and white women. Analysis of variance showed that the described angle was larger in black than in white people and larger in black women than in black men. This implies that needle insertion for caudal anesthesia would require different angles in the two races. The height of the caudal canal was greater in black than in white people. In black women the height was, on the average, 6 mm compared with 5 mm in white women. These numbers are in agreement with the results by Trotter, but several investigators who measured large numbers of black and white people (alive or as skeletons) showed that the black population was less varied in physical dimensions than the white population. These studies dealt primarily with features of the face and pelvis. Some morphologic features of black people are very stable, and are expressed even when mixed with other races.

What causes the structural differences of the sacrums of the two races is not known. A pronounced lumbar lordosis in black people has been well described, and we assume that this hyperlordosis is connected with a slightly greater forward tilt of the pelvis. However, the forward tilt is not simply a matter of posture, but rather is determined by structural features of the vertebrae and the pelvis.

The results of our measurements may have clinical implications. On the average, in the black patient, the insertion of a needle for caudal anesthesia should be a little easier, because the caudal canal is higher and the needle will be at a steeper angle than in white patients.

REFERENCES

Cardiac Arrhythmias during Coronary-artery Operations with Halothane or Enflurane Anesthesia

HAMPTON D. WILLIAMS, M.D.,* AND LAW SONE, JR., M.D.†

Halothane and enflurane, two commonly used halogenated anesthetic agents, have been shown to potentiate ventricular arrhythmias. In this study a retrospective evaluation was undertaken to compare the incidences of ventricular arrhythmias occurring during coronary-artery operations with halothane and with enflurane anesthesia. The influence of halogenated anesthetics upon cardiac electrical activity has been attributed to numerous factors, including endogenous and ex-
Table 1. Incidences of Arrhythmias and Distribution of Patients by Age and Sex

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of Patients</th>
<th>Age (Years)</th>
<th>Patients with Arrhythmias*</th>
<th>Per Cent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>Range</td>
<td>Per Cent</td>
</tr>
<tr>
<td>Group I.</td>
<td>92</td>
<td>55.9</td>
<td>37-78</td>
<td>87</td>
</tr>
<tr>
<td>halothane</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group II.</td>
<td>113</td>
<td>57</td>
<td>36-73</td>
<td>82.3</td>
</tr>
</tbody>
</table>

* Difference not significant; X² = .32.
† Difference significant, P < 0.02; X² = 6.05.

ogenous catecholamines, carbon dioxide accumulation, oxygen content, arterial blood pressure, heart rate, electrolyte status, drug administration, and anesthetic concentration and depth. Enflurane seems to have less potential than halothane for arrhythmia production in the face of exogenous epinephrine injection. Implications have thus been made that during halothane or enflurane anesthesia in which no exogenous catecholamine is administered, a difference in the potentials for ventricular arrhythmias could be observed. To date, however, no one has demonstrated any difference between the occurrences of arrhythmias in man undergoing surgical procedures with the two anesthetics.

Methods

A retrospective analysis was made of two groups of patients with angiographically proven coronary-artery disease who underwent coronary-artery revascularization at this medical center. Patients eliminated from the study were those who underwent additional simultaneous surgical procedures such as valve replacement or ventricular aneurysmectomy. Uniformity of medical management and recording of data in the two comparison groups were enhanced by including in the study only those patients whose anesthesia was administered by one of three anesthesiologists and whose revascularization was performed by one of four surgeons. The three anesthesiologists are members of anesthesiology group, and the four surgeons are members of a cardiovascular surgery group, so that a high degree of procedural standardization existed throughout the study. There was no change in surgical or cardiopulmonary bypass techniques during the study.

Group I consisted of 92 patients who underwent revascularization during the first six months of 1976 and received halothane as the primary anesthetic agent. Group II consisted of 113 similar patients anesthetized with enflurane during the first six months of 1977.

Every patient had continuous monitoring of the electrocardiogram, arterial pressure, central venous pressure, and esophageal temperature. Electrolytes were measured during cardiopulmonary bypass and serial arterial blood-gas determinations were performed before, during and after bypass. Patients were considered to need treatment for ventricular arrhythmias when they manifested: 1) frequent ventricular premature beats (>6/min) or multifocal ventricular contractions; 2) ventricular tachycardia; or 3) ventricular fibrillation. These patients were treated with various combinations of lidocaine, propranolol, potassium chloride, procainamide, and electrical countershock. The observation of any of the above-mentioned arrhythmias and the institution of treatment qualified a patient for categorization in the group manifesting ventricular arrhythmias. Regardless of subsequent arrhythmic activity a patient was tabulated only once. Not included in the "arrhythmia" group were patients who had isolated extrasystoles or patients who had brief runs of extrasystoles that spontaneously abated before treatment could be instituted. Except for those necessitating treatment to allow termination of bypass, arrhythmias occurring during cardiopulmonary bypass were not included in the study.

Preoperative medication consisted of an anticholinergic, a narcotic, and a sedative such as diazepam, droperidol, or hydroxyzine. A few patients in each group experienced hypertension and tachycardia associated with chest pain or diaphoresis prior to induction of anesthesia. These patients were treated with sodium nitroprusside, propranolol and, in some cases, diazepam until they were normotensive and asymptomatic before induction of anesthesia. While administering oxygen, anesthesia was induced with sodium thiopental, followed by introduction of the respective inhalational agent, which was administered for approximately 5 min. Muscular relaxation was achieved with pancuronium, .08 mg/kg, or gallamine, 1 mg/kg, and, in some patients, succinylcholine, 1-1.5 mg/kg. The trachea was sprayed with 4 ml or 4 per cent lidocaine and, after 1 min, orotracheal intubation was performed. Anesthesia was maintained with halothane, 0.5-3 per cent, or enflurane, 1-3 per cent, with a semiclosed circle absorber system. Ventilation was controlled throughout the procedure, and additional muscle relaxant was administered as needed to maintain diaphragmatic immobility.

During cardiopulmonary bypass, anesthesia was maintained with 0.5 per cent halothane or 1.0 per cent enflurane delivered into the pump-oxygenator and additional muscle relaxant as required. Normothermia was maintained during bypass, although
cardiac hypothermia was achieved using iced Ringer's solution. Ventricular fibrillation was produced with hypothermia or alternating current. Following completion of the distal anastomoses, the heart was electrically defibrillated. After completion of the proximal anastomoses and establishment of normal cardiac rhythm and output, cardiopulmonary bypass was withdrawn.

Differences in the distribution of male-to-female patients in each group and the incidence of ventricular arrhythmias in each group were subjected to $2 \times 2$ contingency-table chi-square analysis, with $P < 0.05$ considered significant.

**RESULTS**

The average age and range of ages in years in each group is given in table 1. The ratio of male-to-female patients in each group is also shown; the difference was not significant.

The incidence of ventricular arrhythmias necessitating treatment was found to be 18 of 92 patients (19.6 per cent) in the halothane group. This can be compared with the finding of eight of 113 patients (7.1 per cent) with arrhythmias in the enflurane group. This difference is significant ($P < 0.02$).

**DISCUSSION**

In the presence of halogenated anesthetics there are many possible factors that could alter cardiac electrical activity and lead to arrhythmia production. The exact mechanism of ventricular arrhythmias during halothane or enflurane anesthesia is not certain. Early theories dealing with halothane proposed that the anesthetic agent sensitized the myocardium to the effects of epinephrine, leading to enhancement of automaticity at a ventricular pacemaker site. Later studies have shown that with halothane, ventricular automaticity is actually suppressed. This has led to the suggestion that altered conduction by a re-entry of excitation mechanism through the atrioventricular (AV) node is causative. Recent studies by Atlee et al. concerning AV nodal conduction have shown that enflurane and halothane have in common prolongation of AV nodal conduction time; however, enflurane had a sparing effect upon His–Purkinje and ventricular conduction times, which were shown to be prolonged by halothane. This was proposed as a possible basis for the clinical impression of fewer ventricular arrhythmias occurring during enflurane than during halothane anesthesia.

There are numerous indications in the medical literature that ventricular arrhythmias occur less often with enflurane than with halothane anesthesia. This difference has not been previously demonstrated in man without the administration of exogenous catecholamines. A retrospective analysis was made of two similar groups of patients undergoing coronary-artery revascularization with halothane and with enflurane anesthesia. Significantly fewer arrhythmias occurred in the enflurane group.

**REFERENCES**