmethode. Zervikale Vagal-Sektion elimini-
ierte nicht die durch TCE verursachte Tachypnoe.

ESTRUCTURA DE LA RESPIRACION Y DE
LAS RESPUESTAS AL DIOXIDO DE CARBONO
DURANTE LA ANESTESIA POR
TRICLOROETILENO EN EL GATO

SUMARIO

Mediante una traqueostomía en gatos adultos anestesiados
por medio de 0,7%; 1% y 1,5% de tricloroetileno (TCE)
inspirado, se midieron el volumen respiratorio ($V_T$), la
ventilación por minuto ($V_E$), la duración de la inspiración
y de la expiración, así como el $P_{\text{aco}}$. Al principio de la
inspiración, estaba ocluida a intervalos la cánula traqueal y se
midió la presión traqueal para evaluar la fuerza de contracción
del músculos respiratorios. La anestesia mediante 0,7% de
TCE se acompañó de un aumento de $V_E$, una reducción del
$V_T$ y de un incremento marcado de la frecuencia respiratoria
y del ritmo del flujo inspiratorio medio, pero los valores del
$P_{\text{aco}}$, no se alteraron de manera significativa para con los de
animales conscientes. También era mayor la ventilación
durante la anestesia con un 1% de TCE que la de los animales
conscientes. Un 1,5% de TCE dio lugar a un $P_{\text{aco}}$
sensiblemente mayor que en los animales conscientes. Todas
las concentraciones de TCE provocaron una reducción de la
respuesta ventilatoria al dióxido de carbono, medido según
el método de estado estable. La sección vagal cervical no
eliminó la taquímia causada por el TCE.