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Editorial Comment

Cardioversion and the Digitalized Patient*

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Transthoracic synchronized direct current discharge or cardioversion was introduced nearly 25 years ago to restore sinus rhythm in patients with diverse tachyarrhythmias (1). This simple, expeditious and nearly complication-free method has withstood the test of time.

The likelihood of serious adverse reactions is related more to the underlying mechanism being treated than to the electrical discharge itself. Systemic and pulmonary embolism are among the more serious complications and are encountered in about 1% of those who undergo cardioversion of atrial fibrillation without anticoagulant drug prophylaxis. In rare instances, cardioversion induces or aggravates preexisting pulmonary congestion. Immediately after the procedure, arrhythmias are common, usually consisting of atrial, junctional or ventricular ectopic beats that disappear within several minutes. Malignant arrhythmias are the result of improper synchronization or excessive electrical energy, generally in patients with severe degrees of myocardial dysfunction.

Shortly after the introduction of cardioversion, the report of two deaths (2,3) raised the concern that the digitalized patient may be susceptible to electrical shock-induced arrhythmias. Both patients were overdigitalized and the electrical discharge provoked irreversible ventricular fibrillation. Animal experiments (4) have confirmed that digitalization enhances the arrhythmogenic effect of transthoracic shock. In normal dogs digitalized to near toxicity, the median energy for inducing ventricular tachycardia is lowered from 400 to 0.2 J, a 2,000-fold reduction. The arrhythmia provoked by electrical discharge in the digitalized heart is identical in configuration and rate to that resulting from excess digitalis in the absence of electric shock. When the electrical threshold is tested during incremental digitalization, increased susceptibility to ventricular tachycardia requires administration of approximately 85% of a toxic dose of ouabain.

In the digitalized heart, pacemaker stimuli to either right or left ventricular endocardium elicit repetitive ventricular responses with as little as 1 to 2 μJ (5). In the normal heart, such responses are difficult to induce except with extraordinarily high currents of about 5 to 6 J; this represents a difference of six orders of magnitude in energy level. With advancing degrees of digitalization, the repetitive response increases in the number of successive cycles, resulting ultimately in sustained ventricular tachycardia. The phenomenon of repetitive ventricular response has been elicited in diverse mammalian species as well as in human beings (6). There can be little doubt that digitalis drugs sensitize the heart to electrical shock-induced ventricular arrhythmias.

Clinicians accept this fact, but have been hard pressed to identify the subject at risk. The practice has been to discontinue digitalis drugs for 1 day or more before cardioversion. This approach has a disadvantage for the patient who is dependent on digitalis for ventricular rate control. Stopping digoxin for 1 day or more unduly accelerates the heart rate, especially after initiation of quinidine therapy. If cardiac reserve already is compromised, discontinuation of glycosides may provoke decompensation, impede restoration of sinus rhythm and, even if cardioversion is immediately successful, the normal mechanism may not persist.

Role of the Digoxin Blood Level

The study of Mann et al. (7) in this issue is, therefore, of some interest. Their thesis is that if the digoxin blood level is in a therapeutic range (0.5 to 1.9 ng/ml), cardioversion does not provoke ventricular ectopic activity even after high levels of applied shock. This confirms the earlier report of Ditchey and Karliner (8), who found no increase in ventricular ectopic activity among 21 patients with serum digoxin levels ranging from 0.1 to 3.0 ng/ml (mean 1.6). Of note was that among their eight patients with modestly elevated serum digoxin concentrations (<2.0 ng/ml), ventricular premature beats remained unchanged in pre- and post-shock recordings. The study by Ditchey and Karliner may have been confounded by the fact that their patients, unlike those of Mann et al., were receiving quinidine. However, quinidine pretreatment has not been shown to protect against postcardioversion ventricular arrhythmias (9).

A single variable such as the serum digoxin concentration may not predict safety from postcardioversion arrhythmias. Indeed, in digitalized patients, no correlation exists between the serum digoxin level and proximity to digitalis intoxi-

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cation when assessed by means of the ultrarapid-acting cardiac aglycone, acetylstrophanthidin (10). The serum digoxin concentration does not provide information concerning myocardial sensitivity to glycoside-provoked ventricular arrhythmias. Cardiac sensitivity is determined by a multiplicity of variables, including myocardial functional impairment, presence and extent of ischemia, age of the patient, serum potassium and magnesium concentrations, the state of adrenergic tone and a host of other factors. It is, therefore, unlikely that the serum digoxin level in the individual patient will predict development of cardioversion-induced arrhythmias. One would have to concur with the conclusion of Ditchey and Karliner "that within reasonable limits, serum digoxin levels are of little value in predicting the risk of electrical cardioversion in patients apparently without digitalis toxicity."

Clinical Recommendations

How then is one to deal with the digitalized patient with atrial fibrillation who is to undergo cardioversion? Twenty years ago, it was already known that the patient who developed malignant ventricular arrhythmias after cardioversion presented with findings suggestive of digitalis overdosage before cardioversion (11). Now we know that the patient with atrial fibrillation who exhibits paroxysms of regularized rhythm, bradycardia or early junctional or idioventricular escape with carotid sinus massage has a 50% chance of developing ventricular arrhythmias after cardioversion. In such cases, one can either delay cardioversion or titrate the energy of the discharge. The latter procedure assures the safety of cardioversion. The starting energy is adjusted to the anticipated likelihood of overdigitalization. When this possibility is high, one begins with 10 J and progresses stepwise to 50, 100, 200 J and so forth (12). If ectopic activity emerges that can be subdued with lidocaine, it is safe to proceed to the next level of energy. Attention to these methodologic details, in combination with a relaxed patient who is reassured about the safety of the procedure, diminishes the likelihood of serious complications. Adhering to these principles, we have encountered no major untoward effects in more than 1,000 cardioversions for chronic atrial fibrillation.

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