Direct current shock and antidysrhythmic drugs

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A review is given of 457 episodes of atrial fibrillation that occurred in 318 patients and were treated by DC shock. Antidysrhythmic drugs, such as quinidine, procainamide, and propranolol, given singly or in combination, were used concomitantly in 389 instances, and DC shock alone was given in 68 instances. The combined effects of quinidine and DC shock, and of procainamide and DC shock were studied in the experimental animal.

Combined DC shock and drug therapy gave a higher conversion rate than DC shock alone, and a statistically significant difference was found in respect of the group of patients receiving procainamide and propranolol together ($p < 0.01$).

Antidysrhythmic drugs failed on the whole to reduce the incidence of DC shock-induced dysrhythmias. However, the incidence of certain digitalis and DC shock-induced dysrhythmias was significantly less when propranolol and procainamide were given as pretreatment than when procainamide or quinidine was given alone ($p < 0.01$).

In animal experiments, quinidine had no protective action against digitalis and DC shock-induced ectopic tachycardias.

Clinical and experimental observations suggest that the cardiotoxicity of these drugs may be enhanced by DC shock. Immediate or delayed post-shock rhythm disorders can be drug related and, therefore, great caution should be exercised in the use of antidysrhythmic drugs in conjunction with DC shock therapy.

In a previous paper (Szekely *et al.*, 1969) we reported on the effects of the concomitant use of digitalis and direct current (DC) shock, and presented clinical and experimental evidence, in agreement with others (Lown, Kleiger, and Williams, 1965; Kleiger and Lown, 1966; Oram, 1967), that DC shock potentiates the cardiotoxicity of digitalis. In the present communication, our aim is to evaluate clinical and experimental observations on the concomitant use of various antidysrhythmic drugs and DC shock.

Method, clinical groups, and experimental material

(A) Clinical Five hundred and fifty consecutive episodes of ectopic tachycardia treated by DC shock have been reviewed. Of these, a total of 457 episodes of atrial fibrillation occurring in 318 patients form the basis of the present observations. 241 patients had rheumatic heart disease, 30 had coronary heart disease with or without hypertension, 4 had congenital heart disease, 11 had thyrotoxicosis, 1 had chronic pulmonary heart disease, and there were 31 patients with no firm clinical diagnosis, and a number of these were

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suspected to have cardiomyopathy. In 389 instances digitalis, quinidine, procainamide, and propranolol were used singly or in combination at the time of DC shock therapy, and in the remaining 68 instances no antidysrhythmic drugs were used at all. The method of DC shock treatment was the same as described before (Szekely, Batson, and Stark, 1966; Szekely *et al.*, 1969).

(B) Experimental A total of 30 experiments was carried out in cats weighing between 1.4 and 3.5 kg. The animals were anaesthetized with intraperitoneal sodium pentobarbitone, 45–50 mg./kg. The trachea was cannulated, and artificial ventilation was given intermittently with a respiratory pump. The arterial blood pressure was continuously recorded by a mercury manometer which was attached to the carotid artery. A standard lead II or occasionally lead III electrocardiogram was recorded throughout the experiment. Quinidine and procainamide were given by injection into the femoral vein. DC shocks were delivered at an energy level setting of 40 to 60 joules.

Clinical observations

The 457 episodes of atrial fibrillation treated by DC shock were divided into several

TABLE 1 *Direct current shock and concomitant drug therapy in atrial fibrillation: underlying pathology and results*

Underlying pathology		Concomitant drug therapy													
		Quinidine		Quinidine and propranolol		Procainamide		Procainamide and propranolol		Propranolol		No drug therapy		Total	
		AF No.	SR re-stored (%)	AF No.	SR re-stored (%)	AF No.	SR re-stored (%)	AF No.	SR re-stored (%)	AF No.	SR re-stored (%)	AF No.	SR re-stored (%)	AF No.	SR re-stored (%)
Rheumatic heart disease	A	26	88	5	100	74	84	23	96	3	100	21	85	152	87
	B	50	90	8	100	76	84	48	96	4	100	25	85	211	89
	Total	76	89	13	100	150	84	71	96	7	100	46	85	363	88
Coronary heart disease		0	0	0	0	15	80	4	100	1	100	11	73	31	81
Thyrotoxicosis		0	0	0	0	8	100	3	66	1	100	0	0	12	92
Cor pulmonale		0	0	0	0	0	0	0	0	0	0	1	0	1	0
Congenital heart disease		4	100	0	0	4	50	1	100	0	0	1	100	10	80
Lone atrial fibrillation		3	33	0	0	15	60	12	75	1	0	9	55	40	60
Total		83	88	13	100	192	82	91	92	10	90	68	78	457	85

A pre-surgical; B post-surgical; AF atrial fibrillation; SR sinus rhythm.

groups according to the concomitant drug therapy. The nature of the underlying pathology in the various drug groups and the immediate results are shown in Table 1. In the rheumatic group, 152 episodes of atrial fibrillation were treated partly in patients who underwent cardiovascular surgery subsequently and partly in patients who have not had surgical treatment to date (Group A), and 211 episodes were treated at varying

time intervals after cardiac surgery (Group B).

Quinidine 83 episodes of atrial fibrillation occurring in 66 patients were studied in this group. On 23 occasions the patients received quinidine sulphate, 1 g. daily, and on 60 occasions a long-acting quinidine preparation (Kinidin durules) was used, 800 to 1200 mg. daily, 3 to 5 days before electroconversion.

TABLE 2 *Direct current shock and concomitant drug therapy in atrial fibrillation: immediate results and post-shock dysrhythmias*

Drugs used	No. of episodes treated	Sinus rhythm restored	Nature and incidence of post-shock dysrhythmias											
			Atrial extrasystoles		Atrial tachycardia		Nodal rhythm		Ventricular extrasystoles		Ventricular tachycardia		AV block	
			No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Quinidine	With digitalis	22	20	90	3	13.5	4	18	5	23	9	41	3	13.5
	No digitalis	61	53	87	7	11.5	2	3.5	2	3.5	9	15	2	3.5
	Total	83	73	88	10	12	6	7	7	8.5	18	22	5	6
Quinidine and propranolol, no digitalis	13	13	100	2	15	0	0	1	7.5	1	7.5	0	0	0
Procainamide	With digitalis	115	96	84	13	11	15	13	10	8.5	35	30	8	7
	No digitalis	77	61	80	8	10	3	4	3	4	9	11.5	3	4
	Total	192	157	82	21	11	18	9.5	13	7	44	23	11	6
Procainamide and propranolol	With digitalis	60	57	95	6	10	4	7	4	7	10	17	5	8.5
	No digitalis	31	27	87	3	10	1	3	1	3	3	10	1	3
	Total	91	84	92	9	10	5	5.5	5	5.5	13	14	6	6.5
Propranolol, no digitalis	10	9	90	1	10	0	0	1	10	1	10	0	0	0
No drug therapy	68	53	78	7	10	2	3	1	1.5	3	4.5	0	0	0
Total	457	389	85	50	11	31	7	28	6	80	17.5	22	5	5

On 22 occasions the patients received digitalis in full or reduced maintenance doses. On 61 occasions digitalis was discontinued 3 to 5 days before DC shock treatment. The immediate results together with the incidence and nature of post-shock dysrhythmias in the two groups are shown in Table 2. Sinus rhythm was restored in 90 per cent and 87 per cent of the cases, respectively. The average serum quinidine level was 3.2 mg./l. at the time of attempted conversion, as estimated according to the method described by Brodie and Udenfriend (1943). The average energy which achieved conversion was 140 joules. The over-all incidence of atrial extrasystoles was 12 per cent, and of short runs of atrial tachycardia or persisting atrial tachycardia 7 per cent. Nodal rhythm was observed in 8.5 per cent, ventricular extrasystoles in 22 per cent, and ventricular tachycardia or aberrant conduction simulating ventricular tachycardia in 6 per cent of the cases. There was 1 episode (1.2%) of partial heart block persisting for 2 hours. However, the incidence of most of these dysrhythmias was less when digitalis was not used, as seen in Table 2. The serum potassium which was determined in the great majority of the patients, ranged from 3.6 to 4.5 mEq/l. the average being 4.0 mEq/l. in both digitalis and non-digitalis groups.

The immediate post-shock dysrhythmias were of short duration and disappeared as a rule spontaneously. In 2 instances of atrial tachycardia and one instance of ventricular tachycardia a further DC shock was applied successfully. In 2 instances of bradycardia, one sinus in origin and one from nodal rhythm, intravenous atropine resulted in an increase in the heart rate. These two episodes of bradycardia were associated with significant arterial hypotension, 80/60 and 75/50 mm. Hg, respectively.

There were 2 serious late complications: a 54-year-old woman with rheumatic heart disease developed a fatal syncope due to ventricular fibrillation 6 days after atrial defibrillation while on quinidine sulphate 1 g. daily and digoxin 0.25 mg. daily. Another patient, a 42-year-old woman with rheumatic heart disease who had a successful mitral valvotomy 9 years earlier and developed atrial fibrillation only 2 weeks before, had a syncope due to ventricular fibrillation 24 hours after atrial defibrillation. At this stage she had been on quinidine sulphate (Kinidin durules), 800 mg. daily, for 5 days. The serum quinidine level was 6.1 mg./l. on the day the syncope occurred. A post-conversion electrocardiogram taken 22 hours after atrial

defibrillation and 2 hours before the onset of ventricular fibrillation showed deeply inverted T waves and slight prolongation of the QT interval suggesting myocardial injury and quinidine effect (Fig. 1). The serum transaminase level was raised to 85 units/ml. Quinidine was discontinued and propranolol substituted. The patient made a good recovery and she is still in sinus rhythm 12 months after the episode.

Quinidine and propranolol In 13 instances in 9 patients quinidine sulphate 800 mg. daily and propranolol 30 to 60 mg. daily were used 3 to 5 days before elective electroconversion (Table 2). Sinus rhythm was restored in all 13 cases. The average serum quinidine level was 3.5 mg./l. The average energy necessary for conversion was 150 joules. Atrial extrasystoles were observed immediately after conversion in 15 per cent, nodal rhythm in 7.5 per cent, and ventricular extrasystoles in 7.5 per cent of the cases. In this group, serum potassium levels ranged from 3.8 to 4.8 mEq/l., averaging 4.2 mEq/l.

On 2 occasions, the heart rate immediately after DC shock was unduly slow for a few seconds, suggesting sino-atrial node depression.

Procainamide Included in this group are 192 episodes of atrial fibrillation occurring in 171 patients. The patients received procainamide, 3 g. daily, 3 to 5 days before electroconversion. On 115 occasions the patients continued on a maintenance dose of digitalis and on 77 occasions digitalis was discontinued 3 to 5 days before electroconversion, or not used at all (Table 2). Sinus rhythm was restored in the two groups in 84 and 80 per cent of the cases, respectively. The average energy necessary to restore sinus rhythm was 125 joules. The over-all incidence of atrial extrasystoles was 11 per cent, of atrial tachycardia 9.5 per cent, of nodal rhythm 7 per cent, of ventricular extrasystoles 23 per cent, and of ventricular tachycardia 6 per cent. There were 3 episodes of partial AV block (1.5%), lasting a few seconds, 30 minutes, and 3 hours, respectively. Here again, the incidence of post-shock dysrhythmias was higher when digitalis was given as well, as seen in Table 2. Serum potassium levels ranged from 3.8 to 4.4 mEq/l., averaging 3.9 mEq/l. in the digitalis group, and 4.0 mEq/l. in the non-digitalis group.

Procainamide and propranolol There were 77 patients who received on 91 occasions 2 g. procainamide and 30 to 80 mg. propranolol daily for 3 to 5 days before electroconversion. On 60 occasions the patients continued with a maintenance dose of digoxin, and on 31 occasions digitalis was discontinued 3 to 5 days before electroconversion. The results are shown in Table 2. Sinus rhythm was restored in 95 and 87 per cent of the cases, respectively. The average energy necessary for conversion was 115 joules. Of the various post-shock dysrhythmias, atrial extrasystoles occurred in 10 per cent of the cases, atrial tachycardia in 5.5 per cent, nodal rhythm in 5.5 per cent, ventricular extrasystoles in 14 per cent, and ventricular tachycardia in 6.5 per cent of the

cases. There was 1 transient episode of partial AV block, and 2 episodes which were considered to represent sino-atrial block. When the patients were divided into 2 groups, namely those who had digitalis as well, and those in whom digitalis was discontinued or not used at all before electroconversion, the incidence of post-shock dysrhythmias was higher in the former group. Serum potassium levels were between 3.5 and 4.4 mEq/l., with an average of 4.0 mEq/l. in both digitalis and non-digitalis groups.

Propranolol Ten patients received propranolol, 60 to 80 mg. daily, for 5 days before electroconversion (Table 2). Sinus rhythm was restored in 90 per cent of the patients. The average energy level necessary for con-

FIG. 1 (A) Lead II, atrial fibrillation. DC shock, 80 joules. Slow nodal rhythm immediately after shock. Normal sinus rhythm 8 seconds later. (B) Leads I, II, III, V₁, V₂, V₄, V₇. Inverted T waves and slight prolongation of QT interval 22 hours after DC shock. (C) Lead II, 2 hours after (B). Transient ventricular fibrillation causing syncope followed by runs of ventricular ectopic beats interrupted by sinus complexes. (D) Same leads as in (B), 3 weeks later. Inversion of T waves disappeared.

version was 190 joules. Of post-shock dysrhythmias, atrial extrasystoles occurred in 10 per cent, nodal rhythm in 10 per cent, and ventricular extrasystoles in 10 per cent of the patients. Serum potassium levels were between 3.6 and 4.2 mEq/l., with an average of 4.0 mEq/l.

In 2 patients who received 80 mg. propranolol daily, post-shock sinus bradycardia occurred, which was abolished by 1 mg. atropine in one, and in the other patient the heart rate increased spontaneously.

Direct current shock without concomitant drug therapy On 68 occasions in 63 patients DC shock was used without concomitant drug therapy (Table 2). Sinus rhythm was restored in 78 per cent of the cases by an average energy of 130 joules. The incidence of post-shock atrial extrasystoles

was 10 per cent, of atrial tachycardia 3 per cent, of nodal rhythm 1.5 per cent, and of ventricular extrasystoles 4.5 per cent. Serum potassium levels ranged from 3.8 to 4.4 mEq/l., with an average value of 4.1 mEq/l.

Experimental observations

First a synchronized DC shock of 40 to 60 joules was delivered in all 30 animals before the administration of drugs. The results of the combined use of DC shock and drugs were evaluated in all animals, including those in whom the initial DC shock produced electrocardiographic changes.

Quinidine Twenty-six experiments form the basis of these observations. After the initial shock of between 40 to 60 joules, quinidine was injected into the femoral vein as quinidine hydrochloride in doses

FIG. 2 Cat, weight 2.4 kg. Lead II. (1) Initial tracing. (2) After 4 mg./kg. of quinidine, note wider QRS complex. (3) and (4) Continuous tracing. DC shock, 60 joules. (5) One minute later, slower heart rate, further widening of QRS complex, transient lengthening of PR interval, with occasional AV block.

equivalent to 2 mg./kg. of quinidine sulphate at 5-minute intervals. When a quinidine effect appeared in the electrocardiogram, mostly a widening of the QRS complex, further DC shocks of the same energy were delivered, and also further injections of quinidine were given about 5 minutes before each shock.

In 14 out of 20 experiments the combined use of quinidine and DC shock resulted in progressive widening of the QRS complex and/or partial or complete heart block. In 6 of these 14 experiments, DC shock did not increase or alter in any way the quinidine-induced intraventricular or atrioventricular conduction disorder. In 8 experiments, however, the immediate post-shock electrocardiograms frequently showed further transient

or persisting widening of the QRS complex or increase in the AV block, as compared with the immediate pre-shock electrocardiograms. Illustrative tracings are shown in Fig. 2 and 3. In the other 6 experiments the administration of quinidine and DC shock resulted in ectopic impulse production: ventricular tachycardia on 4 occasions, ventricular fibrillation on 1 occasion, and ventricular extrasystoles on 1 occasion. In 3 of these 6 experiments the initial DC shock before the first quinidine injection resulted in conspicuous ST segment depression, T wave inversion, and ST segment elevation, respectively. An illustrative tracing is shown in Fig. 4. Of the 14 experiments in which the combined use of quinidine and DC shocks resulted in signs of cardiac depression,

FIG. 3 Cat, weight 1.5 kg. Lead II. (1) Initial tracing. (2) and (3) DC shock, 40 joules; no change. (4) After 4 mg./kg. of quinidine. Deep S waves and widening of the QRS complex. (5) After further 2 mg./kg. of quinidine, 2:1 AV block. (6) and (7) Continuous tracing. DC shock, 40 joules. Asystole followed by further increase in AV block.

significant ST segment or T wave changes induced by the initial DC shock were observed only on 2 occasions.

In 6 further experiments the administration of a single dose of digoxin 0.25 mg., either early and simultaneously with a quinidine injection or later at the stage of quinidine-induced depression of intraventricular conduction, followed by a DC shock, resulted in transient or persistent ectopic tachycardia on 4 occasions, ventricular fibrillation on 1 occasion, and no change in the quinidine-induced electrocardiographic pattern on 1 occasion. In 2 of this special group of 6 experiments, further DC shocks were given during the stage of ectopic tachycardia, but the ectopic rhythm was not abolished (Fig. 5).

Procainamide Four experiments were carried out in this group. Amounts of 10 to 20 mg./kg. procainamide per dose were injected intravenously and each injection was followed by a DC shock. The procedure was exactly the same as for quinidine. The combined use of procainamide and DC shock resulted in progressive intraventricular and AV conduction defect in all 4 experiments (Fig. 6). In 3 experiments, several immediate post-shock electrocardiograms showed potentiation of the procainamide-induced depression of atrioventricular and intraventricular conduction, and in 1 experiment DC shocks did not increase the procainamide-induced changes.

Discussion

Antidysrhythmic drugs have been used in conjunction with DC shock in an attempt to increase the conversion rate and to prevent early recurrence of the ectopic rhythm. Rossi and Lown (1967) advocated the use of quinidine before electroconversion, being of the opinion that patients receiving quinidine required fewer shocks and lower energy levels to restore sinus rhythm. They also stated that pretreatment with quinidine reduced the incidence of post-shock dysrhythmias and prevented prompt relapse of atrial fibrillation. Hall and Wood (1968) also found that premedication with quinidine reduced the frequency of post-conversion dysrhythmias.

In our whole series of 457 episodes of atrial fibrillation, sinus rhythm was restored by DC shock in 85 per cent of the cases. The conversion rate was 78 per cent in the group of patients not receiving any drugs at all. It was higher in those receiving concomitantly various antidysrhythmic drugs, with a sig-

nificant increase (92%) in the combined procainamide and propranolol group ($p < 0.01$). This latter group and the group of patients not treated concomitantly with drugs were comparable regarding the underlying pathology, especially lone atrial fibrillation which accounted for 13 per cent of all episodes treated in both groups. This is relevant, for, in agreement with the observations of Oram and Davies (1964) and of Resnekov and McDonald (1968), the con-

FIG. 4 *Cat, weight 1.5 kg. Lead II.*
(1) Initial tracing. (2) DC shock, 60 joules. Inverted T waves. (3) After a total of 6 mg./kg. of quinidine. S waves with wider QRS complex. (4) and (5) Continuous tracing. DC shock, 60 joules. Further widening of the QRS complex with transition into ectopic tachycardia, probably ventricular. (6) and (7) Continuous tracing. DC shock, 60 joules. No immediate change. Tachycardia stops about 8 seconds later. (8) 1 minute later, slow ventricular rate with wide QRS complexes. No atrial activity seen.

version rate was in our experience significantly lower in lone atrial fibrillation than when atrial fibrillation had a detectable underlying cause, 60 per cent and 87.5 per cent, respectively ($p < 0.001$). Addition of propranolol to quinidine also resulted in a very much higher than average conversion rate, but the numbers in this group were too small to establish a statistically significant advantage. However, this small group contained no patients with lone atrial fibrillation, and this undoubtedly contributed to the high success rate. When quinidine alone was used as pretreatment, the conversion rate was higher than when procainamide alone was given, 88 per cent and 82 per cent, respectively, but this difference is statistically not significant.

Antidysrhythmic drugs did not reduce the energy requirements necessary for the restoration of sinus rhythm.

In the rheumatic group, which is far the largest, representing 75 per cent of all patients and 78 per cent of all episodes included, the conversion rate was 87 per cent in the combined pre-surgical and non-surgical group, and 89 per cent in the post-surgical group, a difference that is statistically not significant.

We reported previously that the duration of atrial fibrillation was an important factor in influencing the conversion rate, being 98 per cent in those with less than 1 year duration and 82 per cent in those with more than 1 year duration (Szekeley *et al.*, 1966). This trend has remained unchanged. In the present series, the patients in the various groups were comparable with regard to the duration of atrial fibrillation.

In our experience, the concomitant use of antidysrhythmic drugs did not result on the whole in a reduction of post-shock dysrhythmias. Having regard to the importance of digitalis in causing post-shock dysrhythmias (Szekeley *et al.*, 1969), we compared the incidence of post-shock dysrhythmias in patients who received quinidine or procainamide and/or propranolol but no digitalis with that in patients who received no drugs at all, and found that post-shock dysrhythmias were even more frequent in the drug-treated groups of patients. However, when digitalis was continued, the combined use of procainamide and propranolol resulted in a significant decrease in the incidence of post-shock atrial tachycardia and ventricular extrasystoles, as compared with the use of procainamide or quinidine alone ($p < 0.01$). This was especially the case when larger doses of propranolol, 80 mg. daily, were used.

This is of interest in the light of the recent observations of Wittenberg and Lown (1969) who found in animal experiments that pro-

FIG. 5 Cat, weight 2 kg. Lead II. (1) Initial tracing. (2) and (3) Continuous tracing. DC shock, 40 joules. No significant change. (4) After 4 mg./kg. of quinidine and 0.25 mg. of digoxin. Change in ventricular complex. (5) to (7) Continuous tracing. DC shock, 40 joules. Ectopic beats followed by ectopic tachycardia, probably ventricular. (8) Further DC shock, 40 joules. Ectopic tachycardia persists.

FIG. 6 Cat, weight 2.7 kg. Lead II. (1) Initial tracing. DC shock 60 joules. Slight ST segment and T wave changes. (2) After procainamide, 20 mg./kg. Wide QRS complex. (3) and (4) Continuous tracing. DC shock, 60 joules. Further widening of the QRS complex and slowing of ventricular rate.

pranolol, when given in larger than beta-blocking doses, abolished both digitalis-induced dysrhythmias and digitalis-induced sensitization to electrical shock.

Our experimental findings also suggest that quinidine has no protective action against digitalis and DC shock-induced ectopic tachycardia; in fact, quinidine even tended to reduce digitalis tolerance. This observation is in agreement with that previously reported by Rodensky, Kathe, and Wasserman (1960).

At the time of DC shock treatment, no patient had significant hypopotassaemia. Hypopotassaemia is an important factor in the development of post-shock dysrhythmias, as pointed out before by Lown and Wittenberg (1968) and Thind, Blakemore, and Zinsser (1969), but in our experience digitalis caused post-shock dysrhythmias even when the serum potassium levels were normal. DC shock *per se* can cause myocardial potassium loss, as has been shown recently in animal experiments (Regan *et al.*, 1969).

It seems from our clinical and experimental observations that DC shock can increase the cardiotoxicity not only of digitalis, as previously shown (Szekely *et al.*, 1969), but also of quinidine. It is possible that such a potentiation is more likely to occur when acute post-shock myocardial injury ensues. Castellanos *et al.* (1965) pointed out earlier that DC shock enhanced the toxic effect of quinidine in those instances in which there was quinidine cardiosensitivity. Published work

contains a number of well-documented cases of ventricular fibrillation or ventricular standstill in patients treated with quinidine alone or with the combination of quinidine and DC shock (Selzer and Wray, 1964; Oram and Davies, 1964; Davies, Leak, and Oram, 1965; Bjerkelund and Orning, 1968).

The cardiotoxicity of procainamide is perhaps less severe than that of quinidine. In our experience, it caused no serious side-effects. However, Castellanos and Salhanick (1967) observed conspicuous sino-atrial node depression following the combined use of procainamide and DC shock.

Propranolol, when given in larger doses (60 to 80 mg. daily), alone or added to quinidine or procainamide, resulted in post-shock depression of the sino-atrial node in a few cases. Lown (1968) previously reported sino-atrial standstill and cardiac arrest after the combined use of propranolol and DC shock.

Though currently used antidysrhythmic drugs, given singly or in combination in conjunction with DC shock, can increase the conversion rate of atrial fibrillation, they should be used with great caution, if at all, as their cardiotoxicity may be enhanced by DC shock. Lown (1967) has also stated recently that the major hazard of DC shock treatment is related to the use of digitalis being partly responsible for the immediate post-shock dysrhythmias, and of quinidine causing delayed disorders of the cardiac

rhythm. Furthermore, large doses of anti-dysrhythmic drugs can depress pacemaker activity, an effect which becomes unmasked after abolition of the ectopic tachycardia by DC shock as pointed out before by Wagner and McIntosh (1969). The long-term use of antidysrhythmic drugs in maintaining sinus rhythm after atrial defibrillation is now being evaluated and will be the subject of a separate communication.

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