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Anesthetic Management of the Wolff-Parkinson-White Syndrome

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The Wolff-Parkinson-White (WPW) syndrome and its variants are called the pre-excitation syndrome. The anesthetic management of patients with this syndrome is aimed at avoiding tachyarrhythmias. Katz and Kadir advocate minimal circulatory disturbance using a nitrous oxide, oxygen, and narcotic technique. Similarly, on the basis of one case discovered intraoperatively, van der Starre recommended neuroleptanalgesia and avoiding drugs with negative inotropic effects on the heart. Conversely, Kumazawa advised using deep inhalational anesthesia. We have recently anesthetized 13 patients with the pre-excitation syndrome, and our experience supports the latter opinion.

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RESUMÉ OF THIRTEEN CASES

Nine of the 15 episodes of anesthesia in these 13 patients were for surgical treatment of arrhythmias (His- or Kent-bundle divisions) refractory to medical therapy. All patients except Patient 4 were known to have the syndrome, and had had episodes of tachyarrhythmias (table 1).

 Patients 1 and 2 received morphine (1 mg/kg) for induction of anesthesia with the addition of halothane (0.5–1.0 per cent) and N₂O after endotracheal intubation. They had no arrhythmias. Patient 3 received anesthesia with morphine (1 mg/kg), diazepam, 10 mg, N₂O, and pancuronium. Episodes of supraventricular tachycardia appeared soon after incision of the skin, necessitating cardioversion more than ten times. Patient 4 was managed by a similar anesthetic technique, and cardioversion was done for atrial fibrillation, with a rapid ventricular rate. A few days later, after the heart rate was controlled with propranolol, Patient 4 underwent uneventful repair of an abdominal fistula with morphine, N₂O, and d-tubocurarine.

The remaining nine patients received inhalational anesthesia with halothane or enflurane. Blood pressure and heart rate were maintained at or below preoperative values. For all but Patient 10, d-tubocurarine was used. Only Patient 5 had an intraoperative arrhythmia, during cardiac manipulation for vena caval cannulation.

DISCUSSION

Excitation of the heart is diagrammed in figure 1A. The impulse spreads from the sinus node through the atrium, undergoes physiologic delay at the atrio-ventricular (A-V) node, then passes through the His bundle to the Purkinje network. Characteristic of the pre-excitation syndrome is premature activation of a portion of ventricular muscle. The common denominator in all forms is the presence of an anomalous conduction pathway that bypasses the A-V node. The classic form of pre-excitation in Wolff-Parkinson-White syndrome is depicted in figure 1B. The sinus impulse is conducted simultaneously down an anomalous pathway (bundle of Kent) and the normal pathway. The lack of physiologic delay in the anomalous pathway accounts for the short PR interval. Ventricular excitation is a composite of the two impulses; thus, a fusion beat containing a delta wave accounts for the QRS prolongation. Variations of the pre-excitation syndrome include the presence of some, but not all, of the above electrophysiologic features. The Lown-Ganong-Levine (LGL) syndrome

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also has a short PR interval, but with a normal QRS configuration. The underlying mechanism is an anomalous tract, the James pathway, which bypasses the area of physiologic delay in the A-V node and inserts into the His bundle (fig. 1C). Another variant of the pre-excitation syndrome is characterized by a normal PR interval with a delta wave. This results from anomalous fibers (Mahaim fibers) arising below the area of physiologic delay and inserting directly into the ventricular muscle (fig. 1D).

The clinical significance of the syndrome lies in the disabling tachycardias and associated cardiac anomalies. Cardiac anomalies include balloon mitral valve, Ebstein’s anomaly and coronary-artery disease. The most frequently seen arrhythmia in patients with Wolff-Parkinson-White syndrome is a rapid, regular tachycardia of 120–230 beats/min. This tachycardia may be accompanied by chest pain, congestive heart failure, or syncope.† Tachycardias are of the re-entrant type, with antegrade conduction down the normal pathway and retrograde conduction up the anomalous pathway. The arrhythmia most commonly encountered in Lown-Ganong-Levine syndrome is atrial flutter-fibrillation, with rapid conduction over the accessory pathway. Medical therapy modifies the pathways involved in the re-entrant phenomena and reduces the number of ectopic beats.† Quinidine

### Table 1. Operative Procedures and Anesthesia for Patients with the Pre-excitation Syndrome

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Operation</th>
<th>Anesthesia</th>
<th>Arrhythmia</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>63</td>
<td>His-bundle division</td>
<td>Morphine, halothane, N₂O, pancuronium</td>
<td>No</td>
<td>WPW* and coronary-artery disease</td>
</tr>
<tr>
<td>52</td>
<td>His-bundle division</td>
<td>Morphine, halothane, N₂O, pancuronium</td>
<td>No</td>
<td>WPW; rheumatic heart disease and previous mitral valve replacement ×2</td>
</tr>
<tr>
<td>26</td>
<td>Kent-bundle division and mitral annuloplasty</td>
<td>Morphine, diazepam, N₂O, pancuronium</td>
<td>Yes</td>
<td>WPW; countershocked more than ten times for supraventricular tachycardia</td>
</tr>
<tr>
<td>51</td>
<td>Intestinal resection</td>
<td>Morphine, N₂O, pancuronium</td>
<td>Yes</td>
<td>Lown-Ganong-Levine syndrome diagnosed by anesthesiologist intraoperatively</td>
</tr>
<tr>
<td>42</td>
<td>Fistula repair</td>
<td>Morphine, N₂O, d-tubocurarine</td>
<td>No</td>
<td>Atrial fibrillation controlled preoperatively with propranolol and digitalis</td>
</tr>
<tr>
<td>59</td>
<td>His-bundle division</td>
<td>Halothane, N₂O, d-tubocurarine</td>
<td>Yes</td>
<td>WPW; arrhythmia occurred during cardiac manipulation</td>
</tr>
<tr>
<td>64</td>
<td>Kent-bundle division</td>
<td>Halothane, N₂O, d-tubocurarine</td>
<td>No</td>
<td>WPW; supraventricular tachycardia persistent postoperatively</td>
</tr>
<tr>
<td>53</td>
<td>Kent-bundle division</td>
<td>Halothane, N₂O, d-tubocurarine</td>
<td>No</td>
<td>WPW</td>
</tr>
<tr>
<td>31</td>
<td>Kent-bundle division</td>
<td>Enflurane, N₂O, d-tubocurarine</td>
<td>No</td>
<td>WPW</td>
</tr>
<tr>
<td>36</td>
<td>Replacement of ascending aorta and aortic valve</td>
<td>Halothane, N₂O, pancuronium</td>
<td>No</td>
<td>WPW and Marfan’s syndrome</td>
</tr>
<tr>
<td>4</td>
<td>Removal of Wilms’ tumor</td>
<td>Enflurane, N₂O, d-tubocurarine</td>
<td>No</td>
<td>WPW</td>
</tr>
<tr>
<td>3½</td>
<td>Cystoscopy</td>
<td>Enflurane, N₂O</td>
<td>No</td>
<td>WPW</td>
</tr>
<tr>
<td>38</td>
<td>Transphenoidal hypophysectomy</td>
<td>Enflurane, N₂O</td>
<td>No</td>
<td>WPW; 12 ml 1 per cent lidocaine with epinephrine (1/100,000 injected into mucosa) without arrhythmia</td>
</tr>
</tbody>
</table>

* WPW = Wolff-Parkinson-White syndrome.
and procainamide increase the block in the anomalous pathway, reducing the chance of re-entrant tachycardia. This effect on the anomalous pathway also reduces ventricular response in patients with atrial fibrillation. Digitalis and propranolol block the re-entry circuit by increasing the refractory period of the normal pathway. Intravenous digitalis may shorten the refractory period of the accessory pathway and should not be used for atrial fibrillation. Intravenous administration of propranolol is especially useful intraoperatively. Various combinations of the drugs and their dosages should be tailored for each patient. An attempt at surgical interruption of the accessory pathway is considered in selected patients unresponsive to medical therapy. Before operation, electrophysiologic evaluation is performed at cardiac catheterization. At operation, epicardial mapping during cardiopulmonary bypass identifies the anomalous pathway.

Successful anesthetic management depends on avoiding tachyarrhythmias. We continue all preoperative antiarrhythmic medication until the time of operation and use morphine, scopalamine, and diazepam for premedication. The use of atropine has been discouraged by some. Atropine can produce normal A-V conduction with disappearance of the delta wave, as well as disturbing tachycardias. We administered atropine, 0.4 mg, intraoperatively, to two patients, producing 25 per cent increases in heart rate and no conduction change. For induction, Katz and Kadis advise cautious use of thiopental to avoid aberrant conduction. Thiopental, 1–3 mg/kg, was used in 14 of our anesthetics, without conduction problems. Cyclopropane, halothane, enflurane, methoxyflurane, neuroleptanalgesia, and nitrous oxide and narcotic anesthesia have all been used for patients with the pre-excitation syndrome. Glesing recommended anesthetic techniques that do not increase blood catecholamine levels. Kumazawa advised using deep anesthesia with halothane or methoxyflurane. For nine patients, we used halothane and enflurane, which presumably reduced sympathetic outflow, and only one arrhythmia occurred in those patients. For muscle relaxation d-tubocurarine is less likely to cause tachyarrhythmia than is pancuronium.

Intraoperatively, vagal stimulation is infrequently successful in breaking the tachycardia. Intravenous administration of propranolol can be very helpful in slowing tachycardia intraoperatively. When the arrhythmia is associated with hypotension, direct-current cardioversion should be instituted. In two of our patients, arrhythmias were refractory to all therapy, and only after cardiopulmonary bypass and surgical correction did the arrhythmias cease.

Successful anesthetic management of the patient with Wolff-Parkinson-White syndrome or one of its variants is predicated upon understanding the electrophysiologic and clinical manifestations. The anesthetic technique should be tailored to avoid sympathetic stimulation, which may lead to tachyarrhythmias. We used halothane and enflurane to maintain blood pressure and heart rate at or below preoperative values. Although other anesthetic approaches may be suitable, our experience supports the use of moderate to deep levels of inhalational anesthesia to provide stability of rhythm.

References

LDH<sub>5</sub> Changes after Cholecystectomy or Hysterectomy in Patients Receiving Halothane, Enflurane, or Fentanyl

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The LDH<sub>5</sub> isoenzyme fraction of lactate dehydrogenase is felt to be relatively specific for hepatocellular injury.1 Klar et al.2 measured LDH<sub>5</sub> changes in the first 24 hours after elective cholecystectomy and reported this isoenzyme increased more and remained elevated longer after halothane than after methoxyflurane or thiopental–meperidine anesthesia. They suggested their data were consistent with a selective hepatotoxic effect produced by halothane. In contrast, another report failed to document a detrimental effect of halothane when administered to patients undergoing cholecystectomy.3

In view of these conflicting reports, we elected to repeat the study of Klar et al. by again determining LDH<sub>5</sub> values before and after elective cholecystec-
tomies performed with halothane–N<sub>2</sub>O anesthesia. In addition, LDH<sub>5</sub> measurements were extended to include an additional procedure (hysterectomy) and two other commonly used anesthetic drugs (enflurane and fentanyl).

METHODS

Sixty nonobese adult patients undergoing elective cholecystectomy with intraoperative cholangiography (30 patients) or abdominal hysterectomy (30 patients) were studied. The study protocol was approved by the

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