of a nonfunctioning radial-artery catheter. In the other two patients the opposite radial artery could not be used because numerous attempts at percutaneous cannulation had been unsuccessful.

There are several advantages in using this technique of catheter thrombectomy. The collateral circulation to the palmar arch can be demonstrated. A #3 French embolectomy catheter is easy to insert through the arteriotomy made by the 18-gauge Angiocath. After flow in the radial artery has been re-established, the Angiocath is reinserted through the same arteriotomy, avoiding the need for suturing the arteriotomy. A tie is placed around the artery and catheter when necessary to prevent excessive bleeding from the arteriotomy. Catheter thrombectomy of the radial artery is a means of successfully re-establishing radial arterial flow when the radial artery is felt to be the optimal site for blood pressure and blood-gas monitoring.

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Paroxysmal Supraventricular Tachycardia during Anesthesia

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Compared with their ventricular counterparts, paroxysmal supraventricular tachycardias often are considered benign dysrhythmias unless they are prolonged and refractory to standard treatment or occur in the presence of myocardial disease.[1,2] However, during the altered hemodynamic state associated with anesthesia, these dysrhythmias may rapidly produce deleterious cardiovascular effects that necessitate immediate therapy to prevent serious sequelae. This point is illustrated by the following cases.

REPORT OF FOUR CASES

Case 1. A 24-year-old healthy woman was scheduled for cesarean section because of cephalopelvic disproportion. After subarachnoid injection of tetracaine, a healthy infant was delivered while the maternal blood pressure and pulse remained in the range of 110–120/50–65 torr and 95–110 beats/min. As an infusion of oxytocin was started, the pulse abruptly increased to 190 beats/min, and the blood pressure decreased to 100/50 torr. The electrocardiogram (fig. 1) indicated a 1:1 ratio of atrial to ventricular conduction with a regular ventricular rate. Although carotid massage was performed, no change in heart rate was obtained. As the tachycardia continued, the blood pressure decreased to 55/40 torr. Therefore, methoxamine (20 mg, iv, in divided doses) was given, causing the pulse to slow to 100 beats/min and blood pressure to increase to 125/60 torr. Fifteen minutes later, another abrupt increase in pulse to 180 beats/min occurred with a decrease in blood pressure to 90/50 torr. Carotid massage at this time produced a slowing of the rate to 96 beats/min and an increase in blood pressure to 120/65 torr. At this time, results of arterial blood-gas and serum electrolyte determinations were normal. The rest of the patient's hospitalization was uneventful.

Case 2. A 20-year-old woman was scheduled for incision and drainage of an infected hand. After being premedicated with meperidine and atropine, the patient was brought to the operating room and anesthesia was induced with thiopental, nitrous oxide, and halothane. Shortly after induction, the patient's pulse increased abruptly from 92 to 184 beats/min. The electrocardiogram showed rapid regular atrial activity with a 1:1 ratio of atrial to ventricular conduction. Carotid massage combined with edrophonium (20 mg, iv, in divided doses) produced no change in heart rate. Therefore, phenylephrine (2 mg, iv) was given and an abrupt reduction of rate to 98 beats/min occurred. The rest of the anesthetic course was uneventful.

Case 3. After being medicated with scopolamine and secobarbital, a 4-year-old boy was brought to the operating room for herniorrhaphy. Anesthesia was induced with thiopental and maintained with cyclopropane via an endotracheal tube. While the lower abdominal wall was being prepped, a sudden increase in pulse rate from 100 to 176 beats/min and a decrease in blood pressure from 110/70 to 85/60 torr occurred. The electrocardiogram indicated a 1:1 ratio of atrial to ventricular conduction at a fixed rate. After the anesthetic had been discontinued, carotid massage and positive airway pressure were administered, producing an abrupt return of the blood pressure and pulse to normal values. The anesthesia was continued without further cardiovascular instability.

Case 4. Three weeks after a subendocardial infarction, a 60-year-old woman was scheduled for a coronary-artery bypass graft. After receiving diazepam and morphine, the patient was brought to the operating room and an inhalational induction was performed using nitrous oxide and halothane. While anesthesia was maintained with nitrous oxide and halothane, the bypass graft procedure was performed during 55 minutes of extracorporeal circulation. After the extracorporeal circulation had been discontinued, the patient was able to maintain a blood pressure of 120–140/70–90 torr and a pulse of 100–

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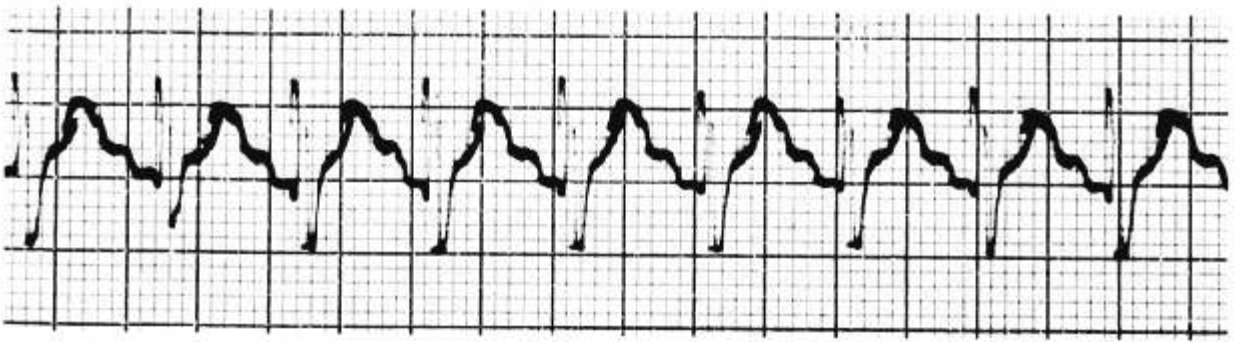

FIG. 1. Lead V_5 of the electrocardiogram obtained from the patient described in Case 1, demonstrating 1:1 ratio of atrial to ventricular conduction during the dysrhythmia.

110 beats/min with the assistance of the intra-aortic balloon pump. Fifteen minutes later, an abrupt increase in pulse to 180 beats/min occurred while the blood pressure decreased to 100/60 torr and the pulmonary wedge pressure increased from 18 to 26 torr. The electrocardiogram revealed a 1:1 ratio of atrial to ventricular conduction at a fixed rate. When carotid massage produced no change in heart rate, neostigmine (1 mg, iv, in divided doses) was given and the pulse slowed to 100 beats/min. The blood pressure and pulmonary wedge pressure returned to pretachycardiac values. The rest of the anesthetic course was uneventful.

DISCUSSION

Paroxysmal supraventricular tachycardia is a more inclusive term used to describe dysrhythmias previously labeled paroxysmal atrial tachycardia, paroxysmal nodal tachycardia, and paroxysmal atrioventricular junctional tachycardia. Although electrocardiographic findings may differ slightly, similarities in clinical presentation, electrophysiologic mechanisms, and therapies of these tachycardias warrant this common designation.[1]

The characteristic clinical feature common to paroxysmal supraventricular tachycardias is their abrupt onset and termination. Ranging from 140 to 220 beats per minute, the rapid heart rate during a typical paroxysm is sustained for a variable period and then suddenly decreases to a rate approximately half the paroxysmal rate. Single, infrequent, or repeated bouts of the tachycardia may occur. Commonly, it occurs in young healthy individuals as well as in those with intrinsic heart disease or systemic illness. An association with thyrotoxicosis and pregnancy also has been found. The hemodynamic consequences of the tachycardia depend on the cause and duration of the dysrhythmia, the ventricular rate, and the previous condition of the heart. A possible decrease in cardiac output secondary to inadequate ventricular filling and an increase in myocardial oxygen requirement coincident with an increase in heart rate may result in heart failure, angina, or ischemia of vital organs.[2]

The characteristic electrocardiogram of paroxysmal supraventricular tachycardia shows a fixed ventricular rate between 140 and 220 beats/min with a 1:1 relationship of atrial to ventricular conduction. P waves may be difficult to recognize at more rapid rates due to superimposition on the preceding T waves. Ventricular complexes and T waves are usually of normal configuration; however, with prolonged tachycardia ST segments may become depressed and T waves inverted. Ventricular aberration may occur and thereby complicate the differential diagnosis between atrial and ventricular tachycardia. Because of the marked differences in prognosis and treatment, an accurate diagnosis is crucial; in addition to ventricular tachycardia, the differential diagnosis includes: paroxysmal atrial tachycardia with block, sinus tachycardia, atrial flutter, and atrial fibrillation.[3]

Since there is no consensus about the most effective treatment of paroxysmal supraventricular tachycardia, full assessment of the possible interactions of the therapeutic modalities with the anesthetic drugs must be made, and a priority of therapy starting with the least dangerous and advancing to more complicated modalities should be established. Usually an attempt to increase vagal tone by carotid massage or a Valsalva maneuver should be tried initially. When unsuccessful alone, these measures combined with an anticholinesterase or an alpha-adrenergic agent may produce the vagotonia necessary to terminate the dysrhythmia. When the cholinergic and pressor effects of these agents are undesirable, propranolol is a useful alternative for interrupting the re-entry phenomenon responsible for the dysrhythmia.[4] However, since there is frequently some heart failure after prolonged tachycardia, the use of this agent may precipitate acute heart failure.[1] Other therapeutic measures that have proved useful in the treatment of paroxysmal supraventricular tachycardia include the use of digitalis, procainamide, electrical cardioversion, and electrical pacing.[5]

The cases presented here represent a spectrum of possible situations in which paroxysmal supra-

ventricular tachycardia can occur in anesthetized patients. As is often the case, exact clinical causes of the dysrhythmias are not clear. In Case 1, there were several possible triggering factors: 1) alterations in myocardial autonomic control secondary to spinal anesthesia; 2) changes in intravascular fluid volume secondary to delivery of the infant; 3) effects of oxytocin on the cardiovascular system. In the patients who received general anesthesia, reflex initiation of the dysrhythmia during light anesthesia in Cases 2 and 3, and direct myocardial damage in Case 4, may have contributed significantly to the precipitation of the dysrhythmia.

Although most dysrhythmias during anesthesia will respond either to a change in anesthetic technique or to cessation of surgical manipulation, the cases presented here emphasize the fact that paroxysmal supraventricular tachycardias during anesthesia may produce deleterious cardiovascular effects that necessitate more rapid restoration of normal rhythm than can be achieved by modifying anesthetic or surgical management. In such situations, the rational use of specific antidysrhythmic therapies is imperative.

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Percutaneous Aortic Catheterization—A Hazard of Supraclavicular Internal-jugular-vein Catheterization

To the Editor:—Unpredictability of peripherally introduced central venous monitoring lines in pediatric patients has led to renewed interest in internal-jugular-vein catheterization. We have employed the supraclavicular approach to the internal jugular vein* to establish guidelines for its use in our patients undergoing operations for congenital heart disease. A new contraindication to this technique has been established.

A Mustard[1] total correction of transposition of the great arteries was performed on a 14-month-old 7.2-kg male infant. Supraclavicular internal-jugular-vein catheterization was attempted after induction of anesthesia and endotracheal intubation. A 20-gauge 1¼-inch Angiocath† was introduced once. Upon stylet removal, pulsatile blood flow was apparent. With heparinization for cardiopulmonary bypass anticipated, the catheter was held in place and monitored by oscilloscope. Shortly thereafter, the arterial trace dampened and blood withdrawal became impossible. After other monitoring was established, the operation was begun. Through a median sternotomy the heart and great vessels were exposed. Dissection revealed a single puncture site with surrounding hematoma formation in the anterior wall of the ascending aorta. The catheter tip, though in close proximity to the puncture, was not in the aorta. Active bleeding from the puncture was not seen.

In complete transposition of the great arteries, the aorta, arising from the right ventricle, is anterior and oriented toward the right thoracic cavity.[2] This anatomic configuration contraindicates right supraclavicular internal jugular vein catheterization in these patients.

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