ACIDOSIS FOLLOWING THORACIC SURGERY

S. K. PANDIT, J. E. GALWAY AND J. W. DUNDEE

SUMMARY

Arterial blood $P_{CO_2}$ and base excess were measured in 155 thoracotomy patients. Pre-, intra- and postoperative samples were analysed. The presence of a mixed respiratory and metabolic acidosis was confirmed. This was related to $P_{CO_2}$ but not to the use of analgesics or halothane during operation. A comparison was made between the effects of thoracotomy and other forms of surgery. It was concluded that thoracotomy per se results in $P_{CO_2}$ values above those normally found for abdominal surgery. The use of parenteral analgesics tends to aggravate the build-up of carbon dioxide, even when small doses are used.

During a study of three muscle relaxants used in thoracic surgery (Pandit, Dundee and Stevenson, 1971) it became apparent that the vast majority of patients had a combined respiratory and metabolic acidosis in the immediate postoperative period. This acidosis occurred irrespective of the type of muscle relaxant used and in spite of good clinical reversal of the neuromuscular block.

Early investigators found that underventilation was a major contributory factor to acidosis and the more profound the anaesthesia the greater was the carbon dioxide retention (Eistien, 1953). The high carbon dioxide level accompanying anaesthesia rapidly resolved at the conclusion of surgery (Holaday, Ma and Papper, 1957). Other factors considered to influence respiratory acidosis were the position of the patient and the use of inefficient anaesthetic circuits where carbon dioxide absorption was erratic, and poor preoperative lung function (Beecher and Murphy, 1950; Swenson, Stalberg-Stenhagen and Beck, 1961).

Although most of the studies were carried out after thoracic surgery, acid-base state after abdominal operations was also studied as various workers (Maier, Rich and Eichen, 1951; Taylor and Roos, 1950) had noticed a more pronounced acidosis in thoracic cases. Patients scheduled for surgery in which spontaneous respiration during anaesthesia was acceptable to the surgeon were also included to ascertain if inadequate reversal of muscle relaxants had a bearing on the subject.

METHOD

All patients included in the trial were visited preoperatively by one of the authors (J.E.G.) at which time they were asked to participate in a simple lung function study. It was explained that this would assist us in our postoperative management of the case. Vital capacity (v.c.) was checked using a Wright respirometer and peak expiratory flow rate (p.e.f.r.) using a peak flow meter. Each patient recorded five maximal attempts for each test. The highest and lowest values were then discarded and an average was calculated for the remaining three readings. This was then noted as the patient’s preoperative control v.c. and p.e.f.r.

In the main study 155 adult patients of one surgical unit scheduled for various thoracic surgery including closed mitral valvotomy, lung resection, and various oesophageal repair procedures were studied. Each patient received a standard preanaesthetic medication


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of pethidine 50 mg and atropine 0.6 mg given intra-
muscularly 45 min before induction. Anaesthesia was
induced with thiopentone 6 mg/kg after which a
standard dose of either tubocurarine, pancuronium
or alcuronium was then given. It was maintained by
nitrous oxide and oxygen (5:2 l./min) on a closed
circuit with a spill-over. Following this levorphanol
2 mg was given intravenously to supplement the
nitrous oxide anaesthesia. Ventilation was maintained
using a Cape-Wain ventilator. The required tidal
volume was determined by noting the chest move-
ment clinically and monitoring an inflation pressure
of 18–25 cm H₂O. Additional doses of muscle relax-
ant were used when indicated but no increments
were given within 30 min from the expected con-
clusion of surgery. If relaxation was inadequate dur-
ing this period, 0.5–1.0% halothane was added to the
anaesthetic gases for as long as necessary. The action
of the muscle relaxant was reversed by a standard
dose of neostigmine 2.5 mg given with atropine 1.2
mg. An additional 2.5 mg dose of neostigmine with
atropine 0.6 mg was given if the reversal was not
considered adequate after 5 min. Adequacy of reversal
was assessed clinically by the head-raising test. After
reversal the patient was transferred to the recovery
room where he breathed oxygen 6 l./min from a
M.C. face mask.

Blood was sampled from the radial artery at the
wrist. The samples were collected in heparinized
plastic syringes and immediately placed in ice and
dispached to the laboratories with a note of the
patient's body temperature; pH and Pco₂,
were later estimated using a standard Astrup
apparatus with a carbon dioxide electrode and base excess was calculated using a nomogram
(Siggaard-Andersen et al., 1960). Estimations were all
carried out within 5–10 min of sampling. In 20
patients arterial blood samples were taken before
induction of anaesthesia and in 82 again 10–15 min
before reversal of the relaxant. From 155 patients
a further blood sample was drawn 10–15 min after
reversal. Blood-gas analysis was again carried out 60
min after this sample was taken. No postoperative
analgesic was given before the first sample was col-
lected. After that time either levorphanol 1 mg or
methadone 5 mg was given intravenously when
indicated to control pain. In patients who received an
analgesic, the collection of the second blood sample
was delayed until at least 45 min had elapsed from
the time of injection. A similar delay was observed
where sodium bicarbonate was given to correct the
acidosis seen in the initial Astrup result.

Three further groups of surgical patients were
investigated in a similar manner, but only one arterial
sample was taken 15 and 30 min after surgery had
been completed. The groups are:

(1) Thirty thoracic surgery cases who had received
a similar premedication and anaesthetic but for
whom a supplementary volatile anaesthetic
agent (halothane) was used instead of the intra-
venous levorphanol.

(2) One hundred and thirty-seven patients who had
undergone major upper abdominal surgery with
thiopentone, nitrous oxide and oxygen, halothane
and muscle relaxant anaesthesia.

(3) Forty patients in whom the surgery was such
that a non-relaxant type of anaesthetic was surgi-
cally acceptable.

RESULTS

Figure 1 is a scattergram of arterial pH figures during
the four periods under study in the main thoracic
surgery series. During the preinduction and pre-
reversal periods the means of pH were within the
normal limits, but in the immediate postoperative
phase an acidosis outside this normal range was
evident. At 15 min mean pH was 7.30 while at 60
min this had risen to 7.33.

![Fig. 1](https://academic.oup.com/bja/article-abstract/45/1/79/258625)
Figure 2 illustrates PaCO₂ levels by an identical means. Preinduction mean PaCO₂ was 39 mm Hg. During surgery the average PaCO₂ was 32 mm Hg and 15 min after surgery it had risen to 50 mm Hg and remained at this level for at least a further 60 min.

Figure 3 depicts the base excess as a scattergram for the same four periods and in addition a further column at 60 min recording only those cases for whom no base correction had been made. Preinduction values showed a mean of −0.4 m.equiv/l. while just prior to reversal it averaged −2.9 m.equiv/l. This fall continued and the mean value was −3.6 m.equiv/l. at 15 min after surgery. Thereafter the base excess rose to within normal limits at 1 hour after surgery. The mean value for the non-corrected group at 60 min was −2.23 m.equiv/l.

These three figures show conclusively that patients suffer from a respiratory and metabolic acidosis following thoracotomy. This acidosis is mainly respiratory but does have a metabolic element.

Table I shows an analysis of the respiratory aspect of the acidosis at 15 min after surgery and shows the effect of the many variable factors. Age, sex, body weight, nature of operation and duration of operation had little effect on the PaCO₂ level. Neither the type...
Table I. Mean and range of Pco₂ obtained 15 min after operation, analysed according to the variables listed.

<table>
<thead>
<tr>
<th>Variables</th>
<th>No. of patients</th>
<th>Pco₂ (mm Hg) 15 min postoperative</th>
<th>Variables</th>
<th>No. of patients</th>
<th>Pco₂ (mm Hg) 15 min postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>Range</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Age: up to 50 years</td>
<td>51-60 years</td>
<td>66</td>
<td>51.5</td>
<td>31-80</td>
<td>40</td>
</tr>
<tr>
<td>over 60 years</td>
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<td></td>
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<td></td>
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<tr>
<td>2. Sex: male</td>
<td>88</td>
<td>51.6</td>
<td>30-79</td>
<td></td>
<td>67</td>
</tr>
<tr>
<td>female</td>
<td></td>
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<td>3. Weight (kg)</td>
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<tr>
<td>up to 60 kg</td>
<td>up to 60 kg</td>
<td>75</td>
<td>52.1</td>
<td>23-83</td>
<td>52</td>
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<tr>
<td>61-70 kg</td>
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<td>over 70 kg</td>
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<td>4. Nature of operation</td>
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<td>operation on lungs</td>
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<tr>
<td>mitral valvotomy</td>
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<tr>
<td>other thoracotomies</td>
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<td>5. Duration of surgery</td>
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<td></td>
<td></td>
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<tr>
<td>(min)</td>
<td>up to 90</td>
<td>54</td>
<td>50.2</td>
<td>31-83</td>
<td>58</td>
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<tr>
<td>91-120</td>
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<td>over 120</td>
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<td>6. Type of muscle relaxant</td>
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<tr>
<td>Tubocurarine</td>
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<td>Pancuronium</td>
<td></td>
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<td>Alcuronium</td>
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<td>7. Total dose of muscle relaxant</td>
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<tr>
<td>1 dose</td>
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<td>2-3 doses</td>
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<td>over 3 doses</td>
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<td>8. Interval between the last dose of relaxant and the reversal (min)</td>
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<td>30-60</td>
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<tr>
<td>61-90</td>
<td></td>
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<tr>
<td>over 90</td>
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nor the total dose of muscle relaxant had any marked effect on the respiratory acidosis. A similar finding can be seen for the interval between the last dose of relaxant and the point of reversal of the relaxant. When patients are grouped according to their prereversal Pₐ₀₀, there is a significant difference (P<0.02) in the postoperative Pₐ₀₀. In the group where Pₐ₀₀ was less than 25 mm Hg there was a mean postoperative Pₐ₀₀ at 15 min of 42.7 mm Hg whereas in the group in which the prereversal Pₐ₀₀ was in excess of 35 mm Hg the postoperative value was 51.8 mm Hg. Simple preoperative lung function tests (vital capacity and peak expiratory flow rate) were not useful in forecasting the Pₐ₀₀ postoperatively. One was surprised to find no significant difference between mean of Pco₂ in patients who had or had not lung tissue resected.

Table II shows data comparing the results recorded above with those from three further groups of patients. The group of thoracotomy patients who received a volatile agent rather than levorphanol 2 mg to supplement anaesthesia had mean postoperative Pₐ₀₀ almost identical to those reported above. The third group, comprising 137 patients who underwent abdominal surgery, had a significantly lower average Pₐ₀₀ at this time than either of the above groups (P<0.0001). Patients having non-relaxant type of anaesthesia had a mean postoperative Pₐ₀₀ significantly lower than the two groups of thoracic patients (P<0.0001) but similar to that found in the abdominal series. Table III shows that those thoracotomy patients who received a systemic analgesic postoperatively had a significant (P<0.05) mean rise of Pₐ₀₀ from 49.5 to 54.3 mm Hg over 45 min,
ACIDOSIS FOLLOWING THORACIC SURGERY

TABLE II. Mean and range of postoperative (15 min) Pco₂ in four groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>No. of patients (yr)</th>
<th>Age (kg)</th>
<th>Pco₂ (mm Hg)</th>
<th>Average postop.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracotomy, intravenous levorphanol, no volatile supplement</td>
<td>155</td>
<td>51.1</td>
<td>60.7</td>
<td>50.1</td>
<td>28-83</td>
</tr>
<tr>
<td>Thoracotomy, volatile supplement, no intravenous analgesic</td>
<td>30</td>
<td>58.7</td>
<td>67.5</td>
<td>51.3</td>
<td>32-78</td>
</tr>
<tr>
<td>Upper abdominal surgery</td>
<td>137</td>
<td>50.2</td>
<td>41.0</td>
<td>28-54</td>
<td></td>
</tr>
<tr>
<td>Trunk and limb surgery</td>
<td>40</td>
<td>45.8</td>
<td>41.4</td>
<td>23-60</td>
<td></td>
</tr>
</tbody>
</table>

TABLE III. Effect of postoperative analgesics (levorphanol 1 mg or methadone 5 mg) on postoperative Pco₂ after thoracic surgery.

<table>
<thead>
<tr>
<th>Postoperative analgesic (systemic)</th>
<th>No. of patients</th>
<th>Postoperative Pco₂ (mm Hg)</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analgesic given</td>
<td>41</td>
<td>15-30 min</td>
<td>49.5</td>
<td>54.3</td>
</tr>
<tr>
<td>Mean</td>
<td>23-72</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>23-78</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No analgesic given</td>
<td>70</td>
<td>60 min</td>
<td>51.5</td>
<td>50.4</td>
</tr>
<tr>
<td>Mean</td>
<td>28-83</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>36-77</td>
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while those patients who did not receive an analgesic showed a mean fall from 51.5 to 50.4 mm Hg over the same period of time.

DISCUSSION

The fact that a mixed respiratory and metabolic acidosis occurs following surgery is well recognized. Early workers (Gibbon et al., 1950; Stead, Martin and Jensen, 1953; Falor, Kelly and Reynolds, 1955) all attributed the postoperative acidosis to hypoventilation secondary to the respiratory depressant effect of the anaesthetic agents used. Results were expressed as carbon dioxide combining power and techniques of estimation and were relatively crude compared to present-day micro methods. Maier, Rich and Eichen (1951) and Taylor and Roos (1950) supported this view and pointed out that the acidosis following thoracic surgery was more pronounced than that seen after abdominal surgery. Although these workers were using assisted ventilation, their findings are supported by those of the present study, when a more pronounced acidosis was also found after thoracic surgery than following abdominal operations.

Swenson, Stallberg-Stenhagen and Beck (1961) found higher postoperative carbon dioxide tensions in patients with poor lung function but this was not confirmed in the present study. The preoperative respiratory measurements carried out were of no value in predicting those patients who would have a marked postoperative respiratory acidosis but this may be due to a lack of sensitivity of p.e.p.r. and v.c. as appropriate tests. It was surprising to find that, although patients who had some resections of lung parenchyma did have slightly higher postoperative Paₐₒ₂ levels than those patients not having a resection, the differences were not significant.

Ngai and Papper (1962) have remarked that "respiratory acidosis leads to metabolic acidosis, there is an increase in the lactic acid concentrations but the magnitude of its increase cannot account for the full extent of the changes in buffer base. The induced metabolic acidosis is probably the result of sympathoadrenal activation. Interference with oxidative processes with consequent accumulation of intermediary metabolites may also occur". Other factors which could contribute to metabolic acidosis include poor tissue perfusion due either to hypotension, hypovolaemia or vascular occlusion and impaired renal function. One cannot speculate usefully on the exact cause of the acidosis in the present study but it is definitely shown to be of mixed respiratory and metabolic origin with a greater respiratory component.

Bunker (1962) and Brewster, Bunker and Beecher (1952) have shown the ability of diethyl ether to induce a metabolic acidosis. Ether was not used in this study but our results show no significant difference between the degree of acidosis in the group given halothane and that in whom supplementation was with intravenous levorphanol.

The authors relied on clinical judgement to assess the adequacy of ventilation during surgery. It is interesting to note that those patients in whom it was possible to achieve a very low Paₐₒ₂ during surgery (below 25 mm Hg) were those for whom the subsequent postoperative Paₐₒ₂ was lowest. As the ventilator settings were made without knowledge of the Paₐₒ₂ one must presume that these findings reflect pulmonary exchange. This fact concurs with the finding of Swenson, Stallberg-Stenhagen and Beck (1961) who showed that higher postoperative levels of Paₐₒ₂ were found in patients with poor lung function, compared with those with good lung function.

It is widely held that pain inhibits adequate respiration and that an analgesic, if administered judiciously, will improve this state. In the present study the authors found that even a small amount of an opiate...
than a resulting significant rise in blood carbon dioxide tension. One of the authors (Galway, 1972) has shown subsequently that this respiratory depression following an opiate is much greater following thoracic surgery than after abdominal or limb operations and that dosage is much more critical in the former group, a fact previously recorded by Loder (1962).

As far as the site of operation is concerned, the results of table II support the findings of Maier, Rich and Eichen (1951) and Taylor and Roos (1950) that thoracic surgery is followed by higher PaCO₂ than after either abdominal or limb surgery. This is probably due to several features, namely mechanical impairment of respiration by stitching and under-water drains; shunting due to inadequate lung inflation and also to pain which originates from the trauma to ribs, musculature, the rib cage, pleura and diaphragm.

From the findings of this study a good case can be made for ventilation of post-thoracotomy patients using a nitrous oxide/oxygen 50% mixture for the first few hours especially when the PaCO₂ during operation is higher than 50 mm Hg, assuming adequate ventilation with effective carbon dioxide removal has failed to reduce the PaCO₂ value.

ACKNOWLEDGEMENTS

This study would not have been possible without the co-operation of the nursing staff in the Recovery Ward and the Biochemical Laboratory, to whom we extend thanks. During this work Dr Galway was supported by grants from the Medical Research Council and the Belfast Hospital Management Committee.

REFERENCES


ACIDOSE APRES CHIRURGIE THORACALE

SUMMAIRE

La PaCO₂ du sang artériel et l'excès de base ont été mesurés chez cent cinquante cinq thoracotomisés. Des échantillons pré-, intra- et post-opératoires ont été analysés. L'existence d'une acidoïde respiratoire et métabolique mixte a été confirmée. Elle était en rapport avec la PaCO₂ mais pas avec l'emploi d'analgesique ou halothane durant l'opération. On a comparé les effets de la thoracotomie et d'autres formes de chirurgie. Il a été conclu que la thoracotomie per se résulte en des taux PaCO₂ au dessus de ceux observés normalement en chirurgie abdominale. L'emploi d'analgésiques parentéraux tend à aggraver la formation d'analydride carbonique, même lorsque de petites doses ont été utilisées.

ACIDOSE NACH THORAXCHIRURGISCHEN EINGRIFFEN

ZUSAMMENFASSUNG

Bei einhundertfünfundfünfzig Thoracotomie-Patienten erfolgte eine Messung des PaCO₂ im arteriellen Blut sowie des Basenanstiegs. Präoperative, intraoperative und postoperative Blutproben wurden analysiert. Das Verhältnis einer gemischten respiratorischen und metabolischen Acidose konnte bestätigt werden. Diese Acidose bezog sich insbesondere auf das PaCO₂, jedoch nicht auf die verminderte Base. Analgetica oder Halothan während der Operation. Es wurde ferner ein Vergleich angestellt zwischen dem Einfluß der Thoracotomie und anderer chirurgischer Eingriffe. Daraus konnte die Schlußfolgerung gezogen werden, daß die Thoracotomie per se zu dem Ergebnis führt, daß die
Acidosis following thoracic surgery

Paco₂-Werte über diejenigen Werte ansteigen, welche man normalerweise bei der Abdominalchirurgie findet. Die Verwendung parenteraler Analgetica scheint aber die Entstehung von Kohlendioxyd noch zu verstärken, auch wenn nur kleine Dosen verwendet werden.

Resumen

La Pco₂ arterial y exceso de base fueron medidos en ciento cincuenta y cinco pacientes de toracotomía. Fueron analizadas muestras pre, intra y postoperatorias. Fue confirmada la presencia de una acidosis mixta respiratoria y metabólica Louis, correlateado con la Paco₂, pero no con el uso de analgésicos o halotano durante la operación. Fue llevada a cabo una comparación entre los efectos de la toracotomía y otras formas de cirugía. Se concluyó que la toracotomía conduce por sí misma a valores de la Paco₂ superiores a los encontrados normalmente en la cirugía abdominal. El uso de analgésicos parenterales tiende a agravar la formación de ácido carbónico, incluso cuando son empleadas dosis pequeñas.

Correspondence

Respiratory effects of pethidine and pentazocine during trichloroethylene anaesthesia

SIR,—A recent paper from Dr Dundee's department (Unni, McArdle and Dundee, 1972) suggests that pethidine is a more effective agent than pentazocine for reducing the tachypnoea due to trichloroethylene. While this may be true, the evidence presented does not support their claim.

The authors refer to inconsistencies when comparing their own results with those of others. These difficulties run throughout the pentazocine literature, but can be reconciled, in part, by understanding the peculiar shape of the dose-effect curve for pentazocine. The curve parallels that for pethidine at the lowest doses, but then demonstrates a ceiling effect at relatively low dosage. It follows that no overall value for relative potency can ever be given for these two drugs, because their relative potencies are constantly changing (Hoffman and DiFazio, 1970).

One would therefore anticipate that a smaller dose of pentazocine would control the tachypnoea effectively. Hoffman and DiFazio (1970), referring to the personal communication from Smith, report that the ceiling effect for the respiratory depression to intravenous pentazocine is at about 60 mg. Hence the comparatively larger dose of pentazocine needed in the present study seems not to be due to the ceiling effect.

The tidal volume after pethidine administration approached the awake control values, but not after pentazocine injection. The higher Paco₂ values and the absence of any significant rise in the tidal volume after pentazocine administration suggest that pethidine is a superior drug for the control of the tachypnoea under these circumstances. As the tachypnoea during trichloroethylene anaesthesia is rather unpredictable, it is difficult to produce two identical groups. We too, could not explain some of the large differences in minute volumes after pentazocine pretreated and non-treated groups.

Some of the values for Pco₂ measured 7–10 min after narcotic are hard to explain. Even had the narcotic made the patient totally apnoeic, the Paco₂ would be expected to have risen by only about 10 mm Hg/min for the first minute, and by 3 mm Hg/min subsequently (Eger and Severinghaus, 1961). A value of 70 mm Hg seen in one of our patients 7–10 min after a value of 38 mm Hg is equivalent to 8 minutes of apnoea, rather than the 6.1 l./min recorded. It is unfortunate that these patients were not intubated to prevent troublesome respiratory obstruction, and that no effort was made to achieve a relatively steady state for both trichloroethylene uptake and carbon dioxide excretion.

The authors refer to inconsistencies when comparing their own results with those of others. These difficulties run throughout the pentazocine literature, but can be reconciled, in part, by understanding the peculiar shape of the dose-effect curve for pentazocine. The curve parallels that for pethidine at the lowest doses, but then demonstrates a ceiling effect at relatively low dosage. It follows that no overall value for relative potency can ever be given for these two drugs, because their relative potencies are constantly changing (Hoffman and DiFazio, 1970).

Additional references


References


