TACHYPNOEA DURING THE ADMINISTRATION OF TRICHLORETHYLENE

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It is generally agreed that a rise in respiratory rate occurs more frequently during the use of trichloroethylene (Trilene) than with any of the other volatile agents in common use in anaesthesia. In this investigation the incidence, predisposing factors, theories of causation, dangers, prophylaxis, and treatment of this troublesome complication have been studied. The investigation is limited to the use of N₂O-O₂-Trilene in anaesthesia, and no cases of analgesia with the drug are included in this series. All subjects received Trilene from the chloroform bottle of a standard Boyle's apparatus. Tachypnoea has been classed as "mild" with respiratory rates of 28–35 per minute, and "severe" with rates of over 35 per minute. For a rise in respiratory rate to be considered a tachypnoea in this study it must have persisted for at least five minutes.

INCIDENCE OF TACHYPNOEA

The maximum respiratory rates observed during 437 administrations of Trilene are shown in table I. In this series about one-third of all classes developed a definite and persistent tachypnoea. These figures are compared with those of Ayre (1944), who recorded this complication in about half of the 105 cases he reported. The significance of the different incidence of increased respiratory rate in the two series will be discussed later.
Maximum respiratory rates recorded during 437 administrations of Trilene. The observations of Ayre (1944) are included for comparison.

<table>
<thead>
<tr>
<th>Respiratory rate per minute</th>
<th>No. of cases</th>
<th>Percentage</th>
<th>Findings of Ayre (1944)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 and under</td>
<td>108</td>
<td>25</td>
<td>13%</td>
</tr>
<tr>
<td>21-30</td>
<td>176</td>
<td>40</td>
<td>44%</td>
</tr>
<tr>
<td>31-40</td>
<td>105</td>
<td>24</td>
<td>33%</td>
</tr>
<tr>
<td>41-50</td>
<td>37</td>
<td>8</td>
<td>6%</td>
</tr>
<tr>
<td>51 and over</td>
<td>11</td>
<td>3</td>
<td>4%</td>
</tr>
</tbody>
</table>

PREDISPOSING FACTORS

Of the predisposing factors of tachypnoea with Trilene, youth seems to be the most important, as can be seen from table II and figure 1. These show relationship of age to maximum respiratory rate in 400 subjects anaesthetized with nitrous oxide-oxygen-Trilene. Respiratory rates of over 28 per minute occurred in 88 per cent of subjects aged 10 years and under, while the figure for those between 31 and 40 years was only 16 per cent. Tachypnoea was also slightly more common in elderly subjects. This association of youth and a tendency to increased respiratory rates is

<table>
<thead>
<tr>
<th>Age in years</th>
<th>Under 28/min.</th>
<th>28-35/min.</th>
<th>Over 35/min.</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 10</td>
<td>12%</td>
<td>33%</td>
<td>55%</td>
<td>30</td>
</tr>
<tr>
<td>11-20</td>
<td>64%</td>
<td>17%</td>
<td>19%</td>
<td>42</td>
</tr>
<tr>
<td>21-30</td>
<td>75%</td>
<td>12%</td>
<td>13%</td>
<td>67</td>
</tr>
<tr>
<td>31-40</td>
<td>84%</td>
<td>10%</td>
<td>6%</td>
<td>78</td>
</tr>
<tr>
<td>41-50</td>
<td>66%</td>
<td>20%</td>
<td>14%</td>
<td>68</td>
</tr>
<tr>
<td>51-60</td>
<td>61%</td>
<td>23%</td>
<td>16%</td>
<td>55</td>
</tr>
<tr>
<td>61-70</td>
<td>52%</td>
<td>28%</td>
<td>20%</td>
<td>42</td>
</tr>
<tr>
<td>71 and over</td>
<td>45%</td>
<td>33%</td>
<td>22%</td>
<td>18</td>
</tr>
</tbody>
</table>

TABLE II

Showing relationship of tachypnoea during Trilene anaesthesia to age of patient. Only percentages of total cases in each age group are shown.
Fig. 1

Histogram showing relationship of maximum respiratory rate during the administration of Trilene to the age of the patient in 400 subjects.

Black = severe tachypnoea (over 35/min.)
Stippled = mild tachypnoea (28-35/min.)
in agreement with the observations of Brittain (1948). In his series of 250 administrations of Trilene for neurosurgery, the only cases of persistent tachypnoea which would not respond to any treatment occurred in children.

The method of induction of anaesthesia as an aetiological factor was studied in 413 patients, the results being shown in table III and figure 2. The highest incidence of tachypnoea occurred when anaesthesia was induced with nitrous oxide-oxygen. Of those who received thiopentone, the greater the induction dose of barbiturate the smaller became the increase in respiratory rate. This finding is related to the previous one concerning age and incidence of tachypnoea. Very young patients form the majority of subjects induced with nitrous oxide-oxygen and a large proportion of those receiving less than 300 mg. thiopentone belonged to the extremes of age. However, irrespective of age, there is a relationship between the method of induction and incidence of tachypnoea with Trilene, as it was soon found that a fairly reliable method of producing a rise in respiratory rate (for experimental purposes) was to induce anaesthesia with nitrous oxide-oxygen or an amount of thiopentone not exceeding 300 mg.

<table>
<thead>
<tr>
<th>Induction dose of thiopentone</th>
<th>Respiratory rates</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nil (N₂O-O₂)</td>
<td>Under 28/min.</td>
<td>38 %</td>
</tr>
<tr>
<td>Under 300 mg.</td>
<td>33 %</td>
<td>38 %</td>
</tr>
<tr>
<td>300-500 mg.</td>
<td>58 %</td>
<td>25 %</td>
</tr>
<tr>
<td>Over 500 mg.</td>
<td>80 %</td>
<td>16 %</td>
</tr>
</tbody>
</table>
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**Fig. 2**

Histogram showing relationship of tachypnœa during Trilene anaesthesia to the induction dose of thiopentone in 413 subjects.

- **Black** = severe tachypnœa
- **Stippled** = mild tachypnœa
RELATIONSHIP OF TACHYPNOEA TO DOSAGE OF TRILENE

Hewer (1946) considers that tachypnoea should always be regarded as evidence of overdosage with Trilene, of which Enderby (1944) considers it to be the earliest sign. Other writers are not so dogmatic, but all are agreed that it is a common sequela of “pushing” Trilene (Culbert, 1943; Macintosh and Bannister, 1947; Brittain, 1948).

The lower incidence of increased respiratory rates recorded in the present series (table I) as compared with the figures of Ayre (1944) could be partly explained by the different dosage of Trilene employed. In the personal series about half of the cases were undergoing neurosurgical operations, and as far as possible the dosage was limited to 1 drachm (3.5 ml.) per hour, as recommended by Ostlere (1948). Ayre, on the other hand, averaged 2–4 drachms (7–14 ml.) per hour.

In two further series of reported cases the same explanation is tenable. Gordon and Shackleton (1943) found that 78 per cent of cases have respiratory rates ranging between 26 and 46 per minute, and 6 per cent showed rates of over 60 per minute. They used on an average 1.5 drachm (5.5 ml.) of Trilene per hour in a closed circuit, which represents a much higher dosage than with a semi-closed technique. One drachm (3.5 ml.) per 5 minutes for short operations, and one ounce (28 ml.) per hour for long operations were the amounts employed by Haworth and Duff (1943). Respiratory rates of over 40 per minute occurred in 80 per cent of their cases.

A relationship between the incidence of tachypnoea and dosage of Trilene could possibly be deduced from table III. The larger the induction dose of thiopentone, the smaller would be the amount of Trilene required to maintain anaesthesia; hence the lower incidence of tachypnoea with
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large induction doses of thiopentone. A similar relationship may hold for table II. The smallest dose of Trilene delivered from the chloroform bottle of the standard Boyle's apparatus may be an overdosage for a child, and to a lesser extent for aged subjects.

It is not true, however, to say that "pushing" Trilene will result in tachypnoea in all patients. In the experiments to which reference is made later it was desired to have respiratory rates of 40 and over per minute. To obtain this, anaesthesia was induced with either nitrous oxide-oxygen or small doses of thiopentone, and nitrous oxide-oxygen was often bubbled through Trilene for periods up to 10 minutes. The resulting respiratory rates recorded in 66 such cases are shown in table IV. Comparing tables I and IV leaves no doubt that the larger the amount of Trilene administered to the patient, the greater is the likelihood of an increase in respiratory rate. Reverting to the original classification, 4 of the 66 cases (6 per cent) showed no tachypnoea and 11 (17 per cent) responded with only a mild tachypnoea (28-35 per min.). Two cases of respiratory arrest occurred; the rates were under 35 per minute in both subjects at the time when respiration ceased.

Two types of cases encountered during this study do not
come under any of the above predisposing causes. In the first type very little Trilene had been given and anaesthesia was too light when the operation commenced. There was an immediate rise in respiratory rate in response to the towel clips or skin incisions. The resulting tachypnoea persisted throughout the operation, even though anaesthesia was ultimately deepened. The only effect of giving more Trilene was to convert rapid deep respiration to rapid shallow respiration. The second type of subject appeared to have a true sensitivity to Trilene. Tachypnoea developed immediately Trilene administration was started, irrespective of dosage. As long as the smallest trace of the agent remained in the anaesthetic mixture the rapid respiration persisted. Three such patients were encountered, all of the 21-40 age group, and on each occasion it was necessary to abandon the use of Trilene.

If an overdose of a drug is considered to be that amount which produces toxic reactions, and if tachypnoea is regarded as a toxic manifestation of Trilene, it is quite true to say that in all subjects tachypnoea is evidence of an overdose of Trilene. A few subjects appear to be hypersensitive and the smallest amount of Trilene is an overdose to them. On the whole, however, in adults it has been found that tachypnoea occurs only when Trilene is “pushed”. The converse does not hold true and all subjects do not react to an overdosage of Trilene by an increase in respiratory rate.

THEORIES OF CAUSATION OF TACHYPNOEA

The earliest reported theory is that of Culbert (1943). He postulated that when the amount of Trilene in the blood reached a certain level a histotoxic anoxia resulted, which manifested itself in the rapid shallow breathing of oxygen lack. He based his view on the fact that increasing the
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Oxygen in the anaesthetic mixture reduced the respiratory rate, and advised that pure oxygen should be administered to all subjects receiving Trilene. This beneficial effect of increasing the percentage of oxygen in the anaesthetic mixture has not been found to be reliable, and in the few cases in whom it resulted in a decrease in the respiratory rate, the fall was only 4–6 respirations per minute and was not maintained. Moreover, Gordon and Shackleton (1943) recorded a very high incidence of tachypnoea when Trilene was administered with pure oxygen. Once tachypnoea is established anoxia may play a part in maintaining it, but it cannot be said that histotoxic anoxia is the sole cause of the increase in respiratory rate that accompanies the administration of Trilene.

Whitteridge and Bülbbring (1944, 1946) have studied changes in the activity of pulmonary receptors during anaesthesia and their influence on respiratory behaviour. Two sets of afferent fibres are concerned in the Hering-Breuer reflex control of respiration, viz. stretch receptors which are stimulated by expansion of the alveoli and cut short inspiration, and separate receptors which are stimulated by deflation and initiate inspiration. All volatile anaesthetics increase the excitability of the pulmonary stretch receptors and this increased excitability is largely responsible for the reduction in depth of respiration. The deflation reflexes which produce acceleration of breathing are briefly stimulated and then paralysed by ether, whereas they are stimulated throughout the exposure by Trilene. Whitteridge and Bülbbring concluded that the increased respiratory rate during the administration of Trilene is probably due to a characteristic action of this drug which cuts short both inspiration and expiration.

This theory is criticized by Johnstone (1951), who believes that a persistent degree of bronchoconstriction may
follow the inhalation of Trilene, leading to retention of carbon dioxide with consequent increase in the respiratory rate. Evidence in support of this view is supplied by the effect of pethidine in abolishing the tachypnoea. He suggests that pethidine reduces the respiratory rate by dilating the bronchi and bronchioles, thus facilitating the elimination of carbon dioxide. He ignores the depressant effect of pethidine on respiration, especially when given intravenously (Mushin, 1951; Pearce, 1951), as a possible cause of the decrease in respiratory rate. Another action of pethidine is its ability to inhibit laryngeal and pharyngeal reflexes (Ruben and Andreassen, 1951) which are of vagal origin. Cutting the vagus causes respiration to become slow and deep (as after pethidine), presumably because both the stretch receptors and deflation reflexes are abolished. It is probable that at least a part of the action of pethidine in reducing the tachypnoea due to Trilene is due to its depressant action on the Hering-Breuer reflex, to the increased activity of which Whitteridge and Bülbbring attributed the increase in respiratory rate.

In an attempt to elucidate this problem, the effects of various drugs of known action were tried on subjects with an established tachypnoea. With two exceptions (amyl nitrite and ether), the drugs were given intravenously and the concentration of Trilene being administered to the patient was not changed until the observations were completed. To make a fair comparison of the various drugs, none was administered to subjects with a respiratory rate of under 40 per minute. Table V summarizes the results obtained with the various drugs.

The findings with pethidine confirm those of Johnstone (1951). His theory is not substantiated by the effects of the other bronchial dilating drugs. Aminophylline (theophylline ethylene diamine) is the most potent of these (Hawkins
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### Table V

**Showing effect of various drugs in reducing the respiratory rate during the administration of Trilene.**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>No. of cases</th>
<th>Resp. rate reduced</th>
<th>Duration Max. of effect effect (min.)</th>
<th>Average reduction in respiratory rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pethidine</td>
<td>25 mg.</td>
<td>16</td>
<td>16 (100%)</td>
<td>5 25 23 per min.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>20 mg.</td>
<td>30</td>
<td>30 (100%)</td>
<td>7 31 12 &quot;</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>1-1 gr.</td>
<td>6</td>
<td>6 (100%)</td>
<td>5 90 28 &quot;</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(8-10 mg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procaine HCl</td>
<td>100-200 mg.</td>
<td>32</td>
<td>20 (62%)</td>
<td>24 6 8 &quot;</td>
<td></td>
</tr>
<tr>
<td>amide</td>
<td>100-200 mg.</td>
<td>10</td>
<td>7 (70%)</td>
<td>7 25 6 &quot;</td>
<td></td>
</tr>
<tr>
<td>Aminophylline</td>
<td>250 mg.</td>
<td>6</td>
<td>0 (0%)</td>
<td>— — —</td>
<td></td>
</tr>
<tr>
<td>Aleudrin</td>
<td>0.01-0.5 mg.</td>
<td>10</td>
<td>2 (20%)</td>
<td>2 14 6 &quot;</td>
<td></td>
</tr>
<tr>
<td>Amyl nitrate</td>
<td>3-5 minims</td>
<td>12</td>
<td>8 (66%)</td>
<td>3 5 10 &quot;</td>
<td></td>
</tr>
<tr>
<td>Ether</td>
<td>6</td>
<td>4</td>
<td>6 (66%)</td>
<td>3 9 12 &quot;</td>
<td></td>
</tr>
<tr>
<td>Antistin</td>
<td>100 mg.</td>
<td>6</td>
<td>0 (0%)</td>
<td>— — —</td>
<td></td>
</tr>
</tbody>
</table>

and Schild, 1951), but observations of its action are valueless because of its respiratory stimulant effect (Sperling, Weisman, and Papermaster, 1942). Isopropyl noradrenaline, which has about ten times the bronchial dilating action of adrenaline, was only effective in reducing the respiratory rate in 20 per cent of cases. Doses greater than 0.5 mg. produced a transient tachycardia and rise in blood-pressure. In a few cases, not recorded in table V, in whom this rise in blood-pressure occurred, there was a fall in respiratory rate coincident with, and of similar duration to, the pressure changes. Amyl nitrite and the addition of a high concentration of ether vapour would seem from table V to be fairly effective but, in actual fact, the respiratory rate was only reduced by them in subjects who coughed or held their breath in response to the irritant vapours.

In view of the anti-histamine action of pethidine (Yonkman, 1948) it was considered advisable to try the effect of
Antistin, which, although not a true bronchial dilator, has been used to counteract the bronchospasm produced by curare (Foregger, 1950). Its failure to reduce the respiratory rate on any of the 6 occasions it was used makes it unlikely that the anti-histamine action of pethidine plays any part in its effect on Trilene tachypnoea.

Morphine, which has a similar though less depressant action than pethidine on the respiratory centre, is equally effective in reducing the respiratory rate during Trilene anaesthesia. Its action on the bronchial tree is one of constriction rather than dilatation. The reduction of tachypnoea by morphine and pethidine varies in degree and duration with the respiratory depressant effects of the doses of the two drugs injected, and it is reasonable to assume that their central action on the respiratory centre is the common factor.

The remaining drugs, procaine hydrochloride and procaine amide, abolished the tachypnoea in about two-thirds of the cases in whom they were used. The amide, which is longer acting than the hydrochloride, has a more prolonged effect in reducing the respiratory rate. They are too unreliable to be of use clinically; even in cases of severe tachypnoea the reduction in respiratory rate which they caused was much less than with either morphine or pethidine. The findings with the procaine drugs are of no help in elucidating the cause of the rise in respiratory rate during Trilene anaesthesia as they have a slight bronchial dilating action and slight depressant effect on respiration, as well as being local analgesics which could paralyse the pulmonary receptors in the lungs.

The findings reported above are all in agreement with the view that the occurrence of tachypnoea during the administration of Trilene is due to stimulation by the drug of the pulmonary stretch and deflation receptors. Bronchial
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dilating drugs are ineffective in reducing the respiratory rate, and it is highly improbable that bronchial constriction and retention of carbon dioxide is a causative factor.

DANGERS OF TRILENE TACHYPNOEA

One usually considers the main hazard of Trilene to be its effect on the cardiovascular system. Increases in respiratory rate are generally thought to be an annoying, but comparatively harmless, complication of the administration of Trilene. Gordon and Shackleton (1943), Hewer (1946) and Brittain (1948), however, have all drawn attention to the possibility of the shallow rapid respiration causing anoxia. There are no recorded observations as to the extent of this anoxia and its relationship to the respiratory rate.

Quantitative studies of respiratory volumes and arterial blood oxygen content were performed on 8 patients undergoing operation of ligation of bilateral varicose veins. Respiration rates during the administration of Trilene ranged from 14–54 per minute. All subjects received the same flow of gases throughout the investigation, viz. 6 litres of nitrous oxide and 2 litres of oxygen per minute. Minute respiratory volume was recorded by a Glover spirometer and blood oxygen content estimated by the colorimetric method of Exton, Schattner, Korman, and Rose (1945).

Figure 3 shows the changes recorded in minute and tidal volumes at various respiratory rates. For the sake of simplicity, values are expressed as a percentage of those at the respiratory rate of 20 per minute. The consistent rise in minute volume and fall in average tidal volume are what one would expect during tachypnoea with Trilene.

The fall in arterial blood oxygen content associated with these respiratory disturbances is shown in figure 4. As the respiratory rate is doubled from 20–40 per minute, on an
average the oxygen content of arterial blood fell 25 per cent, and trebling the respiratory rate reduced it by one-third. From these figures it is clear that in the average patient cyanosis would not occur with respiratory rates of under 60 per minute. This is in agreement with the findings
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Arterial blood oxygen content at various respiratory rates in 7 subjects who developed tachypnoea during the administration of Trilene.

Of Gordon and Shackleton (1943), who only observed cyanosis with extreme increases in respiratory rate.

In anaemic subjects the fall in oxygen saturation of the blood which occurs with moderate increases in respiratory rate could well be dangerous. The absence of cyanosis gives a false sense of security even in normal patients, and to allow a respiratory rate of 40 or over per minute to persist for long periods during Trilene anaesthesia is quite clearly undesirable. This is of prime importance when Trilene is used in anaesthesia for neurosurgery. The
slightest degree of anoxia manifests itself by a rise in intracranial pressure and increased venous oozing which is so detrimental to such operations (Hunter, 1952).

**PROPHYLAXIS AND TREATMENT**

In a very small number of adult subjects (less than one per cent) the administration of minute amounts of Trilene results in an increase in respiratory rate. If tachypnoea occurs immediately the drug is introduced into the anaesthetic mixture, it is wise to consider the patient to be sensitive to Trilene and abandon its use. In the majority of cases the onset of tachypnoea can be prevented by using minimal amounts of Trilene (1 dr. (3.5 ml.) per hour). This can best be achieved in adults by the use of an adequate induction dose of an ultra-short acting barbiturate. It may be necessary to give a small supplementary dose of barbiturate five minutes after induction, as by this time diffusion to non-nervous tissues will have taken place and the blood concentration be low. Using the chloroform bottle of the Boyle’s apparatus it is often impossible to keep the dosage low enough if the drug is administered continuously. Intermittent administration has proved successful as the low volatility of Trilene and its subsequent low rate of excretion permit satisfactory anaesthesia to be continued by nitrous oxide-oxygen for long periods after Trilene has been cut off. In children this is particularly desirable, for most vapourizing bottles deliver a concentration of Trilene which is an overdose to a child. Theoretically at least, basal narcosis with rectal thiopentone or hexobarbitone should reduce the incidence of tachypnoea in children. Personal experience of this is too small to allow any conclusions to be drawn.

Once tachypnoea is established, the respiratory rate will generally fall if the concentration of Trilene vapour is
Reduced. It takes some time for this to happen and it may be desirable to reduce the respiratory rate more rapidly because of sub-oxygenation of the patient. Of the drugs shown in table V, pethidine is the most effective, the safest,
and the one most likely to be available without delay. Tachypnoea will be reduced in 2 minutes by 50 mg. pethidine but this dose is frequently followed by apnoea (Johnstone, 1951). To find the safest dose of pethidine, respiratory volumes and arterial blood oxygen content were measured in a series of patients with tachypnoea, before and after doses of pethidine varying from 10–40 mg. The respiratory depression following doses greater than 25 mg. caused a greater decrease in blood oxygen than resulted from the tachypnoea. The most satisfactory results were obtained with 20 mg. pethidine (fig. 5). On an average the effect of this amount reaches its maximum in 5–7 minutes and lasts for 30–35 minutes. Once the pethidine has exercised its full effect there is no necessity to administer Trilene for at least 20 minutes. The dose of pethidine can safely be repeated should a further rise in respiratory rate occur, but should this be necessary it may be as well to continue with nitrous oxide-oxygen-pethidine and abandon the use of Trilene.

As mentioned previously the effect of the procaine drugs is too unreliable for them to be of any clinical value in this respect. Procaine hydrochloride is not entirely free from danger as the following case will show:

A male subject, aged 16, was for exploratory craniotomy for suspected cerebral tumour. Anaesthesia was induced with 500 mg. thiopentone and topical application of Xylocaine to pharynx and larynx using the technique recommended by Hunter (1951). Anaesthesia was continued with endotracheal nitrous oxide-oxygen-Trilene. The respiratory rate was 36 per minute, 65 minutes after induction. Fifty mg. procaine reduced the rate to 32 in 2 minutes when a further 50 mg. were given. Within one minute the respiratory rate was 48 per minute, breathing was stertorous and movements of the head under the towels indicated that the patient was having a convulsion. This was immediately controlled by 50 mg. thiopentone which, incidentally, reduced the respiratory rate to 24 per minute.
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This patient had suffered from epileptiform convulsions for the past few months due to the cerebral tumour. His threshold for convulsions was presumably low and 100 mg. procaine given intravenously over a period of 2 minutes was sufficient to initiate a major fit. At the beginning of the operation 40 ml. of 1 per cent procaine (400 mg.) with 1:200,000 adrenaline was injected into the scalp without any untoward effects. The absorption of this solution would be reduced by the adrenaline and the blood level never reached a sufficiently high level to cause a convulsion. Furthermore, it was given soon after the thiopentone, which would raise the threshold to convulsions.

CONCLUSIONS

1. Tachypnoea occurs in about one-third of all subjects to whom Trilene is administered.

2. It is more common in children and the aged.

3. The larger the dose of thiopentone used to induce anaesthesia the less is the likelihood of an increase in respiratory rate with Trilene.

4. Tachypnoea should be regarded as a sign of over-dosage with Trilene.

5. Failure of bronchodilator drugs to abolish tachypnoea is at variance with the view that the increase in respiratory rate is due to bronchoconstriction and retention of carbon dioxide.

6. Findings reported are in agreement with the theory that tachypnoea is due to increased sensitization of the pulmonary stretch and deflation reflexes by Trilene.

7. Minute volume is increased, average tidal volume reduced and arterial blood oxygen level reduced with increases in respiratory rate. The fall in blood oxygen may be detrimental to the patient.
8. Tachypnoea can be prevented in the majority of subjects by the use of minimal doses of Trilene.

9. Pethidine, in doses not exceeding 25 mg. for an adult, will abolish tachypnoea for 30–35 minutes.

10. Intravenous procaine is unreliable and even dangerous as a treatment for tachypnoea during the administration of Trilene.

SUMMARY

1. The incidence of rise in respiratory rate following the use of Trilene is reported. Predisposing factors of age of the subject and induction dose of thiopentone have been studied. A relationship has been shown between the dose of Trilene and the occurrence of tachypnoea.

2. Theories of causation of the increase in respiratory rate have been reported and experimental evidence produced to substantiate them.

3. The effect of tachypnoea on respiratory volumes and arterial blood oxygen content has been studied.

4. The prophylaxis and treatment of tachypnoea during the administration of Trilene are discussed.

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REFERENCES

Tachypnoea during Administration of Trichlorethylene 23