ARRHYTHMIA AND ORAL SURGERY: INDUCTION OF ANAESTHESIA WITH ALTHESIN

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SUMMARY

Induction of anaesthesia with Althesin appears to offer a significant protective effect against the cardiac arrhythmias which may follow the stimulus of oral surgery.

The high incidence of cardiac arrhythmias during oral surgery has received considerable attention in recent years (Kaufman, 1965, 1966; Ryder, 1971; Alexander, 1971; Thurlow, 1972; Rollason and Hall, 1973). Dodds and Twissell (1972) showed that the recently introduced intravenous anaesthetic Althesin protected the cat heart against arrhythmias induced by the combination of halothane and adrenaline. Warren (1972) considered that the drug had an anti-arrhythmic action on the heart in patients undergoing outpatient dental extractions.

The purpose of this paper is to present evidence that induction of anaesthesia with Althesin has a protective effect against arrhythmias induced by the stimulation of oral surgery in man.

METHODS

One hundred male inpatients were studied. Their ages ranged from 13 to 62 years and the age distribution was almost identical with that reported in a previous study (Alexander, 1971). All were physically fit and had a normal cardiovascular system on clinical examination. The commonest operation was removal of impacted wisdom teeth (48 patients), the remainder had multiple extractions or removal of unerupted or supernumerary teeth. Nine patients underwent interdental wiring for fractured jaw. The majority of operations lasted from 60 to 90 min and the patients were supine.

Premedication was atropine 0.6 mg i.m. given 1 hour before surgery.

Anaesthesia was induced with Althesin 0.075 ml/kg injected intravenously at a rate of approximately 0.5 ml/sec, followed by suxamethonium 50 mg to aid nasotracheal intubation. The throat was packed. Anaesthesia was maintained with nitrous oxide 5 litre/min and oxygen 3 litre/min, with halothane (2% reducing to 1% or less) from a Fluotec vaporizer, via a Magill circuit. Ventilation was spontaneous on recovery from the suxamethonium.

Arterial systolic pressure was recorded using a sphygmomanometer cuff and radial pulse palpation, before induction of anaesthesia and as soon as the patient was settled following intubation. Cardiac rhythm was monitored continuously with a Cardiocorder oscilloscope and intermittently recorded using a Miniwriter pen recorder (Cardiac Recorders Ltd). Standard lead II was used throughout.

RESULTS

The results are summarized in table I. Sinus tachycardia after induction was notable, the mean increase in heart rate being 48 beats/min, representing an average increase of 60% (SD 53.3) over control values. In 73 patients the heart rate increased to 120 beats/min and in 24 of these a further increase to 150 beats/min or more was seen. Arterial systolic pressure showed a mean increase of 10.1 (SD 19.5) mm Hg (range —30 to +60 mm Hg).

E.c.g. changes immediately after induction were seen in 15 patients, with a decreased amplitude or disappearance of the T wave lasting from 15 sec to 5 min. Junctional (“nodal”) rhythm was noted in 4 patients. In 1 patient both the P and T waves disappeared, the latter reappearing after 1 min.

During anaesthesia, atrioventricular dissociation (junctional or “nodal” rhythm) occurred in 33 patients in association with either breathing the higher concentrations of halothane or surgical stimulation. Twenty-nine patients responded to the stimulus of surgery with a tachycardia of 120 beats/min and in 4 the heart rate increased to 150 beats/min or more.
TABLE I.  Rhythm changes during anaesthesia in 100 patients receiving anaesthesia with Althesin.

<table>
<thead>
<tr>
<th>Arrhythmias</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia (beats/min)</td>
<td></td>
</tr>
<tr>
<td>&gt;120</td>
<td>73</td>
</tr>
<tr>
<td>&gt;150</td>
<td>24</td>
</tr>
<tr>
<td>During intubation</td>
<td></td>
</tr>
<tr>
<td>Fusion beats (also during extubation)</td>
<td>1</td>
</tr>
<tr>
<td>During anaesthesia and surgery</td>
<td></td>
</tr>
<tr>
<td>A-V dissociation (junctional or “nodal” rhythm)</td>
<td>33</td>
</tr>
<tr>
<td>Ventricular ectopic (also observed preoperatively)</td>
<td>1</td>
</tr>
<tr>
<td>During surgical stimulation</td>
<td></td>
</tr>
<tr>
<td>A-V dissociation with occasional bigeminy</td>
<td>2</td>
</tr>
<tr>
<td>Junctional beats</td>
<td>1</td>
</tr>
<tr>
<td>Junctional ectopic (total less than 20)</td>
<td>3</td>
</tr>
<tr>
<td>Junctional ectopic (total more than 20) with</td>
<td></td>
</tr>
<tr>
<td>tachycardia and ventricular aberration</td>
<td></td>
</tr>
</tbody>
</table>

One patient developed ectopic complexes during induction of anaesthesia, and in a further 10 patients abnormal rhythms were observed during surgery. The majority of these were junctional in origin (table I), and in 3 patients, each of whom had more than a total of 20 ectopic beats, these were associated with tachycardia (120 or more beats/min) and ventricular aberration (fig. 1, A and B). In 1 patient an attempt to abolish the arrhythmia by increments of Althesin (two doses each of 1 ml) changed the rhythm temporarily from a junctional escape rhythm to junctional beats alternating with ectopic complexes (fig. 2, A and B).

![Fig. 1](image1.png)

**Fig. 1.** (A) Bigeminal junctional ectopies during removal of a wisdom tooth in a 20-year-old male. The ectopic complexes show aberrant intraventricular conduction. Rate 120/min.

(B) The same patient during removal of a second wisdom tooth. The arrow marks the start of a short period of bidirectional tachycardia, rate 150/min. There is increased aberration. F is a fusion beat. The last four complexes are of normal configuration although only the last two are preceded by P waves with short and varying P-R intervals.

![Fig. 2](image2.png)

**Fig. 2.** (A) Junctional escape rhythm with two junctional ectopies marked E. Rate 120–150/min. The arrow denotes 1 ml Althesin injected i.v. After 6.6 sec the rhythm changes in one cycle length to (B) junctional beats alternating with ectopic complexes; rate 105/min. The ectopies exhibit varying aberration. (A) and (B) are continuous. A further increment of Althesin 1 ml after 1 min. failed to restore normal rhythm.

**DISCUSSION**

Induction of anaesthesia with Althesin under the conditions of this study (endotracheal intubation aided by suxamethonium) led to a small mean increase in arterial systolic pressure and a marked increase in heart rate (60% over control values). In 15 patients there was a decreased amplitude or disappearance of the T wave of the electrocardiogram for short periods of time, a change ascribed to the induced tachycardia.

Althesin appears to confer a significant protective effect upon the heart against arrhythmias induced by oral surgery if the present data are compared with an incidence of 23% in a previous study by the author (Alexander, 1971), where thiopentone, methohexitone or propanidid were used as induction agents ($\chi^2=5.95$, P<0.05). This protective effect becomes highly significant if the number of patients having a total of 20 or more ectopic beats (3% and 17% respectively) is considered ($\chi^2=12.84$, P<0.001).

Although Cundy (1973) found Althesin capable of restoring normal rhythm in an anaesthetized patient with an established arrhythmia, this treatment failed in the one patient in whom it was tried in this study.

The results of this investigation again confirm the increased automaticity of the atrioventricular junctional tissues during oral surgery under light halothane anaesthesia. It has been noted frequently that the increase in heart rate in response to surgery reaches a frequency of 120 or more beats/min before ectopic rhythms occur. These are often associated with aberrant interventricular conduction which gives rise to complexes which may be mis-
taken for ventricular extrasystoles (Schamroth, 1971).

Rosenbaum, Elizari and Lazzari (1969), have shown that alternating block of the bundles of the His-Purkinje system can be rate-dependent. The majority of their patients had diffuse myocardial damage or strong digitalis effect, but it would appear that similar conditions can pertain in the healthy heart during reflex sympathetic stimulation and light halothane anaesthesia, the junctional tissues being excited while the conducting tissues are inhibited (Alexander, Bekheit and Fletcher, 1972). These are suitable conditions for re-entry of impulses into the junctional tissue with perpetuation of the abnormal rhythm (Moe and Mendez, 1973).

The significance of these arrhythmias is still not clear. Morton and Bekheit (1974, personal communication) consider that junctional ectopic rhythms in the healthy young heart are benign and that fast heart rates can be tolerated for hours or even days. It seems difficult to believe that such abnormal rhythms can be harmless under all circumstances, particularly since outside anaesthesia they occur most commonly in hearts which are diseased or suffering from some form of toxicity. A definitive answer will require further observation and investigation.

Althesin, although used merely for induction of anaesthesia, seems to confer increased stability to the conducting tissues of the heart during the ensuing surgery. Its use, under the circumstances of this study, was accompanied by a very marked induction tachycardia which must put a severe strain on a heart with limited powers of compensation.

ACKNOWLEDGEMENTS

I am grateful to Dr S. Bekheit of the Cardiological Investigation Department of the Belfast City Hospital for her help in interpreting the electrocardiograms and to Mr C. McKay for access to his patients.

REFERENCES


