ST-segment Elevation with Elective DC Cardioversion

PATRICK K. C. CHUN, M.D., JAMES E. DAVIA, M.D., AND DENNIS J. DONOHOE, M.D.

SUMMARY  Elective direct-current cardioversion was performed in three patients with atrial fibrillation. Transient ST-segment elevation on monitored leads, lasting seconds, was recorded after cardioversion in all three patients. LDH, CPK isoenzymes, and myocardial scintigraphy did not reveal myocardial damage. Elective cardioversion should be performed with caution, for the potential for cardiac damage cannot be ignored.

ELECTIVE direct-current (DC) cardioversion of certain types of cardiac arrhythmias is an accepted and generally safe therapeutic procedure. Complications, however, have included serious arrhythmias, emboli and pulmonary edema. An unusual complication is the manifestation of transient ST-segment elevation.1-8 Previous investigators, however, have not assessed the extent of myocardial damage because of lack of available and refined techniques, i.e., MB-CPK fractionation and infarct-avid imaging.5-12 In this report we discuss three cases of transient ST-segment elevation after elective DC cardioversion and present a review of the literature. Careful technique during the cardioversion as well as completeness in the evaluation to exclude any evidence for myocardial damage are emphasized.

Case Reports

Case 1

The patient was a 22-year-old male who was in excellent health until 1½ years before admission, when he noted the onset of gradual fatigue. In October 1978 he was seen for a sprained ankle. The physical examination was normal except for an irregular pulse with a rate of 50 beats/minute. The ECG revealed atrial fibrillation with a slow ventricular response. The chest x-ray, echocardiogram, complete blood count, SMA-20, T3 and T4 were normal.

Because of the possibility of latent sick sinus syndrome, a prophylactic temporary pacemaker was positioned in the right ventricle before cardioversion. Dosages of i.v. diazepam (15 mg) and sodium pentothal (500 mg) were given as premedication. The Lifepak 6 (Physio-Control) cardioversion equipment was used. Gauze pads soaked in saline were used as the conductive interface between the chest and defibrillator paddles. A sequence of increasing stored energy levels were delivered: 5, 10, 20, 50, 100, 200 and 400 J. Cardioversion was not accomplished until the highest energy level available was delivered. The electrical discharges were delivered at approximately 1½-minute intervals. The patient’s blood pressure was stable throughout the procedure. However, marked ST-segment elevation was manifested transiently on a lead II rhythm strip immediately after the elective cardioversion with 200 and 400 J (fig. 1). His CPK rose to a high of 6000 U/l (normal 95 U/l), SGOT 214 U/l (normal 72 U/l) and LDH 666 U/l (normal 203 U/l). The 12-lead ECG after cardioversion was normal. His CPK isoenzymes were all MM fraction and his LDH isoenzymes fractions V greater than II, greater than I, greater than III, greater than IV. The gated radionuclide angiogram showed an ejection fraction of 65%. The technetium-99m stannous pyrophosphate (99mTc-PYP) myocardial scintigram and the thallium-201 (201TI) scintigram were normal except for the intense radionuclide concentration over the right superoanterior chest wall and left lateral rib cage, where the defibrillator paddles had been positioned (fig. 2). Subsequent His bundle electrogram and sinus node recovery time studies were normal.

Case 2

The patient was a 24-year-old male who was in excellent health until February 1977, when he had the onset of nausea and weakness and an ECG revealed atrial fibrillation with a ventricular rate of 65 beats/minute. Treatment with digoxin and quinidine sulfate was unsuccessful and he was converted by DC cardioversion to a normal sinus rhythm. He remained asymptomatic until August 1977, when he developed exertional chest pain and was again found to have atrial fibrillation. His physical examination was normal except for an irregular pulse. His complete blood count, SMA-20, T3, T4, chest x-ray, echocardiogram and treadmill test were normal. A temporary pacemaker was inserted before DC cardioversion. Sodium pentothal, 500 mg i.v., and fentanyl, 1.5 ml i.v., were given for sedation. After 50, 100 and 200 J were delivered 2 minutes apart with the Lifepak 6 and saline pads, his rhythm converted to normal sinus rhythm. His blood pressure remained stable during the procedure. However, ST-segment elevation developed at 100 and 200 J (fig. 3). His subsequent ECG was normal and showed no changes of an evolving infarct. His CPK rose to a high of 2807 U/l, SGOT 119 U/l, and LDH 313 U/l. The CPK isoenzymes were all MM, and his LDH isoenzymes V
greater than II, greater than I, greater than III, greater than IV. His gated radionuclide angiocardiogram revealed an ejection fraction of 67% and the $^{99m}$Tc-PYP scan and $^{201}$TI scan were normal. Subsequent His bundle and sinus node recovery time studies after cardioversion were also normal.

Case 3

The patient was a 68-year-old female with severe mitral insufficiency who had undergone mitral valve replacement with a #31 Hancock bioprosthesis in March 1977. Perioperatively she sustained an inferior myocardial infarction. Her postoperative course was complicated by sternal dehiscence followed by respiratory insufficiency, necessitating a tracheostomy. She also developed left ventricular failure requiring pressor and aortic balloon counterpulsation therapies. After discharge she developed several episodes of atrial flutter with 2:1 atrioventricular conduction alternating with episodes of rapid atrial fibrillation and associated with congestive heart failure. After an arrhythmic episode on January 9, 1979 failed to convert to sinus rhythm with quinidine and digoxin therapy, she was admitted for elective cardioversion.

A physical examination before cardioversion revealed clear lungs, a grade II/VI mitral regurgitant murmur at the apex, and trace edema in the extremities. Her complete blood count, SMA-20, T3, T4, and digoxin level were normal. The chest x-ray showed cardiomegaly with a Hancock ring present. The echocardiogram showed mild left atrial enlargement with the Hancock prosthesis in position.

Sodium pentothal, 500 mg i.v., and diazepam, 10
mg i.v., were administered for sedation. Energy levels of 10, 20, 50, 100, 200, and 400 J delivered approximately 1½ minutes apart with the Lifepak 6 and saline pads failed to restore normal sinus rhythm. Transient ST-segment elevation was present with 200 J and 400 J energy levels (fig. 4). Her subsequent ECG persisted in showing atrial fibrillation. The CPK rose to 348 U/l, SGOT 25 U/l, and LDH 294 U/l. Her CPK isoenzymes were MM, and LDH isoenzymes II greater than III, greater than I, greater than V, greater than IV. The gated radionuclide angiogram revealed inferior wall hypokinesis and an ejection fraction of 34%. The 99mTc-PYP scan was negative and the 201Tl scan was consistent with an antecedent inferior infarct.

**Discussion**

Complications of DC cardioversion may be anticipated in approximately 14.5% of patients, including complications from the anesthesia, e.g., hypotension, cardiac irritability and depression, arrhythmias, acute pulmonary edema, emboli, and electrical damage to the heart and its surrounding structures, e.g., intercostal and pectoral muscles and the lungs.

Electricity can injure a variety of tissues, including the myocardium. The extent of injury is a function of shock strength, the impedance of the heart, ribs and skin, acetylcholine and catecholamines liberated, the use of paste vs saline pads and creams, the size of the paddle electrodes, and the amount of pressure applied. Thoracic windows, anteroposterior vs apex-base position, time interval between discharges, prior transthoracic DC shocks, body weight-dose relationship, acid-base and electrolyte balance, and the circumstances underlying the arrhythmias are additional variables affecting the extent of injury. Every patient, therefore, requires individual assessment to determine the optimal approach to cardioversion.

ST-segment elevation in humans after energy delivery has usually been attributed to such drugs as digitalis or quinidine or perhaps pericardial injury or variant angina. In dogs, the extent of ECG changes and elevation in CPK levels as well as mortality were dependent on the shock strength, although none of these three variables correlated well with one another. Transient ECG changes were very frequent after countershock regardless of the amount of myocardial damage documented. Thus, ST-segment elevation was considered a nonspecific finding, although the question of myocardial damage associated with ST-segment elevation in humans has not been evaluated. Frequently, only lead II or V1 is monitored, and ST-segment elevation in other leads would be missed.

Only 20 cases of ST-T-segment changes after DC cardioversion have been reported. Only six patients were said to have definite ST-segment elevation, but all were poorly documented. Our patients show that transient ST-segment elevation after DC cardioversion does not necessarily indicate myocardial injury. However, when ST-segment elevation is seen, further
evaluation with isoenzymes and scintigraphy should be continued to exclude necrosis. The potential for cardiac damage cannot be ignored, although there are reports of patients receiving more than 7500 J within 16 hours without evidence at autopsy of injury.\textsuperscript{23, 24} Cardioversions should be performed cautiously, especially in an elective setting. Effort should be made to consider the ideal energy dose for countershock in each patient, enough to cardiovert but not enough to cause myocardial injury. Use of paste, large paddles and the anteroposterior electrode position, with a minimum of 3 minutes between shocks, is recommended.\textsuperscript{20}
References

ST-segment elevation with elective DC cardioversion.
P K Chun, J E Davia and D J Donohue

Circulation. 1981;63:220-224
doi: 10.1161/01.CIR.63.1.220

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/63/1/220

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/