

Direct Current Shock and Digitalis

A Clinical and Experimental Study

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The application of direct current (DC) shock is now an established method of treatment of ectopic tachycardias. However, experience has shown that the concomitant use of certain cardiac drugs can lead to undesirable complications and create new problems in the management of the patient. Several studies have already referred to the use of digitalis (Gilbert and Cuddy, 1965; Lown, Kleiger, and Williams, 1965; Kleiger and Lown, 1966; Szekely, Wynne, and Batson, 1966b; Lown, 1967; Castellanos *et al.*, 1967) in association with DC shock therapy. It is the purpose of this paper to evaluate clinical and experimental observations on the effects of the combined use of digitalis and DC shock. Clinical and experimental observations on the concomitant use of various antiarrhythmic drugs and DC shock will be the subject of a separate communication.

METHOD, CLINICAL GROUPS, AND EXPERIMENTAL MATERIAL

(A) *Clinical.* Five hundred consecutive episodes of ectopic tachycardia treated by DC shock have been reviewed. Of these, 414 episodes of atrial fibrillation occurring in 289 patients form the basis of the present observations. Of the 289, 218 patients had rheumatic heart disease, 30 coronary heart disease with or without hypertension, 4 congenital heart disease, 9 thyrotoxicosis, 1 had chronic pulmonary heart disease, and there were 27 patients with no firm clinical diagnosis, several of whom were suspected to have cardiomyopathy. In 369 episodes of atrial fibrillation the following drugs were used singly or in combination at the time of DC shock therapy: digitalis, quinidine, procainamide, and propranolol. In the remaining 45 episodes no drugs were used at all. The method of DC shock therapy and the results of the first 200 episodes have already been reported in a previous paper (Szekely, Batson, and Stark, 1966a). Only the method of anaesthesia has since been modified. After abandoning pentothal, methohexitone

80–100 mg. had been used and, more recently, propanidid 300–500 mg. Anaesthesia has been maintained when necessary by nitrous oxide.

(B) *Experimental.* The results of 53 experiments carried out in cats weighing between 1.4 and 3.5 kg. have been evaluated. 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 electrocardiogram was recorded throughout the experiment. Digoxin was given by injection into the femoral vein. Synchronized DC shocks to fall on the R wave were delivered at an energy level setting of 40 to 60 joules. Experimental data obtained in our laboratory and serving as controls regarding the effects of digoxin alone and of DC shocks alone were available from previous and current studies.

CLINICAL OBSERVATION

The 414 episodes of atrial fibrillation were divided into three groups according to the dose of digoxin the patients received at the time of DC shock therapy, and irrespective of any other drugs used (Table). Group I, comprising 103 episodes, continued on an adequate maintenance dose of digoxin until the day of elective electroconversion (usually 0.25 mg. or 0.5 mg. daily). In Group II, comprising 94 episodes, the maintenance dose of digoxin was reduced by 50 per cent during 3–5 days before shock treatment. In Group III, with 217 episodes, digoxin was discontinued 3–5 days before electrical treatment. Also included in Group III are patients in whom digitalis was not used at all. The immediate results of DC shock therapy and the incidence of post-shock arrhythmias in these three groups are shown in the Table. Sinus rhythm was restored in 86 per cent of all the episodes, with only slight variation in the three groups. The over-all incidence of atrial extrasystoles was 11 per cent, and of short runs of atrial tachycardia or persisting atrial tachycardia necessitating a further shock 7 per cent. Nodal rhythm lasting from a few seconds to several hours was observed in 6 per cent

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TABLE
DIGITALIS AND DIRECT CURRENT SHOCK IN ATRIAL FIBRILLATION

Groups of patients	No. of episodes treated	Sinus rhythm restored		Nature and incidence of post-shock arrhythmias				
		No.	Percentage	Atrial extra-syst.	Atrial tachycardia	Nodal rhythm	Ventric. extra-syst.	Ventric. tachycardia
Group I: Maintenance dose of digoxin continued	103	91	88	12 (12%)	18 (18%)	14 (14%)	35 (35%)	11 (11%)
Group II: Maintenance dose of digoxin halved	94	82	87	10 (11%)	5 (5.5%)	5 (5.5%)	19 (20%)	5 (5.5%)
Group III: Maintenance dose of digoxin discontinued or digitalis not used at all	217	181	84	24 (11%)	6 (3%)	6 (3%)	21 (10%)	6 (3%)
Total	414	354	86	46 (11%)	29 (7%)	25 (6%)	75 (18%)	22 (5%)

of the cases. The over-all incidence of ventricular extrasystoles was 18 per cent, and in about a quarter of them a bigeminal rhythm was observed, lasting from a few seconds to 30 minutes. Short runs of ventricular tachycardia or aberrant conduction simulating ventricular tachycardia appeared in 5 per cent of the episodes. When the incidence of post-shock arrhythmias was analysed separately in the three groups it was noted that atrial extrasystoles occurred equally frequently in all three groups. However, atrial tachycardia, nodal rhythm, ventricular extrasystoles, and ventricular tachycardia were observed after DC shock about half as frequently in Group II as in Group I, and again half as frequently in Group III as in Group II.

An example of the potentiation of digitalis cardiotoxicity by DC shock is shown in Fig. 1. This patient received a full maintenance dose of digoxin until the day of electroconversion.

When the cases in the three groups were subdivided according to the concomitant use of quinidine or procainamide or propranolol, it was found that the incidence of post-shock arrhythmias was not significantly influenced by these drugs, and depended mainly on whether or not digitalis was still given at the time of DC shock (Szekely *et al.*, 1968).

EXPERIMENTAL OBSERVATIONS

First a synchronized DC shock of 40 to 60 joules was delivered. If an elevation of the S-T segment or T wave changes occurred, the animal was excluded from this group. A few animals in which ectopic beats appeared after the initial shock were also excluded. Thus 30 animals were available for the study of the combined effects of digitalis and DC shock. Between 0.125 mg. and 0.5 mg. of digoxin was injected intravenously and 5 minutes later a DC shock of the same order was delivered. If no multifocal extrasystoles or ectopic tachycardia appeared, the procedure, namely the administration of a similar dose of digoxin followed by a DC shock, was repeated until the appearance of such an ectopic rhythm. In this way an ectopic mechanism consisting of supra-ventricular tachycardia on 4 occasions, ventricular tachy-

cardia on 18 occasions, ventricular fibrillation on 1 occasion, and of multifocal ventricular extrasystoles with or without bigeminy on 7 occasions was produced after not more than three electric shocks (in addition to the first pre-digitalis shock), and after a total dose of digoxin which never exceeded 0.25 mg./kg. This amount of digoxin represents 50 per cent of the average dose of digoxin that was necessary to produce a similar ectopic mechanism when given alone without DC shocks, as ascertained in previous experiments in over 50 cats in our laboratory. When repeated DC shocks of the same order were given alone at 5-minute intervals up to 10 shocks as tested in 15 other animals, ectopic impulse production was observed in only 6 of them: occasional atrial and/or ventricular extrasystoles appeared in some after the first or second shock, but multifocal extrasystoles or an ectopic tachycardia were not seen before the sixth shock.

In 17 of the ectopic tachycardias induced by digitalis and DC shock, further shocks were given without further digitalis: ventricular fibrillation occurred after one further shock on 8 occasions, asystole on 1 occasion, and no further change was noted after several shocks on 8 occasions. In no case was sinus rhythm restored by DC shock. On the other hand, propranolol was often effective in this situation. Illustrative tracings are shown in Fig. 2 and 3.

In 8 additional experiments in which ventricular tachycardia was produced by digitalis alone, a DC shock produced ventricular fibrillation on 6 occasions, asystole on 1 occasion, and no change on 1 occasion. In 3 of the 6 episodes of ventricular fibrillation thus produced, further electric shocks were given but sinus rhythm could not be restored: in 2 ventricular fibrillation persisted and in 1 asystole was produced.

DISCUSSION

The present observations show that contrary to the experience of Stern (1965) the conversion of atrial fibrillation to sinus rhythm by DC shock is not rendered less effective by adequate digitalization. However, digitalis was found to be the most

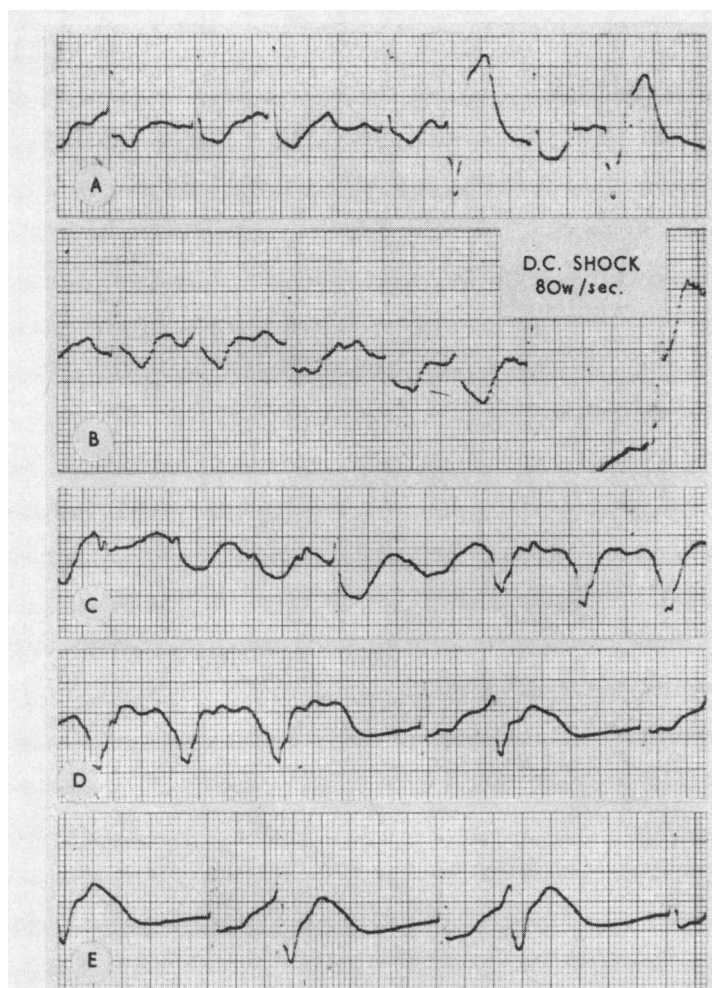


FIG. 1.—(A) Atrial fibrillation with intermittent extrasystolic bigeminy. (B) DC shock, 80 joules. (C) to (E) Continuous tracing after DC shock. Short episode of ventricular tachycardia followed by persistent bigeminal rhythm for 30 minutes. Atrial fibrillation persists.

important causative factor in the production of post-conversion arrhythmias. When digitalis was omitted 3 to 5 days before electroconversion, the incidence of post-shock arrhythmias was greatly reduced. These observations are in agreement with previous studies (Gilbert and Cuddy, 1965; Castellanos, Lemberg, and Fonseca, 1965; Kleiger and Lown, 1966; Szekely *et al.*, 1966b; Lown, 1967). Ventricular fibrillation has been reported in digitalized patients after DC shock therapy (Rabbino, Likoff, and Dreifus, 1964; Robinson and Wagner, 1965). Gilbert and Cuddy (1965) pointed out that it was hazardous to convert electrically a patient from atrial fibrillation or flutter to sinus rhythm when manifestations of digitalis intoxication are

already present. To determine the role of digitalis, Kleiger and Lown (1966) analysed 100 consecutive patients with atrial fibrillation who were subjected to electroconversion. Of the 18 patients who showed a ventricular ectopic mechanism after electric shock, 17 manifested abnormalities in the pre-conversion electrocardiogram suggestive of digitalis intoxication, and only 41 did so of the 82 patients who were free from ventricular arrhythmias after atrial defibrillation. Lown (1967) observed that digitalized patients were especially likely to develop post-shock arrhythmias, when the serum potassium was low before electroconversion. He expressed the view that the electrical discharge caused injury of the cell membrane and thus led to loss of potas-

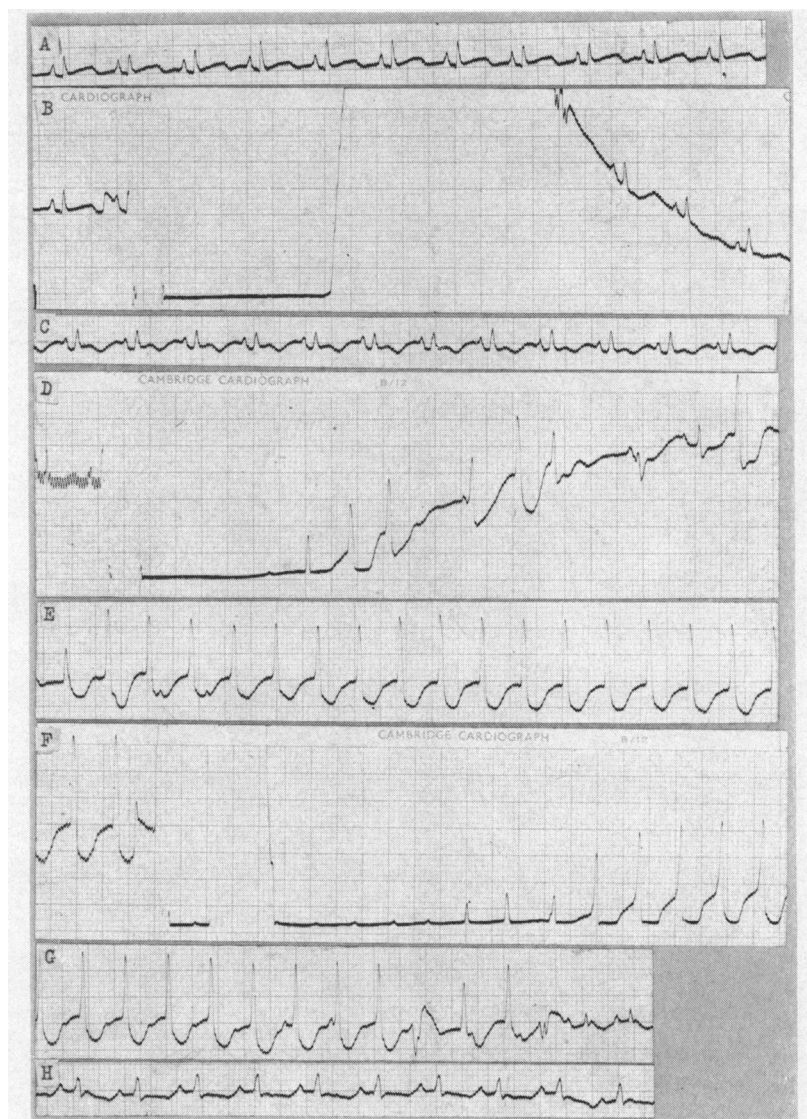


FIG. 2.—Cat, weight 2 kg. (A) Initial tracing. (B) DC shock, 40 joules. No change. (C) After 0.125 mg./kg. of digoxin. Transient T wave inversion. (D) and (E) After DC shock, 40 joules. Ectopic beats followed by ectopic tachycardia. (F) Ectopic tachycardia not abolished by further DC shock. (G) and (H) Sinus rhythm restored by propranolol.

sium from the cell and in this way it potentiated the toxic action of digitalis. Oram (1967) also stated that there was good evidence that DC shock therapy potentiates the toxic effect of digitalis possibly due to transient cellular damage, and that digitalis intoxication might become manifest in sinus rhythm when not evident in atrial fibrillation.

Lown *et al.* (1965) have previously reported in dogs that after digitalization the electrical threshold for ventricular tachycardia was reduced. In our

experience, the combined use of digitalis and DC shock in cats resulted in multifocal ventricular extrasystoles and/or ectopic tachycardias after a digitalis dose which never exceeded 50 per cent of the average dose of digitalis that was necessary to produce an ectopic rhythm when given alone without an electric shock. Our experimental observations also showed that DC shock was ineffective in digitalis-induced ectopic tachycardias, and also potentially dangerous because it often resulted in

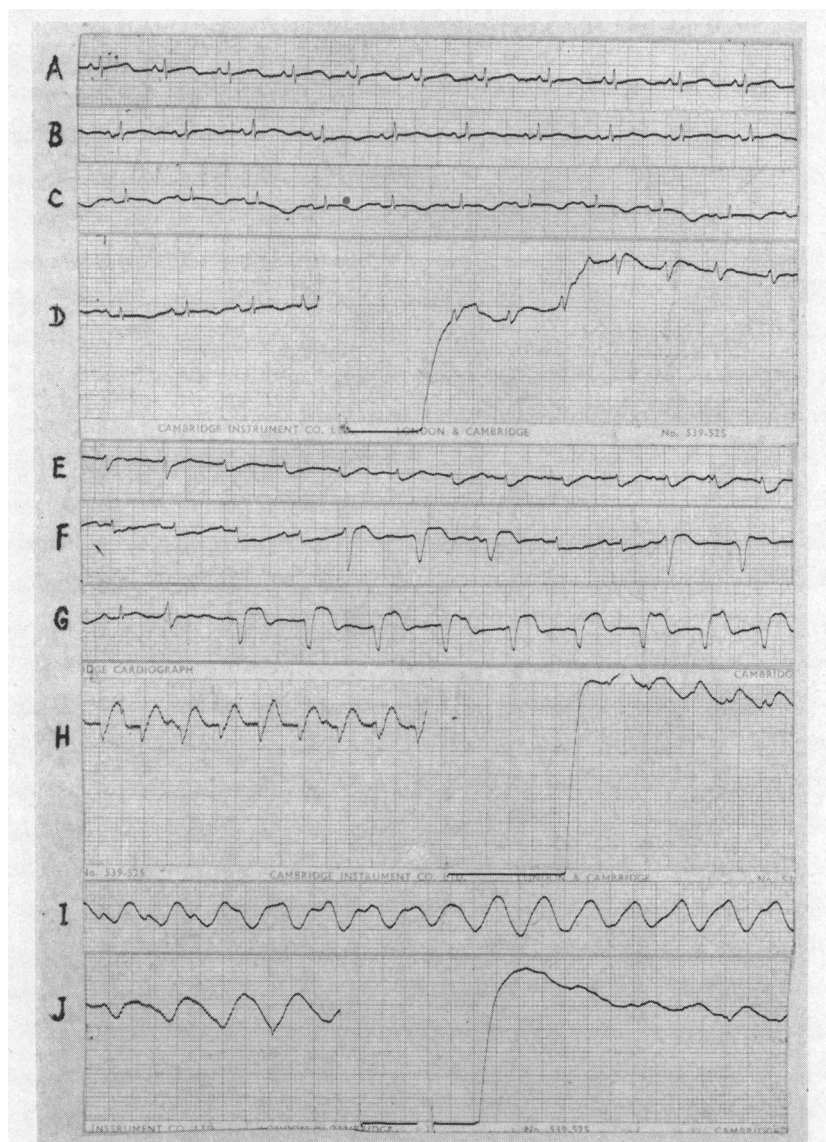


FIG. 3.—Cat, weight 2.5 kg. (A) Initial tracing. (B) and (C) After 0.1 mg./kg. of digoxin. Inverted T waves. (D) to (G) Ectopic tachycardia after DC shock, 40 joules. (H) and (I) Ventricular fibrillation after a further DC shock. (J) Ventricular fibrillation persists after further DC shock.

ventricular fibrillation. These observations are in agreement with those of Lown *et al.* (1965) and of Katz and Zitnik (1966) who also concluded on the basis of experimental studies that DC shock was not only less effective but also hazardous in digitalis-induced ectopic tachycardias and therefore contra-indicated in their treatment.

SUMMARY

The combined effects of digitalis and DC shock

were studied in man and in the experimental animal.

The conversion rate of atrial fibrillation to sinus rhythm by DC shock was not influenced by the concomitant use of digitalis. However, digitalis was the most important factor in the causation of post-conversion arrhythmias.

Experimental data are presented which show that DC shock potentiates digitalis cardiotoxicity, and that DC shock is in digitalis-induced ectopic tachycardias not only ineffective but also potentially

dangerous because it can lead to ventricular fibrillation.

It is advisable to omit digitalis a few days before attempted electroconversion. Digitalis-induced ectopic tachycardia should not be treated by DC shock.

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