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Case Reports

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY PERFORMED ON WOMAN WITH A CARDIAC PACEMAKER

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ABSTRACT

A 73-year-old woman with a sick sinus syndrome and a 3-year-old ventricular demand pacemaker underwent extracorporeal shock wave lithotripsy without incident. There was no damage or malfunction of the pacemaker during or acutely after this therapy. The lithotriptor had no difficulty in sensing all paced or spontaneous ventricular complexes. (*J. Urol., 140: 1510–1511, 1988*)

During the initial clinical experience with extracorporeal shock wave lithotripsy (ESWL†) cardiac arrhythmias were observed in 80 per cent of the patients.[1] These cardiac dysrhythmias were curtailed significantly by synchronizing the lithotriptor with the ventricular (QRS) complex on the surface electrocardiogram, resulting in delivery of the shock wave during the refractory period of the cardiac cycle.[2] The HM3 lithotriptor† senses the R-wave and delivers the shock wave 20 msec. later. A set refractory period is used to prevent rapid firing due to electrical noise or an unexpected tachycardia.

Despite these precautions, the presence of a cardiac pacemaker has been considered a contraindication to ESWL[3] due to the possibility of electrical interference from the spark gap used to produce the shock waves or damage to the pulse generator from the shock waves themselves. In recent studies the effects of ESWL on pacemaker functions in vitro have been investigated.[4–7] Adverse effects could not be produced even when the pulse generator was positioned directly at the focal point of the lithotriptor. Based on these preliminary data, we elected to proceed with ESWL in a patient with a permanent pacemaker.

CASE REPORT

P. R., a 73-year-old woman, presented with left renal colic and a history of intermittent urinary tract infections. She had long-standing hypertension and a sick sinus syndrome with intermittent atrial fibrillation. A permanent ventricular demand pacemaker‡ was implanted in the left infraclavicular region 3 years previously because of symptomatic sinus pauses and a slow response in atrial fibrillation. History also was significant for cerebral vascular disease requiring a carotid endarterectomy. Medications included propranolol, diltiazem, dipyridamole and disopyramide.

Physical examination revealed an elderly, mildly obese woman in no distress. Blood pressure was 110/70 and pulse was 82 and regular. There were scattered bi-basilar rales. The cardiovascular examination was notable for the absence of jugular venous distension and normal carotid upstrokes. The apical impulse was displaced 2 cm. laterally and it was slightly sustained. S4 was appreciated, and S1 and S2 were normal. There was no edema of the extremities.

A preoperative electrocardiogram revealed sinus rhythm with frequent ventricular paced beats. There was appropriate sensing by the pacemaker and 100 per cent capture. A marked first degree atrial-ventricular block was present. Left ventricular hypertrophy was noted and there were inferolateral Q waves consistent with an old myocardial infarction. Mild QT prolongations were believed to be consistent with disopyramide therapy. A preoperative pacing system examination revealed a paced rate of 72 beats per minute and a pulse duration of 0.46 msec. Capture threshold was 0.15 msec., with a sensing threshold greater than 1 (the least sensitive setting). The pacemaker was reprogrammed to a lower rate of 50 beats per minute to allow a greater amount of time in sinus rhythm and the programmed sensitivity was decreased from 4 to 2. Informed written consent was obtained before ESWL. A cardiologist (J. J. L.) familiar with the pacing system stood by with a programmer.

During lithotripsy the patient was in normal sinus rhythm at rates between 65 and 75. No abnormal behavior of the pacing system was observed. Magnet application during lithotripsy demonstrated normal asynchronous (V00) functioning of the pulse generator. The R-wave sensing circuitry of the lithotriptor had no difficulty in sensing all paced and spontaneous complexes (see figure). During asynchronous pacing, when pacing artifacts fell in the absolute refractory period of the ventricle and did not depolorize the heart, on no occasion were they inappropriately sensed by the lithotriptor. The renal calculi were fragmented successfully with a total of 1,800 shocks. A pacemaker examination after ESWL revealed no changes in rate, pulse width, capture threshold or sensitivity.

DISCUSSION

Individuals with cardiac pacemakers have been considered to be inappropriate candidates for ESWL. In vitro studies with numerous models of cardiac pacemakers have shown that ESWL with an HM3 lithotriptor resulted in no spurious reprogramming or damage to the appliance.[4–7] However, no data are available concerning lithotripsy in pacemaker patients. It is ironic that individuals with significant cardiac disease have not been treated with this relatively noninvasive modality.

Our patient had a sick sinus syndrome and a 3-year-old ventricular demand pacemaker implanted into the left infraclavicular region. There was no evidence of pulse generator

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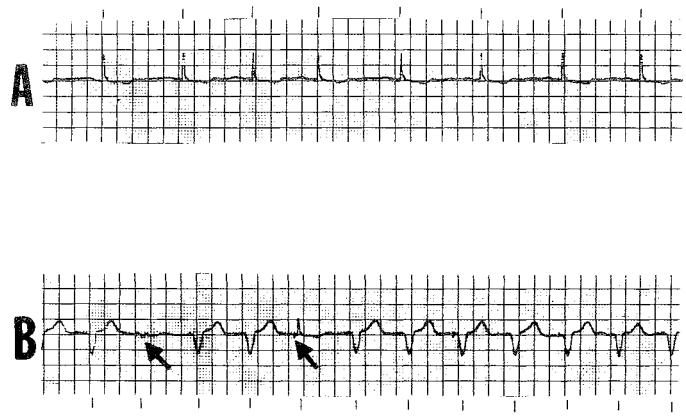
† Dornier Medical Systems, Inc., Marietta, Georgia.
‡ Model 254-01, Intermedics, Foster City, Calif.



Electrocardiogram (lead II) during ESWL. Tic marks indicate timing of shocks. *A*, during sinus rhythm with pacemaker in demand mode. *B*, during asynchronous pacing (V00). Note appropriate sensing of fusion beat (left arrow) by lithotriptor, which is not triggered early by pacing artifact that falls at onset of native QRS (right arrow).

malfunction during ESWL and no change in the pacing threshold or lead sensitivity immediately following the procedure. In addition, pulse generator programming was not changed by exposure to shock waves. The lithotriptor accurately detected R-waves of native and paced origins, and it was not falsely triggered by the pacing artifacts. It is possible that pacing artifacts of larger amplitude might trigger the sensing circuitry of the lithotriptor. Provided that pacemaker function was normal, this would cause the shock wave to occur at the onset of the paced QRS complex and would not result in ectopia. Dual chamber and motion-sensitive, rate-responsive pacing systems have been observed to pace at their programmed upper rate limit during exposure to shock waves in vitro.

Definitive recommendations regarding ESWL in pacemaker patients must await more clinical data. However, based on the aforementioned preliminary data we propose guidelines with regard to permanent pacemakers. A cardiologist familiar with the pacing system and emergency temporary pacing techniques should be present during the procedure. The pacing system should be examined before and after ESWL. Dual chamber (DDD) and activity sensing pulse generators should be reprogrammed temporarily to VV1 during ESWL to prevent pacing at the upper rate limit during the procedure. Patients who are pacemaker-dependent should have a temporary external pacemaker applied before the procedure. It would seem prudent to institute this safe, noninvasive therapy as a backup until more experience has been accumulated in this subset of patients undergoing lithotripsy. Knowledge of the cardiac history, the type and function of the pacemaker, and how to intervene effectively is paramount in treating such individuals. With this knowledge a relatively noninvasive therapy can be used in this patient population to avoid a more invasive procedure.

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