CLINICAL STUDIES OF INDUCTION AGENTS

XXX: VENOUS SEQUELAE FOLLOWING ETHANOL ANAESTHESIA

BY

M. ISAAC AND J. W. DUNDEE

SUMMARY

The effect of a rapid infusion of 5–10 per cent (w/v) solution of ethanol on veins was studied in about 400 patients. The infusion of up to 550 ml was given rapidly over 4–5 minutes. An unacceptably high incidence of pain and of sequelae followed the use of 7 and 10 per cent (w/v) solutions while 5 and 6 per cent (w/v) carried the lowest incidence of side effects. These latter were too dilute for use in intravenous anaesthesia, and the 8 per cent w/v (10 per cent v/v) caused no higher incidence of sequelae than propanidid. Therefore it is acceptable for clinical use.

The use of intravenous ethanol for anaesthesia has been in the past condemned following the high incidence of venous complications, mainly thrombosis in the vein used for infusion (Adams, 1944). To a large extent this is attributable to the high concentrations of ethanol employed by these previous workers. In a recent paper (Dundee, Isaac and Clarke, 1969) it was shown that the anaesthetic potentialities of this drug could be exploited by employing low concentrations but with rapid infusion rates. However, in the light of previous reports of venous complications the effect of this drug on veins was studied and to obtain comparable data this study was based on the method employed by Hewitt and colleagues (1966).

METHOD

A standard 18 s.w.g. infusion needle was used following local infiltration with lignocaine and 80–120 ml/min of ethanol was infused up to a total of 550 ml generally made up in Hartmann's (Ringer-lactate) solution. At the end of the infusion the needle was withdrawn and firm pressure was applied at the site of venepuncture with a sterile swab and strapping.

The following observations were made:

(a) The occurrence of pain on infusion at the site of puncture or in the arm. This was graded as mild (elicited only on direct questioning) or severe, where the patient complained spontaneously.

(b) The state of the vein was studied 24 and 48 hours postinfusion and the criteria of Hewitt and colleagues (1966) were employed, noting the presence or absence of

(i) tenderness on palpation of the vein;
(ii) thrombosis in the vessel lumen.

From these signs the venous sequelae were classified as:

phlebitis (tenderness without thrombosis);
thrombosis (without phlebitis);
thrombophlebitis (combination of both).

These sequelae were graded as localized (under 2.4 cm) or extended.

RESULTS

Table I shows the results from the first 260 patients studied. Varying concentrations of ethanol were employed at random in this series and the majority of these observations were made personally by the authors.

The highest incidence of pain on infusion was observed with 10 per cent (w/v) solution and, surprisingly, the next highest incidence was noted with 7 per cent (w/v). These two concentrations also produced the highest incidence of detectable venous sequelae.

Subsequent to this study, 8 per cent (w/v) of ethanol only has been used routinely with less detailed follow-up and with more observers. The data from these cases are summarized in table II.

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TABLE I

<table>
<thead>
<tr>
<th>Concentration w/v</th>
<th>Percentage pain on infusion</th>
<th>No. of cases</th>
<th>Percentage sequelae</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild</td>
<td>Severe</td>
<td>Phlebitis</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>7.2</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>7</td>
<td>8.4</td>
<td>9</td>
<td>27</td>
</tr>
<tr>
<td>8</td>
<td>10</td>
<td>10</td>
<td>29</td>
</tr>
<tr>
<td>10</td>
<td>12</td>
<td>12</td>
<td>22</td>
</tr>
</tbody>
</table>

Effects of infusion of approximately 500 ml ethanol on veins (sequelae followed up to 48 hours postoperatively).

TABLE II

Complications associated with rapid infusion of up to 550 ml of 8 per cent (w/v) ethanol.

<table>
<thead>
<tr>
<th>Number of patients</th>
<th>233</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain on infusion</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>45</td>
</tr>
<tr>
<td>Severe</td>
<td>6</td>
</tr>
<tr>
<td>Phlebitis</td>
<td>18</td>
</tr>
<tr>
<td>Thrombosis</td>
<td></td>
</tr>
<tr>
<td>Localized</td>
<td>2</td>
</tr>
<tr>
<td>Extended</td>
<td>-</td>
</tr>
<tr>
<td>Thrombophlebitis</td>
<td></td>
</tr>
<tr>
<td>Localized</td>
<td>3</td>
</tr>
<tr>
<td>Extended</td>
<td>-</td>
</tr>
</tbody>
</table>

It was observed that when pain on infusion did occur it was generally very transient and had usually passed off by 1 minute. In only one case out of a total of 400 did the pain persist to the extent of having to abandon the infusion.

DISCUSSION

No explanation can be found for the paradoxical findings in table I, in which 7 per cent solution caused more pain on infusion and sequelae than the 8 per cent. However, results clearly show that 7 and 10 per cent solutions are best avoided in clinical practice. Although pain on infusion and sequelae were less frequent with the more dilute solutions (5 and 6 per cent) clinical experience has shown that it is often difficult to achieve blood anaesthetic levels of alcohol with these and thus the 8 per cent is preferred for routine use. This has the additional advantage of being simple to prepare by adding 55 ml of absolute alcohol to 500 ml of perfusate.

Comparing the findings from the use of 8 per cent solution with those reported by Hewitt and associates (1966) and O'Donnell, Hewitt and Dundee (1969), it would appear that this concentration causes a higher incidence of venous sequelae than do clinical doses of thiopentone or methohexitone, but that the incidence is similar to that noted after injection of propanidid.

ACKNOWLEDGEMENTS

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REFERENCES


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SOMMAIRE

L'effet sur les veines de l'infusion rapide d'une solution à 5-10 pourcent (w/v) d'éthanol a été étudié chez environ quatre-cent patients. L'infusion, pouvant atteindre un volume de 550 ml, a été administrée rapidement en 4-5 minutes. La fréquence des douleurs et autres séquelles était inacceptablement élevée après l'emploi de solutions à 7 et 10 pourcent (w/v), tandis qu'elle était moins élevée avec 5 et 6 pourcent (w/v). Ces dernières solutions étaient trop diluées pour permettre l'usage en anesthésie intraveineuse, et la solution à 8 pourcent (w/v) (10 pourcent v/v) ne causait pas plus de complications que ne le fait propanidid. Elle est donc cliniquement utilisable.
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moderate hypocapnia should be maintained after discontinuing first the halothane and subsequently the nitrous oxide preferably until the pre-operative level of consciousness has been regained. This may take several hours. Under no circumstance can it be justified to "wake up" the patient using a rebreathing system for letting carbon dioxide accumulate. Controlled hyperventilation during recovery from anaesthesia is especially important in head injury cases where risks of vomiting and aspirating gastric contents are increased.

(4) The blood pressure decreases with the combined effect of chlorpromazine, barbiturates, halothane, and hypocapnia. Especially in the initial phase of anaesthesia, and if surgery is not carried out, the blood pressure may fall quite markedly. As is the general practice in most clinics a moderate level of hypotension (at a blood pressure level of approximately 80 per cent of the pre-operative or the assessed normal level) is acceptable. Development of lower pressures is counteracted by fluid administration (we use plasma or glucose 6 per cent in an amount of 1–2 litres if no bleeding occurs) and with hypertonic urea (we use 0.75 to 1.00 g/kg).

No comments will be given here regarding the use of the profound hypotension used for certain types of neurosurgery. Such lowering of the blood pressure is, when necessary, easily achieved by means of postural tilt and the occasional use of hypotensive agents; it should as a matter of principle be of as brief duration as possible and preferably combined with moderate hypothermia (30–32°C). A discussion of the various measures used for using brain bulk also lies beyond the scope of this discussion.

(5) A timely warning which was explicitly made in the Editorial concerns brief halothane anaesthesias in patients with intracranial space-occupying lesions. In such anaesthetics, often used in conjunction with neuroradiological studies, precisely the same precautions as discussed above must be taken, namely: careful induction; intubation, controlled moderate hypocapnia maintained during and after anaesthesia until adequate recovery has been reached and with proper precautions against vomiting and aspiration; as the "stimulus" of surgery is absent, hypotension is apt to be relatively more pronounced and it must be counteracted by fluid administration and/or hyperosmotic therapy. Without proper attention to these problems a most dangerous triad of intracranial hyper-tension, hypercapnia and (despite the hypercapnia) systemic hypotension can arise. This condition may well aggravate the clinical condition of critically ill patients, i.e. precisely those patients who cannot tolerate any further brain tissue damage. Often the neuroradiological procedure is blamed for what really is due to the anaesthetic technique used and its complications (including aspiration of gastric contents). In addition, it may be affirmed, as the Editorial also mentioned, that an angiographic study made during a state of cerebral vasodilatation (as during halothane combined with normal or high PaO2 values) may be falsely interpreted as normal. This is so because the blood flow decreases in the damaged tissue (e.g., around a tumour or in an area of brain contusion) due to the above-mentioned "intracerebral steal" effects.

It is our conclusion that low doses of halothane (0.5 per cent) can be used safely in neurosurgery, but only when used in combination with other techniques. In particular the risks of brief inadequate anaesthesias can hardly be overemphasized. Safer drugs and procedures will undoubtedly be developed. At present, however, none of the alternative modes of conducting routine neuro-anaesthesia can be stated to be of assured superior value to the mode described here. And, more important, the somewhat complex pattern of considerations which the neuro-anaesthetist must be able to comprehend and make use of (cerebral blood flow, intracranial pressure, systemic blood pressure, PaO2, etc.) is undoubtedly here to stay. To have focused attention thereon is a valuable contribution of the Editorial of April 1969.

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REFERENCES
