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Supraventricular Tachycardia Associated with Extracorporeal Shock Wave Lithotripsy

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At the end of 1984, the Food and Drug Administration (FDA) approved the use of extracorporeal shock wave lithotripsy (ESWL) as a means of treating renal calculi. While undergoing this treatment patients are immersed in a warm water bath. Then, under fluoroscopic control, shock waves produced by underwater electrical discharge (spark-gap generator) are focused on the stone.[1] Repetitive bombardment by the shock waves results in the shattering of the stone. The small fragments can then be excreted in the urine. The treatment requires no surgery and hospitalization is brief.[2–6]

During the early stage of ESWL development, the energy caused ventricular dysrhythmias.‡ The lithotripter was modified so that there was an 83-ms delay between the R wave of the ECG and the shock. Thus, the shock wave struck the patient while the ventricles were refractory to stimulation. In our experience with more than 1,200 patients, premature ventricular contractions are rare. However, premature supraventricular contractions occur in about 10% of patients. Occasionally these premature contractions will result in short runs (2–4 beats) of supraventricular tachycardia (SVT) (fig. 1). These arrhythmias spontaneously subside with the momentary cessation of shocking. We describe two patients who developed sustained SVT that required treatment.

REPORT OF TWO CASES

Case 1. A 55-yr-old woman was admitted for the treatment of nephrolithiasis. She was moderately obese, (weight 70 kg; height 155 cm), but in otherwise good general health. Her arterial blood pressure on admission was 150/100 mmHg, heart rate 80 beats/min. The remainder of her physical examination was within normal limits. The ECG and serum potassium were normal.

She received no premedication on the ward. Preanesthesia arterial blood pressure was 130/70 mmHg and heart rate was 120 beats/min. Diazepam, 5 mg , was given iv. The epidural block was inserted without difficulty; a total of 17 ml of 2% lidocaine with 1:200,000 epinephrine was given. A T-4 sensory level developed. Her vital signs remained stable for the next 40 min with a normal sinus rhythm. The patient was then placed in the gantry and immersed in water. After approximately 200 shocks she developed an SVT. The R wave rate, as counted by the physiologic monitor, was 280 beats/min. Her peripheral pulse rate, as recorded on a pulse oximeter, was 140 beats/min. Arterial blood pressure was measured as 160/80 mmHg.

The patient was removed from the treatment bath. She did not respond to initial carotid massage or edrophonium 10 mg iv. Her heart rate, however, slowed to 110 beats/min after receiving 2.5 mg of verapamil iv.

The lithotripter treatment was canceled. In the recovery room her arterial blood pressure remained stable at 160/110 mmHg, heart rate was 120–130 beats/min. A 12-lead ECG was read as sinus tachycardia. There was no evidence of ischemia or preexcitation. On questioning by a consulting cardiologist, she admitted to having five previous episodes of "palpitations" in the past 10 yr. The last episode was 18 months earlier. All episodes were self limited, and none lasted longer than 30 min.

The patient returned approximately 1 month later. In preparation for the definitive treatment, the patient was given diazepam 10 mg orally for premedication. In the induction room, her arterial blood pressure was measured as 155/70 mmHg and her heart rate was 100 beats/min. The epidural blockade was performed using 18 ml 2% lidocaine with 1:200,000 epinephrine. She was given 3 mg propranolol in divided doses over the next 45 min. Her heart rate remained at 90–100 beats/min. She was also given incremental doses of diazepam (total dose 10 mg) over the next 1 h. During the treatment she developed three runs of SVT that were of 2–4 beats in duration and self limited. The stone was shattered, and the patient was discharged from the hospital on the following day.

Case 2. A 73-yr-old, 50-kg woman with bilateral renal lithiasis was admitted for lithotripsy treatment. Arterial blood pressure was 156/98 mmHg and heart rate 100 beats/min. The ECG was read as sinus tachycardia but an old, inferior myocardial infarction could not be excluded. The chest roentgenogram showed cardiomegaly. Nifedipine was given overnight (10 mg q 6 h). After premedication with 5 mg diazepam orally, arterial blood pressure on admission to the lithotripsy unit was 155/100 mmHg and heart rate 100 beats/min. The epidural anesthetic was inserted using a total of 12 ml of 2% lidocaine with 1:200,000 epinephrine. Although arterial blood pressure decreased to 140/90 mm/Hg, heart rate remained at 100 beats/min. She was

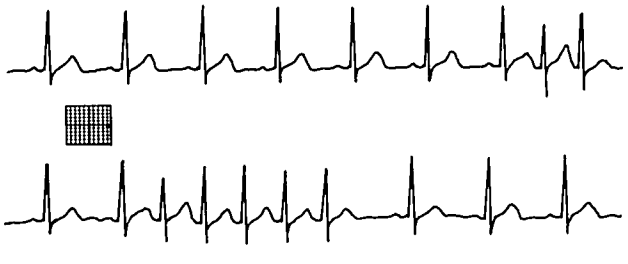

FIG. 1. Short runs of SVT during ESWL in an otherwise-healthy, 45-yr-old man. Surface ECG lead I, continuous tracings. Paper speed = 25 mm/s.

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‡ Chaussy, C. Personal communication.

taken to cystoscopy where a ureteral stent was advanced to the stone. During the cystoscopy her vital signs remained unchanged. An additional 6 ml of 2% lidocaine with 1:200,000 epinephrine was injected *via* epidural catheter prior to transporting her to the lithotripter tub approximately 30 min after the placement of the initial block. The sensory level rose from T-11 to T-6.

After treatment with 77 shocks, the patient developed an SVT at a rate of 210 beats/min. Arterial blood pressure during the tachycardia was 130/80 mmHg. Verapamil 2.5 mg was given three times before the heart rate returned to 100 beats/min. Arterial blood pressure decreased following the verapamil treatment to 100/55 mmHg. Multiple boluses of phenylephrine 25–50 μg were given iv over the next 25 min before the arterial blood pressure stabilized at 135/80 mmHg.

Because the position of this patient's stone made ureteral obstruction a likely complication, the urologist urged that the treatment be completed. A 12-lead ECG showed no change from the preoperative tracing. The vital signs were normal and the patient was in no distress. We elected to proceed. The patient was reimmersed in the bath approximately 45 min after the aborted first attempt. After initiating the shocks she again developed an SVT with a rate of 185 beats/min. The tachycardia responded to iv verapamil, total dose 7.5 mg. Again, phenylephrine was used to treat the decrease in systolic blood pressure that followed the verapamil treatment. The treatment proceeded with the application of 2,400 shocks to the right stone and 1,400 shocks to the left stone. The treatment took 105 min to complete. An additional 6 ml of 1% lidocaine with 1:100,000 epinephrine was given epidurally. During this time, arterial blood pressure was stable at 120/70 mmHg and heart rate 60–75 beats/min. Postoperatively, the patient had no cardiac complications, and she was discharged on the fourth postoperative day.

Discussion

Both of these patients had a rapid heart rate in the immediate preanesthetic period. While the first patient was judged to be apprehensive, the second exhibited no outward signs of anxiety. She was totally relaxed and uncomplaining during the performance of the epidural blockade. She dozed during the cystoscopic procedure and after being placed in the treatment tub.

While the use of epinephrine with the lidocaine could have been a contributing factor to tachycardia, neither patient had an increase in heart rate with the injection of the epinephrine-containing solution. The first patient's heart rate slowed from 120 to 90 beats/min following the block. The second patient had no change in heart rate, even though her systolic pressure slowly decreased from 155 to 140 mmHg. In both patients, the SVT occurred precipitously after the initiation of treatment.

The first patient had a history of previous attacks of palpitation. None had been documented as SVT, and the preoperative ECG was read as normal (without evidence of ventricular preexcitation). With SVT, this patient developed 2:1 electromechanical dissociation (electrical rate 280 beats/min, pulse rate 140 beats/min) and she suffered no hemodynamic consequences. This unusual situation would not have been detected had we only monitored the heart tones or peripheral pulse.

Premature electrical stimulation of the atria, and possibly mechanical energy associated with ESWL, can initiate reentrant atrial tachyarrhythmias, including paroxysmal supraventricular tachycardia (PSVT), atrioventricular (AV) nodal reentrant tachycardia, reciprocating tachycardia due to reentry over a retrograde-conducting concealed accessory pathway, and atrial flutter.[7] The extremely rapid ventricular rate in the first patient makes either of the first two mechanisms unlikely (the rate of tachycardia should have been less than 250 beats/min), and atrial flutter the more likely mechanism.[8] However, for there to have been 1:1 AV conduction, the patient would have had to have a rapidly conducting, accessory conduction pathway. Such could be suspected (history of palpitations), yet her preoperative ECG was read as "normal," *i.e.*, without evidence for ventricular preexcitation (short P–R interval, delta wave). Despite the mechanism for the tachycardia, verapamil was effective drug therapy. It should be noted that verapamil may be effective therapy for atrial flutter, particularly when of recent onset.[9] Low-energy DC cardioversion, however, is preferred.[8,9] The mechanism for tachycardia in the second patient was most likely PSVT or reciprocating tachycardia due to reentry over a concealed accessory pathway. With either of these two mechanisms for tachycardia, maneuvers or drugs (edrophonium) to increase vagal tone, or intravenous verapamil, would be the initially preferred management.[8,9] Prompt termination of tachycardia following verapamil in the second patient provides further evidence for the mechanism of tachycardia.

In summary, we describe anecdotal observations of two patients who developed SVT while being treated for renal lithiasis with ESWL. Both patients responded to indicated management (intravenous verapamil) and were unharmed by the episode. We must await further study to determine whether the dysrhythmias were triggered by the shock waves and, if so, to what extent the triggering of SVT can be prevented. A possible solution might be to alter the timing of the shock waves in relation to the atrial and AV nodal refractory (vulnerable) periods.

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Bronchoscopic Administration of Nebulized Racemic Epinephrine to Facilitate Removal of Aspirated Peanut Fragments in Pediatric Patients

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Aspiration of foreign bodies, such as peanuts or other foods, causes significant morbidity and mortality in children.[1–8] When these organic foreign bodies have been in the airway for a prolonged period of time, mucosal edema develops that traps the fragments, and bronchoscopic extraction may be difficult or impossible. Diluted racemic epinephrine is frequently nebulized to reduce laryngotracheal edema associated with laryngotracheobronchitis (LTB)[9,10] and after tracheal extubation.[11,12] We used an extremely dilute solution of nebulized racemic epinephrine, administered directly through the bronchoscope, to reduce mucosal swelling and thus facilitate extraction of firmly impacted peanut fragments from three small children.

Report of Three Cases

Case 1. A 2½-yr-old boy was admitted with a history suggestive of peanut aspiration. Rigid bronchoscopy under general anesthesia was performed and repeated the next day. Although the peanut fragment was visualized in the right lower lobe (RLL) bronchus, it could not be extracted because of severe swelling of bronchial mucosa. The child developed RLL pneumonia, and fluids and clindamycin administration iv did not result in clinical improvement. He was scheduled for a third rigid bronchoscopy under general anesthesia to be followed by right lower lobectomy if the peanut could not be removed.

The child was anesthetized with enflurane in N_2O/O_2. Direct laryngoscopy was performed, a 5.0-mm rigid bronchoscope introduced, and ventilation controlled. Anesthesia was maintained with enflurane in oxygen given through the sidearm of the bronchoscope. Examination of the tracheobronchial tree was normal except for the RLL bronchus, where marked erythema and swelling were noted. An impacted peanut fragment was visualized in the RLL bronchus, but the mucosal edema was so extensive that extraction was impossible despite multiple attempts by several individuals. A solution of racemic epinephrine (0.1 ml of 2.25% in 5 ml of saline) was nebulized through the bronchoscope and given directly into the RLL bronchus (fig. 1). Arterial blood pressure and heart rate remained unchanged, and no dysrhythmias were noted. Rapid resolution of the edema occurred, and the peanut fragment was then readily extracted. Enflurane was discontinued, and ventilation was controlled through the sidearm of the bronchoscope until spontaneous ventilation was sufficient to permit tracheal extubation. The child had an uneventful recovery.

Case 2. A 2-yr-old girl presented with a 10-week history of productive cough after possible aspiration of a peanut. Physical examination and chest roentgenogram showed left lower lobe (LLL) and lingular bronchopneumonia. She was treated with antibiotics and scheduled for bronchoscopy.

Anesthesia was induced with halothane in N_2O/O_2. After surgical anesthesia was achieved, isoflurane was substituted for halothane and N_2O discontinued. Bronchoscopy was performed with a rigid 4.0-mm bronchoscope and continued for 90 min. Ventilation was controlled through the sidearm of the bronchoscope. The left main bronchus was totally occluded with a purulent exudate. Racemic epinephrine (0.1 ml of a 2.5% solution in 5 ml of normal saline) was nebulized through the bronchoscope directly at the occluded left main bronchus (fig. 1). A total of three doses of nebulized racemic epinephrine were administered during the 60-min bronchoscopy. The exudate became liquefied, the diameter of the bronchus increased, erythema was reduced, and a peanut fragment was extracted. Arterial blood pressure and heart rate remained unchanged, and no dysrhythmias were noted. Isoflurane was discontinued and the trachea was extubated. The patient had an uneventful recovery and was discharged home within 24 h of bronchoscopy.

Case 3: A 16-month-old girl was scheduled for bronchoscopy under general anesthesia 2 weeks after a suspected peanut aspiration. She was febrile despite treatment with erythromycin and had a physical examination and chest roentgenogram suggestive of right middle lobe (RML) foreign body.

Anesthesia was induced with halothane in N_2O/O_2, isoflurane was subsequently substituted for halothane, and N_2O was discontinued. Rigid bronchoscopy was performed, revealing purulent exudate and erythema of the RML bronchus. Racemic epinephrine (0.1 ml of 2.25% solution in 5 ml normal saline) was nebulized into the bronchus through

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