References

1. Parker FB Jr, Marvasti MA, Bove EL: Neurologic complications following coronary artery bypass. The role of atherosclerotic emboli. Thorac Cardiovasc Surg 1985; 33:207-9

2. Mills NL, Everson CT: Atherosclerosis of the ascending aorta and coronary artery bypass. Pathology, clinical correlates, and operative management. J Cardiovasc Thorac 1991; 102:546-53

3. Hessel EA: Cardiopulmonary bypass circuitry and cannulation techniques, Cardiopulmonary Bypass Principles and Practice. Edited by Gravlee GP, Davis RF, Utley JR. Baltimore, Williams and Wilkins, 1993, p 66

4. Murry CE, Jennings RB, Reimer KA: Preconditioning with ischemia: A delay of lethal cell injury in ischemic myocardium. Circulation 1986; 74:1124-36

5. Lawson CS: Preconditioning in man: Progress and prospects. J Mol Cell Cardiol 1995; 27:961-7

6. Flameng W: Intermittent ischemia of the heart. Transplant Proc 1995; 27:2791-2

7. Haessler R, Kuzume K, Wolff RA, Kuzume K, Chien GL, Davis RF, Van Winkle DM: Adrenergic activation confers cardioprotection mediated by adenosine, but is not required for ischemic preconditioning. Coronary Artery Dis 1996; 7:305-14

8. Yellon DM, Alkhulaifi AM, Pugsley WB: Preconditioning the human myocardium. Lancet 1993; 342:276-7

9. Walker DM, Walker JM, Pugsley WB, Pattison CW, Yellon DM: Preconditioning in isolated superfused human muscle. J Mol Cell Cardiol 1995; 27:1349-57

10. Lönn U, Petersen B, Granfeldt H, Casimir-Ahn H: Coronary artery operation with support of the hemopump cardiac assist system. Ann Thorac Surg 1994; 58:519-23

11. de Bruijin NP, Croghwell N, Reves JG: Hemodynamic effects of esmolol in chronically b-blocked patients undergoing aortocoronary bypass surgery. Anesth Analg 1987; 66:137-41

12. Belardinelli L, Linden J, Berne RM: The cardiac effects of adenosine. Prog Cardiovasc Dis 1989; 32:73-97

13. Camm AJ, Garratt CJ: Adenosine and supraventricular tachycardia. N Engl J Med 1991; 325:1621-9

14. Baker AB, Bookallil MK, Lloyd G: Intentional asystole during endoluminal thoracic aortic surgery without cardiopulmonary bypass. Br J Anaesth 1997; 78:444-8

15. Lerman RB, Wesley RC, Belardinelli L: Electrophysiologic effects of dipyridamole on atrioventricular nodal conduction and supraventricular tachycardia. Role of endogenous adenosine. Circulation 1989; 80:1536-43

Anesthesiology
1998; 88:830-2

Unintended Supraventricular Tachycardia Induced by Extracorporeal Shock Wave Lithotripsy

Dinna B. Billote, M.D., Ram M. Challapalli, M.D.,† Robert B. Nadler, M.D.‡*

DESPITE the development of different modes and newer models of lithotriptors, cardiac arrhythmias continue to be reported as a complication of extracorporeal shock wave lithotripsy (ESWL).[1-6] The utilization of an electrocardiographic (ECG)-triggered mode, when compared with respiratory-triggered or nontriggered ESWL, significantly reduces the incidence of arrhythmias.[1-2] ECG-triggered lithotriptors eliminate ESWL-induced ventricular tachyarrhythmias as the delivered mechanical shock is synchronized to the terminal portion of the QRS complex during the absolute refractory period of ventricles. However, because of a potential for atrial stimulation, supraventricular tachyarrhythmias may still occur.[3] After the performance of more than 3,600 cases of ESWL at our institution, we present a case of a reproducible supraventricular tachycardia caused by the Dornier HM3 Lithotriptor (Germering, Germany).

* Clinical Instructor, Department of Anesthesiology.

† Fellow, Section of Electrophysiology, Department of Cardiology.

‡ Assistant Professor of Urology and Head, Section of Endourology and Stone Disease, Department of Urology.

Received from the McGaw Medical Center of Northwestern University, Chicago, Illinois. Submitted for publication August 27, 1997. Accepted for publication November 17, 1997.

Address reprint requests to Dr. Billote: Department of Anesthesiology, McGaw Medical Center of Northwestern University, 303 E. Superior Street, Passavant 360, Chicago, IL 60611-3053. Address electronic mail to: dbillote@nmff.nwu.edu

Key words: Arrhythmia; ESWL; renal calculus; shock wave release.

Case Report

A 71-yr-old man, recently diagnosed with a left renal calculus, planned to undergo cystoscopy and ureteral stent placement followed by ESWL. Other than a history of hepatitis, the medical history was unremarkable. He exercised regularly and denied any history of palpitations, angina, syncope, or dyspnea with exertion. On presenta-

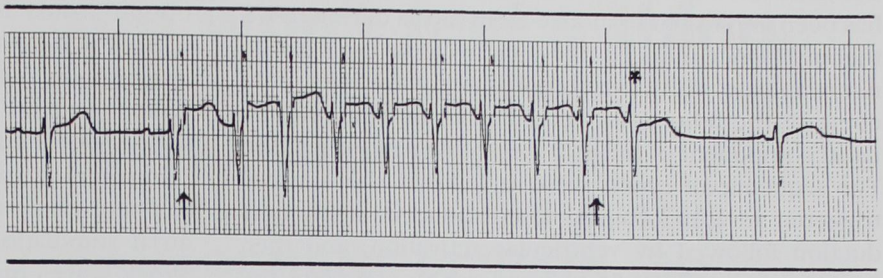

Fig. 1. ESWL-induced supraventricular tachycardia. Arrows indicate abrupt onset and offset of shock wave release. Shock waves are released shortly after the QRS complex during ECG-triggered lithotripsy. After nine shocks, the operator terminates shock wave release. The next complex after ceasing shock wave release (indicated by an asterisk) appears similar in rate and morphology as previous complexes during the SVT. Its supraventricular morphology indicates normal conduction of an atrial capture beat produced by the last ESWL shock wave. The complexes before and after shock wave release are sinus beats. Lead III, paper speed = 25 mm/s and size = 2 cm/mV.

tion, the patient was noted to be thin (height, 168 cm; weight, 60 kg) and in apparent excellent health. His vital signs on admission were a blood pressure of 110/70 mmHg and a pulse of 78 beat/min. The remainder of his physical examination was unremarkable. His ECG revealed sinus rhythm with 1° atrioventricular block at a rate of 50 beat/min.

The patient received midazolam 2 mg, ampicillin 2 g, and gentamycin 100 mg, intravenously as preoperative medication. After preoxygenation, propofol, 100 mg, fentanyl, 50 μg, and rocuronium, 40 mg, were given intravenously for induction, and general anesthesia was maintained using isoflurane 1.0–1.5 vol %, N_2O (1 l/min) and O_2 (1 l/min). He remained hemodynamically stable in sinus rhythm (60 beat/min) during cystoscopy and ureteral stent placement. Approximately 40 min later, the patient was transferred during general anesthesia to the lithotripsy suite for ESWL. During positioning onto the stretcher, sinus bradycardia developed (45 beat/min). The patient received intravenous glycopyrrolate, 0.2 mg, and his heart rate increased to 55 beat/min.

With the initiation of shock wave release, a narrow complex tachycardia of sudden onset was noted on the monitor (fig. 1). After a few beats, the tachycardia stabilized at a rate of 150 beat/min, and the possibility of a monitor artifact was excluded based on pulse oximetry readings. The supraventricular tachycardia (SVT) terminated immediately with cessation of shock wave release. Blood pressure during tachycardia was 170/110 mmHg. Lithotripsy was reattempted four additional times with similar results. The induced SVT varied in duration depending on the number of shocks delivered. The patient received 59 shocks at 20 kV before the procedure was aborted with the kidney stone remaining intact. The patient emerged from general anesthesia, neuromuscular blockade was reversed with neostigmine 2 mg, and glycopyrrolate 0.4 mg, intravenously and then he was extubated and transferred to the recovery room. The patient remained hemodynamically stable (blood pressure, 160/100 mmHg; heart rate, 60 beat/min), and no more arrhythmias were observed.

Given that the patient remained hemodynamically stable during SVT, it was decided that ESWL could be safely reattempted at a future date. The planned protocol remained unchanged except for a few provocative maneuvers designed to elucidate the mechanism of SVT. The patient returned 1 week later for definitive treatment of his renal calculus with no changes in his medical history or clinical presentation. The patient received the exact anesthetic regimen as previous except that intravenous atropine, 1 mg, was given in place of glycopyrrolate. The patient's renal stone remained in the same location, and the patient was positioned in the stretcher in the same manner as his previous operation. The patient again developed SVT during shock wave release; however, compared with the previous procedure, onset of tachycardia did not precisely coincide with shock wave release. Similar to the previous operation, SVT reliably terminated by ceasing shock wave release. Fifty-nine episodes of SVT that ranged in duration from 1.5 s to 16 s (4–40 beats) at a rate of 150 beat/min occurred during ESWL. Modification of the shock wave release pattern to 1:2 prevented the induction of SVT, but there was evidence of ESWL-triggered atrial capture with alternate beats (fig. 2). The patient received 2,400 shocks at 20 kV with satisfactory stone fragmentation. At the end of the procedure, the patient received neostigmine 3 mg, and glycopyrrolate 0.6 mg, intravenously and then was extubated without complication. His recovery was uneventful, and the patient was discharged to home that same day.

Discussion

The typical entities that have been known to cause SVT in adults are reentrant tachycardia (AV node reentry

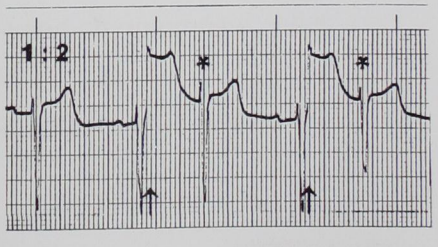

Fig. 2. 1:2 ECG-triggered shock release lithotripsy. Arrows indicate shock waves released on every second beat dependent upon the 1:2 synchronization sequence selected by the operator. Each complex after shock wave release (indicated by an asterisk) is premature, suggesting ESWL-triggered atrial capture with normal conduction. Lead III, paper speed = 25 mm/s and size = 3 cm/mV.

or atrioventricular reentry *via* an accessory AV connection) and atrial tachycardia.[7] Each of these mechanisms has been implicated to induce tachycardia during ESWL therapy.[3] Once induced, these tachycardia typically do not terminate with cessation of shock wave release. In contrast, the lithotriptor served as an integral and unusual component of the SVT mechanism in our patient. The sequence of events were as follows: shock wave-triggered atrial mechanical stimulation, AV nodal conduction followed by ventricular activation, and then R wave-triggered shock wave release to complete the tachycardia circuit. Features that strongly support this mechanism for tachycardia are: (1) dependence on 1:1 shock wave release for tachycardia maintenance and (2) grouped beating caused by ESWL-triggered atrial capture during 1:2 shock wave release synchronization (fig. 2). The premature timing of the QRS complex after shock wave release is a result of ESWL-triggered atrial capture. The upper rate limit of the tachycardia depended on AV nodal conduction. The variability of SVT induction during the second operation was probably a result of intermittent atrial capture.

Several considerations are apparent in this case. Most importantly, the presence of this arrhythmia does not necessarily require terminating ESWL. If a patient presents without significant cardiac risk factors and remains hemodynamically stable intraoperatively, ESWL may be continued without consequence. However, if the patient develops hemodynamic instability or if the development of cardiac ischemia is a significant concern during prolonged SVT, several options are available. Simple repositioning of the patient may prevent supraventricular capture and thereby prevent tachycardia. Another option would be to change the shock wave release pattern to a ratio other than 1:1. Finally, pharmacologic intervention with β-adrenergic or calcium channel blocking agents may be tried with the purpose of slowing AV nodal conduction.

In summary, we present a case of a readily reproducible and easily manipulated SVT during ESWL. Direct atrial stimulation by the release of R wave-triggered shock waves proved to be an integral part of the SVT mechanism.

References

1. Ector H, Janssens L, Baert L, Geest H: Extracorporeal shock wave lithotripsy and cardiac arrhythmias. Pacing Clin Electrophysiol 1989; 12:1910–7

2. Vandeursen H, Tjandramaga B, Verbesselt R, Smet G, Baert L: Anaesthesia-free extracorporeal shock wave lithotripsy in patients with renal calculi. Br J Urol 1991; 68:18–24

3. Walts L, Atlee J: Supraventricular tachycardia associated with extracorporeal shock wave lithotripsy. ANESTHESIOLOGY 1986; 65:521–3

4. Lingeman J, Newman D, Siegel Y, Eichhorn T, Parr K: Shock wave lithotripsy with the Dornier MFL 5000 lithotriptor using an external fixed rate signal. J Urol 1995; 154:951–4

5. Zeng Z, Lindstedt A, Roijer A, Olsson B: Arrhythmia during extracorporeal shock wave lithotripsy. Br J Urol 1993; 71:10–6

6. Kataoka H: Cardiac dysrhythmias related to extracorporeal shock wave lithotripsy using a piezoelectric lithotriptor in patients with kidney stones. J Urol 1995; 153:1390–4

7. Wathen M, Klein G, Yee R, Natale A: Classification and terminology of supraventricular tachycardia: Diagnosis and management of the atrial tachycardia. Cardiol Clin North Am 1993; 11:109–20