before anesthetic induction reliably elevated the gastric fluid pH to far above 2.5. This desirable effect must be weighted against the possible hazards of increased gastric fluid volume in patients pre-treated with an antacid. Routine antacid therapy may be reasonable to consider in view of the fact that 16–17 per cent of fasted patients awaiting elective operations were found to be at risk should aspiration have occurred (i.e., gastric fluid pH below 2.5 and volume of more than 20 ml). Certainly, antacids would seem indicated when a cuffed endotracheal tube is not placed or difficulty and prolonged anesthetic induction or tracheal intubation is anticipated.

REFERENCES

Anesthesiology

Wolff–Parkinson–White Syndrome during Anesthesia

P. J. A. van der Starre, M.D.*

A major problem in anesthetic practice is the diagnosis and treatment of cardiac arrhythmias, especially when they arise for the first time during general anesthesia. Since it was first described in 1930, the Wolff–Parkinson–White syndrome remains one of the most interesting and, at the same time, one of the most difficult of the cardiac arrhythmias to treat. We report a case in which this syndrome arose entirely unexpectedly during general anesthesia, and offer some recent considerations of the subject.

REPORT OF A CASE

A healthy 22-year-old man was scheduled for arthroscopy of the right knee with general anesthesia. There was a history of mild asthmatic bronchitis, but the patient had no complaint of any kind except signs and symptoms related to his knee. Clinical examination, chest x-ray and routine laboratory investigations revealed no abnormality. An ECG was not obtained because the patient was less than 30 years of age and there was no specific indication for it.

Premedication: Atropine, 0.5 mg, and promethazine, 50 mg were given.

Induction: When the patient was connected to Lead 1 of the ECG, before induction of anesthesia, as is our practice, some supraventricular extrasystoles were seen, probably due to an accessory pathway (fig. 1A). The patient denied feeling palpitations or any abnormality of his heart beat. Blood pressure was normal. Induction was commenced with Clemastine, 2 mg, followed by

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Table 3. Gastric Fluid pH and Volume (Mean ± SE) for Those Patients within the Same Study Group According to pH below or above 2.5 or Volume above or below 20 ml

<table>
<thead>
<tr>
<th></th>
<th>pH Below 2.5</th>
<th>Above 2.5</th>
<th>More Than 20 ml</th>
<th>Less Than 20 ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>1.6 ± .08</td>
<td>4.9 ± .3</td>
<td>42 ± 6</td>
<td>10 ± 2</td>
</tr>
<tr>
<td></td>
<td>(n = 47)</td>
<td>(n = 26)</td>
<td>(n = 20)</td>
<td>(n = 55)</td>
</tr>
<tr>
<td>Morphine–atropine</td>
<td>1.8 ± .08</td>
<td>4.6 ± .4</td>
<td>61 ± 16</td>
<td>8 ± 3</td>
</tr>
<tr>
<td></td>
<td>(n = 45)</td>
<td>(n = 32)</td>
<td>(n = 20)</td>
<td>(n = 55)</td>
</tr>
<tr>
<td>Morphine–glycopyrrolate</td>
<td>1.9 ± .09</td>
<td>5.2 ± .2</td>
<td>55 ± 10</td>
<td>8 ± 3</td>
</tr>
<tr>
<td></td>
<td>(n = 37)</td>
<td>(n = 38)</td>
<td>(n = 17)</td>
<td>(n = 58)</td>
</tr>
</tbody>
</table>

n = number of patients.

Table 4. Gastric Fluid pH and Volume (Mean ± SE) for Those Patients within the Same Study Group Manifesting pH below 2.5 and Volume above 20 ml

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>Volume (ml)</th>
<th>Number of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>1.6 ± .06</td>
<td>47 ± 9</td>
<td>12</td>
</tr>
<tr>
<td>Morphine–atropine</td>
<td>1.8 ± .1</td>
<td>70 ± 15</td>
<td>13</td>
</tr>
<tr>
<td>Morphine–glycopyrrolate</td>
<td>1.7 ± .08</td>
<td>60 ± 14</td>
<td>12</td>
</tr>
</tbody>
</table>

propanidil, 450 mg, and succinylcholine, 40 mg, iv. The lungs were inflated with a mixture of N₂O/O₂ and 3 per cent halothane. A cuffed endotracheal tube was inserted. Spontaneous ventilation soon returned. Sinus tachycardia was seen immediately after induction (fig. 1B), quickly followed by the intermittent appearance of a Wolff–Parkinson–White syndrome (fig. 1C). As soon as this was observed, the halothane was discontinued and the lungs were inflated with 100 per cent oxygen, since myocardial hypoxia was considered to be a possible cause. When the syndrome persisted, lidocaine, 60 mg, iv, was injected. Normal sinus rhythm then returned (fig. 1D). Fentanyl, 0.1 mg, and alcuronium bromide, 10 mg, iv, were injected, and the patient was ventilated with a respirator with a mixture of N₂O/O₂, 3:1, at a rate of 18/min and a tidal volume of 500 ml, under capnographic control.

The arthroscopy commenced. Gradually the ECG tracing began to show an abnormality that more and more resembled a form of Wolff–Parkinson–White syndrome, with typical delta waves (fig. 2A). The blood pressure remained normal. Again, the Wolff–Parkinson–White syndrome was seen intermittently (fig. 2B) and was abolished by lidocaine, 50 mg, iv.

At the end of the operation, when the patient was breathing 100 per cent oxygen spontaneously, sinus tachycardia appeared (fig. 2C). Since other cardiovascular indices were normal, the trachea was extubated and the patient was transferred to the recovery room.

A full ECG was recorded and showed intermittent Wolff–Parkinson–White syndrome, type A, with normal ventricular frequency (fig. 3). The patient was observed overnight in the intensive care unit. The ECG showed no abnormality the next day.

**FIG. 1.** ECG tracings obtained before induction of anesthesia (A), immediately after induction, (B, C), and after injection of lidocaine (D).

**FIG. 2.** ECG tracings obtained during arthroscopy, suggesting Wolff–Parkinson–White syndrome (A, B), and after operation (C).

**DISCUSSION**

The Wolff–Parkinson–White syndrome was first described as a clinical entity in 1930. Since then, many articles on the subject have been published, but there are relatively few in the anesthesiologic literature. This may be because the condition is recognized and treated before anesthesia in most cases. In our case it appeared only after induction, and was unsuspected.

The syndrome is best defined as one in which all or some portion of the ventricular muscle is activated earlier, in relation to atrial events, than would be expected had the impulse reached the ventricle by way of the normal atrioventricular conduction system. The typical ECG findings include a short PR interval (0.12 sec or less) and a prolonged QRS interval (0.11 sec or more) due to a delta wave. The presence of a delta wave is of particular importance to the anesthesiologist, who usually uses only one ECG lead. It consists of a slow-rising, slurred QRS complex.

Traditionally, the syndrome is classified into types A and B, depending on the direction of the delta wave. Superficially, type A resembles right bundle-branch block, right ventricular hypertrophy and posterior myocardial infarction. Type B may closely resemble left bundle-branch block and left ventricular hypertrophy. In type A premature activation of the left ventricle occurs and in type B, premature activation of the right ventricle. The incidence of the Wolff–
Parkinson-White syndrome has been estimated as 1.5 per thousand. It occurs more frequently in men than in women. The most significant feature, clinically, is the occurrence of a supraventricular arrhythmia, in 75 per cent of cases a supraventricular tachycardia. This is also described as paroxysmal atrial tachycardia, re-entrant tachycardia, and circus movement tachycardia.

Different arrhythmias, such as atrial flutter or fibrillation (20 per cent) and ventricular tachycardia, flutter and fibrillation, may occur. Sometimes these arrhythmias are fatal. There has been much speculation concerning the possible underlying mechanism of the Wolff-Parkinson-White syndrome. The most recent theory is that it occurs as the result of premature activation of a portion of the ventricles as a result of an anomalous AV conduction via an accessory pathway directly from the atrium to the ventricles. In most cases this pathway is the Kent bundle. Anatomic studies recently revealed that embryologic faults in partitioning the atrium from the ventricle, with persisting muscular bridges, underlie many cases of Wolff-Parkinson-White syndrome. The cardiac anomaly most frequently associated with the syndrome is Ebstein's anomaly.

With these considerations in mind, therapy should be adapted accordingly. A patient who is known to have a Wolff-Parkinson-White syndrome and who is receiving drug therapy should not have the therapy discontinued until the day of operation. Depending on the underlying mechanism, propranolol, pro-cainamide, and quinidine are drugs of choice for long-term therapy. Several new drugs including verapamil, Ajmaline, and Amiodarone, also appear to be effective. When a supraventricular tachyarhythmia with anomalous conduction occurs during general anesthesia, lidocaine given intravenously seems to be the treatment of choice, because it depresses the anomalous pathway. When the supraventricular tachycardia shows a normal QRS complex, propranolol should be used, because it depresses the normal AV pathway. Digitalis should not be used, when it is possible to avoid it, because it may accelerate the anomalous conduction of the syndrome.

In urgent situations, such as supraventricular tachycardia with extremely rapid response, especially with atrial fibrillation or flutter, with anomalous conduction, direct-current shock is the treatment of choice. Vagal maneuvers such as carotid sinus stimulation can be attempted first, but they are of doubtful value, not always possible, and may waste valuable time.

With regard to the anesthetic technique, care should be taken not to use any drug or anesthetic agent that will produce tachycardia or a negative inotropic effect upon the heart. Scopolamine is preferable to atropine as a vagolytic drug for premedication. Of the available hypnotic agents for induction, propanidid, as used in the case reported, is contraindicated, because of vagolytic action and a negative inotropic effect. Etomidate would seem to be the drug of choice because circulation and myocardial...
function remain unaltered. Diazepam is an alternative possibility. The use of halothane as a maintenance drug is questionable. It certainly has a negative inotropic effect, but Pantera claims to have succeeded in treating supraventricular tachycardia during general anesthesia by using increasing concentrations of halothane.

Gallamine should be avoided because it can cause disproportionate tachycardia. Pancuronium bromide and d-tubocurarine would seem preferable. We consider anesthetic techniques that give perfect cardiovascular stability to be the best choices for patients who have the Wolff–Parkinson–White syndrome. We have found this to be true for patients with every kind of cardiovascular abnormality when drugs such as droperidol (0.3 mg/kg) and fentanyl (0.07 mg/kg) are used in neuroleptanalgesia. Another technique recently developed in our neurosurgical department, in which very large doses of fentanyl or sufentanil are used after induction with etomidate, appears to have similar advantages.

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REFERENCES