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Anesthetic Management of the Wolff-Parkinson-White Syndrome

ALBERT R. SADOWSKI, M.D.* AND JOHN R. MOYERS, M.D.†

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 Kadis[1] advocate minimal circulatory disturbance using a nitrous oxide, oxygen, and narcotic technique. Similarly, on the basis of one case discovered intraoperatively, van der Starre[2] recommended neuroleptanalgesia and avoiding drugs with negative inotropic effects on the heart. Conversely, Kumazawa[3] advised using deep inhalational anesthesia. We have recently anesthetized 13 patients with the pre-excitation syndrome, and our experience supports the latter opinion.

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 N_2O after endotracheal intubation. They had no arrhythmias. Patient 3 received anesthesia with morphine (1 mg/kg), diazepam, 10 mg, N_2O, 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, N_2O, 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 1*A*. The impulse spreads from the sinus node through the atrium, undergoes physiologic delay at the atrioventricular (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 1*B*. 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

*Resident, Department of Anesthesia.
†Assistant Professor, Department of Anesthesia.
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Address reprint requests to Dr. Moyers.

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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. 1*C*). 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. 1*D*).

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.[4] 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.[4,6] Quinidine

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

	Age (Years)	Operation	Anesthesia	Arrhythmia	Remarks
Patient 1	63	His-bundle division	Morphine, halothane, N_2O, pancuronium	No	WPW* and coronary-artery disease
Patient 2	52	His-bundle division	Morphine, halothane, N_2O, pancuronium	No	WPW; rheumatic heart disease and previous mitral valve replacement ×2
Patient 3	26	Kent-bundle division and mitral annuloplasty	Morphine, diazepam, N_2O, pancuronium	Yes	WPW; countershocked more than ten times for superventricular tachycardia
Patient 4	51	Intestinal resection	Morphine, N_2O, pancuronium	Yes	Lown-Ganong-Levine syndrome diagnosed by anesthesiologist intraoperatively
		Fistula repair	Morphine, N_2O, *d*-tubocurarine	No	Atrial fibrillation controlled preoperatively with propranolol and digitalis
Patient 5	42	His-bundle division	Halothane, N_2O, *d*-tubocurarine	Yes	WPW; arrhythmia occurred during cardiac manipulation
Patient 6	59	Kent-bundle division	Halothane, N_2O, *d*-tubocurarine	No	WPW; superventricular tachycardia persistent postoperatively
		Kent-bundle division	Halothane, N_2O, *d*-tubocurarine	No	
Patient 7	64	Kent-bundle division	Enflurane, N_2O, *d*-tubocurarine	No	WPW and cardiomyopathy
Patient 8	53	His-bundle division	Enflurane, N_2O, *d*-tubocurarine	No	WPW
Patient 9	31	His-bundle division	Enflurane, N_2O, *d*-tubocurarine	No	Lown-Ganong-Levine syndrome; two previous cesarean sections done uneventfully with spinal anesthesia
Patient 10	36	Replacement of ascending aorta and aortic valve	Halothane, N_2O, pancuronium	No	WPW and Marfan's syndrome
Patient 11	4	Removal of Wilms' tumor	Enflurane, N_2O, *d*-tubocurarine	No	WPW
Patient 12	3½	Cystoscopy	Enflurane, N_2O	No	WPW
Patient 13	38	Transsphenoidal hypophysectomy	Enflurane, N_2O	No	WPW; 12 ml 1 per cent lidocaine with epinephrine (1/100,000 injected into mucosa) without arrhythmia

* WPW = Wolff-Parkinson-White syndrome.

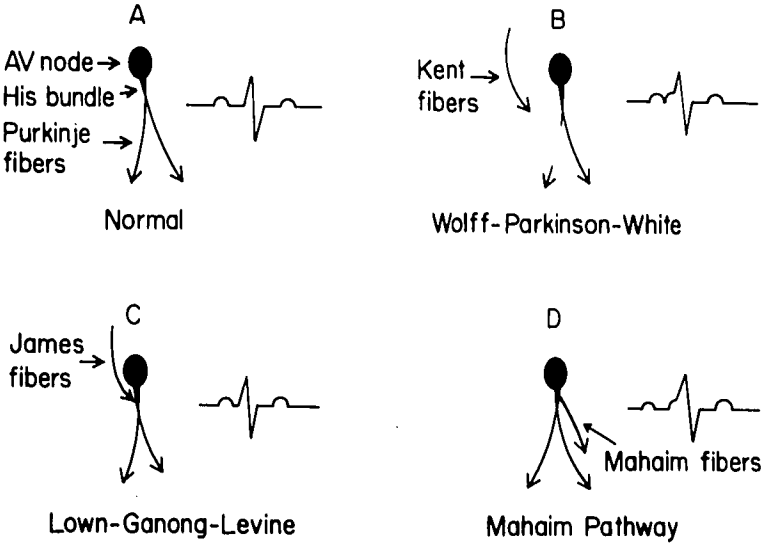

FIG. 1. Conduction in the normal (*A*), Wolff-Parkinson-White (*B*), Lown-Ganong-Levine (*C*), and Mahaim (*D*) pathways.

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 propanolol 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, scopolamine, and diazepam for premedication. The use of atropine has been discouraged by some.[1,5] Atropine can produce normal A-V conduction with disappearance of the delta wave,[6] as well as disturbing tachycardias.[3] 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.[1] Thiopental, 1–3 mg/kg, was used in 14 of our anesthesias, 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.[2,3,5,7,9] Iga[8] recommended anesthetic techniques that do not increase blood catecholamine levels. Kumazawa advised using deep anesthesia with halothane or methoxyflurane.[3] For nine patients, we used halothane and enflurane, which presumably reduced sympathetic outflow,[10,11] and only one arrhythmia occurred in those patients. For muscle relaxation *d*-tubocurarine is less likely to cause tachyarrhythmia than is pancuronium.[12]

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.

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LDH_5 Changes after Cholecystectomy or Hysterectomy in Patients Receiving Halothane, Enflurane, or Fentanyl

OSCAR VIEGAS, M.D.,* AND ROBERT K. STOELTING, M.D.†

The LDH_5 isoenzyme fraction of lactate dehydrogenase is felt to be relatively specific for hepatocellular injury.[1] Klar *et al.*[2] measured LDH_5 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_5 values before and after elective cholecystectomies performed with halothane–N_2O anesthesia. In addition, LDH_5 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 Indiana University School of Medicine Human Research Committee. No patient had a history of hepatic disease, was taking drugs known to alter hepatic function, or had previously received a halogenated anesthetic. Preanesthetic medication was with diazepam, 5–10 mg, orally, and atropine, 0.4 mg, im, 60 to 90 min prior to operation. Anesthetic induction was with *d*-tubocurarine, 40 μg/kg, followed by thiamylal, 4 mg/kg, and succinylcholine, 1.5 mg/kg, to facilitate tracheal intubation. Anesthesia was maintained with 60 per cent inspired nitrous oxide and 0.5–1 per cent halothane (20 patients), 0.5–1.5 per cent enflurane (20 patients), or fentanyl (20 patients). Fentanyl was administered iv as a 100–150 μg loading dose and supplemented with 50 μg every 15 min until approximately 30 min before the anticipated completion of operation. Each anesthetic group consisted of ten patients undergoing cholecystectomy or hysterectomy. Patients were randomly assigned to anesthetic groups, with the exception of those receiving halothane for cholecystectomy. These patients were not studied until completion of the other study groups and, therefore, represent ten consecutive patients. Pancuronium was administered to all patients to provide intraoperative muscle relaxation. Intravenous fluid administration consisted of 5–8 ml/kg/hr lactated Ringer's solution. Blood replacement was not necessary in any patient.

Samples of venous blood for LDH_5 determinations were obtained the evening before operation (control) and then one and 24 hours postoperatively. Results were analyzed by analysis of variance, and $P < 0.05$ was considered significant.

* Assistant Professor.
† Professor and Chairman.
Received from the Department of Anesthesia, Indiana University School of Medicine, Indianapolis, Indiana 46223. Accepted for publication May 1, 1979.
Address reprint requests to Dr. Viegas.