Cardioversion

Ten-year Cleveland Clinic experience

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Direct current electric shock has been widely used in the past 2 decades to convert supraventricular and ventricular dysrhythmias to sinus mechanism. Initial reports stressed the benign nature of this treatment. Subsequently, it was demonstrated that cardioversion could lead to potentially serious complications. Much has been learned about indications, contraindications, and prevention of complications in utilization of direct current electric shock for the treatment of cardiac dysrhythmias. Our experience of more than 10 years with cardioversion at The Cleveland Clinic Foundation is discussed.

Historical considerations

The use of electrical energy for termination of ventricular fibrillation dates back to the turn of the century. In 1899, Prevost and Battelli[1] applied electrical current directly to the hearts of dogs to induce ventricular fibrillation, and at the same time observed that similar electrical discharges were capable of terminating ventricular fibrillation. In 1940, Wiggers[2] confirmed the effectiveness of electrical current in defibrillating the exposed heart after a series of extensive investigations. These studies resulted in the first successful defibrillation of the human heart by Beck et al[3] in 1947; the patient recovered

completely. Alexander et al,[4] in 1961, were the first to employ A-C electricity electively in patients with coronary artery disease to terminate an episode of ventricular tachycardia which was found to be resistant to antiarrhythmic drugs. This successful transthoracic use of alternating current shock led to a new era in the treatment of cardiac dysrhythmias. After extensive experiments in the animal laboratory, synchronized direct current cardioversion to avoid the vulnerable period in the cardiac cycle was developed. Since then, this method has been universally accepted for the termination of most supraventricular and ventricular dysrhythmias. In 1967, a classic paper by Lown[5] was published.

Materials and methods

Between 1964 and 1974, elective synchronized direct current electric cardioversion was applied in 1,002 instances in 894 patients at the Cleveland Clinic. The success of cardioversion and its complications were correlated with patients' clinical features (etiology of organic heart disease, duration of dysrhythmia, roentgenographic findings, and the type of drug therapy, including anticoagulation. The incidence of systemic embolization, before and after cardioversion, was specifically studied in this group of patients in relation to the use of anticoagulant therapy before application of cardioversion. The study group consisted of 894 patients 499 men (56%) and 395 women (44%). Five hundred fifty-one patients (62%) had rheumatic valvular heart disease. The predominant valvular lesions among this group were mitral stenosis (52%), aortic regurgitation (22%), aortic stenosis (15%), and mitral regurgitation (11%). This study represents a special group of patients, since 840 (94%) had had cardiac surgery prior to cardioversion. Most of these patients had undergone valvular heart surgery (mitral valve replacement, aortic valve replacement, mitral commissurotomy). Only 182 patients (20%) of this group had arteriosclerotic heart disease. The method and technique used in cardioversion was essentially the same as described by Lown.[5–7] Digitalis therapy was discontinued 24 to 48 hours before cardioversion in the majority of patients. Quinidine or procainamide hydrochloride (Pronestyl) was used prior to cardioversion in patients with chronic atrial fibrillation, and in 523 instances of cardioversion in this study. In 326 instances (32%) of cardioversion, either quinidine or procainamide hydrochloride therapy was continued after successful reversion of the dysrhythmia to sinus rhythm. Similarly, digitalis therapy was continued after successful cardioversion in most of the patients with chronic atrial fibrillation or flutter. Anticoagulant therapy had been instituted in 243 instances (24%) of cardioversion in patients with predominantly mitral valve replacement, aortic valve replacement, or mitral commissurotomy.

Cardioversion was applied under light anesthesia with methohexitol sodium (Brevital) administered intravenously. Synchronized direct current electric countershock was used in nearly all patients. The energy applied ranged from 50 watt seconds to 300 watt seconds. Lower levels of electrical energy were applied at first, and higher levels of energy were used only if the initial countershock failed.

Follow-up data were available on 705 patients (79%). These follow-up data were determined by reviewing the records of the patients, direct communication with the patients and the referring physicians.

Analysis of results

Successful cardioversion. Sinus mechanism was restored by DC cardioversion in 874 attempts (87%). In these successful instances of cardioversion, 729 patients (83%) had atrial fibrillation, in contrast to 139 patients (16%) who had atrial flutter. Among the successful group, 693 patients (79%) received either quinidine or procainamide hydrochloride prior to cardioversion. The success rate was higher among patients with atrial flutter, since 97% of the instances of atrial flutter were converted to sinus mechanism, in contrast to 85% conversion to sinus mechanism in patients with atrial fibrillation. Although the patients with ventricular tachycardia were few, the success rate was 100% in obtaining sinus mechanism with the first attempt at cardioversion (*Table 1*).

Unsuccessful attempts. In 128 attempts (13% of cases), cardioversion failed to restore sinus mechanism. Most of these patients had moderate to severe left atrial enlargement and long-standing atrial fibrillation. Ninety-six instances (75%) of failure of cardioversion were in patients with moderate to severe left atrial enlargement. Only 32 instances (25%) of failure of cardioversion were in patients with only mild left atrial enlargement, or no left atrial enlargement. One hundred twenty-four failures of cardioversion (97%) were in patients with atrial fibrillation; only four failures of cardioversion (3%) were among patients with atrial flutter. Concerning the duration of the atrial dysrhythmia, 52 instances (41%) of failure occurred in patients with atrial fibrillation of longer than 5 years. Ninety-three instances (73%) of failure to restore sinus mechanism by cardioversion occurred in patients with atrial dysrhythmia of longer than 1 year. One hundred eight attempts (84%) that failed to restore sinus mechanism by cardioversion occurred in patients with previous mitral valve replacement or open mitral commissurotomy, in contrast to failure in two patients (1.6%) of those who had aortic valve replacement. In 33 attempts (26%) which failed, the patients had one or more previous cardioversions for similar dysrhythmia. Nearly all patients in the failure group were treated with digitalis until 1 or 2 days prior to cardioversion. There was no significant difference concerning precardioversion medical treatment, since nearly equal numbers of patients in whom failure to convert dysrhythmia to sinus mechanism were treated with quinidine and procainamide hydrochloride. In 99 instances (77%) of failure, quinidine and procainamide hydrochloride were used before cardioversion.

Table 1. Success rate of cardioversion; atrial fibrillation vs. atrial flutter

Dysrhythmia	No. attempts	No. successes	%
Atrial fibrillation	853	729	85
Atrial flutter	143	139	97

Postcardioversion thromboembolism

Anticoagulant therapy prior to cardioversion was employed in 243 instances (24%) in which direct current

countershock was applied for conversion of dysrhythmia. In 218 cases (90%), an anticoagulant was used because the patient had undergone mitral valve replacement, aortic valve replacement, or had previous systemic embolization. Ninety-four patients (38.7%) treated with anticoagulants prior to cardioversion had previous systemic embolization. In 41 instances (17%) of anticoagulant use prior to cardioversion, some sort of systemic embolization occurred during the long-term follow-up. Six patients in the group that received anticoagulants (2.5%) sustained cerebral or peripheral embolization in the Ist week after cardioversion. In contrast, among the patients in whom anticoagulant therapy was not used prior to cardioversion (759 instances, 76%), systemic embolization occurred in 153 instances (20%) during the long-term follow-up. Twenty patients (2.7%) who had not received anticoagulants sustained systemic embolization during the first week after cardioversion.

The comparison of these two groups that had and had not received anticoagulants prior to cardioversion showed no statistically significant difference in the prevalence of systemic embolization during the first week after cardioversion. Furthermore, of the group of patients in whom cardioversion had failed to restore sinus mechanism, a similar number of patients sustained systemic embolization during the first week after cardioversion was attempted, three patients (2.3%) in 128 instances of failure of cardioversion. Similarly, in 30 of 128 instances of failure of cardioversion (23%), systemic embolization occurred in long-term follow-up. Systemic embolization occurred in 194 (19%) of the total number cases of direct current cardioversion attempts after the countershock was given during the long-term follow-up. Twenty-three of these patients (12%) were found to have left atrial thrombus either at angiography or at the time of surgery. In six of 23 patients (26%) with left atrial thrombus, systemic embolization occurred within the first week after cardioversion. In 62 of 194 instances of systemic embolization after cardioversion (32%), the patients had a history of previous cerebral or peripheral embolization prior to cardioversion. Five of 26 patients who sustained systemic embolization during the first week after successful cardioversion died as a result of massive cerebral infarction. The majority of the survivors who sustained cerebral embolus during the first week after cardioversion had moderate to severe disability as the result of brain infarction. Of 194 instances of thromboembolism in long-term follow-up after cardioversion, 94 patients had mitral valve replacement and 40 had open mitral commissurotomy. Another 22 instances of thromboembolism occurred in patients in whom aortic valve replacement had been performed. It is therefore quite appropriate to assume that in most instances, thromboembolism had occurred because of basic valvular heart disease or prosthetic valve replacement (*Tables 2 and 3*).

Mortality and morbidity

Mortality. The incidence of death among patients who underwent cardioversion was studied in two groups: instances of successful conversion of the dysrhythmia to sinus mechanism, and those instances of failure to convert the dysrhythmia. In addition, first week mortality and long-term

Table 2. Incidence of early (less than 1 month) thromboembolic phenomenon after reversion to sinus rhythm

	Incidence	Rhythm		Anticoagulation		Atrial thrombus
		AF	AFL	Before CV	After CV	
24 hr	6 (16%)	5	1	2	2	1
24 hr to 1 wk	20 (54%)	17	3	4	15	5
1 wk to 1 mo	11 (30%)	9	1	1	4	1
Total	37 (4%)	31 (84%)	5 (14%)	7 (19%)	21(57%)	7 (19%)

Total incidence of early emboli (1 month) is 4% (37 of 874 instances) in successful and unsuccessful DC cardioversion. Total number of cardioversions, 874.

Table 3. Incidence of systemic embolism in all patients

Type of treatment	No. of cases	Incidence in less than 1 wk	Incidence in long-term follow-up (up to 10 yr)
Anticoagulation prior to successful cardioversion	243 (24%)	6 (2.5%)	41 (17%)
No anticoagulation prior to successful cardioversion	759 (76%)	20 (2.7%)	153 (20%)
All patients with unsuccessful cardioversion	128 (13%)	3 (2.3%)	30 (23%)

mortality were separated, because long-term mortality undoubtedly would be related to the nature of the patient's heart disease. However, death during the first week may have some relation to the cardioversion. No deaths occurred in the first week after cardioversion among 128 instances of unsuccessful attempts. In 874 successful attempts, 10 patients died during the first week after cardioversion. As mentioned before, five of these patients died as the result of massive brain infarction. Two patients died as the result of ventricular tachycardia deteriorating to ventricular fibrillation. These ventricular dysrhythmias occurred 1 and 2 days after cardioversion, and were not apparent immediately after the procedure. The cause of death in another two patients was not known; one of these patients was found dead in bed 2 hours after cardioversion, and the second patient was found dead in bed the following day. Finally, one patient died suddenly of ventricular fibrillation the day following cardioversion, and autopsy showed evidence of myocardial necrosis.

Morbidity. Less serious arrhythmias were sparsely recorded and, therefore, no accurate instances of these dysrhythmias can be given. In occasional cases, premature ventricular contractions, runs of bigeminy, premature atrial contractions, and multifocal supraventricular ectopic beats (wandering pacemaker) have occurred among this large group of patients. No serious burn or other significant complications were found in these patients as the result of applications of direct current electric countershock.

Conclusion

This study outlines the results and possible complications of direct current electric countershock applied in 1,002 instances in 894 patients during

1 decade at The Cleveland Clinic Foundation. It should be emphasized that this mode of therapy was applied in a special group of patients, the majority of whom had valvular and rheumatic heart disease, and more specifically most of them had undergone valvular heart surgery (mitral valve replacement, aortic valve replacement, and mitral commissurotomy). The mortality and morbidity in this group of patients undoubtedly reflect the manifestations of their primary heart disease in most instances. The incidence of systemic embolization after restoration of sinus mechanism was not found to be significantly different between the group of patients that had received anticoagulants prior to cardioversion and the group of patients that had not received anticoagulants prior to cardioversion. Furthermore, the group of patients in whom sinus mechanism was not restored had an incidence of systemic embolization similar to those patients with successful reversion to sinus mechanism.

Although rare, serious ventricular dysrhythmias as well as disabling or fatal systemic embolization have been observed early after cardioversion in this group of patients studied. Since the ventricular dysrhythmias occurring rarely after cardioversion are basically preventable, careful monitoring of the patient after cardioversion is recommended. Hypopotassemia and digitalis therapy immediately before cardioversion may enhance the chance for dysrhythmia occurring immediately after cardioversion. Application of very low levels of electrical energy (25- to 50-watt seconds) is recommended when there is a question of digitalis excess.

Cardioversion by direct current electric countershock remains the most effective modality in treating many dysrhythmias. It is most effective in patients with atrial flutter and ventricular tachycardia.

Cardioversion is quite effective in patients with atrial fibrillation and supraventricular tachycardia. In patients with ventricular tachycardia, and rapid supraventricular tachycardia capable of producing significantly reduced cardiac output, cardioversion has been life-saving. Drug therapy, such as digitalis, propranolol, procainamide hydrochloride, and quinidine, depending on the type of dysrhythmia, remain good adjunct therapy in preventing repeated episodes of dysrhythmia. Based on this study, the routine use of anticoagulants prior to cardioversion cannot be recommended.

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