CARDIAC ARRHYTHMIA INDUCED BY PNEUMOENCEPHALOGRAPHY

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SUMMARY
Cardiovascular collapse associated with pneumoencephalography (PNE) has been reported but there has been no prospective study of its nature and cause. We have recorded prospectively the e.c.g. of 82 unselected patients, with no cardiovascular or metabolic disease, undergoing PNE under general anaesthesia. The frequency of arrhythmia following air injection was 60%; bradycardia 22%; ventricular ectopic beats 26%; nodal rhythm or sinus tachycardia 11%. Cardiovascular collapse occurred in three patients; two with “torsades de pointes” and one with bigeminy and q.r.s. block. Arrhythmia was more frequent in patients with a pituitary tumour and intracranial hypertension (91%). Eight postoperative control PNE examinations were uneventful. Three of four patients with frontal lobe tumours and four of seven with posterior fossa tumours exhibited arrhythmia.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age</th>
<th>Disease</th>
<th>Anaesthesia (spontaneous breathing)</th>
<th>Postoperative course</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8 months</td>
<td>Hydrocephalus</td>
<td>Halothane-N₂O</td>
<td>Recovered but died 4 days later of G.I. haemorrhage</td>
</tr>
<tr>
<td>2</td>
<td>6 weeks</td>
<td>Meningoencephalocele</td>
<td>Halothane-N₂O</td>
<td>Recovered</td>
</tr>
<tr>
<td>3</td>
<td>1 month</td>
<td>Meningocele</td>
<td>i.m. ketamine</td>
<td>Recovered (permanent brain damage)</td>
</tr>
<tr>
<td>4</td>
<td>3 months</td>
<td>Subarachnoid haemorrhage</td>
<td>Halothane-N₂O</td>
<td>Died</td>
</tr>
<tr>
<td>5</td>
<td>20 months</td>
<td>Hydrocephalus</td>
<td>Halothane-N₂O</td>
<td>Recovered</td>
</tr>
<tr>
<td>6*</td>
<td>47 yr</td>
<td>Epilepsy</td>
<td>Thiopentone halothane-N₂O</td>
<td>Recovered—G.I. haemorrhage</td>
</tr>
<tr>
<td>7†</td>
<td>66 yr</td>
<td>Subarachnoid haemorrhage</td>
<td>Thiopentone halothane-N₂O</td>
<td>Died</td>
</tr>
<tr>
<td>8‡</td>
<td>54 yr</td>
<td>Syringomyelia</td>
<td>Thiopentone halothane-N₂O</td>
<td>Died</td>
</tr>
</tbody>
</table>

* Arteriosclerosis and coronary disease.
† Respiratory insufficiency (obstructive).
‡ Patient receiving imipramine. Myelography.

Cardiac arrhythmia following pneumoencephalography (PNE) has been reported (Abeles and Schneider, 1935; Bick and Epstein, 1943; Saidman and Eger, 1965; Bordiuk et al., 1969; Burch and Breaux, 1974), but there has been no prospective study to determine the nature, frequency or cause of these changes.

In 6-years, out of a total of 2380 PNE examinations, five infants and three adults developed cardiovascular collapse and cardiac arrest following the injection of air; there were four deaths and one patient sustained permanent brain damage (table I).

We have now studied this problem prospectively.

PATIENTS AND METHODS
Eighty-two unselected patients (age range 1 month to 70 yr; mean 42 yr) were monitored carefully during the procedure. All were free from cardiovascular, respiratory or metabolic disease and had a normal e.c.g.

Before the induction of anaesthesia, e.c.g. was recorded (standard and peripheral leads) and then observed on an oscilloscope. Further recording was made whenever an abnormality was noted. The presenting conditions are shown in table II.
TABLE II. Presenting conditions

<table>
<thead>
<tr>
<th>Disease</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pituitary adenoma</td>
<td>12</td>
<td>14.2</td>
</tr>
<tr>
<td>Other pituitary tumours</td>
<td>9</td>
<td>11.1</td>
</tr>
<tr>
<td>Optic chiasma lesion</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>9</td>
<td>11.1</td>
</tr>
<tr>
<td>Frontal lobe tumour</td>
<td>4</td>
<td>4.8</td>
</tr>
<tr>
<td>Rhinorrhoea</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>Posterior fossa tumour</td>
<td>7</td>
<td>8.5</td>
</tr>
<tr>
<td>Arnold–Chiari malformation</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Pituitary insufficiency</td>
<td>3</td>
<td>3.7</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>9</td>
<td>11.1</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Cerebrovascular accidents</td>
<td>5</td>
<td>6.1</td>
</tr>
<tr>
<td>Encephalomyelitis</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>Cerebral atrophy</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>Cerebellar atrophy</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Disseminated multiple sclerosis</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Others (normal pneumoencephalogram)</td>
<td>11</td>
<td>13.4</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Anaesthesia

Premedication included atropine (except in one patient who had glaucoma), usually combined with a sedative such as diazepam. In the adults, anaesthesia was induced with a dose of thiopentone sufficient to obtund the eyelash reflex and intubation of the trachea was performed after the injection of suxamethonium, using a cuffed non-kinking tube. Anaesthesia was maintained with either halothane in a mixture of nitrous oxide in oxygen using a Magill system with spontaneous breathing, or with nitrous oxide and oxygen combined with a non-depolarizing neuromuscular blocking drug (tubocurarine or pancuronium) and a narcotic analgesic (fentanyl or pheno- peridine) with IPPV. In two infants, anaesthesia was induced with halothane and ventilation was spontaneous through an Ayre’s T-piece.

Air was used as the contrast medium and was injected in divided doses under radiological control using an image intensifier, the patient being in the sitting position. Special care was taken to maintain adequate ventilation, when the patient was in the head-down position.

Air embolism has been noted as a possible complication of PNE (King and Otenasek, 1948; Jacoby et al., 1959; Gardner, 1971). The possibility of major and clinically significant air embolism having occurred in our patients was excluded by the use of a praecordial stethoscope.

RESULTS

The overall frequency of cardiac arrhythmia was 31.6% (table III), 25.6% of ventricular origin and 6.1% were nodal rhythms. In some instances, more than one type of arrhythmia occurred in the same patient. In most cases, the arrhythmia had no clinical consequences, except for three who developed cardiovascular collapse; two with “torsades de pointes” (Krikler and Curry, 1976), and one with bigeminy and q.r.s. block. If we add to these bradycardia (22%) and tachycardia (4.9%) the total frequency of abnormality was almost 60%.

Associated factors

The underlying disease. Thirty patients had pituitary tumours or hydrocephalus. Twenty-two of them had intracranial hypertension as assessed by c.s.f. pressure measurement following lumbar puncture; 20 of these exhibited abnormalities (91%). Eight patients, including some in whom a shunt had been inserted for the relief of hydrocephalus, exhibited no e.c.g.

<table>
<thead>
<tr>
<th>E.c.g.</th>
<th>Total</th>
<th>%</th>
<th>Halothane spontaneous ventilation</th>
<th>Controlled ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Normal</td>
<td>33</td>
<td>40.2</td>
<td>25</td>
<td>43.1</td>
</tr>
<tr>
<td>Bradycardia (−25%)</td>
<td>18†</td>
<td>22.1</td>
<td>9</td>
<td>15.6*</td>
</tr>
<tr>
<td>Tachycardia (+25%)</td>
<td>4†</td>
<td>4.9</td>
<td>3†</td>
<td>5.2</td>
</tr>
<tr>
<td>Ventricular ectopic beats; idioventricular rhythm</td>
<td>21</td>
<td>25.5</td>
<td>19</td>
<td>32.7**</td>
</tr>
<tr>
<td>Nodal rhythm</td>
<td>5</td>
<td>6.1</td>
<td>2</td>
<td>3.4</td>
</tr>
<tr>
<td>T-wave changes</td>
<td>1†</td>
<td>1.2</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>100%</td>
<td>58</td>
<td>100%</td>
</tr>
</tbody>
</table>

* Significant (P < 0.005); ** significant (P < 0.02).
† Other abnormalities.
abnormality associated with PNE in the period after operation.

The arrhythmia was particularly frequent in patients with frontal lobe (3/4) and posterior fossa (4/7) tumours.

Anaesthesia. Arrhythmia of ventricular origin was more frequent in patients receiving halothane anaesthesia; bradycardia was more common in those undergoing IPPV.

Air injection. Arrhythmia was more frequent following the first injection of air which corresponds to the largest volume (20–40 ml in the adult). There appeared to be a direct relationship between the volume of air injected, the intracranial pressure and the occurrence of arrhythmia. In five patients, the removal of air, cerebrospinal fluid, or both, led to immediate recovery and a normal e.c.g.

Position. Arrhythmia was more frequent when the patient was placed in the head-down position to fill the posterior horns with air. This position results in a considerable increase in c.s.f. pressure.

CASE REPORTS
We present two typical cases of this series.

Case 1
A 35-yr-old female had diabetes insipidus and treated pituitary insufficiency. Skull radiograms revealed a normal sella tursica. Premedication was with diazepam 10 mg and atropine 0.5 mg i.m. Anaesthesia was induced with thiopentone, suxamethonium was given for tracheal intubation, and the patient was allowed to breathe spontaneously and 1% halothane in a mixture of nitrous oxide in oxygen.

Following the first injection of air, arterial hypotension occurred (80 mm Hg systolic) followed by respiratory arrest, then cardiovascular collapse. Idioventricular rhythm (“torsades de pointes”) was noted. Recovery followed manual control of ventilation with 100% oxygen, the administration of plasma expanders and the infusion of bicarbonate solution. There was no necessity for the removal of air (fig. 1).

Case 2
A 1-month-old child had hydrocephalus. Premedication was with atropine 0.25 mg i.m. Anaesthesia was induced with halothane, the trachea was intubated and the child breathed spontaneously using the Ayre’s T-tube. Following the third injection of air, alterations of the ST segment were noted on the e.c.g. There were Paredee-like waves, maximal within 3 min. Decompression resulted in immediate recovery. Throughout the events described, the patient was in the same sitting position. Unfortunately, no praecordial e.c.g. leads were recorded. It is therefore impossible to tell whether the alterations corresponded to an axis shift or were of ischaemic origin (fig. 2).

![Fig. 1. 35-yr-old female with craniopharyngioma. Halothane anaesthesia serum electrolyte values normal.](https://academic.oup.com/bja/article-abstract/50/8/833/249623/506883246823)
Disturbances of the central nervous system are known to be capable of inducing cardiac arrhythmia. Experimental head injury (McLaurin and Scott, 1975; Evans et al., 1976) increased intracranial pressure (Jachuck et al., 1975) and electrical stimulation of some areas of the brain (Weinberg and Fuster, 1960; Manning and Cotten, 1962; Porter, Kamikawa and Greenfoot, 1962; Lown and Verrier, 1976) have been shown to affect the heart rate and cause disturbances of the e.c.g. In clinical practice, emotion (Lown et al., 1976), the intracranial effects of hypertension (Connor, 1968; Jachuck et al., 1975), central nervous system disease (Burch, Collogough and Giles, 1962; Hugenholtz, 1962; Grossman, 1976), subarachnoid haemorrhage (Harrison and Gibb, 1964; Hersch, 1964; Srivastava and Robson, 1964; Sudhakaran and Menon, 1964; Beaufils, Bousser and Chapman, 1974; Estanol and Marin, 1975; Goldman, Rogers and Rogers, 1975; Van der Ark, 1975) and head injury (Falsetti and Moody, 1966) have been shown also to induce arrhythmia and even ventricular fibrillation. Millar and Abildskov (1968) found abnormal e.c.g. changes in 89 of 118 recordings in 41 patients with disease of the central nervous system.

Stimulation of the orbital cortex, the projection area of the vagus nerve, may affect repolarization of heart muscle (Cropp and Manning, 1960). Stimulation of the hypothalamus may induce various types of arrhythmia (Melville et al., 1963); the anterior area is associated with the parasympathetic system, its stimulation inducing bradycardia and affecting atrio-ventricular conduction. Stimulation of the posterior area causes tachycardia and hyperexcitability of the heart, which can be abolished by stellate ganglion blockade or by beta-blocking drugs (Weinberg and Fuster, 1960; Grossman, 1976; Lown and Verrier, 1976). This subject has been reviewed by Mauck and Hockman (1967).

Pneumoencephalography is known to induce various vasomotor or neurological complications,
such as headache, nausea, sweating and arterial hypotension. This is particularly marked when the procedure is undertaken without general anaesthesia and when the removal of cerebrospinal fluid is complete. Fractional pneumoencephalography has reduced the frequency of these undesired effects, although hypotension remains a problem. The present study highlights the cardiovascular problems. Our patients were free from cardiovascular disease, but it is obvious that special care should be taken in patients with pre-existing heart disease. The results of our investigations suggest that patients with frontal, posterior fossa and sellar tumours are particularly at risk.

It has been shown that halothane (McDowall, Barker and Jennett, 1966; Jennett, McDowall and Barker, 1967; Jennett et al., 1969; Fitch and McDowall, 1971; McDowall, 1975; Lassen and Christensen, 1976), ketamine (Gardner, Olson and Lichtiger, 1971; Gibbs, 1972; Shapiro, Wyte and Harris, 1972) and nitrous oxide (Henricksen and Jorgensen, 1973; Greenbaum et al., 1975) may cause an increase in intracranial pressure, particularly in patients with brain tumours, even when normal values of $P_{CO_2}$ are maintained. In our study, arrhythmia was more frequent in the patients who received halothane, while bradycardia was more frequent in the patients who received myoneural blocking drugs and narcotic analgesics. It is likely, however, that there are many other factors in addition to the techniques of anaesthesia and the presence of a tumour in the brain; the degree of intracranial hypertension is perhaps particularly worthy of mention.

Halothane is widely employed in neurological procedures (Gilbert, Brindle and Galindo, 1966; Way, Smith and Larson, 1974; McDowall, 1972; Hunter, 1975) although some authors have preferred not to employ this drug (McComish and Bodley, 1971; McDowall, 1972; Gordon, 1975). Pirhman and Shapiro (1977) have shown that previous administration of thiopentone prevents the increase in c.s.f. pressure induced by nitrous oxide. Campkin and Turner (1972) found that c.s.f. pressure was greater during halothane anaesthesia for PNE compared with controlled ventilation, and recommended the latter. Saidman and Eger (1965) and Gordon and Greitz (1970) have noted an increase in c.s.f. pressure following the introduction of nitrous oxide in the anaesthetic mixture during PNE. Therefore, there are good reasons to believe that controlled ventilation is preferable if an increase in c.s.f. pressure is to be avoided.

It is particularly notable, however, that in our study the e.c.g. findings were normal until air was injected.

Some authors (Michenfelder, Gronert and Rehder, 1969; McDowall, 1972) recommended sedation alone for PNE. However, the procedure is unpleasant and time-consuming, requiring heavy sedation which may depress both respiration and the circulation more than would occur during light anaesthesia. An additional disadvantage of the use of sedation is that control of the airway cannot be guaranteed.

Philippon, George and Metzger (1974) have shown that the intraventricular pressure increases considerably following the injection of air during PNE. Nitrous oxide has been recommended as a substitute for air because of its greater solubility in blood and other tissues (Saidman and Eger, 1965; Philippart, Thibaut and Bonnal, 1968; Gordon and Greitz, 1970; Campkin and Turner, 1972), although there is one report which implicates nitrous oxide as a cause of cardiac arrest (Collan and Iivanainen, 1969).

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REFERENCES


