CARDIAC RATE AND RHYTHM DURING ANAESTHESIA FOR DENTAL EXTRACTION

A comparison of Halothane, Enflurane and Isoflurane

W. R. CASSON AND R. M. JONES

The occurrence of cardiac arrhythmia during dental extraction under local or general anaesthesia is well documented (Kaufman, 1965, 1966; Hughes et al., 1966; Tuohy, 1968; Rollason and Dundas, 1970; Ryder, 1970). Although the aetiology of these disturbances of rhythm has not been fully elucidated, they are thought to result from an increase in cardiac sympathetic nervous activity, in response to surgical stimulation (Plowman, Thomas and Thurlow, 1974; Alexander and Murtagh, 1979). When compared with halothane-supplemented anaesthesia, the administration of enflurane is associated with less arrhythmia during dental extraction (Wright, 1980), and isoflurane-supplemented anaesthesia is known to be associated with greater stability of rhythm after the injection of adrenaline (Johnston, Eger and Wilson, 1976). We have studied the incidence of cardiac arrhythmia, and compared heart rates, during nitrous oxide in oxygen anaesthesia supplemented with halothane, enfurane or isoflurane.

PATIENTS AND METHODS

Sixty unpremedicated adults (ASA Grade I), scheduled to have dental extractions under general anaesthesia as out-patients, were allocated randomly to three groups to receive halothane, enflurane or isoflurane. Patients were excluded from the study if they were receiving concurrent drug therapy, if they had a history of cardiovascular disease, if they had any cardiac arrhythmia, or if they had a heart rate greater than 120 beat min⁻¹ or less than 60 beat min⁻¹ before the induction of anaesthesia. The age, weight and sex distribution, and operating times were similar in the three groups (table I).

<table>
<thead>
<tr>
<th></th>
<th>Halothane</th>
<th>Enflurane</th>
<th>Isoflurane</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>20</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Male</td>
<td>4</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Female</td>
<td>16</td>
<td>16</td>
<td>12</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>26.30(8.25)</td>
<td>22.65(6.01)</td>
<td>25.55(6.11)</td>
</tr>
<tr>
<td>Range</td>
<td>18–53</td>
<td>19–44</td>
<td>18–45</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>62.48(12.46)</td>
<td>62.40(11.03)</td>
<td>63.05(11.15)</td>
</tr>
<tr>
<td>Operating time (min)</td>
<td>17.15(8.71)</td>
<td>14.35(6.11)</td>
<td>19.30(9.92)</td>
</tr>
<tr>
<td>Range</td>
<td>7–44</td>
<td>6–29</td>
<td>8–45</td>
</tr>
</tbody>
</table>

Once the patient was settled on the operating table, an ECG monitor (5-lead) (Physio-Control Lifepak 6) was connected to the patient, and the cuff of an automatic non-invasive arterial pressure and pulse monitor (Dinamap) positioned on the right arm. After baseline recordings had been obtained a 23-gauge needle was inserted to a vein on the dorsum.
of the left hand, and anaesthesia induced with a dose of thiopentone sufficient to abolish the eyelash reflex. Suxamethonium 1 mg kg\(^{-1}\) was administered to facilitate the passage of a nasotracheal tube and a throat pack was placed in position. Maintenance of anaesthesia was with 66% nitrous oxide and halothane, enflurane or isoflurane (using a recently calibrated, temperature-compensated vaporizer (Fluotec Mark 3, Enfluratec or Fortec, respectively—Cyprane Ltd)) in oxygen, the concentration of the volatile agent being adjusted to give suitable operating conditions (using an inspired concentration between 1% and 3%). A Bain type co-axial breathing system was used, delivering a fresh gas flow sufficient to minimize rebreathing during spontaneous ventilation. Surgery commenced once normal sinus rhythm had been established. The end-tidal carbon dioxide concentration was measured with an infra-red analyser (Hewlett-Packard 47210 Capnometer with 14369A transducer). The electrocardiogram (Standard Lead 2) was displayed continuously until the end of surgery and a record obtained of any rhythm disturbance occurring in response to surgical stimulation. Arterial pressure and heart rate were measured and recorded at 1-min intervals.

### Table II. Incidence of arrhythmias. *Ventricular ectopics or supraventricular ectopics with aberrant conduction*

<table>
<thead>
<tr>
<th></th>
<th>Halothane</th>
<th>Enflurane</th>
<th>Isoflurane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular ectopics</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Coupled beats*</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Multifocal ectopics</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Supraventricular ectopics only</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Nodal rhythm</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>No arrhythmias</td>
<td>14</td>
<td>20</td>
<td>19</td>
</tr>
</tbody>
</table>

### RESULTS

Results are reported as mean (SD) as applicable. Statistical analysis utilized Fisher's exact probability test or Student's \(t\) test as appropriate. There were no significant differences in end-tidal carbon dioxide concentration between the three groups; \(P_{\text{CO}_2}\) did not exceed 6.6 kPa in any patient. The incidences of arrhythmia observed and recorded are shown in table II. The high incidence of arrhythmias (30%) during dental extractions in patients receiving halothane was confirmed: examples are shown in figure 1. Interpretation of this type of arrhythmia is
difficult, particularly the differentiation between supraventricular ectopic beats with aberrant conduction and ectopic beats of ventricular origin (Sandler and Marriott, 1965). A diagnosis of ventricular ectopics was made only if the added beat was immediately followed by either a P wave (that is, there was retrograde conduction from the ventricle) or by a definite compensatory pause. In the halothane group, four patients had coupled beats (in two patients these were of ventricular origin) and one patient had multi-focal ectopic beats at least some of which were of ventricular origin (fig. 1). Only one patient in the isoflurane group had any type of disturbance of rhythm, an occasional supraventricular ectopic beat (fig. 2). There were no arrhythmias observed in the enflurane group. Statistical analysis revealed a significantly higher incidence of arrhythmia in the halothane group compared with the isoflurane ($P < 0.05$) and the enflurane ($P < 0.05$) groups.

The heart rates observed and recorded varied considerably depending on the degree of surgical stimulation. Therefore, we felt that the most meaningful comparison would be made between the groups by comparing the mean heart rates in the three groups and then comparing all the individual readings. There were significant differences between the three groups: heart rates in the isoflurane group (range of means, 74–116 beat min$^{-1}$; overall mean, 101 beat min$^{-1}$; SD 17.8) were significantly higher than in both the enflurane ($P < 0.001$) and the halothane groups. Patients in the enflurane group (range of means, 75–112 beat min$^{-1}$; overall mean, 95 beat min$^{-1}$; SD 14.8) had significantly faster ($P < 0.001$) heart rates than those in the halothane group (range of means, 72–97 beat min$^{-1}$; overall mean, 82 beat min$^{-1}$; SD 15.9). This is represented graphically in figure 3, where the number of observations of a certain heart rate is plotted against that rate. This also demonstrates that the heart rates recorded in the patients receiving halothane showed less variability when compared with the two other groups.

**DISCUSSION**

A number of factors contribute to the disturbances of cardiac rhythm which may occur during oral surgery. These include hypoxia, hypercarbia and increased sympatho-adrenal activity (Tuohy, 1968). Their incidence is decreased by prior infiltration of the operation site with lignocaine (Kaufman, 1965) and by beta-adrenergic antagonists both with (Tolas and Allen, 1970) and without (Rollason and Hall, 1973; Rollason and Russell, 1980) membrane stabilizing activity. Similar disturbances of rhythm have been observed by neuroanaesthetists on manipulation of the fifth cranial nerve during surgery for trigeminal neuralgia (Ballantine and Jackson, 1960; Hunter, 1975). These observations would suggest that increased sympathetic nervous activity or increased release of catecholamines from the adrenal glands, or both, in response to surgical stimulation are involved in the genesis of these arrhythmias. It
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50-,

Halothane
Enflurane
Isoflurane

40-
30-
20-
10-

479

FIG. 3. Comparison of heart rates.

has also been suggested that there is a concurrent increase in parasympathetic activity (Taggart et al., 1976). The incidence of cardiac arrhythmia during general anaesthesia for dental extractions is increased by the administration of atropine (Thurlow, 1972) and when halothane, as opposed to enflurane, is used to supplement nitrous oxide in oxygen anaesthesia (Wright, 1980). None of the patients in our study received an anti-cholinergic agent, none reached an end-tidal carbon dioxide concentration near the so called “arrhythmic threshold” (Black et al., 1959) and all patients breathed 30% oxygen via an endotracheal tube. Although halothane itself has been reported to be associated with an increase in circulating noradrenaline (Joyce et al., 1982) this does not occur after the i.v. induction of anaesthesia (Joyce, Roizen and Eger, 1981). The injected dose of adrenaline which is required to produce ventricular extrasystoles is known to be reduced during halothane anaesthesia compared with both enflurane and isoflurane anaesthesia (Reisner and Lippmann, 1975; Johnston, Eger and Wilson, 1976). Thus, if the cause of arrhythmia during general anaesthesia for oral surgery is indeed an increase in sympathetic adrenal activity, one would expect the incidence to increase during halothane anaesthesia.

Cardiac arrhythmias result from an alteration in automaticity or re-entry of the cardiac impulse. Pratila and Pratilas (1978) have emphasized that the sensitization of the heart to catecholamines which leads to the occurrence of arrhythmias has, as an underlying mechanism, re-entry of the cardiac impulse. The likelihood of re-entry occurring is increased in any situation where there is slowing of conduction resulting in temporary uni-directional block (Jones, Broadbent and Adams, 1984). Of the three volatile agents used in this study, halothane causes more slowing of A-V nodal and His–Purkinje conduction than the other two and appears to be the only one to cause slowing of ventricular conduction (Atlee and Peterson, 1982). Indeed, isoflurane causes the least slowing of A-V nodal conduction and has little or no effect on His–Purkinje conduction. Thus, as Jones (1984a) has noted previously, the conditions for a re-entry arrhythmia are more likely to occur during halothane, as distinct from enflurane and isoflurane, anaesthesia. Our study has confirmed the high incidence (30%) of arrhythmia during dental extractions when halothane is used.

As it appears that ventricular fibre conduction is minimally affected by either enflurane or isoflurane, premature ventricular contractions would be equally unlikely to occur during enflurane and iso-
flurane anaesthesia. Our study has confirmed this electrophysiologically-based prediction (the only disturbance of sinus rhythm recorded being an occasional supraventricular ectopic beat in one patient anaesthetized with isoflurane).

The occurrence of a tachycardia during isoflurane anaesthesia has been noted previously (Skovsted and Saphavichaikul, 1977); this is associated with a decrease in systemic vascular resistance, a decrease in stroke volume and maintenance of cardiac output (Stevens et al., 1971). Two factors may contribute to the tachycardia associated with isoflurane anaesthesia (Jones, 1984b): first, systemic vascular resistance is markedly decreased, and second, baroreflex activity remains relatively intact except at deeper levels of isoflurane anaesthesia (Skovsted and Saphavichaikul, 1977; Seagard et al., 1983; Kotrly et al., 1984). Therefore, one might expect that patients anaesthetized with isoflurane would have a more rapid heart rate than patients anaesthetized with enflurane or halothane, both of which cause significant depression of baroreflexes (Duke, Fowines and Wade, 1977; Morton, Duke and Ong, 1980). One might expect halothane anaesthesia to be associated with the lowest mean heart rate, since there is a suggestion that this agent causes the most significant slowing of S-A node discharge (Merlos et al., 1980). In addition, enflurane might be expected to be associated with an intermediate range of heart rates, since this agent causes less slowing of S-A node discharge than halothane and also significantly depresses baroreflexes. In our study the comparison of heart rates confirmed these expectations.

In summary, both enflurane and isoflurane have been shown to be associated with less cardiac arrhythmia than halothane when used to supplement nitrous oxide in oxygen anaesthesia for dental extractions. Isoflurane was associated with a higher heart rate than the other two agents, and enflurane with a more rapid rate than halothane. Isoflurane may be the agent of choice when enflurane is relatively contraindicated, for instance in patients with epilepsy (Linde et al., 1970).

ACKNOWLEDGEMENTS

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