SYNCHRONISED CAPACITOR DISCHARGE IN THE MANAGEMENT OF CARDIAC ARRHYTHMIAS WITH PARTICULAR REFERENCE TO THE HAEMODYNAMIC SIGNIFICANCE OF ATRIAL SYSTOLE

Thesis submitted for the degree of

DOCTOR OF MEDICINE

by

Leon Reznekov
M.B.Ch.B. (Cape Town), M.R.C.P. (London)

University of Cape Town August 1965

CONTENTS

CONTENTS (continued)

I N T R O D U C T I O N

In December 1962 a boy of 15 years was referred to the National Heart Hospital because of a ventricular tachycardia (Fig.1), which had proved resistant to treatment

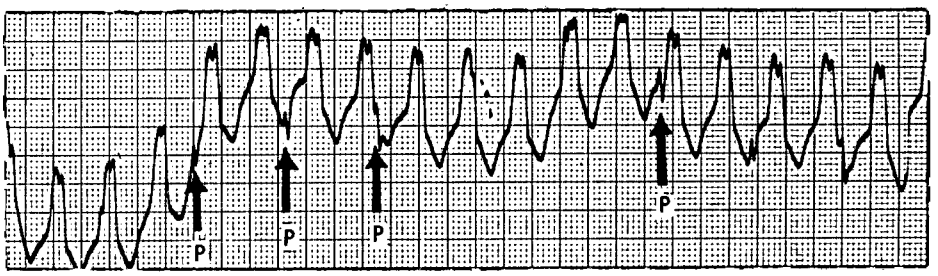


FIG. 1: ECG Oesophageal lead, ventricular tachycardia.

with procaine amide, quinidine, atropine, prostigmine, prednisone and digoxin. A second stage, total correction for tetralogy of Fallot had been successfully performed two years before by Sir Russell Brock and the tachycardia followed a sudden blow on the chest at his school. Further drug treatment to the point of toxicity proved equally unavailing and ten days after the onset of the arrhythmia severe congestive heart failure was present. Sinus rhythm was established, (Fig.2), by a single alternating current discharge of 400 volts to the chest administered under a general anaesthetic (McDonald, Resnekov, & Ross 1963), but

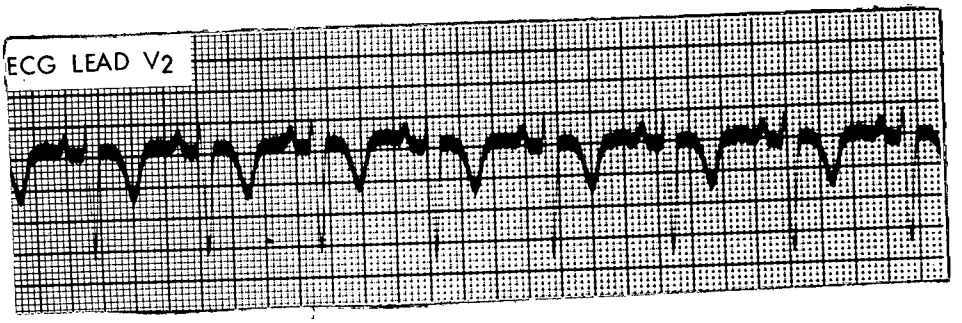


FIG. 2: ECG Lead V_2, Sinus rhythm has returned following an unsynchronised AC shock.

the experience of Zoll and Linenthal (1962) had already demonstrated the hazards of alternating current in the treatment of ectopic rhythms. Apparatus for synchronised capacitor discharge to be used in the treatment of arrhythmias described by Lown, Amarasingham and Neuman (1962) became available in this country in 1963 and preliminary reports were encouraging.

The aim of this work is to evaluate the method of synchronised capacitor discharge in the management of 150 patients with supraventricular and ventricular arrhythmias and to re-appraise the physiological changes at rest and on effort in a representative group of 20 patients studied before and after the conversion to sinus rhythm of atrial fibrillation and flutter.

Chapter 1

HISTORICAL REVIEW

Electrical depolarisation of the heart was first used successfully in man by Beck, Pritchard and Feil (1947) who were able to abolish a long standing ventricular fibrillation by an alternating current shock applied to the surface of the heart. Many investigators had studied the effects on the heart of the passage of an electrical current since Galvani (1737-1798) had demonstrated that the effect of bringing frogs legs into contact with two dissimilar metals was to cause them to jump. Shortly thereafter Gordon (1745) exposed birds, beetles and other small animals to frictional electricity and observed the effect this produced. Early experiments on man included the work of Aldini (1819) who studied the effects of placing electrodes on the hearts of decapitated criminals; Vassalli conducted almost identical experiments at about the same time in Turin.

It was soon realised that the passage of an electrical current through the human body caused an unpleasant sensation when in 1746 two Dutch physicians discharged a Leyden jar

through their bodies (Kouwenhoven 1964). The first fatal electrical accident reported occurred in France in 1879 and this undoubtedly helped to intensify investigation into the effects of the passage of an electrical current on the human body. Hoffa (1850) described the arrhythmia now called ventricular fibrillation.

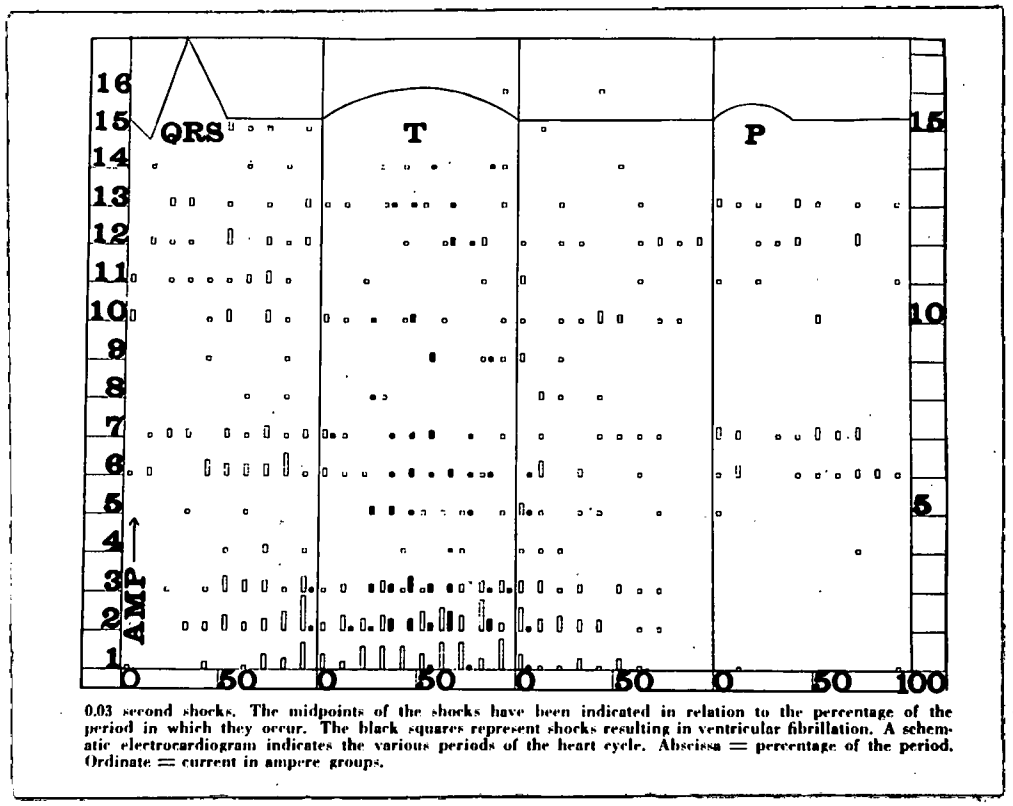
A major step forward was the demonstration by Prevost and Battelli (1899, 1900) that alternating or direct current applied to the surface of the heart could be used to terminate ventricular fibrillation in dogs. It is also of interest that these workers described the use of cardiac massage to keep alive dogs whose hearts were in ventricular fibrillation.

Electrical current was therefore shown to be capable of causing ventricular fibrillation under certain circumstances but also to be capable of restoring sinus rhythm once ventricular fibrillation had been produced. This paradox was obviously worthy of further study. de Boer (1921) demonstrated that in an exsanguinated frog heart ventricular fibrillation could be produced from a single induction shock timed to occur at the end of systole. Duchosal (1931) demonstrated that electrical shocks delivered 0.075 seconds after the R wave appeared particularly

prone to cause ventricular fibrillation. Meanwhile, Langworthy and Kouwenhoven (1930) had demonstrated that direct current shocks with capacitors charged up to 500 volts did not result in lumbar cord haemorrhage and paralysis in rats, unlike alternating current shocks of similar voltage.

As long as the heart was kept well oxygenated Hooker, Kouwenhoven and Langworthy (1933) were able to show that defibrillation of the ventricles could be achieved by the passage of 1 ampère at 130 volts for 0.1 second across the heart of dogs.

The use of capacitor discharge as opposed to alternating current brought into focus the problem of phasing the shock with an appropriate part of the cardiac cycle. King (1934) had demonstrated mainly using the heart of sheep that the greatest chance of ventricular fibrillation occurred with a capacitor discharge of 0.03 or 0.1 second duration timed to occur between 20% and 60% of the onset of the total deflection caused by the T wave (Fig.3) and that at corresponding voltages direct current was less likely to cause ventricular fibrillation than alternating current.



0.03 second shocks. The midpoints of the shocks have been indicated in relation to the percentage of the period in which they occur. The black squares represent shocks resulting in ventricular fibrillation. A schematic electrocardiogram indicates the various periods of the heart cycle. Abscissa = percentage of the period. Ordinate = current in ampere groups.

FIG. 3: The effect on the heart of DC shocks of 0.03 second duration. Black squares represent shocks resulting in ventricular fibrillation.

From: Effect of Electric Shock on Heart Action, B.G. King (1934). (Courtesy of Dr. B.G. King and of the Columbia University, New York).

Nevertheless, Milnor, Knickerbocker and Kouwenhoven as late as 1958 concluded using a capacitor of 25 and 50 microfarads that no vulnerable phase for ventricular fibrillation could be delineated in the cardiac cycle.

Furthermore, ventricular fibrillation could only be produced using voltages above 2,500; these workers were of the opinion that alternating current was safer and more effective than direct current in depolarising the heart in ventricular fibrillation.

Thus far most of the work undertaken was aimed at understanding the effects of the passage of electrical current across biological tissues and of treating the acute arrhythmia, ventricular fibrillation. The twin hazards of electrical current as regards the heart, namely, ventricular fibrillation and cardiac standstill were overcome by the demonstration by Zoll, Linenthal, Gibson, Paul, and Norman (1956) that an alternating current discharge across the closed chest was effective in dealing with ventricular fibrillation on the one hand and that external electrical pacemaking could be undertaken should the second hazard occur, (Zoll, 1952).

The early work of King (1934) and of Ferris, King, Spence and Williams (1936) in delineating a vulnerable phase in the cardiac cycle for ventricular fibrillation in the heart of sheep was confirmed in dogs by Wiggers and Wegria (1940) who demonstrated this to be 27 milliseconds before the end of systole, and by Lown, Kaid Bey, Perlroth

and Abe (1963b) who concluded that this was a property of mammalian heart and therefore would occur in human hearts as well. A period of vulnerability for the atrium was demonstrated by Andrus, Carter and Wheeler (1930) who showed that atrial fibrillation could be produced following a stimulus on the R or S wave of the electro-cardiogram.

Whilst American workers favoured alternating current for defibrillation, Gurvich and Yuniev (1947) and Gurvich (1952) in the Soviet Union as well as Peleška (1957, 1958, 1959) in Czechoslovakia favoured capacitor discharge. It has been known for some time (Kouwenhoven 1964) that an unmodified capacitor discharge is likely to cause ventricular arrhythmias. By the inclusion of an inductance coil in the discharge circuit, however, peak voltage and current are lessened and the duration of discharge lengthened, and Lown, Neuman, Amarasingham and Berkovits (1962) were able to demonstrate that using this type of circuit direct current was more effective than alternating current in the termination of ventricular fibrillation. Furthermore, using the same circuit with the shock phased to occur at a non-vulnerable part of the cardiac cycle direct current depolarisation of the heart in atrial and ventricular arrhythmias would allow the sinus pacemaker to take over;

in other words a safe new method for terminating cardiac arrhythmias was established (Lown, Amarasingham and Neuman, 1962).

Chapter 2

METHODS

This work analyses the use of synchronised capacitor discharge in the planned attempted version to sinus rhythm of 150 patients who had a supraventricular or ventricular arrhythmia. In addition haemodynamic studies were undertaken in 20 patients before and after the establishment of sinus rhythm to determine the haemodynamic significance of atrial systole. The patients were assessed before and after the electrical treatment as follows:-

Clinical Assessment.

All patients were examined personally. A relevant history of disability was obtained and a careful enquiry was made into the duration of the arrhythmia. Specific information was obtained about attempts at drug therapy for the arrhythmia in the past and a note made of the present drug treatment. As almost one half of the patients with atrial fibrillation had had cardiac surgery for rheumatic valvular heart disease, the nature of the operation performed was noted and symptomatic improvement assessed.

A full clinical examination was made in each case. The nature of the arrhythmia was determined clinically if possible. The cardiovascular system was examined to determine the nature and severity of any underlying heart disease and the presence or absence of congestive cardiac failure or failure of the left ventricle. A clinical assessement of the degree of improvement following cardiac surgery was made.

On the day following treatment each patient was examined once more. Apart from rhythm, attention was paid to:

The presence or absence of breathlessness.

The level of the blood pressure.

The height of the jugular venous pressure at 45° above the sternal angle.

The cardiac impulse.

The heart sounds with special reference to the presence or absence of a third heart sound.

Electrocardiogram:

Every patient had a standard 12-lead electrocardiogram recorded to assess the degree of cardiac disability and to determine the nature of the arrhythmia. Where the

arrhythmia was still in doubt the effect of carotid sinus compression was noted and additional leads, especially lead CM_1 or lead V_2, were recorded to determine atrial activity.

The differentiation between ventricular tachycardia and supraventricular tachycardia with bundle branch block, proved difficult at times despite the procedure outlined above. In this circumstance an oesophageal lead ($V_{1.4}$)

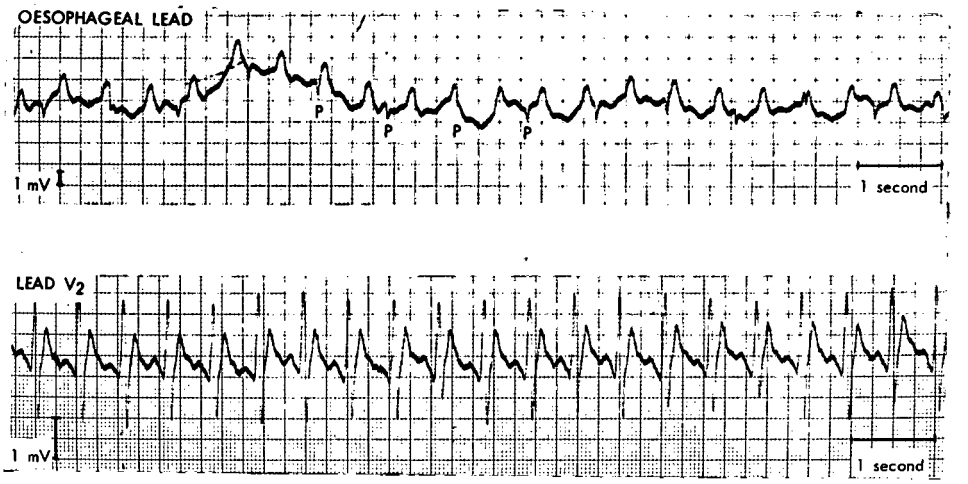


Fig. 4: Ventricular tachycardia.
Oesophageal lead (atrial activity) and lead V_2.
Atrial and ventricular dissociation clearly demonstrated in the oesophageal lead

was recorded at 30 cm., 25 cm. and 20 cm. from the lips. This always demonstrated atrial dissociation from ventricular activity where ventricular tachycardia was the cause.

The electrocardiogram was also analysed for the presence or absence of digitalis segment and T wave change, for ventricular hypertrophy, and for the changes of ischaemic heart disease. The following criteria were adopted (Goldman, 1962):-

Digitalis effect:-

ST segment depression in ventricular epicardial leads, either rounded, concave or as an oblique line descending from the J-point. A low take-off of the T wave.

Shortening of the QT interval.

Some or all of these changes were frequently present in all leads.

Left Ventricular Hypertrophy:-

(i) Praecordial leads -
Ventricular activation time over 0.05 sec. in V_{5-6}. QRS interval prolonged over 0.1 sec. in V_{5-6}. ST segment and T wave inversion V_{5-6}.

(ii) Extremity leads -
Lead aVL - Horizontal heart, changes as for leads V_{5-6}.
Lead aVF - Vertical heart, changes as for leads V_{5-6} (but only when confirmed by the praecordial leads).

(iii) Standard leads -
As for leads aVL and aVF.

Right Ventricular Hypertrophy -

(i) Praecordial leads -
QR - in leads V_1, or V_3R.
Ventricular activation time over 0.03 seconds in leads V_1, or V_3R.
Persistent S wave in leads V_{5-6}.
ST segment depression and T wave inversion in leads V_1, or V_3R.

(ii) Extremity leads -
Lead aVR:- tall R when accompanied by the changes already noted in the praecordial leads.
Lead aVF:- tall R with depressed ST segment and inverted T wave, when accompanied by changes already noted in the praecordial leads.

(iii) Standard leads -
Right axis deviation ($+110^{o}$ or more) depressed ST segment and inverted T wave in leads 2 and 3.

NOTE:- Left ventricular hypertrophy and right ventricular hypertrophy were not diagnosed on the relative size of R and S waves alone.

Ischaemic Heart Disease -

a) Without myocardial infarction
(i) Praecordial leads - ST segment depression with or without T wave inversion in the left praecordial leads V_{4-6}.
(ii) Extremity leads - Leads aVL or aVF - changes as for (i) depending upon the direction of the frontal plane vector. In addition, reciprocal ST segment elevation present in lead aVR.
(iii) Standard leads - as for (ii). When present in lead aVL changes were seen in lead 1; if in lead aVF, they were seen in leads 2 and 3.

b) With myocardial infarction
ST segment (elevation or depression) and T wave changes in association with abnormal Q waves.

NOTE:- The changes on the electrocardiogram were always analysed in association with the clinical history and findings especially when a diagnosis of ischaemic heart disease was considered.

A 12-lead electrocardiogram was repeated following the electrical treatment. In patients now in sinus rhythm particular attention was paid to: Rate, morphology of P waves, the P-R interval and the presence or absence of a wandering pacemaker. All electrocardiograms were examined for atrial, nodal or ventricular ectopic beats and for the morphology of the QRS complex and T waves.

Chest Radiographs:

Each patient had a set of three chest radiographs before and after synchronised capacitor discharge - a 6-foot postero-anterior view, a laterial view (R) and a penetrated antero-posterior grid view at 40 inches to indicate the size of the left atrium. The chest radiographs were repeated one day following the electrical conversion and in certain patients two days thereafter where there had been a significant alteration in heart size shown on the chest X-ray taken after the shock.

The overall size of the heart was estimated by the cardiothoracic ratio (Danzer, 1919). The maximum transverse diameter of the heart and width of the thorax were measured. The percentage ratio of the transverse diameter of the heart to the width of the chest was calculated. Although

enlargement of the heart is frequently the cause when this ratio exceeds 50% this interpretation will be fallacious if the diaphragm is high or low or if the thoracic cage is abnormally wide and shallow or narrow and long (Comeau and White, 1942). Furthermore the size of the heart as determined by the radiographs depends on the part of the cardiac cycle during which the exposure was made.

Despite these limitations, the cardiothoracic ratio did provide a method of comparison of overall cardiac size and specifically showed whether a significant change in the size of the heart occurred following the treatment with synchronised capacitor discharge.

An attempt was made to grade enlargement of the individual heart chambers from these X-rays. Great difficulty was experienced, however, and ultimately it was concluded that delineation of the borders of the heart chambers was not feasible by this means. Protrusion of the right border of the heart to the right of the sternum as seen in the postero-anterior view suggested enlargement of the right atrium. The presence of abnormal curvature of the spine or abnormality of the sternum however made any accurate assessment difficult, as did shift of the mediastirum from any cause. In the lateral plane

superimposition of the shadow of the right atrium and of the ventricles once more made an accurate assessment of size difficult.

Enlargement of the right ventricle is best recognised by a conventional radiograph in the lateral view as an abnormal shadow in the anterior mediastinum; it was found, however, that it was not possible to grade enlargement of this chamber by this means.

Similarly the posteriorly displaced enlargement of the left ventricle, best appreciated in the lateral view was impossible to grade on the one hand but was also frequently difficult to appreciate owing to superimposition of the shadow of the right and left atria.

It was felt, however, that in the case of the left atrium an attempt could be made to grade the size into, normal (N), slight enlargement (1+), moderate enlargement (2+), severe enlargement (3+). Particular attention was paid to the angle of the left main bronchus which progressively becomes less acute as the left atrium enlarges, and to the shadow of the right border of the left atrium seen within the shadow of the heart. The penetrated grid film in the antero-posterior view was particularly helpful in this

connection. As the left atrium enlarges its right border draws closer to the right border of the right atrium and when very large extends to the right beyond its outer border. In Fig. 5 the method of grading of the left

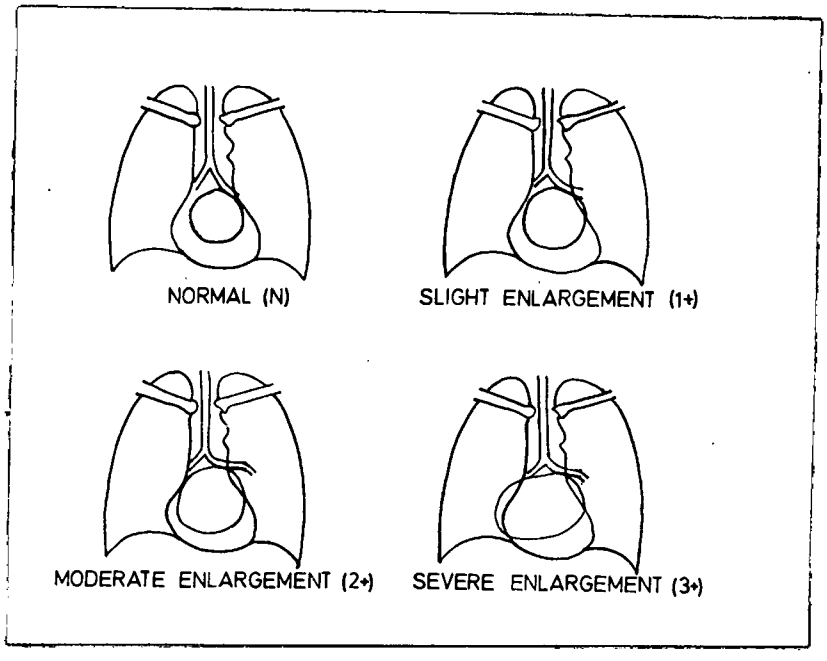


FIG. 5: Schematic representation of the method of grading the size of the left atrium.
N - Normal; 1+, slight enlargement; 2+, moderate enlargement; 3+, severe enlargement.

atrium is shown diagrammatically. It was realised, however, that apparent enlargement of the left atrium would have to be interpreted with some caution depending on whether the radiograph was exposed during systole or diastole.

Whilst an assessment could be obtained of enlargement of the left atrium and of overall enlargement of the heart by chest radiography in the postero-anterior, lateral planes and by a penetrated antero-posterior grid view, routine chest radiography was a disappointing means of diagnosing enlargement of individual heart chambers. In a study of this nature, however, it was felt that it would be unjustified to subject patients to angiocardiography in order to obtain more accurate information of heart size.

Laboratory Data:

The erythrocyte sedimentation rate was measured by a modified Westegren technique (Dawson, 1960), on the day before synchronised capacitor discharge and repeated on the day after.

The serum levels of the glutamic oxaloacetic transaminase was measured by the Sigma Frankel technique

(1956, 1961) and of the lactic dehydrogenase by the technique of Berger and Broida (1957, 1960). Particular note was taken of changes in the serum levels following synchronised capacitor discharge and levels of the glutamic oxaloacetic transaminase which were more than 40 units whereas previously they were below this level were viewed with suspicion. Similarly serum levels of lactic dehydrogenase which rose to more than 600 units were particularly noted.

The sedimentation rate and levels of the serum enzymes were repeated daily until the pre-treatment level returned.

The blood urea and serum electrolytes were measured in all patients receiving diuretic therapy.

The duration of anticoagulant therapy was noted and a thrombotest (Owren 1959) performed to ascertain that the level of control was satisfactory. If necessary the dosage was re-adjusted.

Body Surface Area:

The height and weight of each patient was measured and the body surface area determined from the nomogram prepared according to the formula of Dubois and Dubois (1916).

DRUG THERAPY:

I. Drug Resistant Arrhythmias.

As direct current was a new form of treatment in the management of cardiac arrhythmias, (Lown, Amarasingham, and Neuman, 1962) it was decided that this form of treatment should be restricted at first to those patients whose arrhythmia proved resistant to conventional drug therapy.

Patients were treated initially as follows in an attempt to bring them into sinus rhythm:

1. Patients who had atrial fibrillation:

(a) Not digitalised - Digoxin 0.25 mg each 8 hours for 2 days, subsequently 0.25 mg each 12 hours as a maintenance dose until the ventricular rate had fallen satisfactorily (usually to 70-80 beats per minute) thereafter as for 1(b).

(b) Patients digitalised and on maintenance digoxin -
Quinidine, 1st day test dose of 180 mg followed by 300 mg every 2 hours to a total of 900 mg.
2nd day, Quinidine 600 mg every 2 hours to a total of 1.8 G.
3rd day, Quinidine 900 mg every 2 hours to a total of 2.7 G.
4th day, Quinidine 900 mg every 2 hours to a total of 3.6 G.

All patients being treated with quinidine were admitted to hospital and constant oscilloscopic monitoring of the electrocardiogram was maintained throughout. Precautions were taken so that emergency treatment of cardiac arrest could be undertaken with the minimum delay and medical and nursing staff fully understood the procedure to be adopted in the event of this occurring.

Every patient in this group other than one, (Patient number 15) had been on anticoagulant therapy with satisfactory therapeutic control (Thrombotest 8-15%, Owren, 1959) for at least three weeks before the attempt at drug version.

2. Patients who had atrial flutter:

(a) Digoxin - 0.5 mg every 8 hours until atrial fibrillation occurred or toxic signs developed. In either case digoxin was then withheld. Those patients in whom atrial fibrillation supervened were observed for a further 48 hours to ascertain whether sinus rhythm would occur as the digitalis effect lessened.

(b) (i) Quinidine was given according to the scheme outlined under 1(b) where sinus rhythm failed to follow atrial fibrillation after 48 hours of discontinuing digoxin.
(ii) 3 patients who developed atrial flutter following closure of an atrial septal defect, were given quinidine according to the scheme

outlined in 1(b) even if atrial fibrillation did not occur following heavy digitalisation.

3. Patients who had atrial tachycardia:

(a) Not digitalised (1):

(i) Digoxin 0.5 mg every 8 hours for 3 days.

(ii) Quinidine according to scheme outlined under 1(b) to 2.7 G.

(iii) Procaine amide 250 mg every 6 hours to a total of 20 G.

(b) Patients Digitalised and on maintenance Digoxin (1): Digoxin was continued and quinidine given according to the scheme outlined under 1(b).

II. Remainder:

The remaining patients with atrial fibrillation, atrial flutter, atrial tachycardia or ventricular tachycardia were treated primarily with synchronised capacitor discharge. In many of these patients, however, an attempt had been made to bring about sinus rhythm with quinidine, digoxin, procaine amide or pronethalol. This attempt at drug version was directly under the control of the Physicians who subsequently referred their patients for synchronised capacitor discharge so that the dosages used were not uniform. No claim is made therefore, that the arrhythmias were truly drug resistant.

In all patients digoxin or other digitalis preparations were withheld for one day before the electrical conversion.

Quinidine (300 mg.) was given orally or intramuscularly 1½ hours before synchronised capacitor discharge to a group of patients chosen at random.

Patients in whom sinus rhythm was achieved were divided at random into a group to whom quinidine was given as maintenance therapy and those to whom no quinidine was given. The dosage varied from 180 mg. every 8 hours to 300 mg. every 4 hours and patients were observed for 2-3 days prior to discharge to see whether any toxic symptoms developed. Patients were instructed to stop quinidine immediately and to report as soon as possible with the development of any untoward reaction or with the onset of tachycardia or irregularity of the heart beat.

Digoxin and diuretic therapy were not continued routinely once in sinus rhythm but were maintained in those patients in whom cardiac decompensation was still present.

HAEMODYNAMIC STUDIES:

A representative group of patients with atrial fibrillation or flutter was studied to determine the haemodynamic benefit that could be expected once sinus rhythm had been established.

20 patients were investigated. In 6, heart rate changes and oxygen uptake alone were measured at rest and on graded exercise (Appendix A) and the physical work capacity at a heart rate of 170 beats per minute (PWC 170) determined, by inter- or extrapolation of the linear relationship between heart rate and work load. These studies were undertaken with the arrhythmia present and were repeated one day after the establishment of sinus rhythm.

In the remaining 14 patients cardiac output was measured at rest and on graded exercise and pressure in the pulmonary artery and in the brachial artery was monitored (Appendix B). The PWC 170 was also determined. These studies were done whilst the arrhythmia was present and were repeated one day after the establishment of sinus rhythm. Stroke volume and heart rate were plotted against the identical work load before and after the electrical

conversion. It was found possible therefore, to compare the stroke volume in sinus rhythm and during the arrhythmia at equivalent heart rates. Digitalis was stopped one day before the study and no patient was studied before or after conversion whilst on quinidine.

Exercise was performed using an electrically braked bicycle ergometer, [illegible] and Mattsson (1954), Fig. 6, made by the Elema Corporation. Work loads are preset at the

FIG. 6: Bicycle ergometer (Elema Corporation).

beginning of the experiment and are variable over a very wide range (0 - 2050 kpm; 100 kpm ≡ 16 watts). The patient pedals so as to keep the needle of the revolution counter between 45 and 75 rpm; the work performed does not vary with the revolutions of the pedals between these levels. All measurements were done in the steady state and the position of the patient was constant throughout both at rest and on exercise, namely sitting on the bicycle. Exercise was commenced following a control period of 6 minutes of sitting on the bicycle. Expired air was collected during the last 2 minutes of the control period and the ECG was recorded each 30 seconds but was constantly displayed on an oscilloscope. Six minutes pedalling was performed at each work load and whenever possible the patient was exercised at three separate work loads. Expired air was collected during the last two minutes when heart rate recordings showed that a steady state had been achieved.

Expired air which was collected into a Douglas bag was analysed by a micro method for oxygen and carbon dioxide content, (Scholander, 1947). The volume of the bag was determined by passing the contents through a dry gas meter. A correction of 700 ml representing the dead space of the

bag was made, and volumes were expressed as S.T.P.D. The oxygen uptake of the patient was determined at rest and at each work load. Meticulous attention was paid to ensure that all the expired air was collected in the Douglas bag. The nature of the procedure was carefully explained to the patient and proper fitting and comfort of the mouth piece and nose clip ensured. Trial periods were encouraged to allow the patient to get used to the apparatus and repeated checks were made for air leaks at the mouth or nose.

Heart rate was obtained from the ECG (lead CR5), the number of R waves being counted in 10 seconds, from which the rate per minute was derived.

The 14 patients in whom cardiac output and pressure measurements were also made were studied as follows:- Portex tubing, PG3, 130 cm. long (external diameter 1.02 mm., internal diameter 0.80 mm.) was passed percutaneously into an arm vein through an 18-gauge thin-walled Yale needle and advanced slowly to the pulmonary artery. No X-Ray screening was used and pressure measurements were taken to confirm that the tubing lay in the pulmonary artery. Teflon tubing, TF 10, (external diameter 1.0 mm, internal diameter 0.60 mm.) was passed by the technique described by Seldinger (1953) over a nylon guidewire introduced into the brachial artery on the same side as the

venous catheter through a 19-gauge thin walled Riley needle, and advanced for 6 - 8 inches. Pressure recordings were made using two strain gauges, with a volume displacement of 0.1 cu. mm./100 mm. Hg. (Devices Limited) to ensure an adequate frequency response from the fine bore tubing, and a Sanborn multichannel pen recorder. The tubing, needles and guide-wire used are shown in Fig. 7. Catheterisation was done

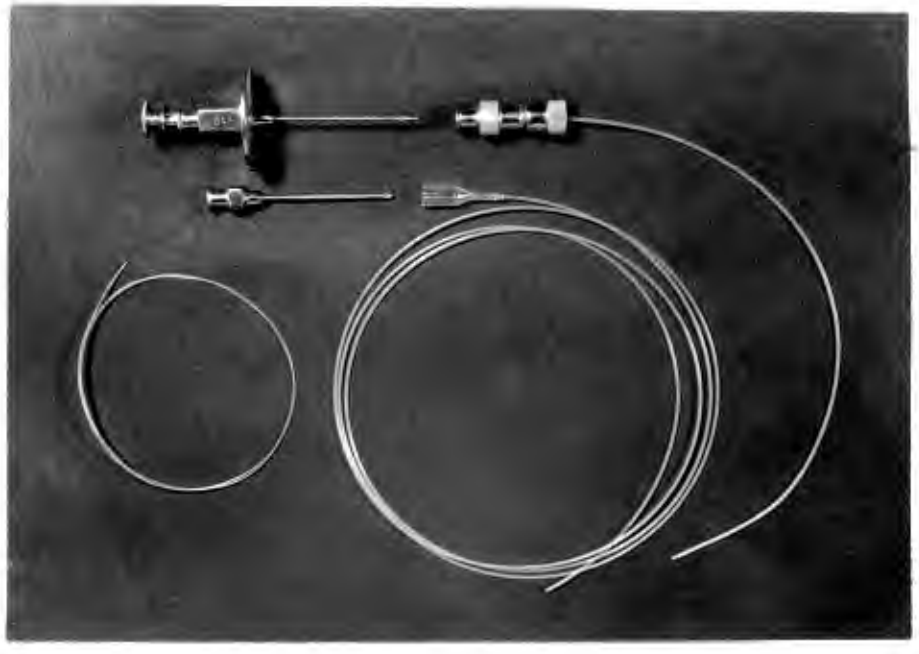


FIG. 7: Venous and arterial catheters, needles and nylon guide-wire.

with the patient lying on an X-Ray table. Once the tubes were in position the patient got off the table and on to the bicycle. Pressure in the pulmonary and brachial arteries was recorded with the electrocardiogram at each 30 seconds; samples of blood from these sites were withdrawn during the last 2 minutes of the 6 minutes of control into oiled glass syringes the dead spaces of which had previously been filled with heparin. Expired air was collected simultaneously with the collection of the blood samples into a Douglas bag. Exercise was then commenced and performed for six minutes at each of 3 work loads (if the condition of the patient permitted). Pressure measurements were recorded with the electrocardiogram each 30 seconds. Once a steady state was achieved expired air was collected during the last two minutes of each work load. Blood samples were collected from the pulmonary and brachial arteries during the collection of the expired air and pressure measurements were recorded with the electrocardiogram at the end of the 6th minute. The general arrangement is shown in Fig. 8. The experiment was repeated one day after the establishment of sinus rhythm.

Pulmonary artery and brachial artery blood was kept heparinised and surrounded by ice until analysed for oxygen content within 1 - 2 hours of the end of the experiment by

Fig. 8: Haemodynamic study.

the manometric method of Van Slyke and Neill (1924). Arterio-venous oxygen difference was obtained and cardiac output derived from this and the oxygen uptake, (Fick, 1870). Stroke volume was calculated and the physical work capacity at a heart rate of 170 per minute determined.

In one patient a different plan was adopted (Appendix C). Formal catheterisation of the pulmonary artery and aorta was done under X-Ray control. Cardiac output was measured in atrial fibrillation sitting on the bicycle at rest and at one work load - the maximum work the patient could undertake. A general anaesthetic was then administered with the patient lying on the catheter room table and sinus rhythm achieved by synchronised capacitor discharge. One hour of recovery from the anaesthetic then followed and the cardiac output studies were then repeated at rest and on one exercise load sitting on the bicycle.

TREATMENT BY SYNCHRONISED CAPACITOR DISCHARGE.

I. THE APPARATUS - THE LOWN CARDIOVERTER

Treatment by synchronised capacitor discharge was done using the Lown Cardioverter (American Optical Company) (Fig. 9) throughout the series.

Basically the apparatus consists of a capacitor of 16 microfarads charged to a direct current voltage by a variable transformer over 10-15 seconds. The capacitor discharges over 2.5 milliseconds through an inductance coil of 100 millihenrys and across the resistance of the body (which varies from patient to patient but is in the order

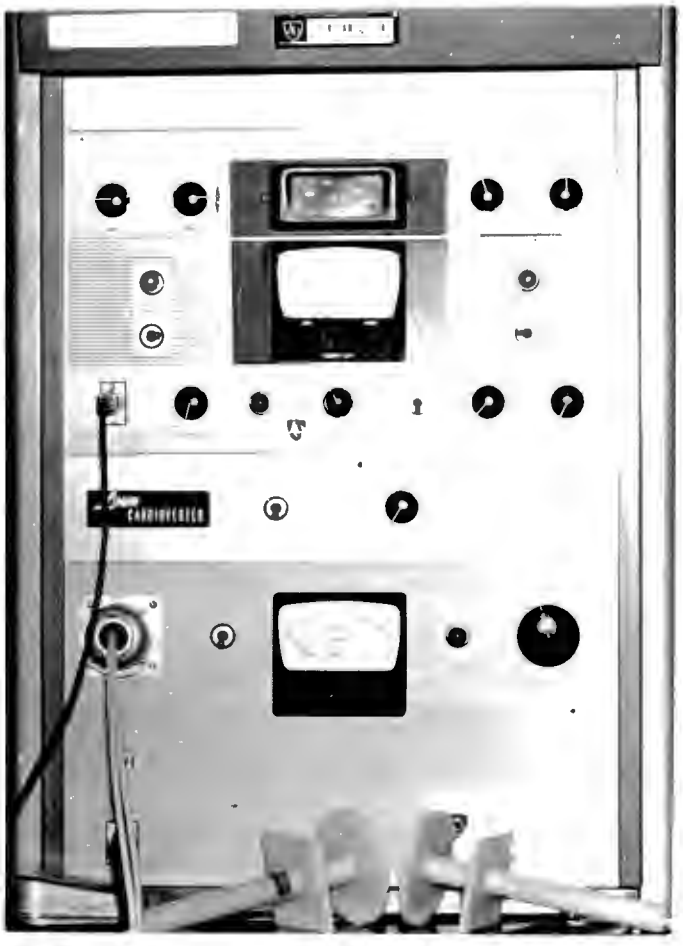


FIG. 9: The Lown Cardioverter.

of 50 ohms) to produce the impulse whose waveform is shown in Fig.10. The energy delivered is a function of the initial direct current voltage and the characteristics of the capacitor and is expressed in joules, or watt-seconds.

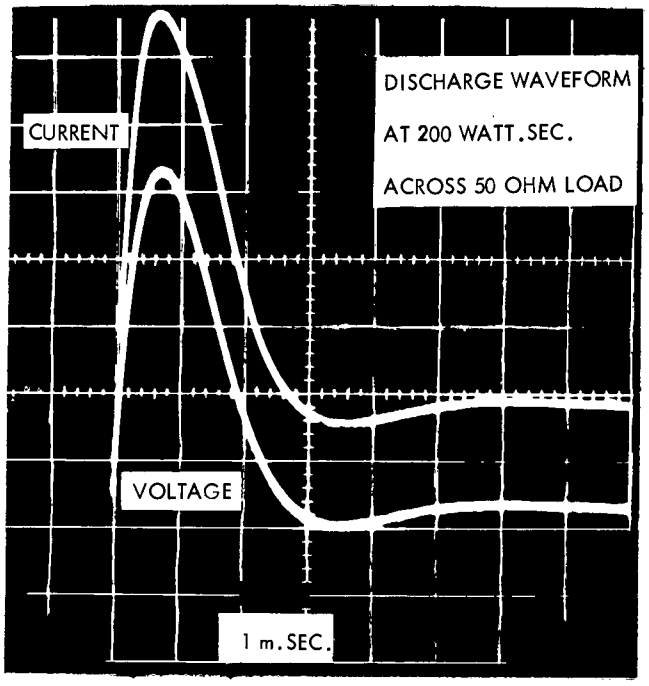


FIG. 10: Current and voltage waveform, Lown Cardioverter.

The discharge is applied at a precise moment in the cardiac cycle and is activated by the R wave of the electrocardiogram or the S wave when R is of insufficient amplitude. Mechanical switching allows a delay of 20 milliseconds following the R or S wave before actual discharge of the

capacitor: the vulnerable phase of ventricular repolarisation is avoided and the risk of ventricular fibrillation abolished. (Fig.11).

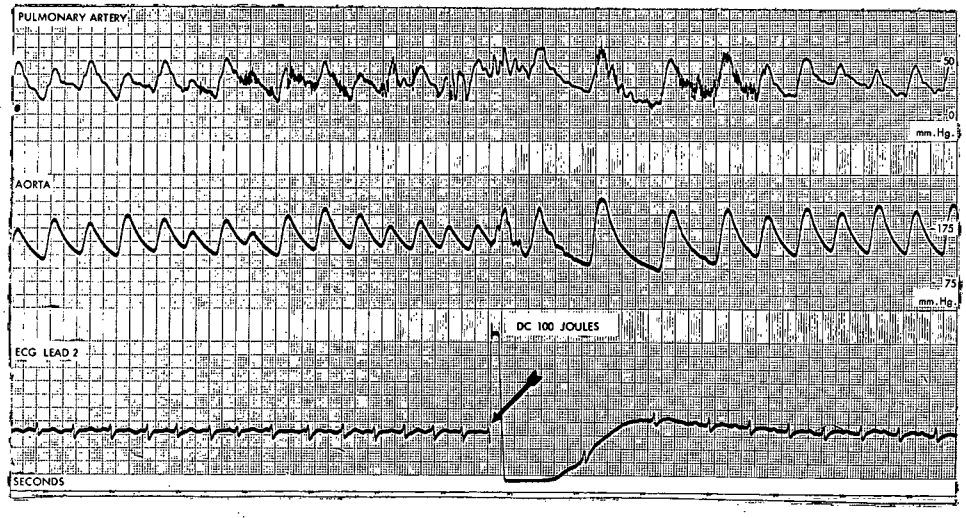


FIG. 11: Synchronised capacitor discharge.
Pressure from the pulmonary artery, and aorta, above, is recorded simultaneously with the electrocardiogram, lead 2, below. Atrial fibrillation is present at the beginning of the record. Sinus rhythm follows a DC shock of 100 joules synchronised to occur on the downstroke of the R wave of the electrocardiogram.
Note the larger pulse pressure in sinus rhythm in both the pulmonary artery and the aorta.

Although the apparatus is provided with a control by which the delay of discharge of the capacitor after the R or S wave can be varied within wide limits (0 to 1.0 seconds) clinical treatment of arrhythmias, other than ventricular fibrillation, demands the minimum delay in all cases. It is essential for the synchroniser to be out of circuit in the treatment of ventricular fibrillation and a switch is provided for this purpose.

In addition, the apparatus provides for constant oscilloscopic monitoring of the electrocardiogram. With the leads connected in the usual way lead 2 of the electrocardiogram is displayed. Alternative connection of the limb leads enables lead 1 or lead 3 to be displayed. A control allowing the sweep-speed of the oscilloscope to be varied from 12.5 to 25 or 50 mm. per second is provided as well as an X-or Y-axis shift control and gain control.

Heart rate can be displayed on a meter sensitive to the R-R time interval of the electrocardiogram. Each R wave can be signalled audibly. For both these parameters to function it is necessary to ensure that the overall sensitivity is such that each R or S wave (reversed polarity) causes a warning light to "flash". With the sensitivity setting too low R or S waves of individual complexes are

missed; with the setting too high the warning light will flash from parts of the cardiac cycle other than the R or S wave, for example the T wave. Great care is taken, therefore, to ensure that the sensitivity control is correctly adjusted. A high rate or low rate setting causes an alarm to sound when either limit is exceeded.

Provision is also made for pacemaking should this be needed, the impulse varying from 0 - 150 volts and the rate setting from 15 - 175/minute. With internal pacemaking up to 15 milliamperes can be delivered from a high impedance source. The impulse lasts 3 milliseconds.

The capacitor discharges between the negative and positive paddles applied to the surface of the chest. The machine is provided with 2 anterior paddles (adult size): 2 anterior paddles (paediatric size) and 1 pair of anterior-posterior paddles (adult size) (Fig.12). Pacemaking can be undertaken through any of the paddles applied directly to the surface of the chest.

The dimensions of the instrument are:
22" x 28" x 12"
and the weight is 120 lbs.
The dimensions of the paddles are:
1. The two anterior paddles: Each, diameter, $3\frac{3}{4}$".
2. The antero-posterior paddles: Anterior, diameter, $3\frac{1}{2}$": Posterior, diameter, 5".
3. The paediatric paddles: Each, diameter, $1\frac{1}{4}$".

FIG. 12: Paddles used to deliver the DC shock.

A - Adult size, anterior.
B - Adult size, antero-posterior.
C - Child, Anterior.

II. METHOD OF TREATMENT BY SYNCHRONISED CAPACITOR DISCHARGE:

Treatment by synchronised capacitor discharge was undertaken in the following way which was kept virtually unchanged throughout the series:-

All patients were explained the nature of their disability and the form of treatment it was proposed to carry out.

Patients were starved routinely for four hours before synchronised capacitor discharge.

Anaesthesia:

Pre-medication when used was 50-75 mg. pethidine intramuscularly in all adults except 5 patients in whom omnopon, 10 mg., was used.

Patients were taken by trolley from their wards to the anaesthetic room of the operating theatre where a full 12-lead electrocardiogram was done. Immediately thereafter anaesthesia was induced by the slow intravenous injection of a sleep dose of 2.5% sodium thiopentone or 1% sodium methohexitone. Once asleep anaesthesia was maintained with 70% nitrous oxide with oxygen administered by face mask and a Magill circuit and, where needed 0.5% halothane was added sparingly.

In gravely ill patients, however, particularly where the cardiac output was low or systemic hypotension present treatment was frequently undertaken in the wards. No pre-medication was used and anaesthesia was induced and maintained with 50 - 60% nitrous oxide, oxygen and 0.5% halothane. Succinyl choline, with controlled respiration was substituted for halothane in any patient who had recently received treatment with a sympathomimetic vasopressor to avoid arrhythmias resulting from the anaesthetic (Johnstone and Nisbet, 1961). Thiopentone or methohexitone were used after induction only if the patient struggled.

Intubation and controlled respiration using succinyl-choline as a relaxant used in the first five patients were discontinued as being unnecessary.

One patient in whom profound circulatory collapse was present was treated without any anaesthetic.

Electrical Conversion:

The electrocardiogram leads of the Lown Cardioverter were attached and the sensitivity adjusted so that only each systole was signalled on the warning light and no other part of the cardiac cycle caused a signal. The heart rate was displayed on the tachometer and the ECG on the oscilloscope.

If the S wave was of greater amplitude than the R wave, the polarity switch was reversed so that synchronisation with the S wave would be obtained. Where both R and S wave were of small amplitude and especially if the T wave amplitude approached the amplitude of R or S (at times it exceeded it) the limb leads were reconnected so that lead 1 or lead 3 was displayed.

Synchronisation of the discharge with the R or S wave of the electrocardiogram was always tested immediately before each treatment session. The paddles through which the capacitor was to discharge were attached to the output socket. The capacitor was charged to 20 joules. The connecting cables of the paddles were brought into physical contact with the electrocardiogram patient cable of the direct-writing electrocardiogram machine. The positive and negative paddles were brought into apposition away from the patient and the capacitor discharged as the electrocardiogram was recorded at a paper speed of 50 mm. per second. The point in the cardiac cycle at which the capacitor discharged was recorded as a distinct artefact on the electrocardiogram (Fig. 13). No instance of failure of synchronisation with the R or S wave occurred, allowing for the inherent 20 milliseconds delay.

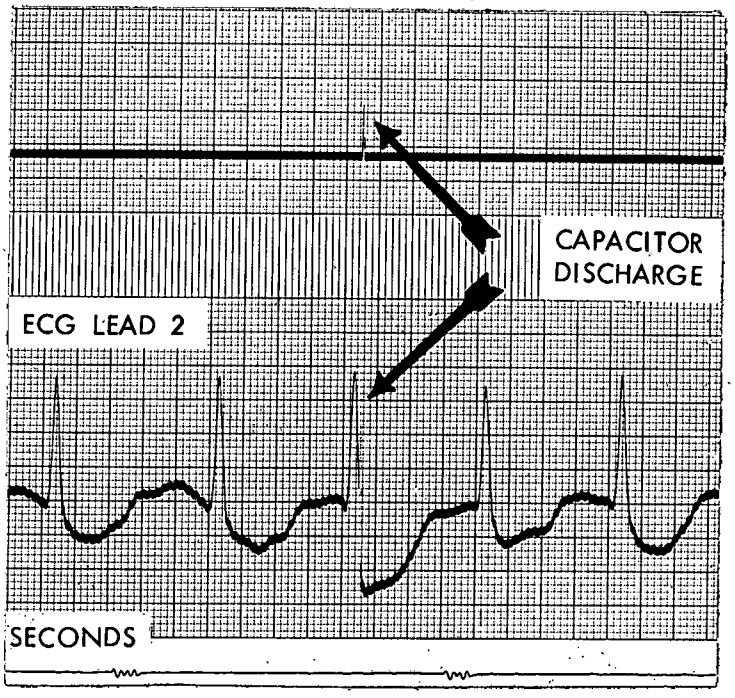


FIG. 13: Test of efficient synchronisation.
ECG lead 2, 50 mm/sec. The artefact caused by discharge of the capacitor is superimposed on the electrocardiogram and is also displayed on the upper channel of the record.

The pacemaker and controls were tested to make sure they were functioning adequately.

The blood pressure was measured by arm cuff.

Either two anterior paddles or an anterior and posterior paddle were used. The skin was prepared by a liberal

application of electrocardiogram jelly well rubbed in. If the two anterior paddles were used, the negative paddle, well covered with electrocardiogram jelly, was placed over the apex beat whilst the anterior, also well covered with electrocardiogram jelly, was placed over the second right interspace just to the right of the sternum (Fig. 14).

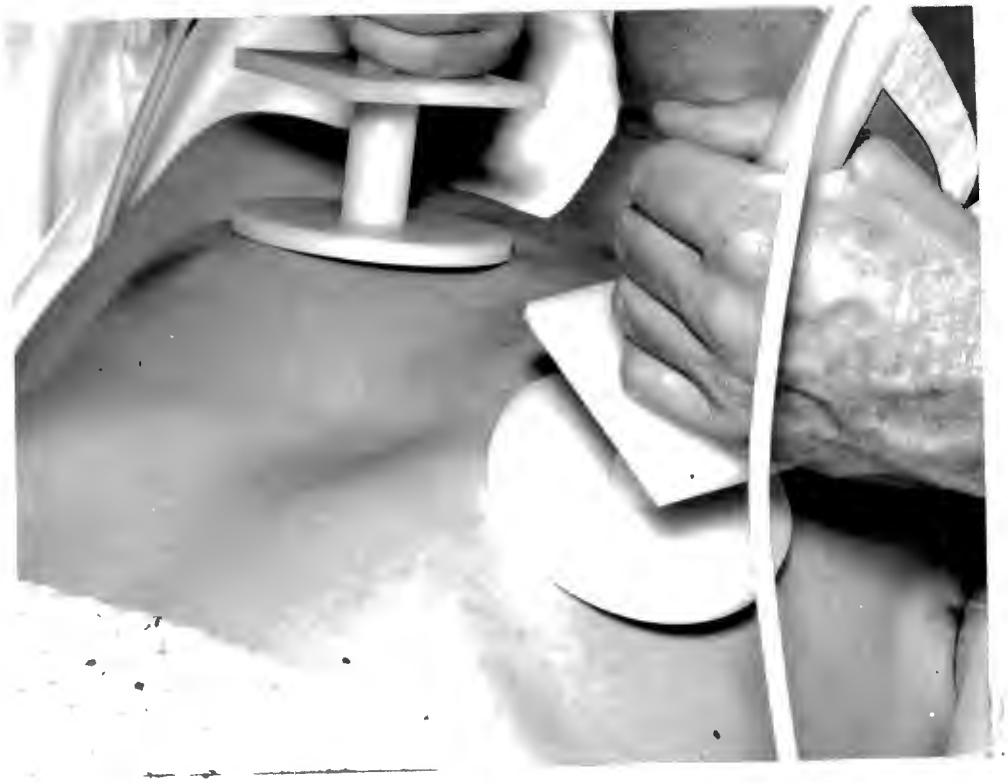


FIG. 14: Synchronised capacitor discharge. Anterior paddles in position.

Using an anterior and a posterior electrode, the flat posterior electrode, well covered with electrocardiogram jelly, was placed between the angle of the scapula and the spine, the patient lying on the paddle as he lay on the rubber mattress of the trolley. The negative anterior electrode was placed over the apex beat as before, (fig.15).

FIG. 15: Synchronised capacitor discharge. Antero-posterior paddles in position.

Great care was taken that no part of the patient's skin was in direct contact with the metal of the trolley on which he was lying. Those who were assisting with the procedure were warned not to touch the patient or his trolley.

The direct writing electrocardiograph was set to record at 25 mm. per second, the anaesthetist momentarily removed the face mask and the capacitor was discharged synchronously with the R or S wave of the electrocardiogram. An initial setting of 100 or 150 joules was used with the two anterior paddles and 50 joules with the antero-posterior paddles. The electrocardiogram was examined immediately after and the rhythm determined. If sinus rhythm was not present the capacitor was charged to deliver a higher energy and the procedure was repeated until maximum energy (400 joules) had been achieved if necessary. In general, two schemes were adopted for the second and subsequent shocks, depending on whether anterior or antero-posterior paddles were being used.

(i) Two anterior paddles - 250 joules, 350 joules, 400 joules.

(ii) Antero-posterior paddles - 100 joules, 200 joules, 300 joules, 400 joules.

A comparison was made of the energy required for the conversion

to sinus rhythm using anterior paddles and antero-posterior paddles. The blood pressure was taken after the second and subsequent shocks.

With the development of sinus rhythm, or on failure to achieve sinus rhythm after maximum energy the anaesthetic was discontinued, and a full 12-lead electrocardiogram was done on every patient.

RECOVERY:

Once awake patients not treated in the wards were either taken to the intensive care unit or returned to their ward. In either case oscilloscopic monitoring of the electrocardiogram was maintained routinely for twenty-four hours and for longer when needed. Blood pressures were recorded by arm cuff every half-hour if hypotension followed the electrical treatment until the blood pressure had returned to its pre-treatment level.

FOLLOW-UP:

Patients were discharged from hospital at the discretion of the referring Physician or Surgeon. A personal follow-up was made whenever possible for a period of up to twenty months and for never less than three months

after treatment. Patients who had moved from London were contacted through their personal Practitioners and a follow-up maintained in that way. Contact was maintained in all but one of the 150 patients of the series.

Patients were divided at random into two groups following treatment - those who were and those who were not taking quinidine as maintenance. The results were analysed statistically.

FURTHER ATTEMPT AT TREATMENT BY SYNCHRONISED CAPACITOR DISCHARGE

1. Patients who Failed to come into Sinus Rhythm:-

A further attempt to achieve sinus rhythm was made in 3 patients in whom the attempt at electrical conversion was unsuccessful.

2. Patients who Reverted to the Arrhythmia:-

A second attempt at synchronised capacitor discharge was undertaken in 17 patients in whom sinus rhythm was maintained only temporarily. This second treatment was undertaken in a similar fashion to the first but quinidine frequently preceded the second attempt when withheld before the first; similarly quinidine was frequently prescribed as maintenance therapy after the second but not after the first attempt.

SUMMARY

1. Details of the method of the clinical assessment of patients before and after synchronised capacitor discharge is given.

2. The electrocardiographic abnormalities before treatment were analysed according to rhythm, digitalis effect, bundle-branch block, ventricular hypertrophy, and changes of ischaemic heart disease. Following treatment particular note was taken of rhythm, ectopic beats, conduction disturbances, and ST segment and T wave change.

3. Overall size of the heart was determined by the cardio-thoracic ratio%. The size of the left atrium was graded. No accurate estimation could be made of the size of the right atrium, right ventricle or left ventricle and the reasons are discussed. The radiographs were analysed before and after treatment.

4. Details are given of the laboratory data used before and after treatment.

5. Body surface area was measured.

6. Details are given of drug therapy used to determine whether atrial fibrillation, atrial flutter and atrial tachycardia were drug resistant.

7. Both before and after synchronised capacitor discharge patients were divided at random into those who were given quinidine and those who were not.

8. Other drug therapy used is detailed.

9. A representative group of 20 patients with atrial fibrillation and flutter were studied to ascertain the haemodynamic benefit in sinus rhythm at rest and on exercise. Details are given.

10. The Lown Cardioverter is described.

11. The method of treatment by synchronised capacitor discharge is described including the anaesthetic techniques employed. Either anterior or antero-posterior paddles were used to deliver the shock and a comparison was made of the energy needed for successful version in either case.

12. Details of the follow-up procedure are given.

13. Further attempts were made at treatment by synchronised capacitor discharge in patients who had failed to achieve sinus rhythm after the first attempt, or in whom sinus rhythm was maintained only temporarily.

Chapter 5

PATIENT MATERIAL

150 patients (Table 1) who had a supraventricular or ventricular tachycardia were treated by synchronised capacitor discharge.

A second planned attempted version using synchronised capacitor discharge was undertaken in 20 patients, 17 of whom had relapsed to their original arrhythmia and in 3 of whom the first attempt had failed. 170 episodes of patient treatment are therefore presented.

The only criterion used for admission to this trial was the presence of a supraventricular or ventricular arrhythmia requiring version to sinus rhythm, and the 150 primary episodes of treatment presented are consecutive. No patient was excluded on the grounds of medical unfitness and indeed, as will be shown later, those patients acutely ill as a result of an uncontrolled arrhythmia formed a most gratifying and successful group.

Patients excluded from this trial fell into two groups:

TABLE 1

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug,	Dose,	RESULT	PREC. QUIN.	SYNCHRONISED ENERGY joules	SYNCHRONISED RESULT
1	F 46	AF	7 mo	Rh. HD Post mitral valvot MI Gr.1.	50	1	AF Dig	6	14	180	Yes	Digoxin Diuretic	Q.	900mg.	Fail Nausea	Yes	50 100	SR
2	M 46	AF	15 mo	Alc.cardiomyop.	54	2	AF.LV+ Dig.V.E.	18	30	160	Yes	Digitalis	Q.	2.70r.	Fail VE ++	Yes	100 150	SR
3	M 55	AF	3 yr	Lone	55	1	AF	5	18	220	Yes	Digitalis	Q.	3.60r.	Fail		100 150 200	SR
4	M 50	AF	16 yr	Rh. HD. Post mitral valvot AI (slight)	49	1	AF Dig	3	24	160	Yes	Digoxin	Q.	2.70r.	Fail Nausea		100 150	SR
5	F 21	AF	6 mo	Lone	40	N	AF Dig	9	18	180	Yes	Digoxin	Q.	2.10r.	Fail Tachycar. VE		100 200 300 350	SR,5 beats SR
6	M 40	A FLUT	12 mo	Post op. ASD	45	N	A Flut 4:1 Dig Inc RBBB	5	47	260	Yes	Digitoxin	Digoxin Q.	2.10r.	Fail Nausea		150	SR
7	M 45	AF	4 yr	Rh. HD. Post mitral valvot MI Gr.1.	47	1	AF. Dig. Inc RBBB	4	60	280	Yes	Digoxin	Q.	240mg	Fail Undywn.	Yes	100	SR
8	F 53	AF	14 yr	Rh. HD. Post mitral valvot MI Gr.1	64	1	AF Dig.	20	14	260	Yes	Digitalis Diuretic	Q.	3.60r.	Fail		150 200 250 350	Fail
9	F 57	AF	3 mo	Alc.cardiomyop.	53	N	AF Dig	7	24	190	Yes	Digoxin	Q.	4.40r.	Fail		150	SR
10	M 52	AF	4 yr	Alc.cardiomyop.	47	1	AF Dig.	2	22	480	Yes	Digoxin	Q.	2.40r.	Fail Nausea	Yes	150 200 250	SR
11	F 33	A FLUT	1 wk	Rh. HD. Post mitral valvot	49	1	A Flut 3:1	11	21	230	Yes	Digitoxin	Q.	4.50r.	Fail		150	SR

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY		ECG	SINUS RHYTHM	BSA
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR%	LA		MAINTAINED FOR	m^2
VR		Yes	8	26	190	50	1	PR 0.16 Dig.	6 mo	1.64
		Yes	6	30	180	52	2	PR 0.24 LV+	4 mo	1.86
	Hypotension Inverted T Pulm.inf.	Yes	9	34	200	55	1	T ↓ V_{4-6}	10 mo	1.81
Atrial pc		Yes	3	24	170	49	1	PR 0.26	90 min	1.80
Atrial pc			7	34	200	40	N	PR 0.10	30 min	1.57
2:1 HB Wenck		Yes	7	47	280	44	N	Inc RBBB	Still present	1.99
Atrial pc		Yes	4	60	320	44	1	Inc RBBB Dig	2 mo	1.81
	Enzyme +		22	42	680	62	1	No change		1.60
Atrial pc		Yes	5	10	430	51	N	PR 0.16	3 mo	1.65
Nodal VT	Enzyme+	Yes	2	38	660	47	1	PR 0.22 LV+	3 mo	1.96
			16	30	190	49	1	PR 0.24	Still present	1.32

TABLE 1 (continued 1)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug, Dose,	DRUG REVERSION RESULT	PREC. QUIN.	SYNCHRONISED ENERGY joules	SYNCHRONISED RESULT
12	22 M	AF	2 mo	Lone	47	1	AF Dig	2	20	-	Yes	Digoxin	Q. 1.4Gr.	Fail V.E.	Yes	150 250 350 400	 SR
13	42 M	AF	17 yr	Lone	57	1	AF Dig	3	21	255	Yes	Digoxin	Q. 2.1Gr.	Fail V.E.	Yes	200 250 350 400 400	 Fail
14	29 M	AF	6 yr	Rh. HD. Post mitral valvot MI Gr. 2	45	2	AF LV+ Dig	12	36	-	Yes	Digoxin	Q. 540mg.	Nausea Fail		150 250 350 400	 Fail SR 20 sec
15	46 M	AF	4 yr	Rh. HD. Post mitral valvot MI Gr. 1	49	1	AF Dig	28	24	220		Digoxin Diuretic	Q. 1.7Gr.	Fail Hypo-tension		150 250	SR
16	43 M	AF	3 mo	Alc.cardiomy.	45	1	AF Dig	1	38	-	Yes	Digoxin	Q. 4.2Gr.	Fail Nausea V.E.		150 250	SR
17	32 M	AF	8 mo	Rh. HD. MI. MS. AI MI Gr. 1	50	2	AF Dig.LV+	2	18	480	Yes	Digoxin Diuretic	Q. 5.2Gr.	Fail Nausea V.E.		150	SR
18	27 F	A FLUT	15 mo	Post op. ASD	50	1	3:1 Inc RBBB	6	28	-	Yes	Digoxin	Q. 2.4Gr.	Fail		150	SR
19	52 M	AF	3 yr	Rh. HD. Post mitral valvot MI Gr. 2	55	2	AF Dig	4	16	400	Yes	Digoxin	Q. 2.2Gr.	Fail Nausea	Yes	150 250	SR
20	47 F	AF	2.5 yr	Post thyrotox.	50	N	AF Dig	3	27	290	Yes	Digoxin	Q. 600mg.	Fail Syncope Tachyc.	Yes	150	SR
21	53 M	AF	2 yr	Alc.cardiomy.	43	1	AF Dig	3	32	490	Yes	Digoxin	Q. 4.2Gr.	Fail	Yes	150	SR
22	55 F	AF	5 mo	Rh. HD. Post mitral valvot MI Gr. 1	50	1	AF Dig LV+	3	18	450	Yes	Digoxin	Q. 5.2Gr.	Fail		150	SR

CAPACITOR DISCHARGE INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER	ESR	ENZYME SGOT	ENZYME LDH	X-RAY CTR%	X-RAY LA	ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
	Enzyme + Gallop	Yes	3	70	-	50	1	PR 0.12	Still present	1.78
	Hypotension Enzyme +		6	60	680	54	1	No change		1.83
	Enzyme +		16	51	-	54	2	No change		1.90
		Yes	32	38	280	49	1	P-R 0.22 P mitrale	Still present	1.50
VB		Yes	1	41	-	44	1	PR 0.20	Still present	1.84
VE		Yes	2	18	510	50	2	PR 0.24 P mitrale	1 mo	1.72
Nodal		Yes	4	31	-	50	1	PR 0.26	1 yr	1.65
		Yes	8	25	290	55	2	PR 0.24 Dig	1 mo	1.92
		Yes	6	24	490	50	N	PR 0.16 Dig	Still present	1.62
		Yes	2	31	360	43	1	PR 0.16 Dig	Still present	2.05
Brady car. At. pc.		Yes	4	18	160	50	1	PR 0.16 Inc RBBB	5 mo	1.52

TABLE 1 (continued 2)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug,	Dose,	RESULT	PREC. QUIN.	SYNCHRONISED ENERGY joules	SYNCHRONISED RESULT
23	42 F	AF	9 mo	Rh. HD. Post mitral valvot	51	1	AF Dig	4	22	260	Yes	Digitaline	Q.	3.6Gr.	Fail	Yes	150	SR
24	42 F	AF	2 mo	Rh. HD. MI Gr. 2 AI Gr. 2	53	2	AF LV+ Dig	5	15	120	Yes	Digoxin	Q.	1.5Gr.	Fail Nausea		150 250	SR
25	40 F	AF	1 yr	Rh. HD. Post mitral valvot MI Gr. 2	54	2	AF Dig	8	18	510	Yes	Digoxin Diuretic	Q.	1.2Gr.	Fail Nausea	Yes	150	SR
26	40 F	AF	8 mo	Rh. HD. Post mitral valvot MI Gr. 1	57	2	AF Dig	41	21	200	Yes	Digoxin	Q.	2.4Gr.	Fail	Yes	150	SR
27	49 M	AF	15 yr	Lone	46	1	AF Dig	3	21	110	Yes	Digitalis	Q.	5.4Gr.	Fail	Yes	150 250 350 400	Fail
28	51 M	A FLUT	18 yr	Lone	48	1	3:1 Dig	2	38	340	No	Digoxin	Q. Proc.Amide Neostig.	1.8Gr. 2.0Gr. 1 mg	Fail	Yes	150	SR
29	50 M	AF	7 yr	Post op. ASD	56	N	AF Dig Inc RBBB	6	10	280	Yes	Digoxin	Q.	2.7Gr.	Fail	Yes	150 250 350 400	SR
30	45 M	A FLUT	13 mo	Post op. ASD	61	2	4:1 Dig Inc RBBB	7	34	220	No	Digitaline	Q. Digoxin	3.8Gr. 1mg	Fail Nausea	Yes	150	SR
31	57 F	A TACHY	7 mo	Rh. HD. Post mitral valvot MI Gr. 2	64	2	2:1 Dig	15	16	160	No	Digoxin	Q.	2.2Gr.	Fail		150	SR
32	56 F	AF	2 yr	Rh. HD. MS. MI. Gr. 2	58	2	AF Dig	4	10	230	Yes	Digitalis	Q.	6.0Gr.	Fail Nausea	Yes	150 250	SR
33	47 F	AF	10 yr	Rh. HD. Post mitral valvot	62	1	AF Dig	13	18	200	Yes	Digoxin	Q.	7.0Gr.	Fail	Yes	150	SR

CAPACITOR DISCHARGE INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER	ESR	ENZYME SGOT	ENZYME LDH	X-RAY CTR%	X-RAY LA	ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
Nodal At. pc.		Yes	3	14	190	51	1	PR 0.14 Dig	11 mo	1.62
		Yes	8	7	400	54	2	PR 0.22 Dig LV+	9 mo	1.64
		Yes	15	15	320	54	2	PR 0.16	Still present	1.48
		Yes	40	27	180	57	2	PR 0.24 Dig	Still present	1.47
	Enzyme + Hypotension Gallop T flat V_6		3	168	800	47	1	T flat V_6		1.64
		Yes	2	18	230	48	1	PR 0.16 Dig	5 mo	1.70
At. pc V.E.		Yes	5	18	300	56	N	PR 0.16 Dig Inc RBBB	20 d	1.68
		Yes	4	32	250	61	2	PR 0.18 Inc RBBB	Still present	2.02
At. pc VE		Yes	18	20	140	64	2	PR 0.20 Dig	14 mo	1.53
VE		Yes	6	40	210	58	2	PR 0.24 P mitrale Dig	1 mo	1.70
		Yes	9	25	180	56	1	PR 0.18 Dig	4 mo	1.68

TABLE 1 (continued 3)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug,	DRUG REVERSION Dose,	DRUG REVERSION RESULT	PRECOX QUIN.	SYNCHRON ENERGY joules	SYNCHRON RES
34	51 M	AF	2 wk	Lone	50	1	AF Dig	2	8	100	Yes	Digoxin	Q.	4.7Gr.	Fail	Yes	150 250	SR
35	42 M	AF	2 yr	Rh. HD. MS. Gr. 2	40	1	AF Dig	4	25	250	Yes	Digoxin	Q.	300mg.	Fail Syncope		150 250	SR
36	44 F	AF	10 yr	Rh. HD. Post mitral valvot MI. Gr. 1	71	2	AF Dig RV+,LV+	21	16	190	Yes	Digitalis	Q.	2.4Gr.	Fail		25 50 100 250 350 400	Fail
37	49 M	AF	1 yr	Rh. HD. Post mitral valvot MI Gr. 1 AI Gr. 1	55	2	AF Dig LV+	5	13	190	Yes	Digoxin Diuretic	Q.	2.7Gr.	Fail Syncope		25 50 150	SR
38	57 M	AF	7 mo	Chr.Isch HD	55	2	AF Dig RBBB Ant.Inf.	7	14	430	Yes	Digoxin Diuretic	Q.	2.7Gr.	Fail Syncope	Yes	50 150	SR
39	28 F	AF	4 yr	Rh. HD. SBE Mitral valve repair MI Gr. 2	55	2	AF Dig LV+, RV+	8	24	285	Yes	Digoxin Diuretic	Q.	1.8Gr.	Fail Nausea	Yes	50 150 250 400	Fail
40	53 M	AF	5 mo	Lone	50	N	AF Dig	7	46	190	Yes	Digoxin	Q.	2.8Gr.	Fail Nausea	Yes	150 250	SR
41	59 M	AF	4 mo	Rh. HD MS Gr. 2	47	1	AF Dig	3	26	210	Yes	Digoxin	Q.	2.4Gr.	Fail Nausea	Yes	50 150 350	SR
42	41 M	A TACHY	4 d	Myoc.Inf.	59	2 ‡	A.tachy Ant.Inf. RBBB	3	30	200	Yes		Digoxin Proc.amid.	1.5mg. 2Gr.	Nausea Fail		150	SR
43	49 M	AF	6 mo	Rh. HD. MS Gr. 2	49	2	AF Dig LV+	7	40	180	Yes	Digoxin	Q.	1.5Gr.	Fail Nausea	Yes	150	SR
44	53 M	AF	11 mo	Lone	48	N	AF Dig	5	30	100	Yes	Digoxin	Q.	4.8Gr.	Fail	Yes	150 250	SR

‡ Pulm. oedema

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY		ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR%	LA			
VT		Yes	6	13	280	48	1	PR 0.16	1 yr	1.98
			8	35	230	41	1	PR 0.18 P mitrale	12 mo	1.84
	Enzyme +		10	65	1200	71	2	No change		1.55
		Yes	6	24	230	55	2	PR 0.16 P mitrale LV+ Dig	19 mo	1.74
		Yes	4	40	340	55	2	PR 0.26 RBBB Ant. Inf.	2 mo Death in S.E.	1.77
			15	33	290	55	2	No change		1.63
At. pc		Yes	4	40	180	49	N	PR 0.20 P mitrale LV+	Still present	2.05
	Pulm. oedema	Yes	3	27	200	52	2	PR 0.18 P mitrale Dig	3 d	1.81
		Yes	6	40	210	51	N	PR 0.16 Ant. inf.	Still present	1.70
At. pc. V.S.		Yes	6	34	200	52	2	PR 0.20 P mitrale	10 mo	1.75
At. pc.		Yes	5	30	120	48	N	PR 0.20 Dig	Still present	2.25

TABLE 1 (continued 4)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	LA	ECG	ESR mm	ENZYME units SGOT	LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug,	Dose,	RESULT	PREC. QUIN.	SYNCHRONIS ENERGY joules	RESUL
45	42 M	AF	5 yr	Lone	47.5	N	AF Dig	4	35	80	Yes	Digitalis	Q.	1.2Gr.	Fail Hypotension Nausea Tinnitus	Yes	150 250 350	Fail
46	34 F	AF	3 mo	Rh. HD. MS.MI.TI. AI. severe	67	2	AF Dig RV+,LV+	7	20	180	Yes	Digoxin Diuretic	Q.	2.7Gr.	Fail Nausea	Yes	150	SR
47	52 M	A FLUT	1 yr	Syst. hyp. MI. Gr. 2	54	2	3:1 Dig LV+	25	20	210	Yes	Digoxin Diuretic	Q.	3.6Gr.	Fail	Yes	150	SR
48	54 M	AF	19 mo	Lone	50	N	AF Dig	4	43	160	Yes	Digoxin	Q.	1.0Gr.	Fail Nausea	Yes	150	SR
49	53 M	A FLUT	2 mo	Lone	44	N	6:1 Dig	5	43	140	Yes	Digoxin	Q.	2.4Gr.	Fail	Yes	150	SR
50	26 M	A FLUT	8 d	VSD PHT	64	3	2:1,3:1 RV+ LV+	5	40	130	No	Digoxin Diuretic	Digoxin	1.5mg.	Toxic		150	SR
51	43 M	AF	2 yr	Rh. HD. MS. Gr. 1	50	1	AF Dig	9	15	160	Yes	Digoxin				Yes	50 150	SR
52	36 F	AF	3 yr	Rh. HD. Mitral valvot MI Gr. 2	60	2	AF Dig LV+	6	23	250	Yes	Digoxin Diuretic	Q.	8.2Gr.	Fail	Yes	150 250	SR
53	48 M	AF	1 yr	Rh. HD. MI severe	61	2	AF Dig LV+	3	16	240	Yes	Digoxin Diuretic	Q.	1.8Gr.	Fail VE	Yes	150	SR
54	45 F	AF	19 mo	Rh. HD. Mitral valvot MI Gr. 1	50	2	AF Dig LV+,RV+	12	10	200	No	Digoxin Diuretic				Yes	150	SR
55	58 F	AF	8 mo	Rh. HD. MI. Gr. 2	60	2	AF Dig LV+	2	22	220	Yes	Digoxin	Q.	1.9Gr.	Fail Nausea	Yes	150 250	SR
56	40 M	AF	3 mo	Rh. HD. MS. MI. AI.	60	2	AF Dig LV+,RV+	9	29	280	No	Digoxin Diuretic				Yes	150 250 350 400	Fail

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY		ECG	SINUS RHYTHM MAINTAINED	BSA
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR%	LA			m^2
	Hypotension Enzyme +		6	50	40	47	N	No change		1.77
		Yes	3	20	180	67	2	PR 0.26 Dig LV+,RV+	1 mo	1.50
		Yes	20	23	190	53	2	PRO.20 LV+	9 mo	1.92
At. pc.			4	32	200	50	N	PR 0.26 Dig	15 hr	1.90
Nodal		Yes	3	41	160	44	N	PR 0.16 Dig	Still present	1.96
			6	59	120	64	3	PR 0.20 RV+ LV+	3 wk	1.65
	Death	Yes	10	30	120	50	1	PR 0.20 Dig	36 hr to VF	1.67
V.E.		Yes	4	30	220	60	2	PR 0.24 Dig LV+	3 mo	1.58
V.E. At. pc.		Yes	5	20	230	60	2	PR 0.18 P mitrale LV+	Still present	1.76
V.E. Nodal		Yes	15	20	210	50	2	PR 0.20 Dig RV+ LV+	3 mo	1.59
V.E.		Yes	5	35	340	60	2	PR 0.24 Dig LV+	1.5 hr	1.53
			13	43	300	60	2	No change		1.73

TABLE 1 (continued 5)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-COAG. Yes/NO	OTHER DRUGS	DRUG REVERSION Drug,	DRUG REVERSION Dose,	DRUG REVERSION RESULT	PREC. QUIN.	SYNCHRONISED ENERGY joules	SYNCHRONISED RESULT
57	42 M	AF	9 yr	Rh. HD. Mitral valvot MI Gr.1. AI	54	2	AF Dig LV+	11	26	260	No	Digoxin				Yes	150 250 350	SR
58	56 F	AF	9 yr	Rh. HD. Mitral valvot	67	3	AF Dig	7	28	180	No	Digoxin					150 250 350 400	Fail
59	56 F	AF	4 mo	Rh. HD. MI Gr. 2	58	2	AF Dig RBBB	2	35	590	Yes	Digoxin	Q.	1.20gr	Fail	Yes	50 150	SR
60	47 F	AF	6 yr	Rh. HD. Mitral valvot MI Gr.1. AI	52	1	AF Dig	9	18	325	Yes	Digoxin Diuretic	Q.	1.20gr.	Fail Nausea	Yes	50 150 250 400	Fail
61	39 M	AF	4 yr	Lone	44	N	AF Dig	2	25	250	Yes	Digoxin	Q.	1.60gr.	Fail		150 250	SR
62	56 F	AF	8 yr	Post op. ASD Still P/S = 2	71	2	AF Dig Inc RBBB	11	24	230	No	Digitalis Diuretic	Q.	1.80gr.	Fail	Yes	150 250 350	Fail
63	51 F	AF	6 wk	Lone	50	1	AF Dig	9	35	500	Yes	Digoxin	Q.	2.40gr.	Fail		150	SR
64	74 M	VT	1 d	Myoc. inf.	57	1	VT	4	>200	>2000	No	Digoxin Diuretic	Q. Proc.amide	2.00gr. 1.40gr.	Fail		150	SR
65	25 M	A TACHY	9 d	VSD WPW	53	N	A.Tachy	17	-	>2000	No		Q. Digoxin	3.60gr. 1.5mg	Fail	Yes	150 250 400 400 400	Fail
66	17 M	A FLUT	3 mo	Dystrop.myoton.	46	N	2:1,3:1 Inc RBBB	4	30	270	No	Digoxin					150	SR
67	45 M	AF	12 yr	Lone	43	1	AF Dig	5	15	400	Yes	Digoxin	Q.	0.40gr.	Fail	Yes	150	SR
68	39 M	AF	3 mo	Cor.Transp. "MI" Gr. 2	51	1	AF Dig	7	18	-	Yes	Digoxin Diuretic					150 250 350	SR

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY		ECG	SINUS RHYTHM MAINTAINED	BSA
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR%	LA		IVS	m^2
		Yes	10	28	260	54	2	PR 0.24 Dig LV+	2 mo	1.86
V.E.			8	26	200	67	3	No change		1.47
V.E.		Yes	2	30	390	56	2	PR 0.20 Dig RBBB	2 wk	1.74
V.E.			12	30	360	52	1	No change		1.51
Readycard.			4	28	200	44	N	PR 0.16 Dig	2 d	2.13
V.E.			8	31	250	71	2	No change		1.56
At. po.			6	19	380	50	1	PR 0.16 Dig	4 d	1.71
			57	180	470	57	1	PR 0.24 RBBB Ant.inf.	3 mo Death in S.R	1.52
			18	-	>2000	53	N	No change		1.86
		Yes	2	37	420	43	N	PR 0.16 Inc RBBB	Still present	1.74
		Yes	5	32	250	43	1	PR 0.16	Still present	1.62
V.E.	Pulm oedema Hypotension		5	30	240	54	1	PR 0.28 Dig	4 mo	1.78

TABLE 1 (continued 6)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug, Dose,	DRUG REVERSION RESULT	PHEN. QUIN.	SYNCHRONISED SHOCK ENERGY joules	SYNCHRONISED SHOCK RESULT
69	51 F	AF	6 yr	Rh. HD. Aortic & mitral valvot AI Gr.2, MI Gr.2	66	3	AF Dig LV+	14	20	230	Yes	Digoxin Diuretic	Q.	7.0Gr.	Fail	Yes	150	SR
70	64 F	A TACHY	3 mo	Lone	50	N	A Tachy 2:1	18	33	420	Yes	Diuretic	Q. Digoxin	3.6Gr. 1.5mg	Fail		50	SR
71	55 F	AF	2 yr	Rh. HD. Mitral valvot MI Gr. 1	59	1	AF Dig	25	20	200	Yes	Digoxin	Q.	1.8Gr.	Fail	Yes	150 250	SR
72	51 M	VT	1 d	Chr.Isch HD	50	2	VT	8	30	220	No	Digoxin	Q. Propranolol	3.6Gr. 5mg.	Fail		50	SR
73	34 M	AF	3 yr	Rh. HD. Mitral valvot MI Gr.1, AI Gr.1	45	1	AF Dig	5	23	260	Yes	Digoxin	Q.	4.0Gr.	Fail	Yes	50 150 250	SR
74	33 F	AF	6 yr	Rh. HD Mitral valvot MI Gr. 1	57	2	AF Dig	3	25	260	Yes	Digoxin	Q.	8.4Gr.	Fail	Yes	50 150 250 350	Fail
75	64 F	AF	2 yr	Post thyrotoxic	55	1	AF Dig	4	29	280	Yes	Digoxin					150	SR
76	56 M	AF	14 yr	Alc.cardiomy.	51	1	AF Dig	6	35	180	Yes	Digoxin	Q.	3.6Gr.	Fail		150 250 350 400	SR
77	14 F	A FLUT	3 mo	Post op. ASD	51	N	3:1 RBBB	3	12	110	No	Digoxin	Digoxin		Fail Nausea		150	SR
78	48 M	AF	20 d	Rh. HD Mitral valvot MI Gr. 1	49	2	AF Dig Inc RBBB	39	15	100	No	Digoxin	Q.	2.2Gr.	Fail		150	SR
79	41 M	AF	10 yr	Rh. HD Mitral valvot MI Gr.1, PVR+	49	1	AF Dig RV+ RBBB	4	38	200	Yes	Digoxin Diuretic					150 250 350 400	SR

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY		ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR%	LA			
		Yes	18	21	320	66	3	PR 0.20 LV+	2 mo	1.47
At. pc.		Yes	10	30	390	50	N	PR 0.16 Dig Inc RBBB	1 d → A tachy → SR spont	1.87
		Yes	20	23	189	59	1	PR 0.16	Still present	1.64
			10	22	180	49	2	PR 0.16 Inf.inf. Ant.isch.	3 hr	1.87
		Yes	6	33	260	45	1	PR 0.18 Dig	Still present	1.68
VF	Hypotension		5	32	170	57	2	No change		1.64
At. pc.			3	28	260	55	1	PR 0.20 P mitrale	Still present	1.54
Wand pacem. At. pc.			4	30	160	52	1	PR 0.22 Dig	1 hr	1.77
			3	16	120	51	N	PR 0.14 RBBB	Still present	1.49
At. pc.			40	14	80	49	2	PR 0.20 Dig Inc RBBB	Still present	1.65
A.Flut. V.E. Bradyc. Wand pacem.			6	38	200	49	1	PR 0.26 Dig RBBB RV+	1.5 hr	1.70

TABLE 1 (continued 7)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH
80	39 F	VT	10 d	Cardiomyop.	50	N	VT	15	41	400
81	49 M	A FLUT	1 yr	ASD Post op.	52	1	3:1 4:1 Inc RBBB Dig	29	17	-
82	60 F	AF	2 yr	Rh. HD Mitral valvot MI-2; AI-1	56	1	AF Dig Inc RBBB LV+	5	22	160
83	46 M	AF	7 mo	Rh. HD Mitral valvot MI-0	55	2	AF Dig	13	24	110
84	49 F	AF	3 wk	Rh. HD Mitral valvot MI-1	59	2	AF Dig	20	23	100
85	56 M	AF	6 wk	Alc.cardiomyop.	49	1	AF Dig	26	23	280
86	42 F	AF	5 mo	Rh. HD Mitral valvot MI-0	58	2	AF Dig	8	20	250
87	46 F	AF	2 yr	Rh. HD Mitral valvot MI-0	63	2	AF Dig Inc RBBB	14	35	240
88	51 M	AF	24 d	Rh. HD Mitral valvot MI-1	47	1	AF Dig Inc RBBB	51	18	180
89	34 M	A FLUT	6 yr	ASD post op.	52	N	3:1 Inc RBBB Dig	20	16	180
90	36 F	AF	2 mo	Rh. HD AOVR, Post op. MI-Gr.2,AI-Gr.2	68	2	AF Dig LV+	20	21	400

ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug,	Dose,	RESULT	SYNCHRONISED PREC. QUIN.	ENERGY joules	RESULT
Yes	Digoxin Diuretic	Q. Proc.amid.	1.8Gr. 2.0Gr.	Fail Nausea		150	SR
Yes	Digoxin	Q. Digoxin	2.8Gr. 2.2mg.	Fail		150	SR
Yes	Digoxin Diuretic	Q.	2.4Gr.	Fail Nausea		150	SR
Yes	Digoxin					150	SR
Yes	Digoxin	Q.	2.7Gr.	Fail		150 250 350	SR
No	Digoxin Diuretic					150	SR
Yes	Digoxin	Q.	2.7Gr.	Nausea Fail		150	SR
No	Digoxin	Q.	1.8Gr.	Fail Nausea		150	SR
Yes	Digoxin	Q. Digoxin Proc.amide Prestigmine	3.6Gr. 2.0mg. 2.0Gr.	Fail		150	SR
No	Digoxin Diuretic	Q.	3.3Gr.	Fail		150	SR

CAPACITOR DISCHARGE INTERVENING ARRYTHES	COMPLICATIONS	QUIN AFTER	ESR	ENZYME SGOT	ENZYME LDH	X-RAY CTR%	X-RAY LA	ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
	Pulm oedema Hypotension Enzyme + Tinvevted	-	12	86	860	51	N	PR 0.18 T ↓ V_{4-6}	Still present	1.86
			30	20		52	1	PR 0.16 Inc RBBB	Still present	1.92
At. pc Wand pace			8	29	140	56	1	PR 0.16 Inc RBBB Dig LV+	Still present	1.46
		Yes	16	18	80	54	2	PR 0.18 Dig	Still present	1.94
At. pc VE			18	26	120	59	2	PR 0.20 Dig	Still present	1.82
Bradyc. At. pc Wand pacem.			15	38	300	49	1	PR 0.24 Dig	Still present	2.12
VE			12	17	270	58	2	PR 0.18 Dig	Still present	1.63
At. pc			12	25	200	63	2	PR 0.26 Dig P mitrale	1 mo	1.77
			63	17	160	47	1	PR 0.24 Dig Inc RBBB	7 mo	1.80
Nodal			24	33	190	52	N	PR 0.20 Inc RBBB Dig	Still present	2.10
Nodal VE	Cer. emb.		18	30	390	63	2	PR 0.18 Dig LV+	6 wk Death in AF 6 mo	1.63

TABLE 1 (continued 8)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR	LA	ECG	ESR mm	ENZYME SGOT	units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug, Dose,	RESULT	SYNCHRONISED SHOCK FREQ. QUIN.	ENERGY joules	RESULT
91	61 M	AF	4 mo	Lone	49	N	AF Dig RBBB	2	40	300	Yes	Digoxin	Q 3.6Gr.	Fail		150 250	SR
92	44 M	AF	3 mo	Cardiomyop.	55	1	AF Dig LV+	6	26	180	Yes	Digoxin				150	SR
93	36 F	AF	11 mo	ASD Post op.;P:S=2	62	1	AF Dig Inc RBBB	3	24	180	Yes	Digoxin	Q. 2.2Gr.	Fail Syncope		150 250	SR
94	38 F	AF	18 mo	Rh. HD Mitral valvot MI-Gr. 1	52	2	AF Dig	18	21	200	Yes	Digoxin	Q. 1.6Gr. Proc.amide 2.0Gr.	Fail Nausea		150	SR
95	26 M	AF	2 yr	Cardiomyop.	50	1	AF Dig	2	26	350	Yes	Digoxin	Q. 3.6Gr.	Fail Nausea		150	SR
96	50 M	AF	1 yr	Rh. HD Mitral valvot MI-Gr. 1	56	2	AF LV+ Dig	14	16	280	Yes	Digoxin Diuretic	Q. 2.0Gr.	Fail		50 150	SR
97	57 M	AF	10 mo	Lone	47	N	AF Dig	3	40	160	No	Digoxin				50 150 250 400	SB 20 sec Fail
98	41 F	AF	14 mo	Rh. HD. Mitral valvot MI-O	57	2	AF Dig	5	20	300	Yes	Digoxin	Q. 3.6Gr.	Fail		50 150	SB
99	44 M	AF	14 yr	Post thyrotox. Familial AF	46	N	AF Dig	2	21	220	No	Digitalis	Q. 2.2Gr.	Fail Nausea		50 150 250 400	Fail
100	56 M	AF	6 mo	Cardiomyop.	50	1	AF Dig	10	25	420	Yes	Digoxin	Q. 3.6Gr.	Fail		150	SR
101	16 F	A TACHY	2 wk	VSD. PHT. Post op.	66	1	2:1	15	61	360	No	Lanat C. Diuretic	Pronethalol	Fail		50	SB
102	49 M	AF	3.5 yr	IHD	46	1	AF Dig Isch.	5	19	230	Yes	Digoxin				150 250 350	SR

CAPACITOR DISCHARGE INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER	ESR	ENZYMS SGOT	LDH	X-RAY CTR%	LA	ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
VE		Yes	4	42	470	49	N	PR 0.16 Dig RBBB	1 mo	1.92
VE			6	30	370	55	1	PR 0.16 Dig LV+	4 mo	2.20
VE			3	17	160	62	1	PR 0.16 Inc RBBB Dig	6 d	1.58
VE			14	25	300	52	2	PR 0.20 Dig	Still present	1.62
At. po			2	29	130	52	1	PR 0.14 Dig	?	1.88
At. po			12	18	300	53	2	PR 0.16 Dig LV+	8 mo	1.81
			3	30	180	47	N	No change		2.10
			10	26	280	57	2	PR 0.26 Dig	1 mo	1.72
			2	30	210	46	N	No change		2.07
			10	27	380	50	1	PR 0.16 P mitrale Dig	1 mo	1.78
			14	66	400	66	1	PR 0.12 Dig LV+,RV+	Died in Hx, 1 wk	1.12
VE	Pulm oedema		5	35	270	50	1	PR 0.12 Dig Isch	Still present	2.14

TABLE 1 (continued 9)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-DIG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug, Dose,	DRUG REVERSION RESULT	PHEC. QUIN.	SYNCHRONIS ENERGY joules	SYNCHRONIS RESULT
103	60 M	AF	5 mo	Rh. HD MI-Gr. 2 MS-Gr. 2	52	2	AF Dig	12	24	290	Yes	Digoxin				150 250	SR
104	31 F	AF	4 yr	Rh. HD. Mitral valvot MI-Gr.1; AI-1	42	N	AF Dig Inc BBBB	10	39	360	Yes	Digoxin Diuretic				150	SR
105	62 M	AF	1.5 yr	Lone	42	N	AF Dig Inc RBBB	10	39	360	Yes	Digoxin Diuretic				150 250	SR
106	53 M	A FLUT	7 mo	ASD Post op.	57	N	2:1,3:1, 4:1 Inc RBBB	13	22	570	Yes	Digoxin	Digoxin Pronethalol	Fail Fail		150	SR
107	8 M	A FLUT	3 yr	ASD Post op.	54	1	2:1,3:1 Inc RBBB	10	28	330	No	Digoxin	Q.	2.0Gr. Fail		50 150	SR
108	44 F	AF	11 mo	Rh. HD. Mitral valvot MI-Gr.1,AI-Gr.1	55	2	AF Dig LV+	4	16	300	No	Digoxin	Q.	2.6Gr. Fail		150	SR
109	19 M	A TACHY	9 d	ASD Post op.	43	N	2:1 Inc RBBB	2	37	210	No	Digoxin	Q. Digoxin	3.6Gr. Fail 2.2mg Nausea		150	SR
110	34 M	AF	1 yr	Rh. HD. Mitral valvot MI-1	52	1	AF Dig	2	23	250	Yes	Digoxin				150	SR
111	44 F	AF	2 mo	Rh. HD. Mitral valvot MI-Gr.1	65	2	AF Dig	3	57	290	Yes	Digoxin	Q.	6.3Gr. Fail Nausea		150 250	SR
112	55 M	AF	3 mo	Rh. HD. Mitral valvot MI-0	52	2	AF Inc RBBB Dig	17	30	210	Yes	Digoxin				150 250	SR
113	2.5 M	A FLUT	2 wk	ASD Post op.	56	N	2:1 Dig	-	-	-	No	Digoxin	Digoxin	Fail		10	SR
114	54 M	AF	2 yr	Lone	44	N	AF Dig	10	35	150	Yes	Digoxin				150 250 350 400	SR 5 beats Fail

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY		ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR	LA			
VF At. pc			10	26	300	52	2	PR 0.18 Dig	6 d	1.75
Nodal			8	43	430	50	1	PR 0.16 Dig	Still present	1.56
VE			10	42	270	42	N	PR 0.20 Dig Inc RBBB	1 wo	2.10
		Yes	12	39	280	57	N	PR 0.16 Inc RBBB	Still present	1.84
Prolonged PR At. pc. VE			10	31	250	54	1	PR 0.24 Inc RBBB	8 wo	1.06
At. pc			4	17	380	55	2	PR 0.16 Dig LV+	5 wo	1.62
Nodal Wand pacem		Yes	4	42	470	46	N	PR 0.18 Dig	Still present	1.72
			2	37	380	52	1	PR 0.16 Dig	6 wo	1.88
		Yes	2	40	270	58	2	PR 0.16 Dig	Still present	1.76
Nodal Bradyc.			17	31	260	46	2	PR 0.24 Inc RBBB	Still present	1.83
Nodal						56	N	PR 0.12 Dig Inc RBBB	Still present	
			6	19	300	42	N	No change		1.84

TABLE 1 (continued 10)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYME units SGOT	ENZYME units LDH	ANTI-ARRH. Yes/no	OTHER DRUGS	DRUG REVERSION Drug, Dose,	DRUG REVERSION RESULT	ARRH. QUIN.	SYNCHRONISED SHOCK ENERGY joules	SYNCHRONISED SHOCK RESULT
115	52 M	AF	3 yr	Rh. HD. Mitral valvot MI-0	52	2	AF Dig	7	19	100	Yes	Digoxin				150	SR
116	60 M	AF	2 yr	Rh. HD AOVD AI-Gr.2;MI-1	41	1	AF Dig LV+	3	19	290	Yes	Digoxin Diuretic				150	SR
117	52 M	AF	5 mo	Rh. HD. AS. AI Gr. 3	57	1	AF Dig RBBB LV+	10	23	180	Yes	Digoxin Diuretic				150	SR
118	63 M	AF	18 mo	Ischaemic HD	53	1	AF Dig Isch	2	2	34	Yes	Digoxin				150 250 350 400	Fail
119	41 F	AF	1 yr	Rh. HD Mitral valvot MI-Gr.2	55	2	AF Dig	20	19	130	No	Digoxin				150	SR
120	60 M	AF	5 mo	Lone	41	N	AF Dig	5	20	-	Yes	Digoxin				150	SR
121	25 F	AF	4 wk	Rh. HD. ACVD Post op.	58	1	AF Dig LV+	6	21	430	Yes	Digoxin				150	SR
122	45 F	AF	3 wk	Rh. HD. Mitral valvot MI-Gr. 1	52	1	AF Dig	3	19	320	No	Digoxin				150	SR
123	44 M	AF	1 yr	Rh. HD. Mitral valvot MI-Gr.2	61	2	AF Dig	2	18	190	Yes	Digoxin				150 250 350	SR
124	62 F	AF	5 yr	Rh. HD. Mitral valvot MI-0	52	2	AF Dig	7	41	680	Yes	Digoxin Diuretic				150 250	SR
125	31 F	AF	9 yr	Rh. HD. Mitral valvot (X2) MI-Gr.1	74	3	AF Dig LV+	40	20	340	Yes	Digoxin Diuretic				150 250 350	Fail
126	155 F	A TACHY	3 hr	WPW AOVD(mild)	49	N	1:1	41	10	220	No					50 100	SR

CAPACITOR DISCHARGE INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER	ESR	ENZYME SGOT	ENZYME LDH	X-RAY CTR%	X-RAY LA	ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
			7	14	210	52	2	PR 0.20 Dig	1 hr	1.73
At. pc			5	14	280	43	1	PR 0.24 Dig LV+	48 hr	1.60
At. pc VE			10	25	190	58	1	PR 0.22 Dig RBBB LV+	2 min	1.72
			4	39	430	53	1	No change		2.10
At. pc			21	33	290	55	2	PR 0.20 P mitrale Dig	Still present	1.53
			4	41	290	41	2	PR 0.20 Dig	5 min	1.88
			18	35	520	58	1	PR 0.20 Dig LV+	Still present	1.80
			3	19	250	52	1	PR 0.14 Dig	Still present	1.42
VE At. pc Prolonged PR	Enzyme +		2	40	680	58	2	PR 0.24	24 hr	2.02
			8	34	450	52	2	PR 0.24 Dig P mitrale	Still present	1.74
VE			40	21	340	74	3	No change		1.56
			36	8	300	43	2	WPW	Still present	1.69

TABLE 1 (continued 11)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ENZYME units SGOT	ENZYME units LDH	ANTI-COAG. Yes/NO	OTHER DRUGS	DRUG REVERSION Drug,	Dose,	RESULT	PREC. QUIN.	SYNCHRONISH ENERGY joules	SYNCHRONISH RESULT
127	65 M	AF	6 wk	Ischaemic HD	43	N	AF Dig Isch	30	420	Yes	Digoxin	Q.	5.6Gr.	Fail		150 250	SR
128	51 M	AF	7 d	Rh. HD. AOVD, post op. Mitr. valvot	64	2	AF Dig LV+	40	480	No	Digoxin					25 50 100	SR
129	59 M	A TACHY	3 d	Ant.Myoc.inf.	57	2	1:1 Ant.inf.	43	600	No	Digoxin	Q. Proc.amide Prostigmine Digoxin	1.5Gr. 4.0Gr.	Fail	Yes	50	SR
130	60 M	AF	6 mo	Cardiomyop.	49	1	AF Dig	26	320	Yes	Digoxin Diuretic					150 250	SR
131	47 F	AF	3 mo	Rh. HD. Mitral valvot MS-Gr.1	47	1	AF Dig	40	1050	Yes	Digoxin Diuretic					150	SR
132	65 F	AF	6 mo	Lone	52	N	AF Dig	35	420	Yes	Digoxin	Q	3.6Gr.	Fail		50 100 200 350	SR
133	61 F	AF	1 yr	Rh. HD. Mitral valvot MI-Gr.1	59	2	AF Dig	18	200	Yes						50 100 200	SR
134	58 M	VT	2 d	Inf.myoc.inf.	64	2	VT	180	740	Yes	Digoxin	Pronethalol		Fail Collapse		50 50	VF SR
135	60 M	AF	5 mo	Lone	51	N	AF Dig	47	280	Yes	Digitalis	Q.	2.8Gr.	Fail		50 150	SR
136	38 M	AF	3 mo	Post thyrotox.	47	1	AF Dig	40	320	Yes	Digoxin					50 100 200	SR
137	53 F	AF	4 mo	Rh. HD. Mitral valvot MI-Gr.1.	59	1	AF Dig	35	450	No	Digoxin					50	SR
138	62 M	AF	7 mo	Ischaemic HD	48	N	AF Dig Old inf. inf.	32	170	Yes	Digoxin Diuretic					50 150 250 400	SR

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY		ECG	SINUS RHYTHM MAINTAINED FOR	BSA m^2
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR%	LA			
VE			4	10	300	43	N	PR 0.18 Dig Inch	Still present	1.96
		Yes	38	43	460	64	2	PR 0.20 Dig LV+	2 hr	1.86
	Saddle embolus	Yes	40	55	620	56	2	PR 0.18 Ant. inf.	2 d	1.60
			5	28	360	49	1	PR 0.20 Dig	Still present	2.00
			40	42	1020	47	1	PR 0.20 Dig	Still present	1.61
VE			16	41	400	51	N	PR 0.18 Dig	1.5 hr	1.70
At. pc			3	16	180	59	2	PR 0.20 Dig	1 mo	1.75
Accel VE	Ac.LVF		56	200	760	64	2	PR 0.20 Inf. Inf. Inch.	2 d Death in SR	1.78
			10	30	210	51	N	PR 0.20 Dig	Still present	1.80
			2	32	320	47	1	PR 0.18 Dig	Still present	1.91
Sand pace At. pc			13	35	500	59	1	PR 0.14 Dig	6 wk	1.36
VE	Pulm oedema Enzyme + V.E.	Yes	13	35	500	53	1	PR 0.20 VE	Still present	1.90

TABLE 1 (continued 12)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR-LA		ECG	RSR mm	ENERGY units SCOT	ENERGY units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug, Dose,		RESULT	SYNCHRONISED PREC. QUIN.	SYNCHRONISED ENERGY joules	SYNCHRONISED RESTORE
139	55 M	AF	1 yr	Lone	56	1	AF Dig	28	54	500	No	Digoxin					50 150 250	NR
140	55 F	AF	9 mo	Rh. HD. Mitral valvot MI-Gr.1	58	2	AF Dig	7	25	320	Yes	Digoxin					50 100 150 250 400	SR 5 beats SR
141	44 F	AF	7 mo	Rh. HD. Mitral valvot MI-Gr.1	56	1	AF	27	35	500	Yes	Digoxin					50 100 150 200	SR
142	55 F	AF	5 yr	Ischaemic HD	48	N	AF Dig Isch.	2	14	200	Yes	Digoxin					50 100 150 200	SR
143	64 M	A TACHY	11 d	IHD Digitalis	56	1	2:1 Dig	9	220	750	Yes	Digoxin	Digoxin		Fail		25 50	SR
144	28 M	AF	6 wk	Lone	43	N	AF	1	24	390	No	Digoxin					50 100 200 300 400	Fail
145	51 M	VT	3 d	Ischaemic HD Old inf.inf.	58	2	VT	4	134	-	No	Digoxin	Digoxin Proc.amide	1.5mg. 5.00	Fail		150	SR
146	52 F	AF	2 wk	Obstr.cardiomy.	54	1	AF Dig LV+	6	36	220	No	Digoxin					50 100 200	SR
147	42 M	AF	2 mo	Rh. HD Mitr, valvot MI-0	52	1	AF Dig	26	32	280	No	Digoxin					150 250	SR
148	47 M	AF	3 wk	Rh. HD. Mitral valvot MI-0	48	1	AF Dig	27	27	500	No	Digoxin					50 100	SR

CAPACITOR DISCHARGE			ESR	ENZYME		X-RAY			SINUS RHYTHM MAINTAINED FOR	BSA m^2
INTERVENING FACTORS	COMPLICATIONS	QUIN AFTER		SGOT	LDH	CTR%	LA	ECG		
Prolonged IR		Yes	36	46	470	53	1	PR 0.16 Dig	Still present	1.98
			9	33	280	58	2	PR 0.16	Still present	1.64
			39	40	560	56	1	PR 0.16 Dig	Still present	1.57
	Pulm oedema		2	20	300	54	1	PR 0.16 Dig	6 hr	1.63
At. pc VS		Yes	12	111	500	56	1	PR 0.18 Old inf. Dig	4 hr	1.54
VS			2	40	450	43	N	No change		2.37
AS		Yes	4	57	-	52	.2	PR 0.20 Old inf. inf.	Still present	1.77
At. pc			5	38	210	50	1	PR 0.20 LV+	3 wk	1.46
			18	35	295	52	1	PR 0.18 Dig	Still present	1.92
			20	31	280	48	1	PR 0.20 M mitrale Dig	Still present	1.67

TABLE 1 (continued 13)

NO.	AGE SEX	RHYTHM	DURATION	UNDERLYING HEART DISEASE	X-RAY CTR %	X-RAY LA	ECG	ESR mm	ENZYMES units SGOT	ENZYMES units LDH	ANTI-COAG. Yes/No	OTHER DRUGS	DRUG REVERSION Drug, Dose,	DRUG REVERSION RESULT	FREQ. AQIN.	SYNCHRONISED ENERGY joules	SYNCHRONISED RESULT
149	56 M	A FLUT	1 yr	Cardiomyop.	52	1	3:1,5:1	20	23	280	No	Digoxin				50	SR
150	49 M	AF	1 yr	Rh. HD, Post op. AOVD	49	1	AF Dig LV+	7	73	390	No	Digoxin				50 100 200 300	SR

Table 1: Clinical details and treatment in 150 patients. Energy delivered with antero-posterior paddles underlined.

Note: Abbreviations used in this and subsequent tables are listed in Appendix D.

CAPACITOR DISCHARGE											
INTERVENING RHYTHMS	COMPLICATIONS	QUIN AFTER	ESR	ENZYME SGOT	ENZYME LDH	X-RAY CTR%	X-RAY LA-		ECG	SINUS RHYTHM MAINTAINED PER	BSA m^2
VS		Yes	50	20	260	52	1	PR	0.18	Still present	1.55
Bradyc.			5	68	350	50	1	PR LV+ Dig	0.20	Still present	1.82

1. Where the arrhythmia was ventricular fibrillation and an unsynchronised direct current shock was used as part of the management of cardiac arrest.

2. Where synchronised direct current shock was used in the treatment of a ventricular or supraventricular arrhythmia arising in the immediate post-operative phase following cardio-pulmonary by-pass or closed mitral valvotomy.

It was felt that the problems presented in the management of cardiac arrest on the one hand, and in the immediate post operative period following major heart surgery on the other, could in no way be equated to the planned version of cardiac arrhythmias as typified by the 150 patients on which this study is based.

I. DRUG RESISTANT ARRHYTHMIAS - Patient Numbers 1 - 50.
Tables 1 and 2.

The first 50 patients were treated initially with drugs in full therapeutic doses, but all failed to come into sinus rhythm. In this group there were 40 patients who had atrial fibrillation, 8 who had atrial flutter and 2 who had atrial tachycardia (Table 2). There were 16 patients who had had a mitral valvotomy performed for mitral stenosis, and a further 8 patients had mitral, aortic, or mitral and

TABLE 2

DIAGNOSIS	ATRIAL FIBRILLATION	ATRIAL FLUTTER	ATRIAL TACHYCARDIA	TOTAL
RHEUMATIC HEART DISEASE				
Mitral stenosis, post. op.	14	1	1	16
Mitral regurgitation, post. op.	1	-	-	1
Mitral stenosis	3	-	-	3
Mitral stenosis regurg.	1	-	-	1
Mitral and AOVD	3	-	-	3
Alcoholic cardiomyopathy	4	-	-	4
Lone	10	2	-	12
ASD, post. op.	2	3	-	5
Post thyrotoxicosis	1	-	-	1
Chronic ischaemic HD	1	-	-	1
Acute myoc inf.	-	-	1	1
Systemic hyp, mitral regurg.	-	1	-	1
VSD, Pulm hyp.	-	1	-	1
TOTAL	40	8	2	50

Table 2: 50 patients in whom the arrhythmia was drug resistant.

aortic valve disease upon which an operation had, or had not been performed. In 12 patients no underlying cause for the arrhythmia could be detected. 5 patients had had an atrial septal defect repaired surgically. 4 patients had alcoholic cardiomyopathy. Less frequent associated heart conditions included, treated thyrotoxicosis, chronic ischaemic heart disease, acute myocardial infarction, systemic hypertension with functional mitral regurgitation and ventricular septal defect associated with severe pulmonary hypertension.

II. ANALYSIS OF THE 150 PATIENTS (TOTAL SERIES)

A ATRIAL FIBRILLATION: (120 Patients). Table 3.

(1) Underlying heart disease: 120 patients presented with atrial fibrillation (Table 3). 51 patients - Numbers

1, 4, 7, 8, 14, 15, 19, 22, 23, 25,
26, 33, 36, 37, 52, 54, 57, 58, 60, 71,
73, 74, 78, 79, 82, 83, 84, 86, 87, 88,
94, 96, 98, 104, 108, 110, 111, 112, 115, 119,
122, 123, 124, 125, 131, 133, 137, 140, 141, 147,
148.

had had a mitral valvotomy performed for mitral stenosis. 4 patients (numbers 90, 121, 128 and 150) had had aortic

TABLE 3

DIAGNOSIS	ATRIAL FIBRILLATION	ATRIAL FLUTTER	ATRIAL TACHYCARDIA	VENTRICULAR TACHYCARDIA	TOTAL
RHEUMATIC HEART DISEASE:					
Mitral stenosis, post.op.	51	1	1	-	53
Mitral and AOVD, post op.	1	-	-	-	1
AOVD, post. op.	4	-	-	-	4
Mitral regurg, post. op.	1	-	-	-	1
Mitral stenosis	4	-	-	-	4
Mitral regurgitation	2	-	-	-	2
Mitral stenosis and regurg.	2	-	-	-	2
Mitral and AOVD	6	-	-	-	6
Alcoholic cardiomyopathy	6	-	-	-	6
Cardiomyopathy	4	1	-	1	6
Lone	22	2	1	-	25
ASD, post op.	4	9	1	-	14
Post thyrotoxicosis	4	-	-	-	4
Chronic ischaemic HD	6	-	-	2	8
Acute myoc. inf.	-	-	2	2	4
Systemic hyp. mitral regurg.	1	1	-	-	2
VSD, Pulm hyp.	-	1	1	-	2
VSD, WPW.	-	-	1	-	1
AOVD, WPW.	-	-	1	-	1
Cor transp. "EX"	1	-	-	-	1
Obstr. cardiomyop.	1	-	-	-	1
Dystrophia myotonica	-	1	-	-	1
Digitalis	-	-	1	-	1
TOTAL	129	16	9	5	150

Table 3: The arrhythmia and underlying heart disease in 150 patients.

valve surgery under cardio-pulmonary by-pass. 1 patient (number 69) had had both mitral and aortic valves operated on under cardio-pulmonary by-pass and 1 patient (number 39) had had an open repair of mitral regurgitation caused by repeated subacute bacterial endocarditis on a valve affected by rheumatic fever. There were 4 patients who had mitral stenosis for which no operation had been performed (numbers 35, 41, 43, and 51). In 2 patients (numbers 55 and 59) mitral regurgitation was the dominant lesion. In 2 further patients (numbers 32 and 103) mitral stenosis with regurgitation were present. 6 patients (numbers 17, 24, 46, 56, 116 and 117) had mitral stenosis associated with rheumatic aortic valve disease. Rheumatic heart disease was associated therefore in 71 out of the 120 patients with atrial fibrillation, of whom 57 had had the natural history of their disease altered by surgery.

The remaining associated heart conditions in the patients with atrial fibrillation were as follows:- 10 patients had cardiomyopathy (numbers 2, 10, 16, 21, 76, 85, 92, 95, 100 and 130) by which is meant enlargement of the heart unassociated with valvular disease, coronary artery disease, cor pulmonale, idiopathic pulmonary hypertension, septal defects or systemic hypertension. Of these, the

first 6 (numbers 2, 10, 16, 21, 76 and 85) were thought to be due to excessive intake of alcohol over a prolonged period. None of these patients had clinical evidence of obstruction to the outflow of the left or right ventricle.

In 22 patients (numbers 3, 5, 12, 13, 27, 34, 40, 44, 45, 48, 61, 63, 67, 91, 97, 105, 114, 120, 132, 135, 139 and 144) no underlying cause for the arrhythmia was detected and these patients have been labelled "lone" (Evans and Swann, 1954), (Table 3).

4 patients (numbers 9, 29, 62 and 93) had been operated on for closure of an ostium secundum atrial septal defect.

4 patients (numbers 20, 75, 99 and 136) had been successfully treated for thyrotoxicosis of whom 3 (numbers 20, 75 and 136) had had a partial thyroidectomy; patient number 99 had been treated medically. No patient had clinical or laboratory evidence of thyrotoxicosis still being present.

In 6 patients (numbers 38, 102, 118, 127, 138, 142) a diagnosis of chronic ischaemic heart disease was made. None of these patients showed electrocardiographic evidence of recent myocardial infarction.

1 patient (number 53) had systemic hypertension associated with mitral regurgitation.

1 patient (number 68) had corrected transposition associated with regurgitation of the systemic atrio-ventricular valve.

1 patient (number 146) had obstructive cardiomyopathy of the left ventricle (sub valve stenosis).

(ii) Age-Range and Sex Incidence: Table 4.

The age range varied from 21 years to 65 years (see Table 4). The majority of patients (73) were between 41 and 60 years old.

There were 70 male and 50 female patients.

(iii) Duration of the Arrhythmia: Table 5.

In 82 patients (see Table 5) atrial fibrillation had been present in periods varying from 1 month to 3 years. The shortest period in which atrial fibrillation had been present before treatment was 7 days (patient number 128); the longest, 17 years (patient number 13, with "lone" atrial fibrillation).

TABLE 4

DIAGNOSIS	21-30 M	21-30 F	31-40 M	31-40 F	41-50 M	41-50 F	51-60 M	51-60 F	61-70 M	61-70 F
RHEUMATIC HEART DISEASE:										
Mitral stenosis, post op.	1	-	2	6	12	17	4	7	-	2
Mitral and AOVD, post op.	-	-	-	-	-	-	1	-	-	-
AOVD, post op.	-	1	1	1	1	-	-	-	-	-
Mitral regurg, post op.	-	1	-	-	-	-	-	-	-	-
Mitral stenosis	-	-	-	-	3	-	1	-	-	-
Mitral regurgitation	-	-	-	-	-	-	-	2	-	-
Mitral stenosis and regurg.	-	-	-	-	-	-	1	1	-	-
Mitral and AOVD	-	-	2	1	-	1	2	-	-	-
Alcoholic cardiomyopathy	-	-	-	-	1	1	4	-	-	-
Cardiomyopathy	1	-	-	-	1	-	2	-	-	-
Lone	2	1	1	-	3	1	9	2	2	1
ASD, post op.	-	-	-	1	1	-	1	1	-	-
Post thyrotoxicosis	-	-	1	-	1	1	-	-	1	-
Chronic ischaemic HD	-	-	-	-	1	-	2	-	3	-
Syst. hyp , mitral regurg.	-	-	-	-	1	-	-	-	-	-
Cor. Transp. "MI"	-	-	1	-	-	-	-	-	-	-
Obstr. cardiomyop.	-	-	-	-	-	-	-	1	-	-
TOTAL	4	3	8	9	25	21	27	14	6	3

Table 4: The age range in years and sex incidence in 120 patients with atrial fibrillation.

TABLE 5

DIAGNOSIS	1-7d	1wk-1mo	1-3mo	3-6mo	6mo-1yr	1-3yr	3-5yr	5-10yr	≥ 10yr
RHEUMATIC HEART DISEASE:									
Mitral stenosis, post op.	-	5	4	6	12	9	4	9	2
Mitral and AOVD, post op.	-	-	-	-	-	-	-	1	-
AOVD, post op.	1	1	1	-	1	-	-	-	-
Mitral regurg., post op.	-	-	-	-	-	-	1	-	-
Mitral stenosis	-	-	-	2	-	2	-	-	-
Mitral regurgitation	-	-	-	1	1	-	-	-	-
Mitral stenosis and regurg.	-	-	-	1	-	1	-	-	-
Mitral and AOVD	-	-	3	1	1	1	-	-	-
Alcoholic cardiomyopathy	-	-	2	-	-	2	1	-	1
Cardiomyopathy	-	-	1	2	-	1	-	-	-
Lone	-	1	4	5	3	4	2	-	3
ASD, post op.	-	-	1	-	1	-	-	2	-
Post thyrotoxicosis	-	-	1	-	-	2	-	-	1
Chronic ischaemic HD.	-	-	1	-	2	1	2	-	-
Cyst. hyp , mitral regurg.	-	-	-	-	1	-	-	-	-
Cor. Transp. "MI".	-	-	1	-	-	-	-	-	-
Obstr. Cardiomyop.	-	1	-	-	-	-	-	-	-
TOTAL	1	8	19	18	22	23	10	12	7

Table 5: The duration of atrial fibrillation before synchronised capacitor discharge in 120 patients.

(iv) Electrocardiogram: Table 6.

Normal (other than the arrhythmia - 2 patients (numbers 3 & 144)

Digitalis effect - was noted in 118 of the 120 patients (Table 6).

Left ventricular hypertrophy - was present in 29 patients.

Right ventricular hypertrophy - was noted in 6 patients, all of whom had rheumatic heart disease.

Biventricular hypertrophy - was present in patients numbers 36, 39, 46 and 56.

Myocardial infarction - patient number 38 showed evidence of an old anterior myocardial infarct and patient number 138 an old inferior myocardial infarct. In addition, patient number 38 had right bundle branch block.

Ischaemic RS-T segments and T wave change - were noted in 4 patients.

Right bundle branch block - was present in 4 patients (numbers 79, 59, 117 and 38) and delayed activation over the surface of the right ventricle not amounting to right bundle branch block was present in 10.

(v) Heart Size: Table 7.

(a) Cardiothoracic Ratio

91 patients had a cardiothoracic ratio between 45 and 59% (Table 7).

18 patients had a cardiothoracic ratio of more than 60% of whom all except three had underlying rheumatic heart disease.

TABLE 6

DIAGNOSIS	DIGITALIS	LVH	RVH	MYOC. INF.	ISCH. SEG.	RBBB	Inc. RBBB
RHEUMATIC HEART DISEASE							
Mitral stenosis, post op.	51	11	3	-	-	1	6
Mitral and AOVD, post op.	1	1	-	-	-	-	-
AOVD, post op.	4	4	-	-	-	-	-
Mitral regurg., post op.	1	1	1	-	-	-	-
Mitral stenosis	4	1	-	-	-	-	-
Mitral regurgitation	2	1	-	-	-	1	-
Mitral stenosis and regurg.	2	-	-	-	-	-	-
Mitral and AOVD	6	6	2	-	-	1	-
Alcoholic cardiomyopathy	6	1	-	-	-	-	-
Cardiomyopathy	4	1	-	-	-	-	-
Lone	20	-	-	-	-	-	1
ASD, post op.	4	-	-	-	-	-	3
Post thyrotoxicosis	4	-	-	-	-	-	-
Chronic ischaemic HD	6	-	-	2	4	1	-
Syst. hyp , mitral regurg.	1	1	-	-	-	-	-
Cor. Transp. "MI"	1	-	-	-	-	-	-
Obstr. cardiomyop.	1	1	-	-	-	-	-
TOTAL	118	29	6	2	4	4	10

Table 6: Electrocardiographic changes before synchronised capacitor discharge in 120 patients with atrial fibrillation.

TABLE 7

DIAGNOSIS	CTR %					LEFT ATRIUM			
	40-44	45-49	50-54	55-59	60	N.	1	2	3
RHEUMATIC HEART DISEASE									
Mitral stenosis, post op.	-	9	16	17	9	-	23	26	2
Mitral and AOVD, post op.	-	-	-	-	1	-	-	-	1
AOVD, post op.	-	1	-	1	2	-	2	2	-
Mitral regurg., post op.	-	-	-	1	-	-	-	1	-
Mitral stenosis	1	2	1	-	-	-	3	1	-
Mitral regurgitation	-	-	-	1	1	-	-	2	-
Mitral stenosis and regurg.	-	-	1	1	-	-	-	2	-
Mitral and AOVD	1	-	2	1	2	-	2	4	-
Alcoholic cardiomyopathy	1	3	2	-	-	-	6	-	-
Cardiomyopathy	-	1	2	1	-	-	4	-	-
Lone	7	6	6	3	-	14	8	-	-
ASD, post op.	-	-	1	1	2	2	1	1	-
Post thyrotoxicosis	-	2	1	1	-	2	2	-	-
Chronic ischaemic HD	1	3	1	1	-	3	2	1	-
Syst.hyp , mitral regurg.	-	-	-	-	1	-	-	1	-
Cor. transp. "HI"	-	-	1	-	-	-	-	1	-
Obstr. cardiomyop.	-	-	1	-	-	-	1	-	-
TOTAL	11	27	35	29	18	21	54	42	3

Table 7: The cardiothoracic ratio and size of the left atrium before synchronised capacitor discharge in 120 patients.

The largest cardiothoracic ratio, 71% was in patient number 62, a lady of 56 years, who had had an ostium secundum atrial septal defect operated on in 1956 but in whom only partial closure had been obtained.

(b) Size of the Left Atrium

This was normal in 21 patients, 14 of whom had "lone" atrial fibrillation (Table 7).

96 patients had slight (1+) or moderate (2+) enlargement of the left atrium and 3 patients had severe (3+) enlargement.

B ATRIAL FLUTTER: (16 Patients).

(i) Underlying heart disease: Table 3.

16 patients presented with atrial flutter (table 3). 1 patient (number 11) had had a mitral valvotomy for mitral stenosis and 1 patient (number 149) had a cardiomyopathy the aetiology of which was not apparent.

2 patients (numbers 28 and 49) were labelled as "lone" atrial flutter as no underlying heart disease was detected in either.

The commonest underlying cause of atrial flutter was surgical closure of an atrial septal defect. 9 patients

(numbers 6, 18, 30, 77, 81, 89, 106, 107 and 113) had had an ostium secundum atrial septal defect closed and the operation was followed in all by atrial flutter.

One patient (number 47) had systemic hypertension associated with mitral regurgitation. One patient (number 59) had a ventricular septal defect in association with reactive and hyperkinetic pulmonary hypertension (Wood, 1958). One patient had atrial flutter and dystrophia myotonica.

(ii) Age Range and Sex Incidence: Table 8.

The age range varied from 2½ years to 53 years (Table 8). The ages of 8 of the 16 patients were between the second and fourth decade.

There were 13 male and 3 female patients.

(iii) Duration of the Arrhythmia: Table 9.

The shortest period that atrial flutter had been present was 7 days (patient number 11) and the longest 18 years (patient number 28). In 5 patients (numbers 149, 6, 81, 106 and 47) the arrhythmia was present for periods varying from 6 months to 1 year.

TABLE 8

DIAGNOSIS	1-5		6-10		11-20		21-30		31-40		41-50		51-60	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Mitral stenosis, post op.	-	-	-	-	-	-	-	-	-	1	-	-	-	-
Cardiomyopathy	-	-	-	-	-	-	-	-	1	-	-	-	-	-
Lone	-	-	-	-	-	-	-	-	-	-	-	-	2	-
ASD, post op.	1	-	1	-	-	1	-	1	2	-	2	-	1	-
Syst.hyp. mitral regurg.	-	-	-	-	-	-	-	-	-	-	-	-	1	-
VSD, Pulm hyp.	-	-	-	-	-	-	1	-	-	-	-	-	-	-
Dystrophia myoton.	-	-	-	-	1	-	-	-	-	-	-	-	-	-
TOTAL	1	-	1	-	1	1	1	1	3	1	2	-	4	-

Table 8: The age range in years and sex incidence in 16 patients with atrial flutter.

TABLE 9

DIAGNOSIS	1-7d	8d-1mo	1-3mo	6mo-1yr	1-3yr	5-10yr
Mitral stenosis post op.	1	-	-	1	-	-
Cardiomyopathy	-	-	1	-	-	1
Lone	-	-	-	-	-	-
ASD, post op.	-	1	1	3	3	1
Syst.hyp, mitral regurg.	-	-	-	-	-	-
VSD, Pulm hyp.	-	1	-	1	-	-
Dystrophia myoton	-	-	1	-	-	-
TOTAL	1	2	3	5	3	2

Table 9: The duration of atrial flutter in 16 patients before synchronised capacitor discharge.

(iv) Electrocardiogram: Table 10.

Normal (other than atrial flutter) - 2 patients (numbers 11 and 113).

Digitalis effect - was noted in 9 patients (Table 10).

Left ventricular hypertrophy - was present in two patients, (numbers 47 and 50).

Biventricular hypertrophy - was present in patient number 50.

Right bundle branch block - was present in patient number 77.

Delayed activation over the surface of the right ventricle not amounting to right bundle branch block was present in 8 patients numbers 6, 18, 30, 66, 81, 89, 106 and 107.

(v) Heart Size

(a) Cardiothoracic Ratio Table 11.

The cardiothoracic ratio lay between 50 - 54% in 7 patients. 5 of these patients (numbers 18, 77, 81, 89 and 107) had had an ostium secundum atrial septal defect closed surgically.

The largest ratio was 64% (patient number 50) who had a ventricular septal defect and pulmonary hypertension.

(b) Size of the Left Atrium table 11.

This was normal in 7 patients of whom 5 (patients numbers 6, 77, 89, 106 and 113) had had an atrial septal defect closed surgically.

TABLE 10

DIAGNOSIS	DIGITALIS	LVH	RVH	RBBB	Inc. RBBB
Mitral stenosis, post op.	-	-	-	-	-
Cardiomyopathy	1	-	-	-	-
Lone	2	-	-	-	-
ASD, post op.	5	-	-	1	7
Syst. hyp., mitral regurg.	1	1	-	-	-
VSD, Pulm hyp.	-	1	1	-	-
Dystrophia myoton	-	-	-	-	1
TOTAL	9	2	1	1	8

Table 10: Electrocardiographic changes before synchronised capacitor discharge in 16 patients with atrial flutter.

TABLE 11

DIAGNOSIS	CTR%					LEFT ATRIUM			
	40-44	45-49	50-54	55-59	>60	N	1	2	3
Mitral stenosis, post op.	-	1	-	-	-	-	1	-	-
Cardiomyopathy	-	-	1	-	-	-	1	-	-
Leno	1	1	-	-	-	1	1	-	-
ASD, post op.	-	1	5	2	1	5	5	1	-
Cyst. hyp. mitral regurg.	-	-	1	-	-	-	-	1	-
VSD, Pulm hyp.	-	-	-	-	1	-	-	-	1
Dystrophic myoton	-	1	-	-	-	1	-	-	-
TOTAL	1	4	7	2	2	7	6	2	1

Table 11: The cardiothoracic ratio and size of the left atrium before synchronised capacitor discharge in 16 patients with atrial flutter.

In a further 6 patients (numbers 11, 149, 28, 18, 81 and 107) slight (1+) enlargement was present. 2 patients (numbers 30 and 47) had moderate (2+) enlargement of the left atrium and 1 patient who had a ventricular septal defect with pulmonary hypertension (number 50) had severe (3+) enlargement.

C ATRIAL TACHYCARDIA: (9 Patients)

(i) Underlying heart disease: Table 3.

One patient (number 31) had had a mitral valvotomy for mitral stenosis (Table 3).

In one patient (number 70) the atrial tachycardia was unassociated with clinically detectable heart disease and was labelled "lone".

One patient (number 109) had had an ostium secundum atrial septal defect closed surgically.

Two patients (numbers 42 and 129) developed atrial tachycardia following a recent myocardial infarction.

Patient number 101 had a ventricular septal defect associated with pulmonary hypertension. One patient (number 65) had a small ventricular septal defect and the Wolff-Parkinson-White Syndrome. In one patient (number 126)

mild aortic valve disease was associated with the Wolff-Parkinson-White Syndrome.

Digitalis intoxication was the precipitating cause in one patient (number 143).

(ii) Age Range and Sex Incidence: Table 12.

The youngest patient with atrial tachycardia was 15 years (patient number 126).

Two patients (numbers 70 and 143) were 64 years old.

There were 5 male and 4 female patients.

(iii) Duration of the Arrhythmia: Table 13.

In 1 patient (number 126) the arrhythmia was present for 3 hours before synchronised capacitor discharge.

The longest duration of atrial tachycardia before treatment was 7 months (patient number 31) but the majority of patients had the arrhythmia present for periods ranging from 1 week to 1 month (patients numbers 109, 101, 65, 143).

(iv) Electrocardiogram: Table 14.

Digitalis effect - was present in 2 patients (numbers 31 and 143).

Left ventricular hypertrophy - was noted on the electrocardiogram of patient number 101.

TABLE 12

DIAGNOSIS	11-20		21-30		41-50		51-60		61-70	
	M	F	M	F	M	F	M	F	M	F
Mitral stenosis, post op.	-	-	-	-	-	-	-	1	-	-
Lone	-	-	-	-	-	-	-	-	-	1
ASD, post op.	1	-	-	-	-	-	-	-	-	-
Acute myoc. inf.	-	-	-	-	1	-	1	-	-	-
VSD, Pulm hyp.	-	1	-	-	-	-	-	-	-	-
VSD, WPW	-	-	1	-	-	-	-	-	-	-
AOVD, WPW	-	1	-	-	-	-	-	-	-	-
Digitalis	-	-	-	-	-	-	-	-	1	-
TOTAL	1	2	1	-	1	-	1	1	1	1

Table 12: The age range in years and sex incidence in 9 patients with atrial tachycardia.

TABLE 13

DIAGNOSIS	<1d	1-7d	1wk-1mo	1-3mo	6mo-1yr
Mitral stenosis, post op.	-	-	-	-	1
Lone	-	-	-	1	-
ASD, post op.	-	-	1	-	-
Acute myoc. inf.	-	2	-	-	-
VSD, Pulm hyp.	-	-	1	-	-
VSD, WPW	-	-	1	-	-
AOVD, WPW	1	-	-	-	-
Digitalis	-	-	1	-	-
TOTAL	1	2	4	1	1

Table 13: The duration of atrial tachycardia before synchronised capacitor discharge in 9 patients.

TABLE 14

DIAGNOSIS	DIGITALIS	LVH	MYOC.INF.	LBBB	RBBB	INC.RBBB
Mitral stenosis, post op.	1	-	-	-	-	-
Lone	-	-	-	1	-	-
ASD, post op.	-	-	-	-	-	1
Acute myoc. inf.	-	-	2	-	1	-
VSD, Pulm hyp.	-	1	-	-	-	-
VSD, WPW	-	-	-	-	-	1
AOVD, WPW	-	-	-	-	-	1
Digitalis	1	-	-	-	-	-
TOTAL	2	1	2	1	1	3

Table 14: Electrocardiographic changes before synchronised capacitor discharge in 9 patients with atrial tachycardia.

Myocardial infarction - evidence of recent myocardial infarction was present in 2 patients (numbers 42 and 129).

Left bundle Branch block - delayed activation over the surface of the left ventricle was present in 1 patient (number 70).

Right bundle Branch block - one patient (number 42) showed this pattern, but in 3 others (patients numbers 109, 65 and 126) delayed activation over the surface of the right ventricle not amounting to complete right bundle branch block was present.

(v) Heart Size:

(a) Cardiothoracic Ratio Table 15.

A cardiothoracic ratio of 50% or more was present in 7 patients.

1 patient had a cardiothoracic ratio of 66% (patient number 101) and 1 patient (number 31), 64%.

(b) Size of the Left Atrium Table 15.

The size of the left atrium was normal in 4 patients (numbers 70, 109, 65 and 126).

Slight enlargement (1+) was present in two patients (numbers 101 and 143) and moderate enlargement (2+) in three patients (numbers 31, 42 and 129).

TABLE 15

DIAGNOSIS	CTR%					LEFT ATRIUM		
	40-44	45-49	50-54	55-59	>60	N	1	2
Mitral stenosis, post op.	-	-	-	-	1	-	-	1
Lone	-	-	1	-	-	1	-	-
ASD, post op.	1	-	-	-	-	1	-	-
Acute myoc. inf.	-	-	-	2	-	-	-	2
VSD, Pulm hyp.	-	-	-	-	1	-	1	-
VSD, WPW	-	-	1	-	-	1	-	-
AOVD, CHF	-	1	-	-	-	1	-	-
Digitalis	-	-	-	1	-	-	1	-
TOTAL	1	1	2	3	2	4	2	3

Table 15: The cardiothoracic ratio and size of the left atrium before synchronised capacitor discharge in 9 patients with atrial tachycardia.

D VENTRICULAR TACHYCARDIA: (5 Patients)

(i) Underlying Heart Disease: Table 3.

One patient (number 80) had cardiomyopathy as the underlying cause (Table 3).

Two patients (numbers 72 and 145) had ventricular tachycardia associated with ischaemic heart disease but unassociated with recent myocardial infarction.

Two patients (numbers 64 and 134) had their arrhythmia in association with recent myocardial infarction.

(ii) Age Range and Sex Incidence: Table 16.

The oldest patient with ventricular tachycardia was 74 years old (patient number 64) and the youngest 39 years (patient number 80).

4 of the 5 patients were male.

(iii) Duration of the Arrhythmia: Table 17.

Ventricular tachycardia was present in all 5 cases for a relatively short time which varied from 1 to 10 days (Table 17).

TABLE 16

DIAGNOSIS	31-40		51-60		71-80	
	M	F	M	F	M	F
Cardiomyopathy	-	1	-	-	-	-
Chronic ischaemic HD	-	-	2	-	-	-
Acute myoc. inf.	-	-	1	-	1	-
TOTAL	-	1	3	-	1	-

Table 16: The age range in years and sex incidence in 5 patients with ventricular tachycardia.

TABLE 17

DIAGNOSIS	1-7d	1wk-1mo
Cardiomyopathy	-	1
Chronic ischaemic HD	2	-
Acute myoc. inf.	2	-
TOTAL	4	1

Table 17: The duration of ventricular tachycardia in 5 patients before synchronised capacitor discharge.

(iv) Electrocardiogram:

Ventricular tachycardia alone was noted on the electrocardiogram in 4 of the 5 patients. Patient number 72 however also showed evidence of digitalis effect.

(v) Heart Size:

(a) Cardiothoracic Ratio: Table 18.

One patient (number 134) had a cardiothoracic ratio of 64% and in the remaining 4 patients this ratio varied from 50 - 59%.

(b) Size of the Left Atrium: Table 18.

The left atrium was graded as normal (N) in patient number 80.

Patient number 64 had slight (1+) enlargement and patients numbers 72, 145 and 134 moderate (2+) enlargement.

III. HAEMODYNAMIC STUDY:

(i) Cardiac output Measurements - 14 patients. Table 19.

(a) Atrial Fibrillation.

3 patients had rheumatic heart disease, one had had a mitral valvotomy for mitral stenosis and one had had an open operation for aortic stenosis. The third patient had

TABLE 18

DIAGNOSIS	CTR%			LEFT ATRIUM		
	50-54	55-59	>60	N	1	2
Cardiomyopathy	1	-	-	1	-	-
Chronic ischaemic HD	1	1	-	-	-	2
Acute myoc. inf.	-	1	1	-	1	1
TOTAL	2	2	1	1	1	3

Table 18: The cardiothoracic ratio and size of the left atrium before synchronised capacitor discharge in 5 patients with ventricular tachycardia.

TABLE 19

HAEMODYNAMIC STUDY, REST AND EXERCISE, IN ARRHYTHMIA AND SINUS RHYTHM

A ATRIAL FIBRILLATION	
Rheumatic heart disease:	
Mitral stenosis, post operative	1
Aortic valve disease, post operative	1
Mitral and aortic valve disease	1
Alcoholic Cardiomyopathy	1
Cardiomyopathy	1
Obstructive cardiomyopathy of left ventricle	1
Lone	3
Chronic ischaemic heart disease	2
Treated thyrotoxicosis	1
B ATRIAL FLUTTER	
Lone	2
TOTAL	14

Age Range 31-65 years (mean 50)

11 male 3 Female

Table 19: 14 patients in whom cardiac output and pressure measurements were made at rest and on exercise before and after the establishment of sinus rhythm by synchronised capacitor discharge.

mitral and aortic valve disease which had not been operated on.

2 patients had cardiomyopathy and alcohol was thought to be the precipitating cause in one. One patient had obstructive cardiomyopathy of the left ventricle (sub valve stenosis).

In 3 patients atrial fibrillation was unassociated with clinically detectable heart disease. 2 patients had ischaemic heart disease without clinical or electrocardiographic evidence of recent myocardial infarction. The remaining patient in this group had had thyrotoxicosis treated and was now euthyroid.

(b) Atrial Flutter:

2 patients were studied in atrial flutter. In both this was unassociated with clinically detectable heart disease.

The mean age of the 14 patients was 50 years (range 31 - 65 years). 11 were males and 3 were females.

(ii) Heart Rate and Physical Work Capacity: 6 patients. Table 20.

6 patients were studied. Atrial fibrillation was

TABLE 20

HEART RATE AND PHYSICAL WORK CAPACITY, IN ATRIAL FIBRILLATION AND SINUS RHYTHM

Mitral stenosis and regurgitation	1
Lone	1
Chronic ischaemic heart disease	3
Treated thyrotoxicosis	1
TOTAL	6

Age Range: 38-62 years (mean 53.5)

4 male 2 female.

Table 20: 6 patients in whom heart rate and physical work capacity were determined before and after the conversion of atrial fibrillation to sinus rhythm by synchronised capacitor discharge.

present in all 6 before synchronised capacitor discharge.

Rheumatic heart disease, resulting in mitral stenosis and regurgitation was present in one patient. One patient was euthyroid following treatment of thyrotoxicosis and in one patient atrial fibrillation was unassociated with clinically detectable underlying heart disease. Ischaemic heart disease without clinical or electrocardiographic evidence of recent myocardial infarction was present in 3 patients.

The mean age of the 6 patients was 53.5 years (range 38 - 62 years). 4 were males and 2 females. These patients formed a representative group of the 150 patients of the total series. Although 12 also appear amongst the 150 patients the remainder were additional. To avoid confusion the patients forming this group will be listed according to their initials throughout.

IV. PATIENTS TREATED BY SYNCHRONISED CAPACITOR DISCHARGE FOR A SECOND TIME

(1) Patients who reverted to their arrhythmia (17)

12 patients reverted to atrial fibrillation and rheumatic heart disease was associated in 6 (Patients numbers

23, 37, 54, 110, 123, 43). In 2, atrial fibrillation was unassociated with clinical evidence of heart disease (Patients numbers 61, 63). Patient number 9 had had an atrial septal defect closed surgically, patient number 68 had corrected transposition and regurgitation of the systemic atrio-ventricular valve, patient number 142 chronic ischaemic heart disease and patient number 146 obstructive cardiomyopathy of the left ventricle (sub-valve stenosis).

3 patients reverted to atrial flutter. Patient number 28 had no underlying heart disease, patient number 50 had a ventricular septal defect and pulmonary hypertension and patient number 107 had had surgical closure of an atrial septal defect performed.

Patient number 129 who had an acute myocardial infarct reverted to atrial tachycardia.

Patient number 72 with chronic ischaemic heart disease reverted to ventricular tachycardia.

(ii) Patients in whom the first treatment failed (3)

2 patients (numbers 60 and 39) had atrial fibrillation associated with rheumatic heart disease in both.

1 patient (number 65) had atrial tachycardia associated with the Wolff-Parkinson-White Syndrmoe and a ventricular septal defect.

SUMMARY

1. 150 patients with atrial fibrillation, atrial flutter, atrial tacaycardia and ventricular tachycardia are presented.

2. The first 50 patients were treated to determine whether the arrhythmia was drug resistant.

3. Details of the numbers of patients with each arrhythmia are given and patients are analysed according to the underlying heart disease, age and sex, duration of the arrhythmia, radiographic size of the heart and electrocardiographic changes.

4. A representative group of 20 patients were studied for haemodynamic benefit in sinus rhythm. In 6 heart rate and physical work capacity were monitored. In 14 cardiac output measurements were made.

5. 20 patients were treated by synchronised capacitor discharge for a second time, 17 of whom had reverted to their arrhythmia; the first treatment had been unsuccessful in 3.

Chapter 4

R E S U L T S

DRUG THERAPY TRIAL:

Patients numbers 1 - 50 failed to achieve sinus rhythm despite the drug therapy given. The drugs were stopped at lower doses than recorded in Chapter 3 with the development of toxic symptoms. Quinidine caused more toxic symptoms than digoxin and every patient in whom quinidine was used was admitted to hospital and the electrocardiogram monitored on an oscilloscope throughout. During the time these 50 patients with arrhythmia resistant to drugs were collected, 2 cases of quinidine arrest occurred; both were resuscitated without sequelae. The first developed arrest 1 hour after the second dose of 300 mg. of quinidine; the second 1 hour following a second dose of 600 mg.

Nausea, vomiting and tinnitus were frequently complained of and quinidine had to be discontinued because of the first two in 17 of the 50 patients. Multifocal ventricular ectopics beats occurred in 6 patients, dizziness or syncope in 4, and hypotension in 2. In 2 patients who

had atrial fibrillation the ventricular rate increased to 180 per minute on quinidine despite full digitalisation and quinidine had to be discontinued. In 12 patients a combination of toxic symptoms to quinidine developed - nausea, multifocal ventricular ectopic beats and dizziness were commonly associated.

I. PATIENTS TREATED SUCCESSFULLY:

DRUG RESISTANT SERIES (Patient numbers 1 - 50). Table 21.

Drug therapy to maximum tolerance failed to bring about sinus rhythm in 50 patients. 43 patients (86%), came into sinus rhythm following synchronised capacitor discharge (Table 21). Successful version was achieved in 33 of 40 with atrial fibrillation. All 8 patients with atrial flutter and both patients with atrial tachycardia were successfully treated.

TOTAL SERIES (Patient numbers 1 - 150) Table 22.

131 patients (87%) were treated successfully with synchronised capacitor discharge (Table 22).

TABLE 21

DIAGNOSIS	ATRIAL FIBRILLATION		ATRIAL FLUTTER		ATRIAL TACHYCARDIA		TOTAL	
	SUCCESS	TOTAL	SUCCESS	TOTAL	SUCCESS	TOTAL	SUCCESS	POSSIBLE
RHEUMATIC HEART DISEASE								
Mitral stenosis, post op.	11	14	1	1	1	1	13	16
Mitral regurg, post op.	0	1	-	-	-	-	0	1
Mitral stenosis	3	3	-	-	-	-	3	3
Mitral stenosis and regurg.	1	1	-	-	-	-	1	1
Mitral and ACVD.	3	3	-	-	-	-	3	3
Alcoholic cardiomyopathy	4	4	-	-	-	-	4	4
Lone	7	10	2	2	-	-	9	12
ASD, post op.	2	2	3	3	-	-	5	5
Post Thyrotoxicosis	1	1	-	-	-	-	1	1
Chronic ischaemic HD.	1	1	-	-	-	-	1	1
Acute myoc. inf.	-	-	-	-	1	1	1	1
Syst. hyp. mitral regurg.	-	-	1	1	-	-	1	1
VSD, Pulm hyp.	-	-	1	1	-	-	1	1
TOTAL	33	40	8	8	2	2	43	50

TABLE 21: Immediate results following synchronised capacitor discharge in 50 patients with drug-resistant atrial arrhythmias.

TABLE 22

DIAGNOSIS	ATRIAL FIBRILLATION SUCCESS	ATRIAL FIBRILLATION TOTAL	ATRIAL FLUTTER SUCCESS	ATRIAL FLUTTER TOTAL	ATRIAL TACHYCARDIA SUCCESS	ATRIAL TACHYCARDIA TOTAL	VENTRICULAR TACHYCARDIA SUCCESS	VENTRICULAR TACHYCARDIA TOTAL	TOTAL SUCCESS	TOTAL POSSIBLE
RHEUMATIC HEART DISEASE										
Mitral stenosis, post op.	44	51	1	1	1	1	-	-	46	53
Mitral and AOVD, post op.	1	1	-	-	-	-	-	-	1	1
AOVD, post op.	4	4	-	-	-	-	-	-	4	4
Mitral regurg, post op.	0	1	-	-	-	-	-	-	0	1
Mitral stenosis	4	4	-	-	-	-	-	-	4	4
Mitral regurgitation	2	2	-	-	-	-	-	-	2	2
Mitral stenosis and regurg.	2	2	-	-	-	-	-	-	2	2
Mitral and AOVD.	5	6	-	-	-	-	-	-	5	6
Alcoholic cardiomyopathy	6	6	-	-	-	-	-	-	6	6
Cardiomyopathy	4	4	1	1	-	-	1	1	6	6
Lone	16	22	2	2	1	1	-	-	19	25
ASD, post op.	3	4	9	9	1	1	-	-	13	14
Post thyrotoxicosis	3	4	-	-	-	-	-	-	3	4
Chronic ischaemic HD	5	6	-	-	-	-	2	2	7	8
Acute myoc. inf.	-	-	-	-	2	2	2	2	4	4
Systemic hyp, mitral regurg.	1	1	1	1	-	-	-	-	2	2
VSD, Pulm hyp.	-	-	1	1	1	1	-	-	2	2
VSD, WPW	-	-	-	-	0	1	-	-	0	1
AOVD, WPW	-	-	-	-	1	1	-	-	1	1
Cor. transp. "HI"	1	1	-	-	-	-	-	-	1	1
Obstruc. cardiomyop.	1	1	-	-	-	-	-	-	1	1
Dystrophia myotonica	-	-	1	1	-	-	-	-	1	1
Digitalis	-	-	-	-	1	1	-	-	1	1
TOTAL	102	120	16	16	8	9	5	5	131	150

Table 22: Immediate results following synchronised capacitor discharge in 150 patients.

1. THE ARRHYTHMIA

A ATRIAL FIBRILLATION:

(i) Underlying Heart Condition:

(a) Rheumatic Heart Disease:

62 patients (87%) with rheumatic heart disease were successfully brought into sinus rhythm.

(b) Cardiomyopathy:

All 10 patients who had atrial fibrillation in association with cardiomyopathy, were successfully treated.

(c) Lone:

16 patients (73%) were successfully brought into sinus rhythm.

(d) Other:

3 of 4 patients who had had surgical correction of an ostium secundum atrial septal defect were treated successfully as were 3 of 4 patients in whom thyrotoxicosis had previously been treated.

5 of 6 patients in whom atrial fibrillation was associated with chronic ischaemic heart disease were successfully brought into sinus rhythm.

The patient who had systemic hypertension and mitral regurgitation, the patient with corrected

transposition and regurgitation of the systemic atrio-ventricular valve and the patient with obstructive cardiomyopathy of the left ventricle (sub-valve stenosis), were successfully treated.

(ii) Age Range and Sex Incidence:

Age by itself did not alter the chances of successful version by synchronised capacitor discharge. Patients in the seventh decade were successfully brought into sinus rhythm as were those in the second decade. Sex incidence also, was not a factor.

(iii) Duration of the Arrhythmia: Figs. 16, 17.

The number of patients treated successfully compared with the duration of atrial fibrillation is shown in Fig.16. When atrial fibrillation was present for up to 1 year there was a 90% chance of successful version with synchronised capacitor discharge (Fig.17), irrespective of the underlying heart disease. When this period was extended to 3 years, the chance of success was 84%; at 5 years it was 80%, and over 5 years, 48%.

Although the longer atrial fibrillation was present the less the chance of success, patient number 4 who had

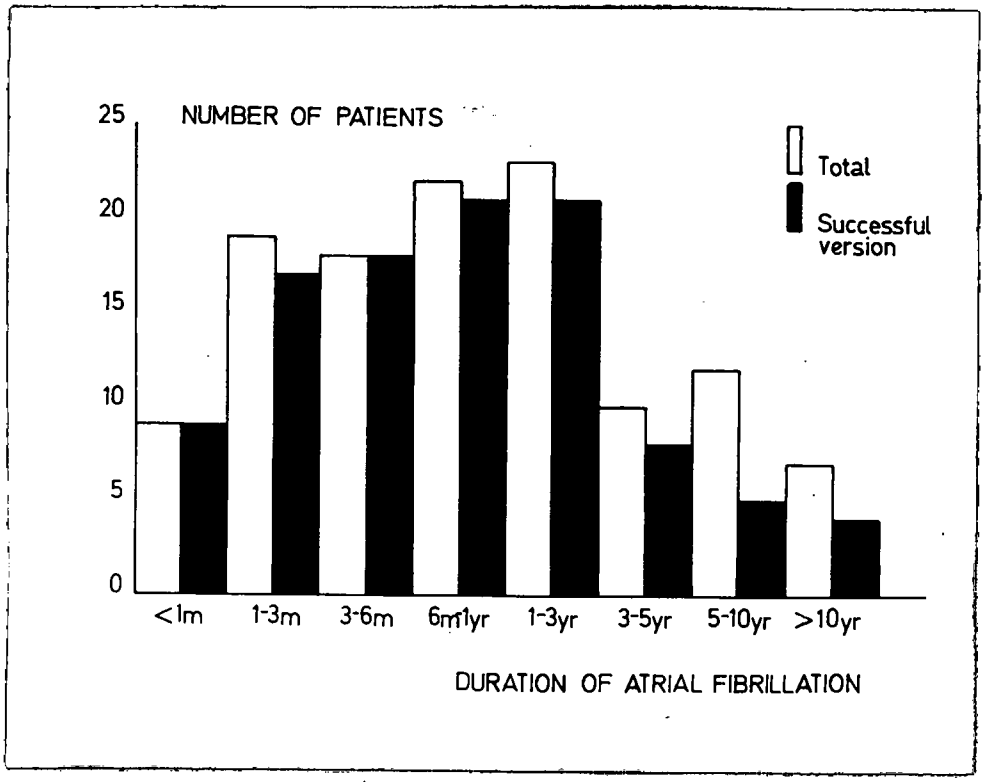


FIG. 16: Duration of atrial fibrillation and number of successful treatments in 120 patients.

had atrial fibrillation for 16 years, patient number 76 (14 years) and patient number 67 (12 years) were successfully brought into sinus rhythm.

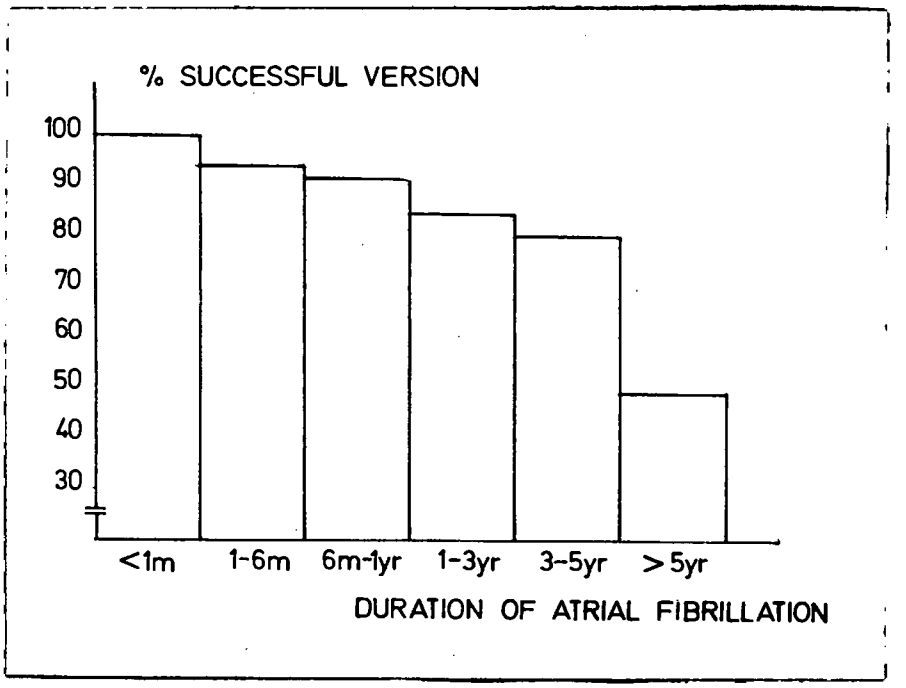


FIG. 17: Percentage successful version and duration of atrial fibrillation in 120 patients.

(iv) Heart Size:

(a) Cardiothoracic Ratio: Figs. 18, 19.

The success rate following synchronised capacitor discharge was inversely related to the cardiothoracic ratio (patients with lone atrial fibrillation excepted) (Fig.18). Once the ratio exceeded 55%, the success

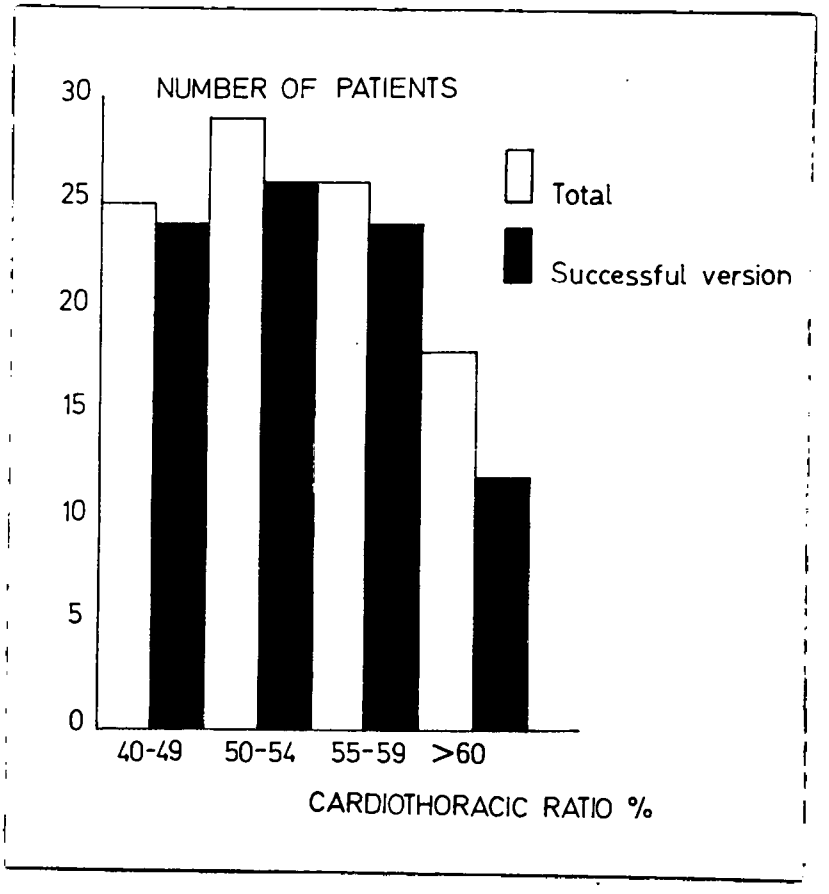


FIG. 18: Cardiothoracic ratio, and number of successful treatments in 90 patients with atrial fibrillation. (excluding 22 patients with lone atrial fibrillation).

rate fell to less than 82% of the 44 patients in this group (Fig.19). This proved to be especially the case in those patients who, following closed mitral valvotomy, were in atrial fibrillation with considerable enlargement of the heart. 5 patients (numbers 85, 92, 87, 110 and 123) out

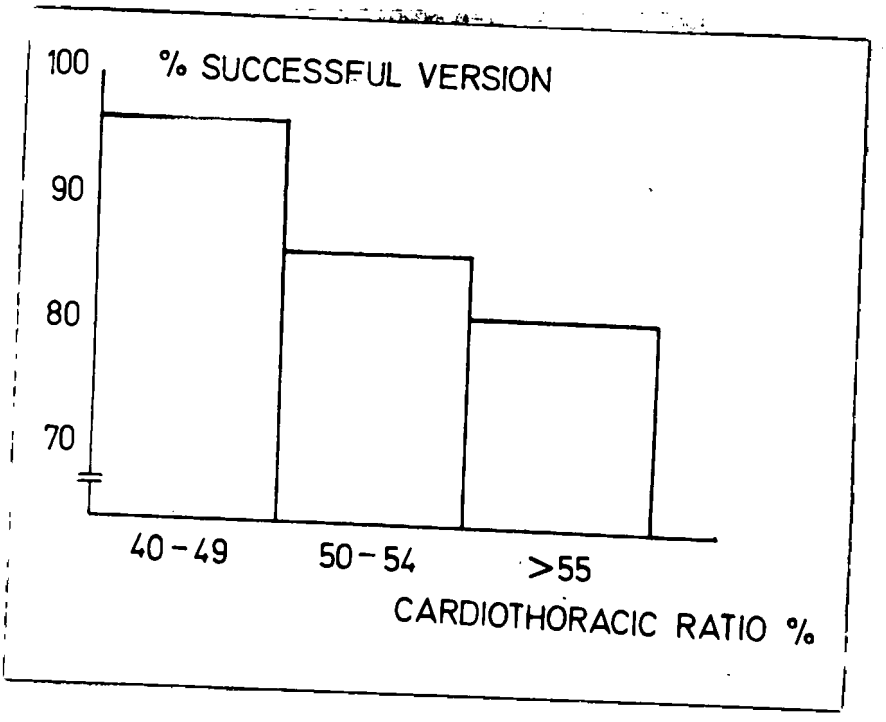


FIG. 19: Percentage successful version and cardiothoracic ratio in 98 patients with atrial fibrillation. (excluding 22 patients with lone atrial fibrillation).

of 9 of this group were brought into sinus rhythm. Nevertheless 12 out of a total of 15 patients in whom the cardiothoracic ratio was greater than 60% did come into sinus rhythm.

An exception to the general rule was found in the 22 patients with lone atrial fibrillation. 8 (patients numbers

5, 61, 67, 105, 120, 12, 44 and 91) out of a possible 13 patients in whom the cardiothoracic ratio varied from 40 - 49% came into sinus rhythm.

(b) Size of the Left Atrium Figs. 20, 21.

Selective enlargement of the left atrium lessened the chances of success (Figs, 20, 21). The patients with lone atrial fibrillation were exceptional. The left atrium was normal in 14 of these patients but only 10 (patient numbers 5, 40, 44, 48, 61, 91, 105, 120, 132, 135) were restored to sinus rhythm.

B ATRIAL FLUTTER

(i) Underlying Heart Disease:

All 16 patients were successfully brought into sinus rhythm.

(ii) Age Range and Sex Incidence:

Age and sex incidence did not lessen the chances of success.

(iii) Duration of Arrhythmia:

Duration of the arrhythmia did not lessen the chances of successful version. Patient number 28 in whom atrial

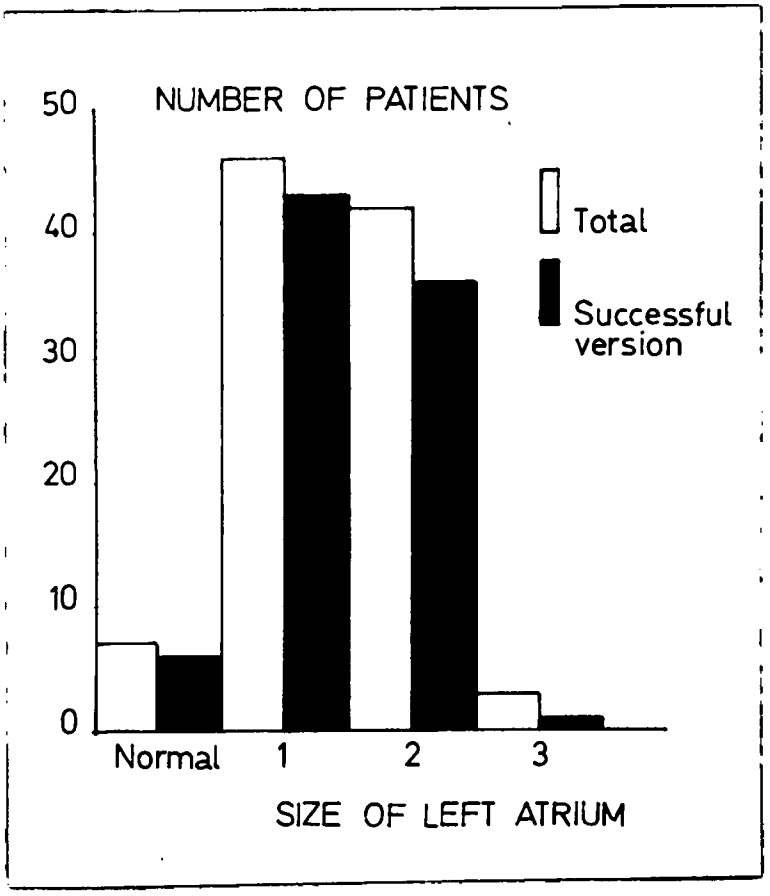


FIG. 20: Size of the left atrium and number of successful treatments in 98 patients with atrial fibrillation. (excluding 22 patients with lone atrial fibrillation).

flutter had been present for 16 years was successfully converted to sinus rhythm.

(iv) Size of the Heart:

(a) Cardiothoracic Ratio:

Enlargement of the heart did not prevent successful treatment.

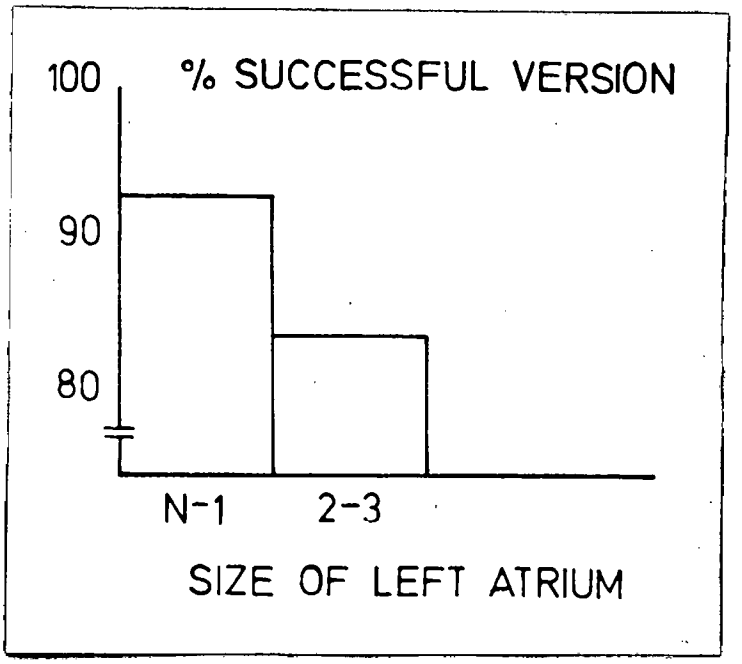


FIG. 21: Percentage successful version and size of the left atrium in 98 patients with atrial fibrillation. (excluding 22 patients with lone atrial fibrillation).

(b) Size of the Left Atrium:

Enlargement of the left atrium did not prevent successful treatment.

C ATRIAL TACHYCARDIA

(i) Underlying Heart disease:

8 patients were treated successfully.

2 patients (numbers 42 and 129) in whom atrial tachycardia followed soon after acute myocardial infarction had their arrhythmia successfully terminated.

(ii) Age Range and Sex Incidence:

Age and sex incidence did not lessen the chances of successful treatment.

(iii) Duration of Arrhythmia:

No patient with long-standing atrial tachycardia presented for treatment.

(iv) Size of the Heart

(a) Cardiothoracic Ratio

Heart size alone did not lessen the chances of successful version.

(b) Size of the Left Atrium:

Moderate (2+) enlargement was present in 3 patients (numbers 31, 42, 129) who were all treated successfully.

D VENTRICULAR TACHYCARDIA

All 5 patients were treated successfully. The age range, overall size of the heart and size of the left atrium did not lessen the chance of successful treatment. The arrhythmia was of short duration in all patients before treatment.

2. QUINIDINE PRECEDING SYNCHRONISED CAPACITOR DISCHARGE

(i) Atrial Fibrillation (120 patients) Table 23.

Synchronised capacitor discharge was preceded by 300 mg. quinidine orally, 60 - 90 minutes before treatment in 41 patients (Table 23). 9 failed to come into sinus rhythm.

Treatment was undertaken without immediate preceding quinidine in 79 patients. 10 failed to come into sinus rhythm.

(ii) Atrial Flutter (16 patients) Table 24.

4 of 16 patients successfully treated were given preceding quinidine (Table 24).

TABLE 23

DIAGNOSIS	QUINIDINE 1½ HOURS BEFORE DC	
	YES	NO
RHEUMATIC HEART DISEASE		
Mitral stenosis, post op.	14 (2)	37 (5)
Mitral and AOVD post op.	1	-
AOVD, post op.	-	4
Mitral regurg., post op.	1 (1)	-
Mitral stenosis	3	1
Mitral regurgitation	2	2
Mitral stenosis and regurg.	1	1
Mitral and AOVD	2 (1)	4
Alcoholic cardiomyopathy	3	3
Cardiomyopathy	-	4
Lone	9 (3)	13 (3)
ASD, post op.	2 (1)	2
Post Thyrotoxicosis	1	3 (1)
Chronic ischaemic HD	1	5 (1)
Syst. hyp , mitral regurg.	1	-
Cor. Transp. "SL"	-	1
Obstr. cardiomyop.	-	1
TOTAL	41 (9)	79 (10)

Table 23: 120 patients with atrial fibrillation treated by synchronised capacitor discharge showing patients whose treatment was preceded by quinidine. The figures in brackets refer to those who failed to come into sinus rhythm.

TABLE 24

DIAGNOSIS	QUINIDINE 1½ HOURS BEFORE DC	
	YES	NO
Mitral stenosis, post op.	-	1
Cardiomyopathy	-	1
Lone	2	-
ASD, post op.	1	8
Syst. hyp, mitral regurg.	1	-
VSD, Pulm hyp.	-	1
Dystrophia myoton.	-	1
TOTAL	4	12

Table 24: 16 Patients who had atrial flutter treated with synchronised capacitor discharge showing patients whose treatment was preceded by quinidine.

(iii) Atrial Tachycardia (9 Patients) Table 25.

Synchronised capacitor discharge was preceded by quinidine in 2 patients (Table 25). Patient number 129 who had acute myocardial infarction was given 300 mg. quinidine 90 minutes before successful treatment. Patient number 65 who had a ventricular septal defect and the Wolff-Parkinson-White Syndrome and was given 600 mg. quinidine one hour before treatment failed to come into sinus rhythm.

In 7 patients synchronised capacitor discharge was not preceded by quinidine. All 7 were successfully brought into sinus rhythm.

(iv) Ventricular Tachycardia (5 patients)

All 5 patients who had ventricular tachycardia successfully treated were not given preceding quinidine.

3. NUMBER OF SHOCKS AND ENERGY REQUIREMENTS:

Sinus rhythm was achieved in 131 out of the 150 patients treated by synchronised capacitor discharge. Two anterior paddles were used in 108 patients and in 23 one anterior and one posterior paddle were used to determine whether conspicuously lower levels of energy resulted (Lown, 1964a).

TABLE 25

DIAGNOSIS	QUINIDINE 1½ HOURS BEFORE DC	
	YES	NO
Mitral stenosis, post op.	-	1
Lone	-	1
ASD, post op.	-	1
Acute myoc. inf.	1	1
VSD, Pulm. hyp.	-	1
VSD, WPW	1 (1)	-
AOVD, WPW	-	1
Digitalis	-	1
TOTAL	2 (1)	7

Table 25: 9 patients with atrial tachycardia treated by synchronised capacitor discharge showing patients in whom treatment was preceded by quinidine. The figure in brackets refers to the patient who failed to come into sinus rhythm.

A. THE ARRHYTHMIA

(i) Atrial Fibrillation (102 Patients) Table 26.

(a) Paddles Anterior:

83 patients were successfully treated with two anterior paddles. In 43, sinus rhythm was achieved after one shock (energy range 50 - 150 joules), in 28 patients, after 2 shocks (energy range 100 - 200 joules), in 7 patients after 3 shocks (energy range 200 - 350 joules), and in 5 patients after 4 shocks (energy range 350 - 400 joules) - see Table 26.

(b) Paddles Antero-Posterior: Table 26.

19 patients were successfully treated with one anterior and one posterior paddle. No patient was brought into sinus rhythm on the first shock (energy range 25 - 50 joules), (Table 26). 5 patients came into sinus rhythm following the second shock (energy range 100 - 150 joules), 8 after the third shock (energy range 100 - 350 joules), 5 after the fourth shock (energy range 200 - 400 joules) and 1 patient after a fifth shock of 400 joules.

TABLE 26

DIAGNOSIS	PADDLES ANTERIOR				PADDLES ANTERO-POSTERIOR				
	NO. OF SHOCKS				NO. OF SHOCKS				
	1	2	3	4	1	2	3	4	5
RHEUMATIC HEART DISEASE									
Mitral stenosis, post op.	23	10	2	1	-	3	3	1	1
Mitral and AOVD, post op.	1	-	-	-	-	-	-	-	-
AOVD, post op.	2	-	-	-	-	-	1	1	-
Mitral stenosis	1	2	-	-	-	-	1	-	-
Mitral regurgitation	-	1	-	-	-	1	-	-	-
Mitral stenosis and regurg.	-	2	-	-	-	-	-	-	-
Mitral and AOVD	4	1	-	-	-	-	-	-	-
Alcoholic cardiomyopathy	1	2	2	1	-	-	-	-	-
Cardiomyopathy	3	1	-	-	-	-	-	-	-
Lone	4	7	1	2	-	-	1	1	-
ASD, post op.	1	1	-	1	-	-	-	-	-
Post thyrotoxicosis	2	-	-	-	-	-	1	-	-
Chronic ischaemic HD	-	1	1	-	-	1	-	2	-
Syst hyp , mitral regurg.	1	-	-	-	-	-	-	-	-
Cor. Transp. "LI"	-	-	1	-	-	-	-	-	-
Obstr. cardiomyopathy	-	-	-	-	-	-	1	-	-
TOTAL	43	28	7	5	-	5	8	5	1

Table 26: The number of shocks needed for successful version of atrial fibrillation to sinus rhythm in 102 patients; in 83 anterior paddles were used, in 19, antero-posterior paddles.

(ii) Atrial Flutter (16 Patients)

(a) Paddles Anterior: Table 27.

13 patients came into sinus rhythm after a single shock of 150 joules. Patient 113 a boy of 2½ years came into sinus rhythm following a single shock of 10 joules. 1 patient came into sinus rhythm after a second shock of 150 joules.

(b) Paddles Antero-Posterior: Table 27.

1 patient came into sinus rhythm after a single shock of 50 joules.

(iii) Atrial Tachycardia (8 patients)

(a) Paddles Anterior: Table 28.

5 patients were brought into sinus rhythm following the first shock. In 4 an energy setting of 150 joules was used and in patient number 101, 50 joules.

(b) Paddles Antero-Posterior: Table 28.

1 patient was brought into sinus rhythm following a shock of energy level setting of 50 joules.

Patient number 126, required a second shock of 100 joules and patient number 143, a second shock of 50 joules.

TABLE 27

DIAGNOSIS	PADDLES ANTERIOR		ANT-POST.
	NO. OF SHOCKS		NO. OF SHOCKS
	1	2	1
Mitral stenosis, post op.	1	-	-
Cardiomyopathy	-	-	1
None	2	-	-
ASD, post op.	8	1	-
Syst. hyp, mitral regurg.	1	-	-
VSD, pulm. hyp.	1	-	-
Dystrophia myotonica	1	-	-
TOTAL	14	1	1

Table 27: The number of shocks needed for successful version of atrial flutter to sinus rhythm in 16 patients; in 15 anterior paddles were used, in 1 antero-posterior paddles.

TABLE 28

DIAGNOSIS	PADDLES ANT.	PADDLES ANT-POST	
	NO. OF SHOCKS	NO. OF SHOCKS	
	1	1	2
Mitral stenosis, post op.	1	-	-
Lone	-	1	-
ASD, post op.	1	-	-
Acute myoc. inf.	2	-	-
VSD, pulm. hyp.	1	-	-
AOVD, PAI	-	-	1
Digitalis	-	-	1
TOTAL	5	1	2

Table 28: The number of shocks needed for successful version of atrial tachycardia to sinus rhythm in 8 patients; in 5 anterior paddles were used, in 3 antero-posterior paddles.

(iv) Ventricular Tachycardia: (5 patients)

Paddles Anterior:

4 patients were brought into sinus rhythm after the first shock (energy range 50 - 150 joules).

1 patient came into sinus rhythm after the second shock of 50 joules.

B. DURATION OF ARRHYTHMIA

(i) Atrial Fibrillation: Table 29.

No correlation was found between the duration of atrial fibrillation and the energy setting needed for successful version to sinus rhythm (Table 29). Patient number 140 who had atrial fibrillation for less than 1 year in relation to rheumatic heart disease required a maximum energy level setting of 400 joules using antero-posterior paddles. In contrast, patient number 4, who had had atrial fibrillation for 16 years, also related to rheumatic heart disease was brought into sinus rhythm with an energy setting of 150 joules.

(ii) Atrial Flutter:

No correlation was found between the duration of the arrhythmia and energy setting needed to bring about sinus rhythm.

TABLE 29

DIAGNOSIS	1-7d	1wk-1mo	1-3mo	3-6mo	6mo-1yr	1-3yr	3-5yr	5-10yr	+10yr
RHEUMATIC HEART DISEASE									
Mitral stenosis, post op.		150, 150, 150, 150, 100	250, 250, 150, 250	100, 100, 250, 150, 150, 50	150, 150, 150, 150, 150, 150, 150, 150, 350, 200, 400, 200	250, 150, 250, 150, 150, 250, 150, 150, 150	250, 150, 150	150, 350, 400	150
Mitral and AOVD post op.								150	
AOVD, post op.	100	150	150		100				
Mitral stenosis				350, 150		250, 150			
Mitral regurg.				150	250				
Mitral sten. and regurg.				250		250			
Mitral + AOVD			250 150	150	150	150			
Alc. cardiomyop.			250 350			350, 150	250		400
Cardiomyopathy			150	150 250		150			
Lone	250		400 100 150	350, 250 250, 150, 350	250, 250	200, 150 250	250		150
ASD, post. op.			150		250			400	
Post thyrotox.			200			150, 150			
Chr. ischaemic HD			250		150 400		350 200		
Syst. hyp HI					150				
Cor. Transp. "HI"			350						
Obstr. cardiomyop.	200								

Table 29: The duration of atrial fibrillation and energy level settings in joules needed for successful version to sinus rhythm in 102 patients. Each figure represents one patient. Energy delivered using antero-posterior paddles is underlined.

(iii) Atrial Tachycardia:

No correlation was found between the duration of the arrhythmia and energy setting needed to bring about sinus rhythm. It must be stressed, however, that this arrhythmia was present for less than one year in all patients.

(iv) Ventricular Tachycardia:

The arrhythmia was present for relatively short periods in all 5 patients; no analysis as regards duration was possible.

C. SIZE OF THE HEART

(i) Atrial Fibrillation

(a) Cardiothoracic Ratio: Table 30.

No correlation was found between the cardiothoracic ratio % and the energy setting required for successful version to sinus rhythm. This is shown in Table 30 where the energy needed in 43 patients who had rheumatic heart disease (post operative mitral stenosis) is detailed and the cardiothoracic ratio shown. The same lack of correlation was found with the remaining patients.

TABLE 30

DIAGNOSIS	CTR%			
	45-49	50-54	55-59	+60
Mitral stenosis, post op.	150, 100, 250, <u>250</u>, 150, 400, 150, 150, <u>100</u>	100, 150, 150, 150, 150, 350, 150, 150, 150, 250, 150, 150, 250, 250	250, 150, <u>150</u>, 250, 150, 150, 150, 150, <u>150</u>, <u>150</u>, 150, 150, <u>400</u>, 200, <u>200</u>, 137.	150, 250, 150 250

Table 30: The cardiothoracic ratio and energy level settings in joules used for successful version of atrial fibrillation to sinus rhythm in 43 patients who had rheumatic heart disease (post operative mitral stenosis). Each figure represents one patient. Energy delivered using antero-posterior paddles is underlined.

(b) Size of the Left Atrium: Table 31.

No correlation was found between the size of the left atrium and the energy settings needed for successful treatment in patients who had rheumatic heart disease (Table 31) or in the remaining patients.

(ii) Atrial Flutter:

(a) Cardiothoracic Ratio:

There was no correlation between the cardiothoracic ratio % and the energy setting required for successful version to sinus rhythm.

(b) Size of the Left Atrium:

There was no correlation between the size of the left atrium and the energy setting required for successful version to sinus rhythm.

(iii) Atrial Tachycardia:

Neither cardiothoracic ratio nor size of the left atrium were related to energy settings needed in the 8 patients who were successfully brought into sinus rhythm.

TABLE 31

DIAGNOSIS	LEFT ATRIUM	
	1	2
Mitral stenosis, post op.	100, 150, 100, 250, 150, 150, 150, 150, 250, 250, 400, 150, 150, 150, 150, 150, 150, 50, 200, 250, 100, 150.	250, 150, 150, 150, 250, 150, 350, 150, 150, 150, 150, 150, 150, 150, 150, 250, 250, 150, 150, 350, 250, 400, 200.

Table 31: The size of the left atrium and energy settings in joules needed for successful version of atrial fibrillation to sinus rhythm in 43 patients whose arrhythmia was associated with rheumatic heart disease (post operative mitral stenosis). Each figure represents one patient. Energy delivered using antero-posterior paddles is underlined.

D. BODY SURFACE AREA

No correlation was found between body surface area and energy level settings for successful treatment.

E. QUINIDINE PRECEDING SYNCHRONISED CAPACITOR DISCHARGE:

Preceding quinidine did not affect the energy levels needed for successful version to sinus rhythm in patients with atrial fibrillation, atrial flutter or atrial tachycardia.

F. PATIENTS REQUIRING MINIMUM AND MAXIMUM ENERGY LEVEL SETTINGS: Table 32.

15 patients were brought into sinus rhythm with energy level settings of up to 100 joules and 15 patients needed an energy level setting of between 300 - 400 joules (Table 32). The majority of patients, 101, were brought into sinus rhythm with an energy setting of more than 100 but less than 300 joules.

As can be seen from Table 32 patients who came into sinus rhythm with low energy level settings tended to have atrial tachycardia rather than atrial fibrillation - 5 out of 8 patients with atrial tachycardia came into sinus rhythm with energy settings of up to 100 joules.

TABLE 32

ENERGY SETTING joules	CASE NO.	ARRHYTHMIA	DURATION	DISEASE	CTR%	LEFT ATRIUM
	1	AF	7 mo	MS post op.	50	1
	7	AF	4 yr	MS post op.	47	1
	70	A. TACHY	3 mo	Lone	50	N
	72	VT	1 d	IHD, chronic	50	2
	101	A. TACHY	2 wk	VSD, PHT	66	1
	113	A.FLUT	2 wk	Post op. ASD	56	N
	126	A. TACHY	3 hr	AOVD, WPW	49	N
50-100	128	A.FLUT	7 d	AOVD, post op.	64	2
	129	A. TACHY	3 d	Ant.Myoc.Inf.	57	2
	134	VT	2 d	Acute Myoc.Inf.	64	2
	135	AF	3 mo	Lone	51	N
	137	AF	4 mo	MS post op.	59	1
	145	A. TACHY	11 d	IHD Chronic	56	1
	148	AF	3 wk	MS post op.	48	1
	149	A.FLUT	1 yr	Cardiomyop.	52	1
	5	AF	6 mo	Lone	40	N
	12	AF	2 mo	Lone	47	1
	29	AF	7 yr	ASD, post op.	56	N
	41	AF	4 mo	MS	47	1
	57	AF	9 yr	MS post op.	54	2
	60	AF	3 mo	Cor.Transp.Pl	51	1
	76	AF	14 yr	Alc. cardiomyop.	51	1
300-400	79	AF	10 yr	MS post op.	49	1
	85	AF	6 wk	Alc. cardiomyop	49	1
	102	AF	3½ yr	IHD	46	1
	123	AF	1 yr	MS post op.	61	2
	132	AF	6 mo	Lone	52	N
	138	AF	7 mo	IHD, chronic	48	N
	140	AF	9 mo	MS , post op.	58	2
	150	AF	1 yr	AOVD, post op.	49	1

Table 32: **30 patients who were brought into sinus rhythm with energy level settings of up to 100 joules, and between 300-400 joules.**

Only 2 of the 16 patients who had atrial flutter and 2 of the 5 patients who had ventricular tachycardia came into sinus rhythm on these modest energy level settings.

All 15 patients in whom the largest energy level settings were needed had atrial fibrillation. In three of this group the arrhythmia was of the "lone" variety. Six patients had rheumatic heart disease, 2 had cardiomyopathy, and two had chronic ischaemic heart disease. One patient had corrected transposition with regurgitation of the systemic atrio-ventricular valve and one patient had had a partial surgical closure of an ostium secundum atrial septal defect. The cardiothoracic ratio varied from 40 - 61% and the size of the left atrium from normal (N) to moderate (2+) enlargement. The duration of the arrhythmia varied from 6 weeks to 14 years and in 8 of the 15 patients was less than one year.

II. PATIENTS WHO FAILED TO REVERT TO SINUS RHYTHM. Table 33.

There were 19 immediate failures out of 150 patients treated by synchronised capacitor discharge. 18 of the patients had atrial fibrillation and 1 patient had atrial tachycardia (Table 33).

TABLE 33

PATIENT NO.	ARRHYTHMIA	DURATION	UNDERLYING HEART DISEASE
8	Atrial fibrillation	14 yr	Mitral stenosis, post. op.
13	Atrial fibrillation	13 yr	Lone
14	Atrial fibrillation	6 yr	Mitral stenosis, post op.
27	Atrial fibrillation	15 yr	Lone
36	Atrial fibrillation	10 yr	Mitral stenosis, post op.
39	Atrial fibrillation	4 yr	Mitral regurgitation, post op.
45	Atrial fibrillation	5 yr	Lone
56	Atrial fibrillation	3 mo	Mitral and Aortic valve dis.
58	Atrial fibrillation	9 yr	Mitral stenosis, post op.
60	Atrial fibrillation	6 yr	Mitral stenosis, post op.
62	Atrial fibrillation	8 yr	Post operative A.S.D.
65	Atrial tachycardia	9 d	VSD. WPW.
74	Atrial fibrillation	6 yr	Mitral stenosis, post op.
97	Atrial fibrillation	10 mo	Lone
99	Atrial fibrillation	14 yr	Post thyrotoxicosis
114	Atrial fibrillation	2 yr	Lone
118	Atrial fibrillation	1.5 yr	Ischaemic HD - Chronic
125	Atrial fibrillation	9 yr	Mitral stenosis, post op.
144	Atrial fibrillation	6 wk	Lone

Table 33: 19 Patients in whom sinus rhythm was not re-established following synchronised capacitor discharge.

A THE ARRHYTHMIA

(i) Atrial Fibrillation:

Sinus rhythm could not be re-established in 18 (15%). Even allowing for the overwhelming preponderance of patients with atrial fibrillation failure in this group was more likely than in any other.

(ii) Atrial Flutter:

There were no failures amongst the 16 patients treated by synchronised capacitor discharge.

(iii) Atrial Tachycardia:

One patient (number 65) who had a ventricular septal defect and the Wolff-Parkinson-White Syndrome failed to come into sinus rhythm.

(iv) Ventricular Tachycardia:

There were no failures amongst the 5 patients treated by synchronised capacitor discharge.

B UNDERLYING HEART DISEASE Table 34.

7 patients had had a closed mitral valvotomy performed for rheumatic mitral stenosis (patients numbers 8, 14, 36, 58, 60, 74 and 125). All except patient number 58 had at

TABLE 34

PATIENT NUMBER	HEART DISEASE	ARRHYTHMIA	DURATION	RADIOGRAPH CTR%	LA SIZE	ECG	COMMENT
8	Mitral stenosis, post op.	AF	14 yr	64	1		MI-1
14	do.	AF	6 yr	54	2	LV+	MI-2
36	do.	AF	10 yr	71	2	LV+, RV+	MI-1
58	do.	AF	9 yr	67	3		
60	do.	AF	6 yr	52	1		MI-1,AI-1
74	do.	AF	6 yr	57	2		MI-1
125	do. *	AF	9 yr	74	3	LV+	MI-1
39	Mitral regurg; post op.	AF	4 yr	55	2	LV++ RV+	MI-2
56	Mitral and AOVD.	AF	3 mo	60	2	LV+ RV+	
13	Lone	AF	13 yr	57	1		
27	do.	AF	15 yr	46	1		
45	do.	AF	5 yr	47.5	N		
97	do.	AF	10 mo	47	N		
114	do.	AF	2 yr	44	N		
144	do.	AF	6 wk	43	N		
62	Post op. ASD	AF	8 yr	71	2	Inc RBBB	ASD still open
99	Post Thyrotox.	AF	14 yr	46	N		Familial AF
118	IHD - chronic	AF	1.5 yr	53	1	Isch seg.	
65	VSD. WPW.	A.Tachy.	9 d	53	N	Inc RBBB WPW	VSD small

* Mitral valvotomy 1956 & 1964

MI - Mitral regurgitation
1 slight
2 moderate
AI - Aortic regurgitation
1 slight

Table 34: 19 Patients in whom synchronised capacitor discharge was unsuccessful to show underlying heart disease duration of arrhythmia radiographic size of the heart and the ECG (when relevant). Patient numbers underlined refer to patients given quinide 60-90 minutes before treatment.

least slight mitral regurgitation (Table 34); patient number 14 had moderate mitral regurgitation and patient number 60 had slight mitral and aortic regurgitation. Enlargement of the left ventricle was shown on the electrocardiogram in patients numbers 14, 36 and 125. The failure rate in patients following closed mitral valvotomy was 14%.

Patient number 39 had had an open operation performed for severe mitral valve regurgitation which had resulted from three attacks of subacute bacterial endocarditis on a mitral valve already damaged by rheumatic fever. Although improved following the operation she was left with moderate mitral regurgitation and the electrocardiogram showed considerable enlargement of the left ventricle.

Patient number 56 had mixed mitral valve disease and aortic regurgitation. There had been no attempt at surgical correction. The valvular lesions were causing considerable embarrassment to the heart and both ventricles could be seen to be enlarged on the electrocardiogram.

There were six patients in whom atrial fibrillation was unassociated with any clinical evidence of heart disease (patients numbers 13, 27, 45, 97, 114 and 144). Most of these patients were asymptomatic other than an awareness of an irregularity of the heart beat especially on effort.

None showed any electrocardiographic change other than due to the rhythm or to digitalis. The failure rate in this group was 27%, the highest in the series.

Patient number 62 had had an operation performed in 1956 for closure of an ostium secundum atrial septal defect. There was clear clinical and radiographic evidence, however (dynamic right ventricle, pulmonary artery systolic flow murmur, fixed wide splitting of the second heart sound and a delayed diastolic flow murmur clinically; pulmonary plethora and enlargement of pulmonary artery and right ventricle on the chest radiograph) - that the defect was still open and that the resultant left to right shunt was not inconsiderable.

Patient number 99 had had thyrotoxicosis treated successfully medically two years before. Clinical and laboratory tests confirmed that he was no longer thyrotoxic. Atrial fibrillation had been present, however, many years before symptoms of thyrotoxicosis developed and one brother and one parent both developed atrial fibrillation at a relatively early age; neither subsequently developed thyrotoxicosis.

Patient number 118 had ischaemic heart disease and suffered angina pectoris on effort. There was no electrocardiographic evidence of recent myocardial infarction.

Patient number 65, was prone to repeated attacks of atrial tachycardia. Although he did have a ventricular septal defect this was not causing any haemodynamic embarrassment to the heart; the arrhythmia was undoubtedly related to an associated Wolff-Parkinson-White Syndrome.

C DURATION OF ARRHYTHMIA Tables 33, 34.

The average duration of the arrhythmia in the 19 patients was 6.5 years (Tables 33 and 34) and in 6 patients exceeded 10 years.

19 patients had had atrial fibrillation for 5 years or over, and 7 for more than 10 years. The immediate failure rate in patients with atrial fibrillation of 5 years duration or over was 63% irrespective of the underlying heart disease and 71% (5 out of 7 patients) when atrial fibrillation was present for more than 10 years.

D HEART SIZE Table 34.

The average cardiothoracic ratio of the 13 patients who failed to come into sinus rhythm was 59% (Table 34)

(excluding 6 patients with lone atrial fibrillation).

Similarly, excluding the 6 patients with lone atrial fibrillation, the size of the left atrium was graded as normal (N) in 2, slightly enlarged (1+) in 4, moderately enlarged (2+) in 6 and considerably enlarged(3+) in 2.

47 patients with atrial fibrillation had a cardiothoracic ratio of 55% or over (Table 7). The immediate failure rate in those was 18% (excluding 3 patients who had lone atrial fibrillation). The immediate failure rate was 33% in the 18 patients who had a cardiothoracic ratio of 60% or more.

E QUINIDINE Table 34.

The giving or withholding of quinidine shortly before treatment was not a factor in the success or immediate failure of synchronised capacitor discharge (Table 34).

III. INTERVENING RHYTHMS: Table 35.

Temporary rhythm disturbances were extremely frequent immediately following synchronised capacitor discharge. There were 107 instances in 83 patients (Table 35). These have been called intervening rhythms and include ventricular ectopic beats, atrial ectopic beats, nodal ectopic beats or

TABLE 35

Ventricular ectopic beats	39
Atrial premature contractions	35
Nodal ectopic beats	13
Wandering pacemaker	7
Prolonged P-R interval	4
Partial heart block	2
Bradycardia	6
Atrial flutter	1
TOTAL EPISODES,	107

Table 35: 107 episodes of intervening rhythms immediately after synchronised capacitor discharge in 65 of 150 patients treated.

rhythm, and less frequently wandering pacemaker, partial heart block, bradycardia and atrial flutter. Multiple intervening rhythms were not uncommon and were found in 24 patients in whom the commonest association was atrial and ventricular ectopic beats. Patients successfully brought into sinus rhythm and those who failed to come into sinus rhythm were both affected.

34 of the 150 patients were receiving diuretic therapy (Table 1). No patient showed clinical evidence of electrolyte disturbance and no patient had shown electrolyte levels outside the normal range. Total body sodium and potassium, however, were not measured.

All patients except five (numbers 42, 70, 97, 126 and 133) were receiving a digitalis preparation as maintenance (Table 1). Patient number 70 and patient number 133 both came into sinus rhythm with frequent atrial premature contractions. Patient number 143 in whom an atrial tachycardia was related to digitalis overdosage came into sinus rhythm which was associated with frequent atrial and ventricular ectopic beats.

(i) Atrial Fibrillation (120 patients) Table 36.

The incidence of intervening rhythms is shown in Table 36. 31 patients developed atrial premature

TABLE 36

DIAGNOSIS	ATRIAL PREMATURE CONTRACTIONS				VENTRICULAR ECTOPIC BEATS				NODAL PREMATURE CONTRACTIONS				PROLONGED P-R INTERVAL			
	ENERGY joules			Q	ENERGY joules			Q	ENERGY joules			Q	ENERGY joules			Q
	< = 150	> 150			< = 150	> 150			< = 150	> 150			< = 150	> 150		
Mitral stenosis, post op.	12	2	2		5	7	5		4	1	2		1	1	-	
Aortic valve disease, post op.	-	-	-		1	-	1		1	-	-		-	-	-	
Mitral stenosis	1	-	1		1	-	1		-	-	-		-	-	-	
Mitral regurgitation	-	-	-		1	1	2		-	-	-		-	-	-	
Mitral stenosis and regurgitation	-	1	-			2	1		-	-	-		-	-	-	
Mitral and Aortic valve disease	2	-	-		2	-	-		-	-	-		-	-	-	
Alcoholic cardiomyopathy	-	3	1		-	2	1		-	1	1		-	-	-	
Cardiomyopathy	1	-	-		1	-	-		-	-	-		-	-	-	
Lone	2	3	3		-	5	1		-	-	-		-	1	-	
Atrial septal defect, post op.	1	1	1		-	3	2		-	-	-		-	-	-	
Post Thyrotoxicosis	1	-	-		-	-	-		-	-	-		-	-	-	
Chronic Ischaemic HD	-	-	-		1	2	-		-	-	-		-	-	-	
Syst. hypertension MI	1	-	1		1	-	1		-	-	-		-	-	-	
Cor.Transposition "MI"	-	-	-		-	1	-		-	-	-		-	-	-	
TOTALS	21	10	9		13	23	15		5	2	3		1	2	-	

DIAGNOSIS	WANDERING PACEMAKER				A.V. ÷ DISSOCIATION				BRADYCARDIA				ATRIAL FLUTTER			
	ENERGY joules			Q	ENERGY joules			Q	ENERGY joules			Q	ENERGY joules			Q
	< = 150	> 150			< = 150	> 150			< = 150	> 150			< = 150	> 150		
Mitral stenosis, post op.	3	1	-		1	-	-		1	2	-		-	1	-	
Aortic valve disease, post op.	-	-	-		-	-	-		-	1	-		-	-	-	
Mitral stenosis	-	-	-		-	-	-		-	-	-		-	-	-	
Mitral regurgitation	-	-	-		-	-	-		-	-	-		-	-	-	
Mitral stenosis and regurgitation	-	-	-		-	-	-		-	-	-		-	-	-	
Mitral and Aortic valve disease	-	-	-		-	-	-		-	-	-		-	-	-	
Alcoholic cardiomyopathy	-	2	-		-	-	-		-	2	1		-	-	-	
Cardiomyopathy	-	-	-		-	-	-		-	-	-		-	-	-	
Lone	-	-	-		-	-	-		-	-	-		-	-	-	
Atrial septal defect, post op.	-	-	-		-	-	-		-	-	-		-	-	-	
Post Thyrotoxicosis	1	-	-		-	-	-		-	-	-		-	-	-	
Chronic Ischaemic HD	-	-	-		-	-	-		-	-	-		-	-	-	
Syst. hypertension MI	-	-	-		-	-	-		-	-	-		-	-	-	
Cor.Transposition "MI"	-	-	-		-	-	-		-	-	-		-	-	-	
TOTALS	4	3	-		1	-	-		1	5	1		-	1	-	

Table 36: Intervening rhythms immediately after synchronised capacitor discharge in 120 patients who had atrial fibrillation.

Q. - Preceded by quinidine.

contractions 9 of whom had had quinidine 60 - 90 minutes before treatment. Energy level settings were 150 joules in 21 patients, and over 150 joules in 10. 3 patients received the direct current energy using antero-posterior paddles.

35 patients developed ventricular ectopic beats. 15 patients had received quinidine 60 - 90 minutes before treatment. Energy level settings used were up to 150 joules in 12 and over 150 joules in 23. Antero-posterior paddles were used in 5. Synchronised capacitor discharge failed to bring about the re-establishment of sinus rhythm in 5.

Nodal ectopic beats were less frequent. 7 instances were recorded and 3 patients had received quinidine 60 - 90 minutes before treatment. Patient number 104 had nodal rhythm for two minutes before the spontaneous establishment of regular sinus rhythm. The energy level setting varied from 150-250 joules, using the two anterior paddles in all.

A wandering pacemaker was found in 7 instances. No patient had received quinidine immediately before treatment. The energy level settings varied from 50-400 joules using two anterior paddles.

Transient prolongation of the P-R interval (in excess of 0.24 seconds) was noted in three instances. Energy

settings used were 150 joules, and 350 joules using two anterior paddles, and 250 joules using antero-posterior paddles. In all 5 the P-R interval became normal during the 24 hours following treatment.

Partial atrio-ventricular dissociation occurred in patient number 108 following synchronised capacitor discharge with an energy level setting of 150 joules using two anterior paddles. Normal conduction returned within five minutes and no specific treatment for the conduction disturbance was needed.

Bradycardia, defined as a slowing of the heart rate to less than 50 beats per minute occurred on six occasions following the re-establishment of sinus rhythm. Energy level settings used were respectively 150, 250, 300, 350, 350 and 400 joules and antero-posterior paddles were employed on 2 occasions. One patient only, had received quinidine 60 - 90 minutes before treatment. Bradycardia was always temporary, and usually lasted only for a few minutes but on one occasion lasted for 2 hours.

One patient (number 79) who had atrial fibrillation developed atrial flutter immediately after the establishment of sinus rhythm.

(ii) Atrial Flutter (16 patients).

Atrial premature contractions occurred in one patient only (number 107) who also showed prolongation of the P-R interval. An energy level setting of 150 joules (two anterior paddles) was used.

Patient number 149 developed frequent ventricular ectopic beats immediately after the re-establishment of sinus rhythm following discharge of an energy level setting of 50 joules (anterior paddles).

4 patients had nodal rhythm or ectopic beats. Nodal rhythm persisted for two hours in patient number 18. All 4 patients except for number 113, who received 10 joules, had an energy level setting of 150 joules.

Prolongation of the P-R interval occurred in patient number 107 who received 150 joules (anterior paddles).

Partial atrio-ventricular dissociation (2:1) followed synchronised capacitor discharge in patient number 6 who received 150 joules (anterior paddles). This lasted only for a few minutes and was followed by normally conducted sinus rhythm.

(iii) Atrial Tachycardia (9 patients)

Atrial premature contractions were noted in three instances. Two patients were treated with energy level settings of 50 joules (antero-posterior paddles) and one patient 150 joules (anterior paddles).

Ventricular ectopic beats were found in two patients following treatment. Patient number 31 was treated with an energy level setting of 150 joules (anterior paddles); patient number 143, received 50 joules (antero-posterior paddles).

Temporary nodal rhythm prior to the establishment of sinus rhythm occurred in patient number 109 (150 joules, anterior paddles).

(iv) Ventricular Tachycardia (5 patients)

Patient number 145 (150 joules, anterior paddles), developed ventricular ectopic beats and patient number 134 (50 joules, anterior paddles), developed nodal ectopic beats. In both the underlying heart disease was chronic ischaemia; neither had had quinidine immediately before treatment.

IV. COMPLICATIONS Tables 37, 38.

There were 21 patients out of 150 treated (14%) who subsequently developed complications thought to be directly

TABLE 37

PATIENT NO.	HEART DISEASE	ARRHYTHMIA	COMPLICATION
3	Lone	Atrial fibrillation	Hypotension. Pulmonary infarction. T wave inversion V_4-V_6
8	Mitral stenosis, post op.	Atrial fibrillation	Enzyme levels raised.
10	Cardiomyopathy, alcoholic	Atrial fibrillation	Enzyme levels raised.
12	Lone	Atrial fibrillation	Enzyme levels raised. Left ventricular third heart sound.
13	Lone	Atrial fibrillation	Enzyme levels raised. Hypotension.
14	Mitral stenosis, post op.	Atrial fibrillation	Enzyme levels raised.
27	Lone	Atrial fibrillation	Enzyme levels raised. Hypotension. T wave flat V_6. Left ventricular third HS.
36	Mitral stenosis, post op.	Atrial fibrillation	Enzyme levels raised.
41	Mitral stenosis	Atrial fibrillation	Increased CTR%. Pulmonary oedema.
45	Lone	Atrial fibrillation	Enzyme levels raised. Hypotension.
51	Mitral stenosis	Atrial fibrillation	Ventricular fibrillation. Death.
68	Cor.Transposition "MI"	Atrial fibrillation	Increased CTR%. Pulmonary oedema.
74	Mitral stenosis, post op.	Atrial fibrillation	Hypotension.
80	Cardiomyopathy	Ventricular tachy.	Enzyme levels raised. Increased CTR%. Pulmonary oedema.
90	Aortic valve dis, post op.	Atrial fibrillation	Changing rhythms. Cerebral embolus. Death

(continued on next page)

PATIENT NO.	HEART DISEASE	ARRHYTHMIA	COMPLICATION
102	Ischaemic HD, chronic	Atrial fibrillation	Increased CTR%. Pulmonary oedema.
123	Mitral stenosis, post MR.	Atrial fibrillation	Enzyme levels raised.
129	Acute myoc. infarction	Atrial tachy.	Saddle embolus.
134	Acute myoc. infarction	Ventricular tachy.	Ventricular fibrillation. Acute pulmonary oedema. Death.
138	Ischaemic HD, chronic	Atrial fibrillation	Enzyme levels raised. Increased CTR%. Pulmonary oedema. Prolonged multi-focal VE*.
142	Ischaemic HD, chronic	Atrial fibrillation	Increased CTR%. Pulmonary oedema.

Table 37: Complications in 21 patients following synchronised capacitor discharge.

HD - Heart sound
VE* - Ventricular ectopic beats.

(ii) Patient number 68 had atrial fibrillation in association with corrected transposition and regurgitation of the systemic atrio-ventricular valve. Sinus rhythm was established following a third shock with an energy level setting of 350 joules; shocks of 150 and 250 joules did not succeed. Hypotension was present for three hours after treatment. Orthopnoea was noted on the following day and the chest radiograph (Fig. 24) showed that in sinus rhythm the cardiothoracic ratio was 54% (51% in atrial fibrillation). Pulmonary oedema was present.

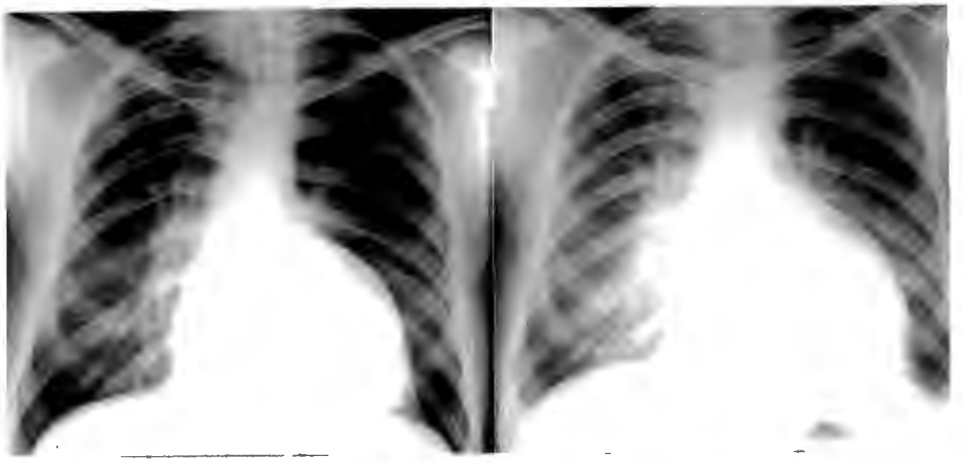


FIG. 24: Chest radiograph of Patient number 68 (corrected transposition). The upper left border of the cardiac shadow is formed by the ascending aorta. On the left the appearance in atrial fibrillation and on the right, in sinus rhythm. In sinus rhythm the heart is larger and pulmonary oedema is shown.

Note:

SGOT - Serum glutamic oxaloacetic transaminase.

LDH - Serum lactic dehydrogenase.

A/P refers to antero/posterior paddles.

related. These complications are listed in Table 37. Only complications other than transient rhythm changes and ring burns have been included under this heading.

1. RING BURNS:

Ring burns occurred in three patients at the position on the skin of the negative paddle (Fig.22). The erythema

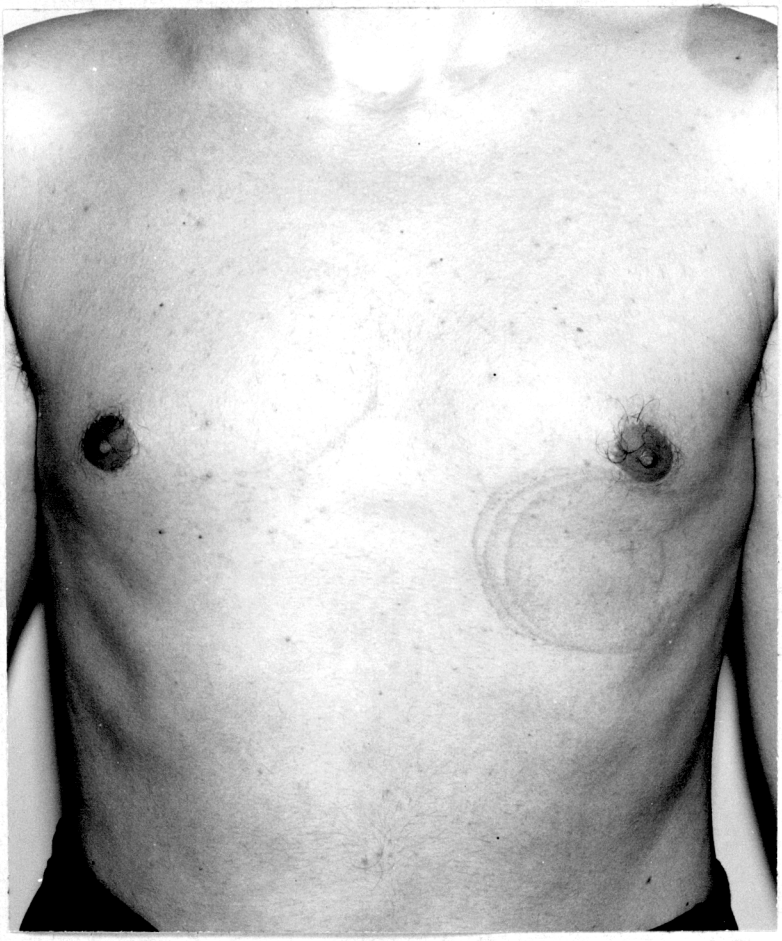


FIG. 22: Ring burns following synchronised capacitor discharge.

faded after 3-4 days. These superficial burns could be prevented by the liberal use of electrode jelly rubbed in well to the skin to lower the electrical resistance.

2. ENZYME CHANGE Table 38.

All patients, with the exception of patient number 113, a boy of 2½ years old, had their serum enzyme levels determined one day before treatment and one day after. Usually the serum levels of the glutamic oxaloacetic transaminase and lactic dehydrogenase were measured in each case but in 10 patients only one was estimated (S.G.O.T. in 9, L.D.H. in 1).

11 patients (numbers 8, 10, 12, 13, 14, 27, 36, 45, 80, 123 and 138) showed a significant rise in enzyme levels following treatment (Table 38). Patients numbers 12, 14, and 45 only had had serum levels of glutamic oxaloacetic transaminase measured, but in the remaining 8, both the S.G.O.T. and the L.D.H. were raised.

Energy level settings of 350 joules or more were used in 9 patients. An energy level setting of 250 joules was used in patient number 10 and 150 joules in patient number 80.

All except patient number 80 (ventricular tachycardia) were treated for atrial fibrillation. No underlying cause for the arrhythmia could be clinically detected in 4 of the patients with atrial fibrillation (numbers 12, 13, 27 and 43). The underlying heart conditions in the remaining patients were, post operative mitral stenosis in four (numbers 8, 14, 36 and 123), cardiomyopathy in two (numbers 10 and 80) and chronic ischaemic heart disease in patient number 138. The raised levels usually returned to the pre-treatment level within one to three days in respect of the S.G.O.T. and three to seven days in respect of the L.D.H.

Elevation of the serum enzyme levels occurred as an isolated complication in four (patient numbers 8, 10, 14 and 123) but was frequently associated with other complications (Table 38).

2. INCREASE IN HEART SIZE AND PULMONARY OEDEMA Table 38.

A significant increase in the post-treatment cardio-thoracic ratio was found in 6 patients (numbers 41, 68, 80, 102, 138, 142) (Table 38). The increase averaged 4 cm. (limits 1 - 6 cm.) and was invariably associated with pulmonary venous engorgement or frank pulmonary oedema.

(i) Patient number 41 had atrial fibrillation and mitral stenosis of a severity which, in the view of the referring physician did not warrant mitral valvotomy. Sinus rhythm was established after the third shock with an energy level setting of 350 joules, a first shock of 50 and a second of 150 joules had been unsuccessful. Regular sinus rhythm was maintained but one day later orthopnoea was present. The cardiothoracic ratio had risen from 47 - 52%, pulmonary oedema was present as well as horizontal lines of engorged intrapulmonary septal lymphatics (Kerley 1933) Fig.23).

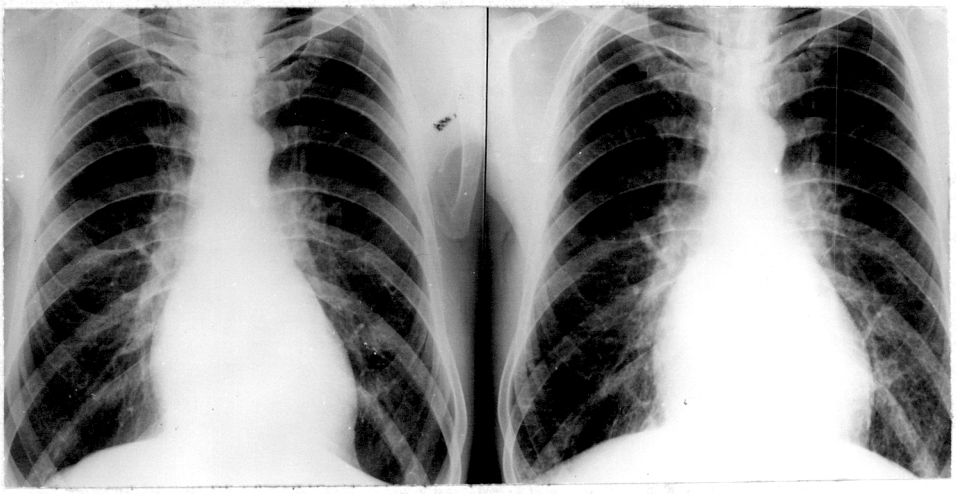


FIG. 23: Chest radiograph of Patient number 41 in atrial fibrillation on the left, and one day after the establishment of sinus rhythm on the right. In sinus rhythm the heart is larger, pulmonary oedema is shown and Kerley's lines are present.

(ii) Patient number 68 had atrial fibrillation in association with corrected transposition and regurgitation of the systemic atrio-ventricular valve. Sinus rhythm was established following a third shock with an energy level setting of 350 joules; shocks of 150 and 250 joules did not succeed. Hypotension was present for three hours after treatment. Orthopnoea was noted on the following day and the chest radiograph (Fig.24) showed that in sinus rhythm the cardiothoracic ratio was 54% (51% in atrial fibrillation). Pulmonary oedema was present.

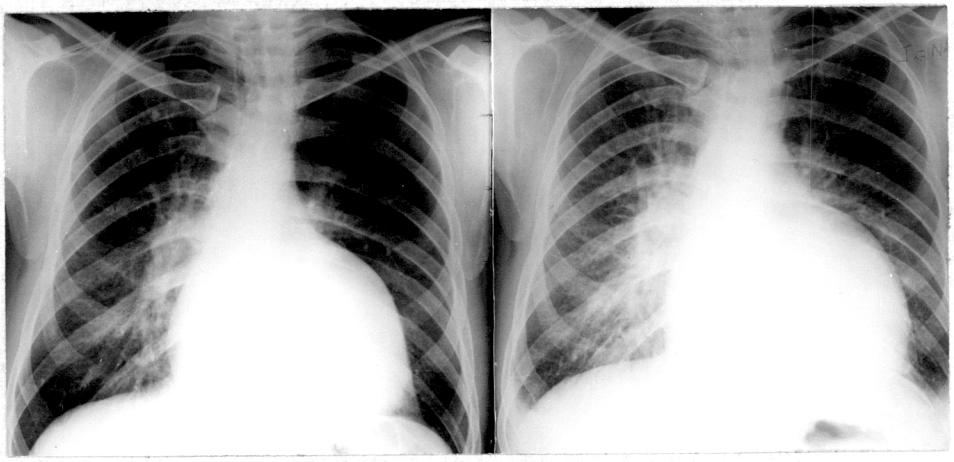


FIG. 24: Chest radiograph of Patient number 68 (corrected transposition). The upper left border of the cardiac shadow is formed by the ascending aorta. On the left the appearance in atrial fibrillation and on the right, in sinus rhythm. In sinus rhythm the heart is larger and pulmonary oedema is shown.

(iii) Patient number 80 had ventricular tachycardia in association with a cardiomyopathy of unknown aetiology. Sinus rhythm was established following a single shock of 150 joules. Hypotension was present for two hours following treatment. The serum levels of the glutamic oxaloacetic transaminase and of the lactic dehydrogenase were elevated on the day following treatment (Table 38). The chest radiograph in sinus rhythm showed gross pulmonary oedema (Fig.25).

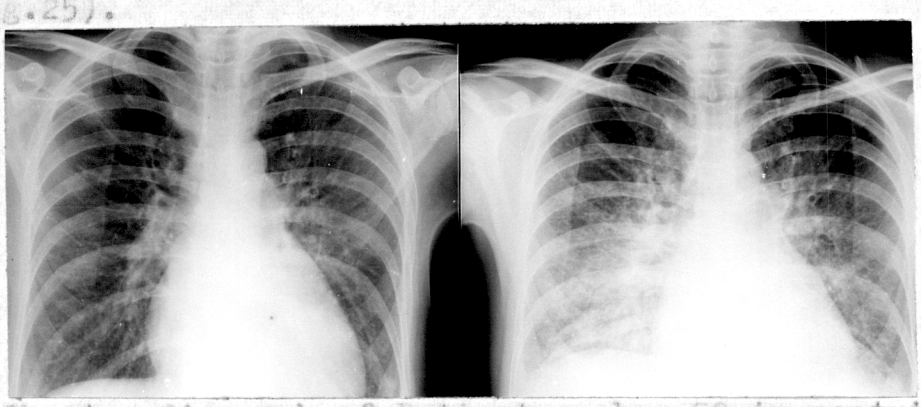


FIG. 25: Chest radiograph of Patient number 80 in ventricular tachycardia on the left, and sinus rhythm on the right. Gross pulmonary oedema is shown in sinus rhythm.

(iv) Patient number 102 had atrial fibrillation in association with chronic ischaemic heart disease. Sinus rhythm was established after a third shock with an energy level setting of 350 joules. Shocks of 150 and 250 joules had not succeeded. One day later the repeat chest radiograph showed the cardiothoracic ratio to be 50% (46% in atrial fibrillation) and slight pulmonary oedema was present. (Fig.26).

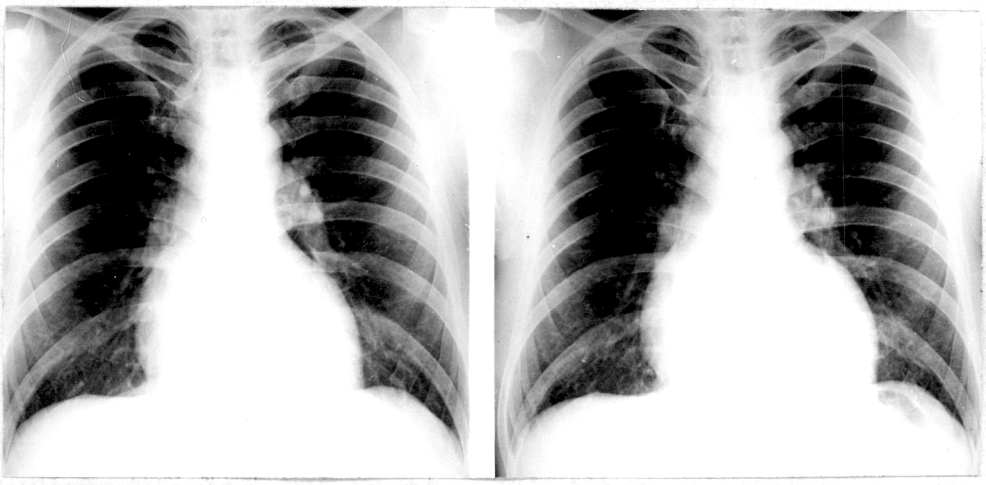


FIG. 26: Chest radiograph of Patient number 102, in atrial fibrillation on the left and sinus rhythm on the right. The CTR is 48% in atrial fibrillation and 53% in sinus rhythm.

(v) Patient number 138 had atrial fibrillation in association with chronic ischaemic heart disease. Sinus rhythm was established following a fourth shock with an energy level setting of 400 joules. Shocks of 50, 150 and 250 joules had not succeeded. All four shocks were delivered using antero-posterior paddles. A repeat chest radiograph showed that the cardiothoracic ratio was 53% in sinus rhythm (48% in atrial fibrillation) and pulmonary oedema was present (Fig.27). A significant increase in the serum levels of glutamic oxaloacetic transaminase and lactic dehydrogenase occurred (Table 38) and persistent multifocal ventricular ectopic beats were recorded (Fig.28).

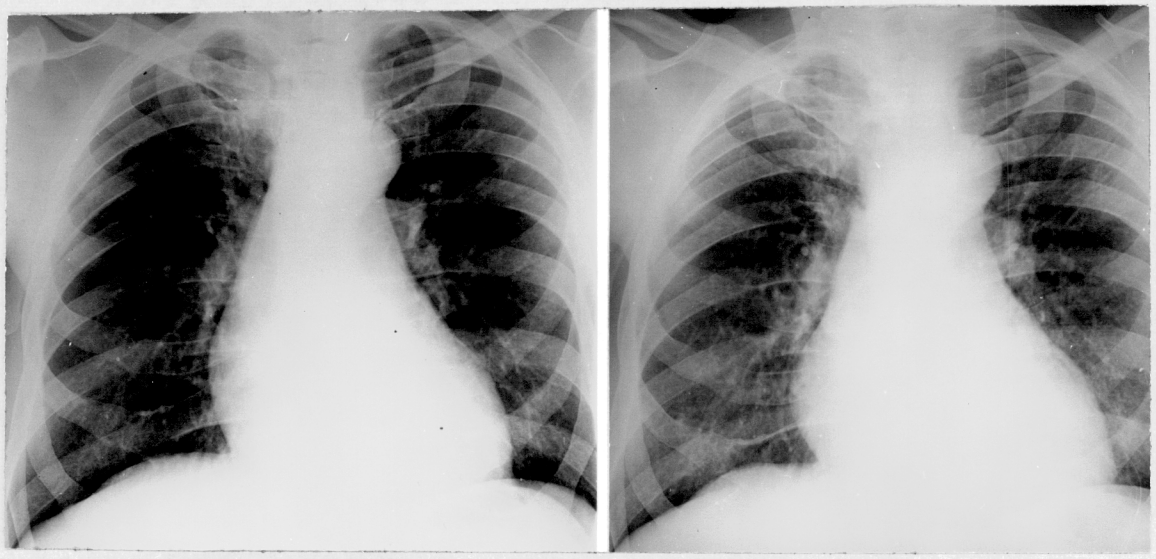


FIG. 27: Chest radiograph of Patient number 138, in atrial fibrillation on the left, and sinus rhythm on the right. The CTR is 48% in atrial fibrillation and 53% in sinus rhythm.

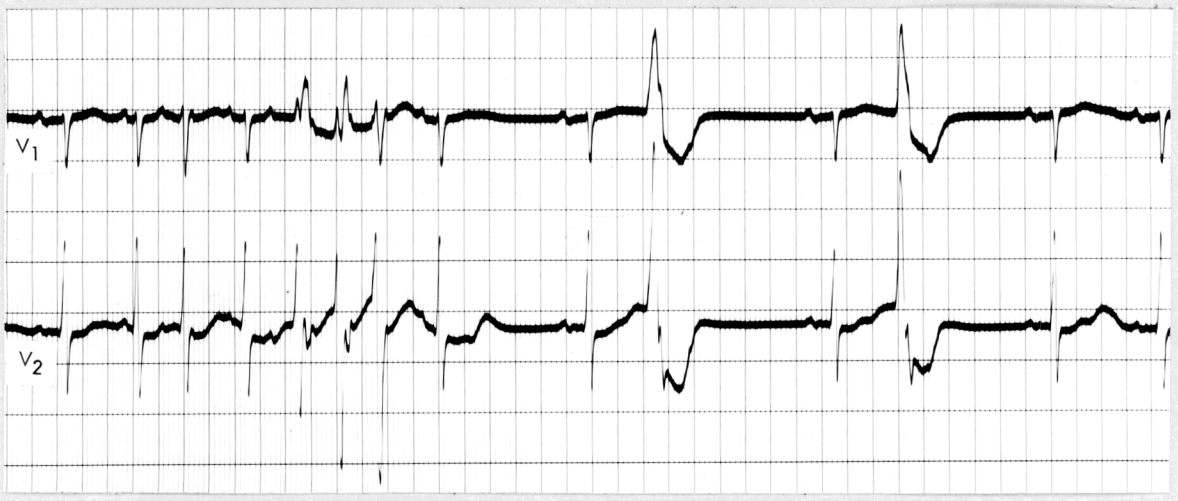


FIG. 28: ECG leads V_1, and V_2 (simultaneous) of Patient number 138 to show atrial premature contractions and multifocal ventricular ectopic beats one day after synchronised capacitor discharge.

(vi) Patient number 142 had atrial fibrillation in association with chronic ischaemic heart disease. Sinus rhythm was established following a fourth shock with an energy level setting of 200 joules (antero-posterior paddles). Shocks of 50, 100, 150 joules had not succeeded. The chest radiograph one day later showed an increase in the cardiothoracic ratio (48% in atrial fibrillation, 54% in sinus rhythm) and pulmonary venous engorgement was present (Fig.29).

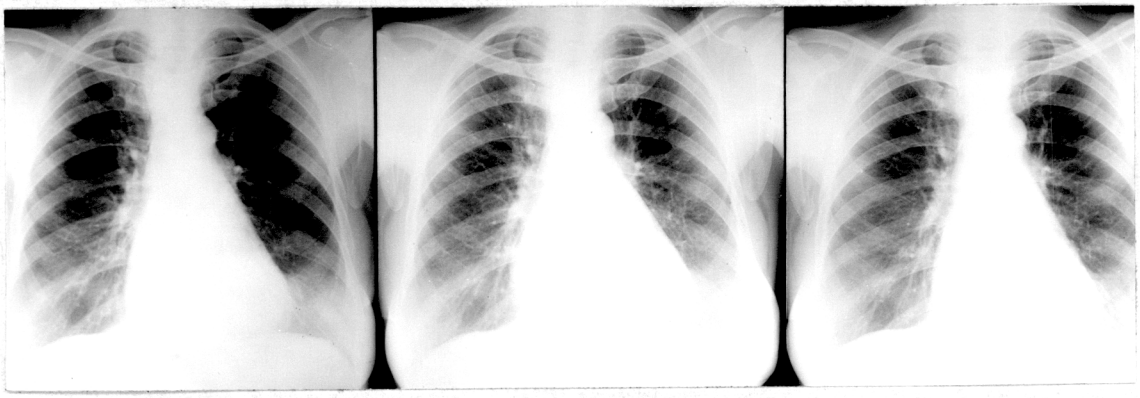


FIG. 29: Chest radiograph of Patient number 142, 1 day before synchronised capacitor discharge on the left, 1 day after (middle) and 2 days after DC shock (right). Note the increase in the size of the heart 1 day after electrical conversion.

The increase in size of the heart in all six patients usually lasted for 1 - 2 days and always responded to bed-rest, re-starting a digitalis preparation and a diuretic. Patient number 138 who in addition had multifocal ventricular ectopic beats (Fig.28) some of which showed the R on T phenomenon (Smirk and Palmer, 1960) was also treated with quinidine, 600 mg. each 6 hours over 48 hours.

3. HYPOTENSION

A systolic blood pressure of 80 mm. Hg. or less lasting for one hour or longer following synchronised capacitor discharge was classified as hypotension.

Hypotension followed the treatment in 7 patients (Tables 37, 38). In all 7, the fall in blood pressure followed the direct current shock and was not present after induction of the anaesthetic and during the time interval that elapsed whilst the patient was anaesthetised but before the shock.

(i) Patient number 3 developed hypotension after a third shock with an energy level setting of 200 joules. Previous shocks of 100 and 150 joules were unsuccessful but did not cause a fall in blood pressure. Hypotension persisted for 4 hours. Pulmonary infarction developed one day after treatment, and in addition the electrocardiogram in sinus rhythm showed inversion of T waves in leads V 4 - 6 which persisted for 2 days. There was no elevation of the serum levels of lactic dehydrogenase or glutamic oxaloacetic transaminase.

(ii) Patient number 13 who received a total of 5 shocks of which the final was at an energy level setting of 400 joules, was hypotensive (B.P. 80/40 mm. Hg.) for one hour after treatment. In addition a significant elevation of the serum enzyme levels occurred.

(iii) Patient number 27 was given a fourth shock with the maximum energy level setting (400 joules). Hypotension which lasted for three hours followed, a considerable rise in the serum enzyme levels occurred (S.G.O.T. 21 - 168 units, L.D.H. 110 - 800 units), the T wave in lead V6 became flat and on auscultation a loud left ventricular third heart sound was noted.

(iv) Patient number 45 was given a third shock with an energy level setting of 350 joules. Hypotension, (B.P. 70/50 mm. Hg.) followed and persisted for three hours. A significant elevation in the serum level of the glutamic oxaloacetic transaminase was associated.

(v) Patient number 68 received a third shock with an energy level setting of 350 joules. Hypotension followed and persisted for three hours; a chest radiograph in sinus rhythm showed pulmonary oedema to be present, as well as a significant increase in the cardiothoracic ratio.

(vi) Patient number 74 was converted from atrial fibrillation to sinus rhythm following a fourth shock with an energy level setting of 350 joules. A blood pressure reading of 80/50 mm. Hg. followed and persisted for two hours.

(vii) Patient number 80 developed hypotension following a single shock of 150 joules for the treatment of ventricular tachycardia. Hypotension persisted for two hours. Pulmonary oedema occurred one day after treatment and a significant elevation of the serum enzyme levels was noted.

Patients numbers 13, 27, 45 and 74 had been given quinidine shortly before synchronised capacitor discharge.

None of the seven patients was given a sympathomimetic drug to raise the blood pressure which always recovered by 4 hours after treatment.

4. ELECTROCARDIOGRAPHIC CHANGES

Significant changes, other than rhythm, were noted in two patients (numbers 3 and 27). (Tables 37 and 38).

(i) Patient number 3 developed inversion of T waves in leads V 4 - 6 which persisted for 2 days.

(ii) Patient number 27 developed a flat T wave in lead V6 (Fig.30) which persisted for one day.

In both patients associated complications were also present (Tables 37 and 38).

5. EMBOLISM

Three patients (numbers 3, 90, 129) developed embolism. (Tables 37 and 38).

(i) Patient number 3 has already been described under Hypotension and Electrocardiographic changes. In addition he developed pleuritic pain on the day following synchronised capacitor discharge and a repeat chest X-ray showed linear atelectrosis on the right, a result of pulmonary infarction (Fig.31). Treatment for atrial fibrillation had been undertaken with satisfactory anticoagulant control. The pulmonary infarcts were small and did not require specific supportive measures.

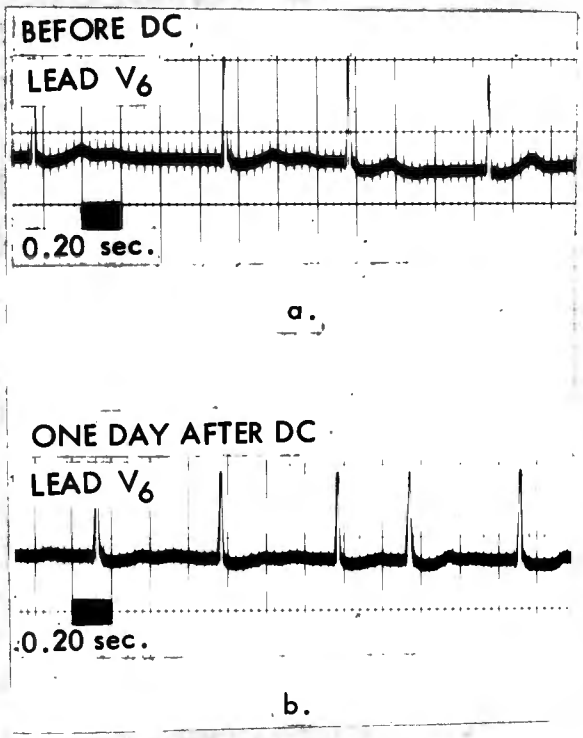


FIG. 30: EOG Lead V_6 a) One day before synchronised capacitor discharge.
b) One day after synchronised capacitor discharge.

Note: Flat T waves following the attempt at electrical conversion.

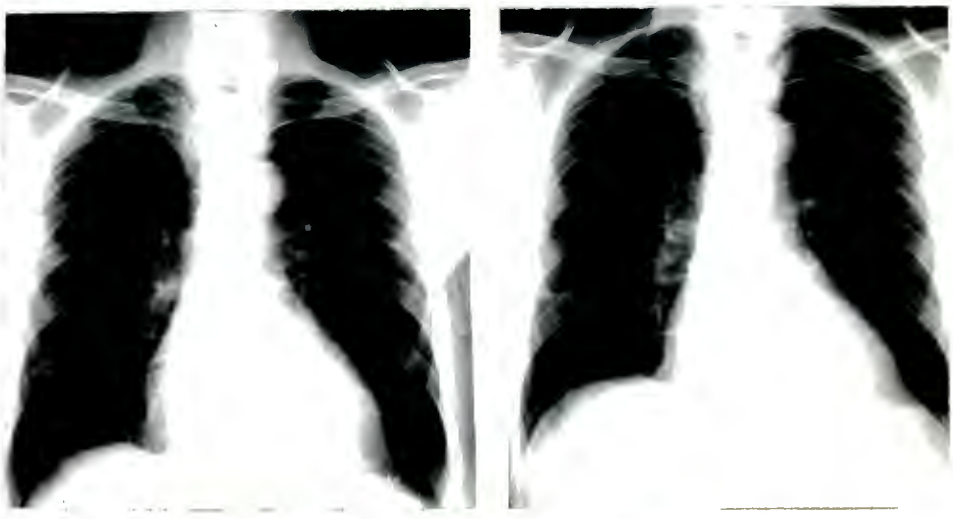


FIG. 31: Chest radiograph of patient number 3 in atrial fibrillation on the left and one day after successful synchronised capacitor discharge on the right. Linear atelectactic shadowing is shown in the right lung in sinus rhythm indicating pulmonary infarction.

(ii) Patient number 30 developed atrial fibrillation following surgery for calcific aortic stenosis performed at another hospital. Attacks of paroxysmal atrial tachycardia occurred 6 weeks after the establishment of sinus rhythm during which she developed a cerebral embolus which resulted in a dense hemiplegia. Death in uraemia occurred six months later. Anticoagulant therapy was not being given when the cerebral embolus occurred.

(iii) Patient number 129 developed atrial tachycardia in association with a recent myocardial infarct. Sinus rhythm was established following a single shock with an energy level setting of 50 joules. Anticoagulant therapy was not being given at the time. Six hours later a saddle embolus occurred at the bifurcation of the aorta. The circulation was re-established, following surgical removal of the embolus two hours later and he made an uneventful recovery.

6. DEATHS FOLLOWING TREATMENT

Death was directly or indirectly related to synchronised capacitor discharge in 3 patients (numbers 51, 90, 134). There were 3 other patients who died following synchronised capacitor discharge (numbers 38, 64, 101) but death was not related to the electrical treatment. These latter patients are discussed under Follow-up.

(i) Patient number 51 had atrial fibrillation in association with mitral stenosis. Sinus rhythm was achieved following a second shock with an energy level setting of 150 joules. His condition in no way gave rise to anxiety after the treatment. Constant oscilloscopic monitoring of the electrocardiogram was undertaken for twenty-four hours as part of the routine post-treatment care and revealed a stable rhythm with no atrial or ventricular ectopic beats. There was no elevation of the serum enzyme levels following the shocks. Maintenance quinidine 300 mg. every 6 hours was prescribed. Ventricular fibrillation developed 36 hours later and failed to respond to emergency resuscitative measures. A post mortem examination (courtesy of Dr. Reginald Hudson) revealed only moderate mitral stenosis (mitral orifice 1.5 sq. cm.) and despite a careful histological examination of the heart muscle and of the conducting tissue, no evidence of structural damage resulting from the direct current shocks could be found (Hudson, 1964).

(ii) Patient number 90 has already been referred to under Embolism. Her death occurred seven months after the re-establishment of sinus rhythm. A cerebral embolus occurred six weeks after the treatment during a paroxysmal attack of atrial tachycardia and subsequently persisting atrial fibrillation recurred. Although aortic valvotomy had relieved aortic stenosis she was left with considerable aortic regurgitation. Progressive renal failure occurred and she died in uraemia.

(iii) Patient number 134 was referred from a hospital at some considerable distance away because of a ventricular tachycardia which had been present for two days, was resistant to drug therapy and had followed an acute myocardial infarct nine days before. On arrival at the National Heart Hospital he was in extremis and in severe left ventricular failure. A rapid ventricular tachycardia was present. Immediate preparations were made for synchronised capacitor discharge without anaesthesia. Cardiac arrest supervened whilst the electrodes were being applied to the limbs despite the fact that ventricular tachycardia still occurred. A single shock with an energy level setting of 50 joules synchronised to avoid the vulnerable phase of ventricular repolarisation was given. Ventricular fibrillation resulted. External cardiac massage and artificial respiration were maintained for one minute and a second shock of 50 joules, which was unsynchronised, was given. Sinus rhythm, regular other than for occasional ventricular ectopic beats followed and a dramatic improvement in the circulation occurred. Acute pulmonary oedema was present which responded to oxygen, intravenous digitalisation, intravenous frusemide and correction of a metabolic acidosis. His general condition continued to improve over 24 hours and regular sinus rhythm was maintained. Cardiac arrest then occurred once more and although electrical beating of the heart was shown on the electrocardiogram the mechanical force of contraction was not sufficient to maintain an adequate circulation and he died despite prolonged attempts to maintain and support the circulation artificially.

7. GALLOP RHYTHM.

Patients numbers 12 and 27 developed a loud left ventricular third heart sound on the day following synchronised capacitor discharge. The phonocardiogram recorded from patient number 12 is shown in Fig.32. The serum enzyme levels were also raised in both patients.

8. OTHER

Patient number 36 developed a fever of 106°F on the night following synchronised capacitor discharge. Blood cultures confirmed a diagnosis of acute bacterial endocarditis (Staph.aureus). Infection was thought to have been introduced at the time of mitral valvotomy 3 weeks previously.

V. HAEMODYNAMIC STUDIES.

The results in the 6 patients studied for heart rate changes and physical work capacity in atrial fibrillation and in sinus rhythm are detailed in Table 39.

The results in the 14 patients who were more extensively studied are detailed in Table 40.

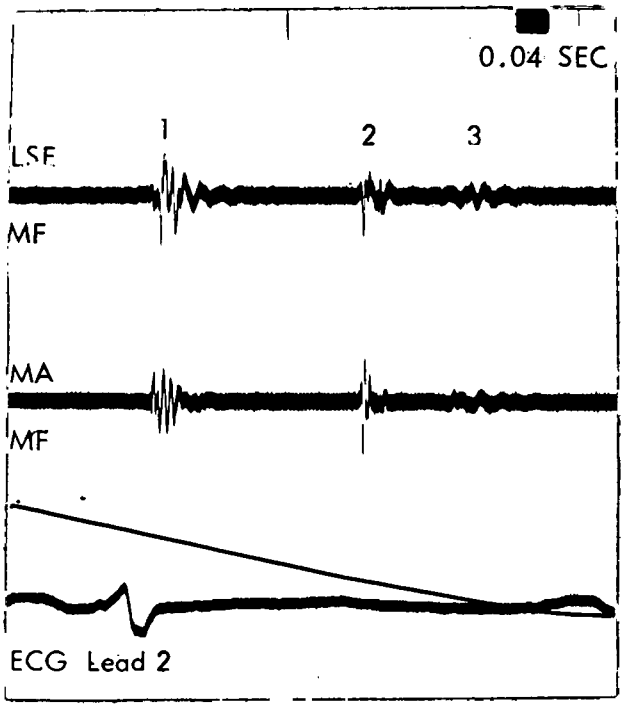


FIG. 32: Phonocardiogram recorded following synchronised capacitor discharge at medium frequency (MF) from the left sternal edge (LSE) and mitral area (MA) synchronous with the ECG, lead 2 and the respiratory cycle. A third heart sound, not present before the electrical conversion is now shown at a time when the serum level of glutamic oxaloacetic transaminase rose from 20 to 70 units.

TABLE 39

PATIENT AGE SEX ARRHYTHMIA HEART DISEASE	EXERCISE kpm/min	ARRHYTHMIA HEART RATE /min	ARRHYTHMIA O_2 UPTAKE ml	ARRHYTHMIA TOTAL WORK kpm	ARRHYTHMIA PWC 170	SINUS RHYTHM HEART RATE /min	SINUS RHYTHM O_2 UPTAKE ml	SINUS RHYTHM TOTAL WORK kpm	SINUS RHYTHM PWC 170
D.F.D.	REST	90	304	-		71	325	-	
38 M	200	150	718	1200	300	84	752	1200	930
AF	400	190	1140	2400		112	1138	2400	
Post thyrotox	600	220	1509	3600/7200		130	1475	3600/7200	
D.E.H.	REST	130	345	-		75	322	-	
57 F	200	180	728	1200	<200	130	718	1200	400
AF Lone	300	220	956	1800/3000		150	968	1800/3000	
J.C.	REST	90	543	-		65	515	-	
49 M	200	110	935	1200	-	75	910	1200	-
AF IHD	400	130	1310	2400/3600		86	1287	2400/3600	
J.L.	REST	100	210	-		73	202	-	
55 F	100	120	456	600		92	478	600	380
AF	200	145	710	1200	280	119	690	1200	
IHD	300	180	955	1800/3600		145	938	1800/3600	
LB	REST	120	510	-		105	522	-	
60 M	200	160	932	1200	360	125	960	1200	600
AF	400	180	1385	2400		150	1485	2400	
MS.MI	600	200	1880	3600/7200		178	1860	3600/7200	
TP	REST	68	362	-		75	378	-	
62 M	200	110	725	1200		95	715	1200	750
AF	400	150	1020	2400	490	120	1038	2400	
IHD	600	195	1365	3600/7200		155	1385	3600/7200	

Table 39: Results of heart rate and the physical work capacity measured in 6 patients first in atrial fibrillation and then repeated in sinus rhythm at rest and on exercise

TABLE 40

PATIENT AGE SEX ARRHYTHMIA DISEASE	EXERCISE kpm/min	ARRHYTHMIA HEART RATE /min	O_2 UPTAKE ml/min	O_2 CONTENT ml/L PA	O_2 CONTENT ml/L BA	AVO_2 DIFF ml	C.O. L/min	S.V. ml	*PRESSURE mm Hg PA	PRESSURE mm Hg BA	TOTAL WORK kpm	PAC 170	HEART RATE /min	O_2 UPTAKE ml	O_2 CONTENT ml/L PA	O_2 CONTENT ml/L BA	AVO_2 DIFF ml	C.O. L/min
DC	REST	100	352	145.8	202.0	56.2	6.3	63	20/15	110/65	-		82	360	144.6	202.8	58.2	6.2
62 M	200	130	726	112.8	202.3	89.5	8.1	62.5	27/15	135/80	1400		100	710	135.4	202.0	66.6	10.7
AF	400	175	1106	88.8	202.8	114.0	9.7	55.5	30/13	145/80	2457	380	120	1090	130.3	202.5	72.2	15.1
IHD	500	195	1310	78.2	203.0	124.8	10.5	54.0	30/17	160/75	3000/6857		128	1290	127.8	202.3	74.5	17.3
	600												138	1450	124.7	202.7	78.0	18.6
D.H.	REST	57	348	105.0	182.0	77.0	5.1	89	25/10	120/80	-		67	342	112.0	181.8	69.8	4.5
49 M	200	100	791	68.1	182.2	114.1	7.0	70	25/8	125/85	1200		92	789	78.3	182.2	103.9	7.6
AF	400	140	1198	51.2	181.7	130.5	9.2	66	28/12	130/80	2400	575	125	1210	63.1	181.9	118.8	10.3
AOVD, post op.	600	180	1580	39.7	182.4	142.7	11.1	62	30/12	140/80	3600/7200		150	1576	59.0	182.4	123.4	12.8
EC	REST	120	380	96.0	179.0	83.0	4.6	37	20/10	150/100	-		88	364	97.1	180.4	83.3	4.3
65 M	200	140	758	71.0	182.0	111.0	6.9	44	25/12	175/110	1233		105	787	75.0	184.0	109.0	7.2
AF	400	160	1312	57.0	184.2	127.2	10.3	64	30/15	210/125	2533	500	130	1330	62.2	185.4	123.2	11.0
IHD	600	180	1880	47.9	186.1	138.2	13.5	75	30/15	210/120	3700/7466		160	1910	54.1	188.1	134.0	14.3
EK	REST	85	170	94	171	87	2.2	26	38/19	130/70	-		68	186	90	171	81	2.3
52 F	100	150	409	61	183	123	3.3	22	45/22	140/70	600	200	110	498	72	183	110	4.4
AF Obst. cardiomy.																		
G.E.	REST	57	386	87.5	161.5	74.0	5.2	91	24/12	110/65	-		72	380	91.4	163.0	71.6	5.3
51 F	100	120	721	60.9	161.7	100.8	6.8	57	28/15	120/60	600		85	726	63.7	163.0	99.3	7.3
AF	200	125	1019	42.5	161.6	119.1	8.5	68	32/15	125/60	1200	1170	100	1037	54.0	163.3	109.3	9.4
Mitral+AOVD	300	130	1350	29.5	161.8	132.3	10.1	78	35/18	130/55	1800/3600		110	1342	47.0	163.1	116.1	11.6

Table 40: Results of haemodynamic studies in 14 patients 12 of whom had atrial fibrillation and 2 atrial flutter; before and after conversion to sinus rhythm by synchronised capacitor discharge, at rest and on exercise.

SINUS RHYTHM

S.V. ml	*PRESSURE mm Hg PA	BA	TOTAL WORK kpm	PWC 170
74.5	23/17	110/70	-	
107	30/15	130/70	1217	
126	30/17	135/75	2500	
135	30/15	135/75	3000	920
135	35/15	150/80	3600 / 10317	
73	28/10	125/70	-	
83	32/12	128/70	1200	
81.6	35/12	130/65	2400	738
85	35/12	135/65	3600 / 7200	
50	25/17	140/95	-	
68.5	25/18	160/110	1300	
91	35/20	200/110	2533	670
119	40/20	210/120	3900 / 7733	
32	40/20	140/70	-	
41	48/23	150/75	600	300
73	23/11	110/60	-	
86	25/12	115/60	600	
95	30/12	125/65	1200	790
105	35/15	135/65	1500 / 3600	

TABLE 40 (continued 1)

PATIENT AGE SEX ARRHYTHMIA DISEASE	EXERCISE kpm/min	ARRHYTHMIA HEART RATE /min	O_2 UPTAKE ml/min	O_2 CONTENT ml/L PA	O_2 CONTENT ml/L BA	AVO_2 DIFF ml	C.O. L/min	S.V. ml	*PRESSURE mm Hg PA	PRESSURE mm Hg BA	TOTAL WORK kpm	PWC 170	HEART RATE /min	O_2 UPTAKE ml	O_2 CONTENT ml/L PA	O_2 CONTENT ml/L BA	AVO_2 DIFF ml	C.O. L/min
G.S.	REST	126	420	143.8	215.0	71.2	5.9	47	20/10	130/85	-		100	426	145.0	200.0	55.0	7.7
50 M	200	130	838	116.4	215.0	98.6	8.5	65	20/10	130/70	1500		104	845	118.0	200.3	82.3	10.3
AF	400	165	1229	108.2	215.4	107.2	11.5	70	30/10	135/70	2867	430	120	1220	106.7	200.8	94.1	13.1
Post thyrotox.	600	200	1608	104.0	215.6	111.6	14.5	72.5	45/20	140/70	3700 / 8067		140	1620	97.3	200.5	103.2	15.8
H.C.	REST	100	355	141.5	198.0	56.5	6.3	63	17/8	130/85	-		85	368	131.2	188.7	57.5	6.4
54 M	200	180	775	94.8	191.0	96.2	8.1	45	30/15	175/115	1300		110	760	96.3	185.6	89.3	8.5
AF	400	190	1125	76.9	190.7	113.8	9.9	58.5	30/20	175/105	2500	<200	135	1115	82.1	189.3	107.2	10.4
Alc. Cardiomy.	600	200	1510	60.7	190.7	130.0	11.6	58	40/25	210/120	3600 / 7400		160	1520	69.8	194.8	125.0	12.4
H.H.B.	REST	80	343	104.7	170.5	65.8	5.2	65	20/10	120/75	-		68	334	107.0	170.0	63.0	5.3
53 M	200	100	780	84.0	174	90.0	8.7	87	20/10	130/70	1200		100	755	95.0	175.0	80.0	9.4
AF	400	122	1190	70.5	170.5	100.0	11.9	97.5	22/10	130/65	2400	810	118	1185	84.5	171.2	86.7	13.7
Lone	600	145	1585	65.8	170	104.2	15.2	104	25/12	135/65	3600 / 7200		134	1580	83.0	172.0	89.0	17.8
J.H.	REST	133	380	143.8	209.3	65.5	5.8	43.5	35/16	130/80	-		88	386	143.5	209.2	65.7	5.9
60 M	200	142	791	105.2	218.8	113.6	7.0	49	35/10	135/80	1500		108	802	110.4	212.2	101.8	7.5
A FLUT	400	155	1191	78.2	219.0	140.8	8.1	52	35/10	140/75	3734	600	120	1190	95.8	214.8	119.0	10.0
LONE	600	170	1585	51.3	221.4	170.1	9.3	55	33/10	140/70	3600 / 8834		135	1578	87.1	218.6	131.5	12.0
K.P.	REST	70	322	80.9	178.4	97.5	3.3	47	20/10	130/80	-		65	318	92.5	189.0	96.5	3.1
36 M	100	90	495	68.4	178.6	110.2	4.5	45	23/12	130/70	600		80	503	82.0	189.3	107.3	4.8
AF	300	110	855	47.8	178.1	130.3	6.4	58	35/15	135/70	1800	900	100	848	66.2	188.8	122.6	6.1
Cardiomyop.	500	130	1170	38.9	178.0	147.1	8.0	61.5	45/18	135/70	2833 / 4233		120	1165	61.0	188.9	127.9	9.2

SINUS RHYTHM

S.V. ml	*PRESSURE mm Hg PA	BA	TOTAL WORK kpm	PWC 170
77	21/6	130/70	-	
99	25/15	135/70	1267	
109	30/18	145/80	2567	915
113	45/25	145/80	3600 7434	
75	25/12	150/85	-	
77	35/22	180/85	1234	
77	45/25	200/100	2433	675
77.5	50/25	225/115	3600 7267	
78	20/10	120/75	-	
94	20/10	120/70	1200	
116	23/12	130/65	2400	1020
135	26/12	130/65	3600 7200	
67	33/15	130/80	-	
72	35/10	130/80	1550	
83	35/10	140/70	2600	660
89	35/10	140/70	3250 7400	
52	20/8	135/70	-	
60	28/12	130/65	600	
69	40/17	135/65	1800	1000
76	48/22	140/70	2333 4733	

TABLE 40 (continued 2)

PATIENT AGE SEX ARRHYTHMIA DISEASE	EXERCISE kpm/min	HEART RATE /min	O_2 UPTAKE ml/min	O_2 CONTENT ml/L PA	O_2 CONTENT ml/L BA	AVO_2 DIFF ml	C.O. L/min	S.V ml	BLOOD PRESSURE mm Hg PA	BLOOD PRESSURE mm Hg BA	TOTAL WORK kpm	PVC 170	HEART RATE /min	O_2 UPTAKE ml	O_2 CONTENT ml/L PA	O_2 CONTENT ml/L BA	AVO_2 DIFF ml	C.O. L/min
						ARRHYTHMIA												
L.D.	REST	50	324	118.5	180.7	62.2	5.2	104	22/10	110/70	-		65	330	117.0	181.0	64.0	5.1
34 F	200	100	748	91.7	180.2	88.5	8.45	84	25/10	120/70	1200		95	742	99.9	181.4	81.5	9.1
AF	400	135	1155	82.2	181.0	98.8	11.7	87	28/12	115/65	2400	600	114	1162	91.7	181.7	90.0	12.9
M.S. Post op.	600	170	1560	77.3	182.3	105.0	14.9	76	30/12	120/65	$\frac{3600}{7200}$		138	1568	88.3	182.1	93.8	16.7
L.L.	REST	110	357	95.8	170.8	75.0	4.75	43	18/8	140/70	-		71	350	100.2	170.2	70.0	5.0
60 M	200	140	875	54.9	170.9	116.0	7.56	54	20/8	150/90	1200		100	882	69.1	170.4	101.3	8.7
AF	400	160	1392	34.7	170.7	136.0	10.2	64	20/8	160/100	2400	500	117	1398	57.3	170.3	113.0	12.3
LONE	600	180	1810	30.8	170.8	140.8	12.9	71.	20/10	165/100	$\frac{3600}{7200}$		135	1830	56.6	170.6	114.0	16.1
M.M.	REST	95	333	131.6	199.0	67.4	4.9	52	15/8	130/90	-		90	349	120.2	196.2	76.0	4.6
51 M	200	115	769	91.2	189.8	98.6	7.8	68	17/10	130/80	1234		102	814	102.6	197.3	94.7	8.6
A FLUT	400	132	1148	104.5	189.7	105.2	10.9	83	10/10	130/75	2467	820	116	1320	94.5	196.8	102.3	12.8
LONE	600	150	1594	85.2	199.2	114.0	14.0	93	22/12	135/75	$\frac{3600}{7301}$		132	1780	89.7	196.4	106.7	16.7
	800												150	2336	80.5	196.7	116.2	20.6
P.Q.	REST	85	370	135.6	199.5	63.9	5.8	67	22/15	130/90	-		60	380	136.7	200.0	63.3	6.0
42 M	200	100	785	96.1	203.5	107.4	7.3	73	22/10	130/90	1250		74	770	103.8	202.3	98.5	7.8
AF	400	112	1157	79.9	210.0	130.1	8.90	78.	22/12	140/90	2470	630	92	1145	85.6	209.8	119.2	9.6
LONE	600	125	1532	66.2	216.2	150.0	10.2	81.	25/12	140/90	$\frac{3850}{7570}$		112	1525	75.2	208.9	133.7	11.4
	800												130	1980	62.7	212.7	150.0	13.2

SINUS RHYTHM

S.V. ml	*PRESSURE mm Hg PA	BA	TOTAL WORK kpm	PWC 170
79	25/12	115/70	-	
96	22/10	115/65	1200	
113	28/12	120/60	2400	850
120	28/12	120/60	3600 7200	
70.5	15/6	145/80	-	
87	18/8	160/100	1200	
100	18/8	165/110	2400	990
119	20/10	170/110	3600 7200	
96	12/8	120/75	-	
84	15/10	120/70	1217	
110	15/8	125/70	2467	
139	20/10	130/65	4050	1050
137	20/10	140/70	4934 12668	
100	25/12	130/90	-	
106	25/12	140/80	1366	
104	28/12	140/90	2450	
102	30/12	135/85	3600	1220
100	30/12	135/85	4800 15216	

* Pressure measured with reference to the sternal cycle

1. HEART RATE (20 patients) Tables 39 and 40.

The heart rate was related in a linear fashion to the work load both during the arrnythmia and in sinus rhythm.

The average heart rate at rest in the 20 patients during the arrhythmia was 93/min and varied from 50 - 133/min (S.D. 24.02). Following the establishment of sinus rhythm the average heart rate was 76/min and varied from 60 - 105/min (S.D. 12.33).

The average heart rate on maximal effort during the arrhythmia in the 20 patients was 173/min and varied from 130 - 220/min (S.D. 28.94).

Once sinus rhythm had been established the heart rate averaged 138/min and varied from 86 - 178/min (S.D. 20.12) when measured at the maximal work load in a steady state.

The difference between the heart rates during the arrhythmia and in sinus rhythm was exaggerated as the work load increased in some but not all the patients. This relationship can be seen in Fig.33 in which the mean heart rates at rest and during exercise is plotted against the work load in the 20 patients.

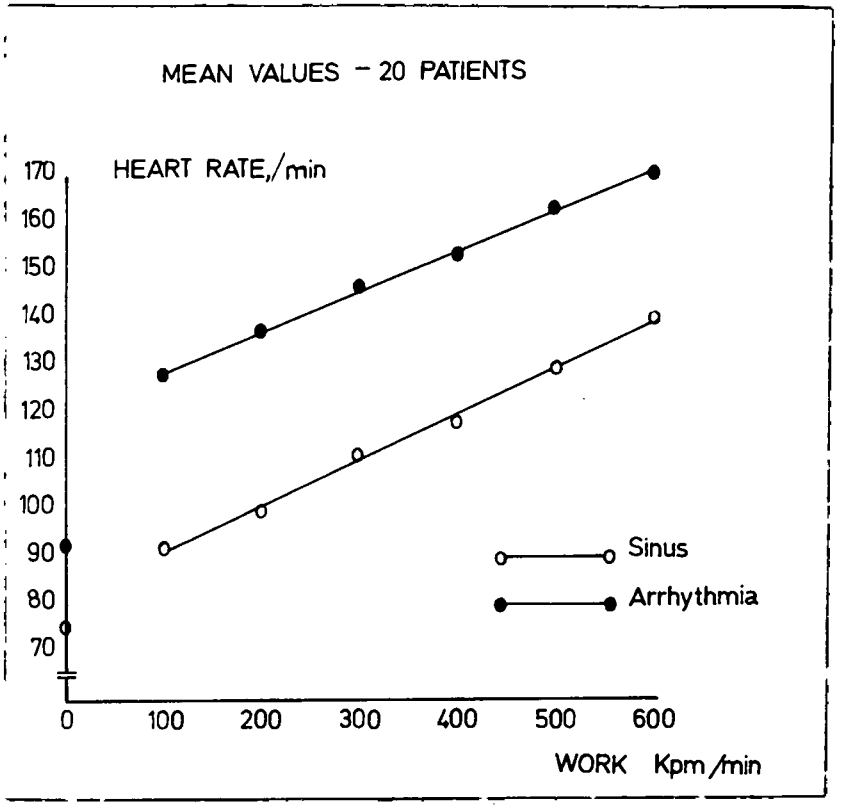


FIG. 33: Mean heart rates in 20 patients at rest and on exercise plotted against work load.

2. PHYSICAL WORK CAPACITY (19 patients) Tables 39 and 40.

The PWC 170 averaged 520 kpm during the arrhythmia and varied from 200 - 1080 kpm (S.D.242.5) in the 19 patients in whom a satisfactory measurement could be obtained (excluding Patient J.C.).

In contrast once sinus rhythm had been established the average PwC 170 in the 19 patients was 783 kpm, and varied from 380 - 1220 kpm (S.D. 248.6).

3. ARTERIO-VENOUS OXYGEN DIFFERENCE: (14 patients) Table 40.

The arterio-venous oxygen difference at rest during the arrhythmia averaged 72.0 ml/L and ranged from 56.2 to 97.5 ml/L (S.D. 8.83). Following the establishment of sinus thythm the average value of the arterio-venous oxygen difference was reduced to 69.7 ml/L. These figures are statistically significant, (S.D, 1.66, t 2.2, $p > 0.02$).

The mean arterio-venous oxygen difference at 200 kpm/min was 103.7 ml/L during the arrhythmia and 93.3 ml/L in sinus rhythm. At 400 kpm/min the corresponding figures were 119.0 ml/L and 104.3 ml/L and at 600 kpm/min 130.7 ml/L during the arrhythmia and 115.4 ml/L in sinus rhythm (Fig.34).

The reduction in the arterio-venous oxygen difference was due to the higher oxygen content of mixed venous blood at rest and on effort in sinus rhythm - Table 40. Significant de-saturation of arterial blood did not occur during exercise in any of the 14 patients studied.

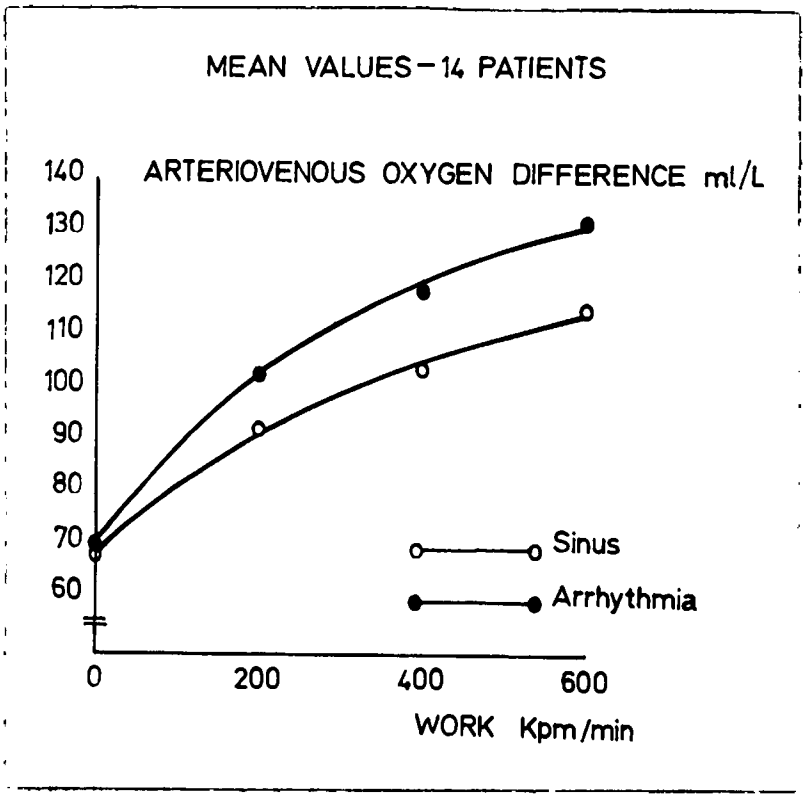


FIG. 34: Mean arterio-venous oxygen difference in 14 patients at rest and on exercise plotted against work load.

4. OXYGEN UPTAKE: (20 patients) Table 39 and 40.

The investigations were performed under strictly comparable conditions during the arrhythmia and in sinus rhythm, and meticulous attention to detail was taken to ensure that all the expired air was collected in the Douglas

bag. The difference in the volume of oxygen uptake at rest and on effort measured during the arrhythmia and sinus rhythm was insignificant. The mean values of oxygen uptake measured in the 20 patients plotted against the work load is shown in Fig.35.

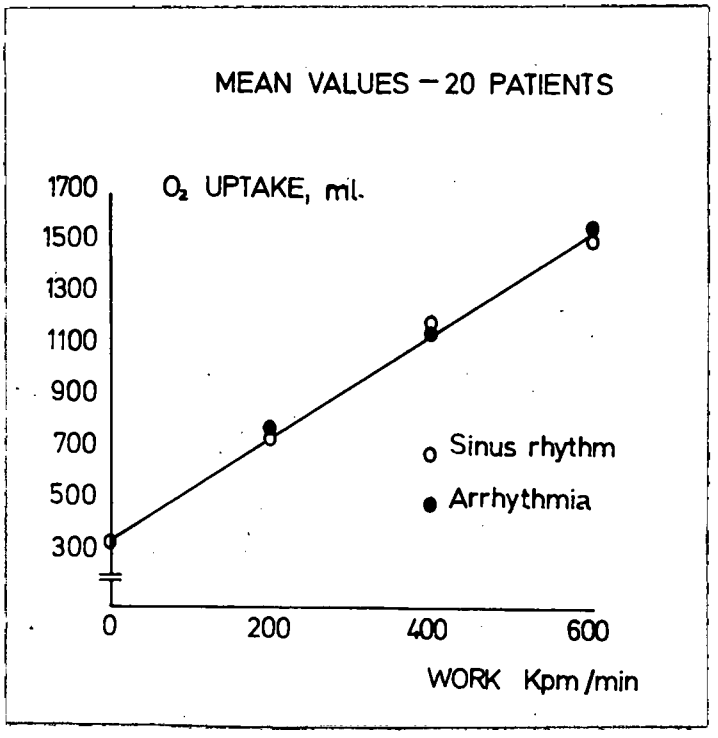


FIG. 35: Mean oxygen consumption in 14 patients at rest and on exercise plotted against work load.

5. CARDIAC OUTPUT: (14 patients). Table 40.

The mean cardiac output at rest during the arrhythmia was 5.04 L/min and following the establishment of sinus rhythm, 5.17 L/min. The difference is not statistically significant.

A significant difference however, occurred with the stress of exercise and was accentuated as the work load increased. The mean cardiac output in the 14 patients at 200 kpm/min was 7.84 L/min during the arrhythmia and 8.79 L/min in sinus rhythm, a difference of just less than 1.0 L/min. Comparable values at 600 kpm/min, however were 12.72 L/min and 14.98 L/min, a difference of 2.26 L/min. These changes are shown graphically in Fig.36.

6. STROKE VOLUME (14 patients) Table 40.

(i) Exercise

The stroke volume averaged 59.8 ml at rest during the arrhythmia and was significantly higher in sinus rhythm (68.4 ml).

The difference became accentuated with the stress of exercise. The stroke volume at 200 kpm/min during the arrhythmia was 64.2 ml and 88.3 ml in a sinus rhythm, a

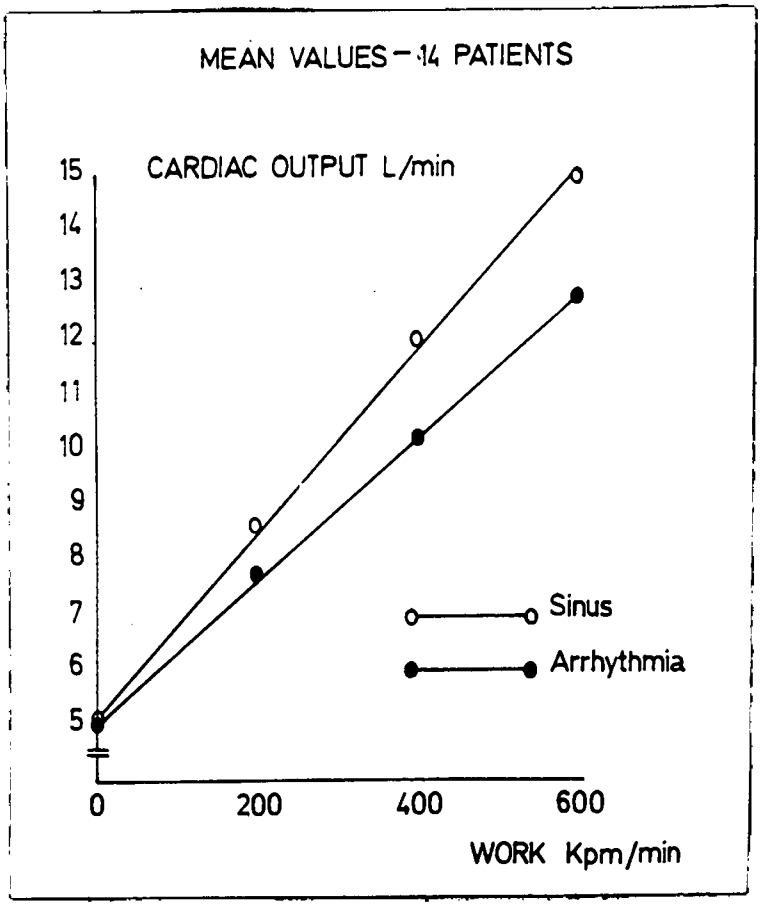


FIG. 36: Mean cardiac output in 14 patients at rest and on exercise plotted against work load.

difference of 24.1 ml; comparable figures at 600 kpm/min were 74.9 ml. and 109.7 ml, a difference of 34.8 ml.

These changes are shown graphically in Fig.37.

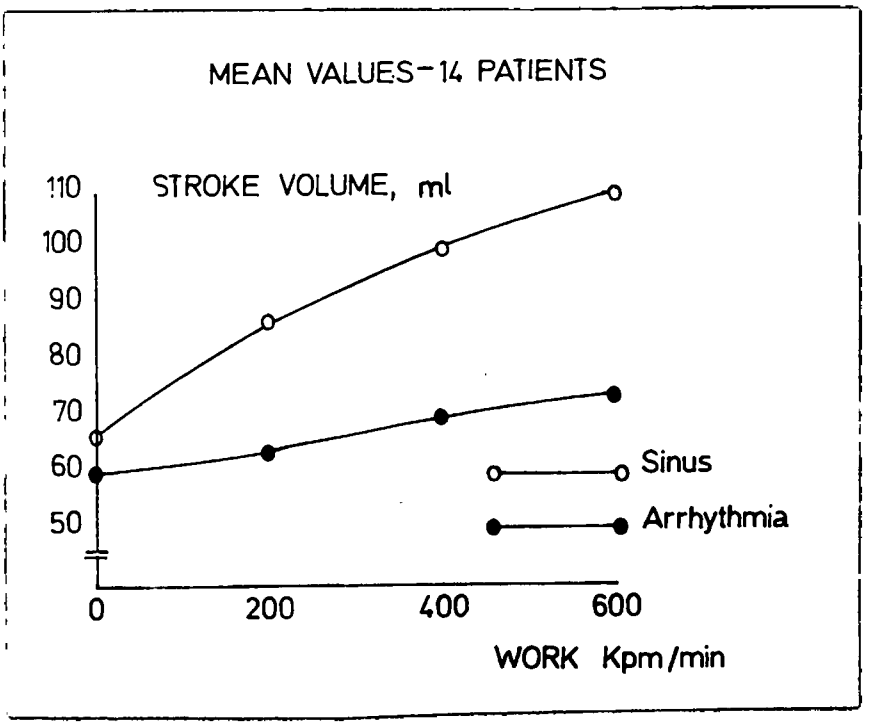


FIG. 37: Mean stroke volume in 14 patients at rest and on exercise plotted against work load.

(xx) Heart rate.

The evidence presented thus far suggests that in sinus rhythm cardiac output and stroke volume are significantly increased during exercise. It has also been demonstrated (Fig.33) that the heart rate is less in sinus rhythm both at

rest and on effort. The beneficial effects on cardiac output and stroke volume in sinus rhythm might therefore be considered to be due to a slower heart rate and consequently an increased diastolic filling time. That this is not entirely so is shown in Fig.38 in which the mean stroke

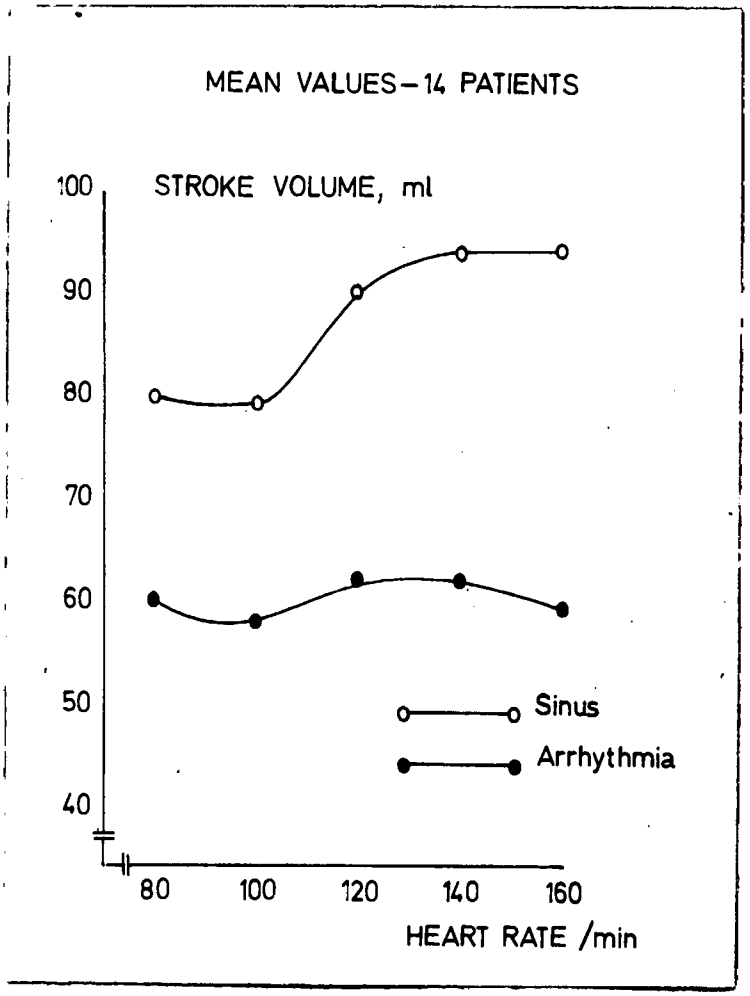


FIG. 38: Mean stroke volume in 14 patients at rest and on exercise plotted against heart rate.

volume is plotted against the heart rate in the 14 patients studied, in sinus rhythm and during the arrhythmia.

The difference between the values in sinus rhythm and during the arrhythmia is that an appropriately timed atrial systole occurs in sinus rhythm. All other parameters, namely steady state, heart rate and exercise performed are identical. The stroke volume rises to a maximum between a heart rate of 110 - 120/min both during sinus rhythm and in the presence of the arrhythmia; the rise, however, is higher in sinus rhythm and furthermore the stroke volume begins to fall off at higher heart rates during the arrhythmia but is held more constant in sinus rhythm.

7. PRESSURE MEASUREMENTS (14 patients) Table 40.

There was no significant change between pulmonary artery and brachial artery pressure at rest and on exercise during the arrhythmia and in sinus rhythm. Typical pressure tracings are shown in Fig.39.

Pressure in the pulmonary artery rose from normal values to 40 or more mm. Hg. in systole on the highest exercise load in patients G.L.S., M.C., E.K., and K.T.

Of considerable interest were patients G.C. and H.C., both of whom developed marked systemic hypertension during

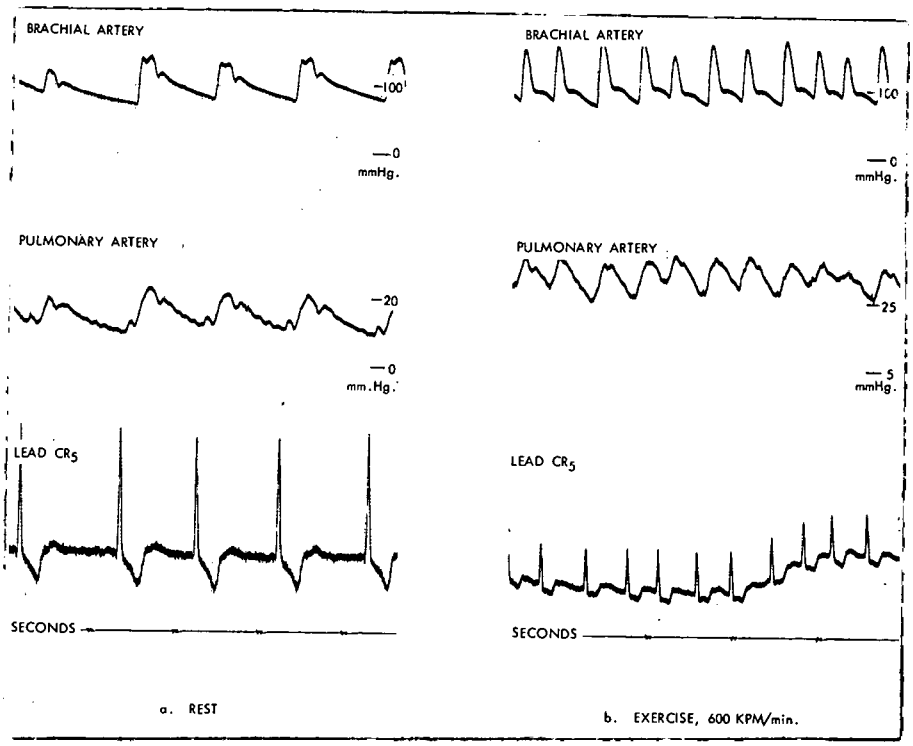


FIG. 39: Pressure recorded simultaneously from pulmonary artery and brachial artery with ECG lead CR_5. On the left at rest, sitting on the bicycle. On the right, during the 6th minute of exercise at 600 kpm/min. The sensitivity of the ECG has been reduced during the exercise record to lessen the effect of respiratory swing.

exercise. Neither had had evidence of hypertension in the past.

The significant mean haemodynamic findings in the patients at rest and on exercise during the arrhythmia and in sinus rhythm are shown in Table 41.

The results in the two patients who were studied in atrial flutter before and after conversion to sinus rhythm were similar to the results in the 12 studied in atrial fibrillation.

VI. FOLLOW-UP STUDIES

A follow-up, extending over from 3 to 20 months has been undertaken in all except one patient of the 131 brought into sinus rhythm. Five deaths have occurred in sinus rhythm further reducing the number of patients to 125.

1. PATIENTS IN SINUS RHYTHM FOLLOWING FIRST TREATMENT (55)

Table 42.

A ATRIAL FIBRILLATION:

40 patients are alive and well and in sinus rhythm. Patient number 38 died in sinus rhythm 2 months after successful synchronised capacitor discharge.

TABLE 41

EXERCISE kpm/min	HEART RATE /min		PWC 170 kpm		AVO_2 DIFF ml/L		CARDIAC OUTPUT L/min		STROKE VOLUME ml	
	AF	SR	AF	SR	AF	SR	AF	SR	AF	SR
REST	93	76			73.0	69.6	5.0	5.2	59.8	69.4
200	131	101			95.7	91.7	7.8	8.8	64.2	88.3
			520	783						
400	153	118			123.5	95.5	10.2	12.0	70.6	100.1
600	178	142			128.0	113.5	12.7	15.0	75.0	110.0

AF - Atrial fibrillation or flutter

SR - Sinus rhythm

Table 41: Mean values during the arrhythmia and in sinus rhythm at rest and on exercise in the patients studied.

TABLE 42

PATIENT NO.	ARRHYTHMIA	HEART DISEASE	DURATION OF ARRHYTHMIA	HEART SIZE CTR%	HEART SIZE LEFT ATRIUM N-3	ECG	MAXIMUM ENERGY LEVEL joules	QUINIDINE DOSAGE AFTER DC. mg	SINUS RYHTHM STILL PRESENT AFTER
15	AF	MS. Post op.	4 yr	49	1		250	180/8 hr	7 mo
25			1 yr	54	2		150	300/6 hr	18 mo
26			8 mo	57	2		150	300/6 hr	16 mo
71			2 yr	59	1		250	180/6hr for 1mo	7 mo
73			3 yr	45	1		250	300/8 hr	5 mo
78			20 d	49	2	Inc RBBB	150	-	15 mo
82			2 yr	56	1	LV+,Inc RBBB	150	-	14 mo
83			7 mo	55	2		150	300/8 hr	14 mo
84			3 wk	59	2		150	-	14 mo
86			5 mo	58	2		150	-	10 mo
94			18 mo	52	2		150	-	12 mo
104			4 yr	50	1		150	-	11 mo
111			2 mo	65	2		250	300/8 hr	9 mo
112			3 mo	52	2	Inc TBBB	250	-	9 mo
119			1 yr	55	2		150	-	9 mo
122			3 wk	52	1		150	-	8 mo
124			5 yr	52	2		250	-	8 mo
131			3 mo	47	1		150	-	6 mo
140			9 mo	58	2		400	-	4 mo
141			7 mo	56	1		200	-	4 mo
147			2 mo	52	1		250	-	7 mo
148			3 wk	48	1		100	-	3 mo
121		AOVD, Post op.	4 wk	58	1	LV+	150	-	9 mo
150			1 yr	49	1	LV+	300	-	3 mo
16		Alc. cardiomy.	3 mo	45	1		250	300/8 hr	19 mo
21			2 yr	43	1		150	300/6 hr	18 mo
85			6 wk	49	1		350	-	14 mo
130		Cardiomyop.	6 mo	49	1		250	-	7 mo
12		Lone	2 mo	47	1		400	300/8 hr	19 mo
40			5 mo	50	N	LV+	250	300/6hr for 6mo	17 mo
44			11 mo	48	N		250	300/6 hr	17 mo
67			12 yr	43	1		150	300/8hr for 1wk	9 mo
135			3 mo	51	N		100	-	5 mo
139			1 yr	56	1		250	300/6 hr	4 mo
20		Post thyrotox.	2½ yr	50	N		150	300/ 6 hr	17 mo
75			2 yr	55	1		150	-	15 mo
136			3 mo	47	1		200	-	5 mo
102		IHD, chronic	3½ yr	46	1	Isch.	350	-	11 mo
127			6 wk	43	N	Isch	250	-	6 mo
138			7 mo	48	N	Inf.infarc.	400	300/6 hr	4 mo

(continued on next page)

TABLE 42 (continued)

PATIENT NO.	ARRHYTHMIA	HEART DISEASE	DURATION OF ARRHYTHMIA	HEART SIZE CTR%	HEART SIZE LEFT ATRIUM N-3	ECG	MAXIMUM ENERGY LEVEL joules	QUINIDINE DOSAGE AFTER DC. mg	SINUS RHYTHM STILL PRESENT AFTER
11	A FLUT	MS. Post op.	1 wk	49	1		150	-	20 mo
149		Cardiomyop.	1 yr	52	1		50	300/8 hr	3 mo
49		Lone	2 mo	44	N		150	300/6 hr	16 mo
6		ASD, Post op.	1 yr	45	N	Inc RBBB	150	300/8 hr	20 mo
30			13 mo	61	2	Inc RBBB	150	300/6 hr	18 mo
77			3 mo	51	N	RBBB	150	-	15 mo
81			1 yr	52	1	Inc RBBB	150	-	14 mo
89			6 yr	52	N	Inc RBBB	150	-	13 mo
106			7 mo	57	N	Inc RBBB	150	300/8 hr	11 mo
113			2 wk	56	N		10	-	9 mo
109	A TACH	ASD, Post op.	9 d	43	N	Inc RBBB	150	180/8 hr	10 mo
42		Ac. myoc. inf.	4 d	59	2	Ant.inf.RBBB	150	300/6 hr	17 mo
126		AOVD, WPW	3 hr	49	N	WPW	100	-	3 mo
80	VT	Cardiomyop.	10 d	50	N		150	-	15 mo
145		IHD, chronic	3 d	58	2		150	300/6 hr 4 mo	4 mo

Only ECG changes other than rhythm or digitalis effect are listed.

Energy levels underlined, delivered using antero-posterior paddles.

Table 42: 55 patients who have remained in sinus rhythm following the first treatment by synchronised capacitor discharge.

(i) Underlying Heart Disease:

22 patients had had a closed mitral valvotomy for mitral stenosis.

2 patients out of 4 who had had an open operation for aortic valve disease and who came into sinus rhythm remained so throughout the follow-up period.

3 patients of 6 with alcoholic cardiomyopathy remain in sinus rhythm.

1 of 4 patients with cardiomyopathy of unknown aetiology remains in sinus rhythm.

16 of 22 patients with lone atrial fibrillation were brought into sinus rhythm; 6 remain in sinus rhythm.

All 3 patients who had been treated for thyrotoxicosis have remained in sinus rhythm.

3 of 5 patients with chronic ischaemic heart disease remain in sinus rhythm.

(ii) Duration of Arrhythmia: Table 43.

35 patients still in sinus rhythm had had atrial fibrillation for up to 3 years before the electrical conversion, and 5 for longer periods (Table 43). Nevertheless, patient number 124 who had atrial fibrillation for 5 years,

TABLE 43

HEART DISEASE	UP TO 1 MONTH	1-3 mo	3-6 mo	6 mo-1 yr	1-3 yr	3-5 yr	Over 5 yr
MS. Post op.	4	2	4	4	5	2	1(5)
ACVD Post op.	-	1	-	-	1	-	-
Alc.cardiomy.	-	1	1	-	1	-	-
Cardiomyop.	-	-	-	1	-	-	-
Lone	-	1	2	1	1	-	1(12)
Post thyrotox	-	-	1	-	2	-	-
IHD, chronic	-	1	-	1	-	1	-
TOTAL	4	6	8	7	10	3	2

Table 43: 40 patients still in sinus rhythm following treatment of atrial fibrillation by synchronised capacitor discharge (first attempt) to show duration of arrhythmia before treatment.

and patient number 67 with lone atrial fibrillation for 12 years are still in sinus rhythm.

(iii) Heart Size: Table 44.

(a) Cardiothoracic Ratio:

17 of the 40 patients still in sinus rhythm had no radiographic enlargement of the heart (Table 44) and in 22 the cardiothoracic ratio was between 50 - 59%

(b) Size of the Left Atrium:

6 patients showed no enlargement of the left atrium, 22 had slight (1+) enlargement and 12 moderate (2+) enlargement (Table 44).

(iv) Electrocardiogram: Table 42.

9 patients showed electrocardiographic changes other than rhythm disturbance or digitalis effect before treatment (Table 42). 4 patients (numbers 82, 121, 150 and 40) showed evidence of ventricular hypertrophy.

B ATRIAL FLUTTER: (10) Table 42.

7 patients who had atrial flutter in association with an ostium secundum atrial septal defect surgically closed, maintained sinus rhythm after successful version, as did one patient who had had a mitral valvotomy, one who had

TABLE 44

HEART DISEASE	CTR%					LEFT ATRIUM		
	40-44	45-49	50-54	55-59	60+	N	1	2
MS. Post op.	-	5	7	9	1	-	10	12
AOVD. Post. op.	-	1	-	1	-	-	2	-
Alc.cardiomy.	1	2	-	-	-	-	3	-
Cardiomyop.	-	1	-	-	-	-	1	-
Lone	1	2	2	1	-	3	3	-
Post thyrotox.	-	1	1	1	-	1	2	-
IHD. Chronic	1	2	-	-	-	2	1	-
TOTAL	3	14	10	12	1	6	22	12

Table 44: 40 patients still in sinus rhythm following treatment of atrial fibrillation by synchronised capacitor discharge (first attempt) to show the cardio-thoracic ratio and size of the left atrium before treatment.

cardiomyopathy and one patient in whom atrial flutter was unassociated with clinically detectable heart disease.

Atrial flutter had been present for one year before treatment in 3 patients (numbers 149, 6, and 81) and for 6 years in patient number 89, but in the remainder (6) atrial flutter had been present for relatively short periods of time.

The cardiothoracic ratio was less than 50% in 3 patients and exceeded 55% in a further 3. In patient number 30, the ratio was 61%.

The radiographic size of the left atrium was normal, (N), in 6 of the 10 patients, was slightly enlarged, (1+), in 3, and moderately enlarged, (2+), in 1.

Electrocardiographic changes were restricted to 6 of the 7 patients who had had an ostium secundum atrial septal defect surgically closed. 5 showed delayed activation over the surface of the right ventricle and 1 (patient number 77) had right bundle branch block.

C ATRIAL TACHYCARDIA (3) Table 42.

3 patients of 8 successfully brought into sinus rhythm remained alive and in sinus rhythm at the end of

the 20 months of follow up. Patient number 101, died in sinus rhythm one week after successful synchronised capacitor discharge.

Patient number 109 had had an ostium secundum atrial septal defect surgically closed. Atrial tachycardia was present for 9 days before treatment, the cardiothoracic ratio was not increased and the left atrium was not enlarged.

Patient number 42 had had a recent anterior myocardial infarction, atrial tachycardia had been present for 4 days before treatment and considerable enlargement of the heart and left atrium were shown radiographically.

Patient number 126 had had atrial tachycardia for only three hours before treatment. She had the Wolff-Parkinson-White Syndrome in association with congenital aortic valve disease, the heart was not enlarged radiographically and the ECG showed only changes of her aberrant conduction.

D VENTRICULAR TACHYCARDIA (2) Table 42.

Two patients (numbers 80 and 145) remain alive and in sinus rhythm at the end of the 20 months of follow up. Patient number 64 died in sinus rhythm 3 months after successful synchronised capacitor discharge.

Patient number 80 who had cardiomyopathy had ventricular tachycardia present for 10 days before treatment. The cardiothoracic ratio was 50% and the left atrium was not enlarged radiographically.

Patient number 145 had ischaemic heart disease with no clinical or electrocardiographic evidence of recent myocardial infarction. Ventricular tachycardia was present for 3 days before treatment; considerable overall enlargement of the heart was present (CTR 58%), and moderate, (2+), enlargement of the left atrium shown radiographically.

2. PATIENTS IN WHOM THE ARRHYTHMIA HAS RECURRED (70) Table 45.

70 patients (56%) of 125 available for long term follow-up studies have reverted to their original arrhythmia (Table 45).

Reversion occurred immediately after the electrical conversion, by the end of the first month or later in the follow up period.

A TIME OF REVERSION

(i) Immediately Following Synchronised Capacitor Discharge (9) Table 46.

There were 9 patients in whom sinus rhythm was maintained only temporarily (2 beats to 2 minutes) following

TABLE 45

PATIENT NO.	ARRHYTHMIA	HEART DISEASE	DURATION OF ARRHYTHMIA	HEART SIZE CTR%	HEART SIZE LEFT ATRIUM N-3	ECG	MAXIMUM ENERGY LEVEL joules	QUINIDINE DOSAGE AFTER DC. mg	SINUS RHYTHM MAINTAINED FOR
1	AF	MS Post op.	7 mo	50	1		100	300mg/8 hr	6 mo
4			16 yr	49	1		150	300mg	90 min
7			4 yr	47	1	Inc RBBB	100	300mg/8 hr	2 mo
19			3 yr	55	2		250	180mg/8 hr	1 mo
22			5 mo	50	1	LV+	150	300mg/8 hr	5 mo
23			9 mo	51	1		150	300mg/8 hr	11 mo
33			10 yr	62	1		150	300mg/6 hr for 3 mo	4 mo
37			1 yr	55	2	LV+	150	300mg/6 hr for 17 mo	19 mo
52			3 yr	60	2	LV+	250	300mg/6 hr	3 mo
54			19 mo	50	2	LV+,RV+	150	300mg/8 hr for 11 wk	3 mo
57			9 yr	54	2	LV+	350	300mg/8 hr	9 mo
79			10 yr	49	1	RV+,RBSB	400	-	1½ hr
87			2 yr	63	2	Inc RBBB	150	-	1 mo
88			24 d	47	1	Inc RBSB	150	-	7 mo
96			1 yr	56	2	LV+	150	-	8 mo
98			14 mo	57	2		150	-	1 mo
108			11 mo	55	2	LV+	150	-	5 mo
110			1 yr	52	1		150	-	6 mo
115			3 yr	52	2		150	-	1 hr
123			1 yr	61	2		350	-	1 d
133			1 yr	59	2		200	-	1 mo
137			4 mo	59	1		50	-	6 wk
69		M+AOVD, Post op.	6 yr	66	3	LV+	150	180mg/8 hr	2 mo
90		AOVD, Post op.	2 mo	63	2	LV+	150	-	6 wk
128			7 d	64	2	LV+	100	300mg	2 hr
35		MS	2 yr	40	1		250	-	12 mo
41			4 mo	47	1		350	300mg/6 hr	3 d
43			6 mo	49	2	LV+	150	300mg/6 hr	10 mo
55		MI	8 mo	60	2	LV+	250	300mg	1½ hr
59			4 mo	58	2	RBBB	150	300mg/6 hr	2 wk
32		MS. MI	2 yr	58	2		250	300mg/8 hr	1 mo
103			5 mo	52	2		250	-	6 d
17		M.+AOVD	8 mo	50	2	LV+	150	300mg/4 hr	1 mo
24			2 mo	53	2	LV+	250	180mg/6 hr	9 mo
46			3 mo	67	2	LV+,RV+	150	300mg/6 hr	1 mo
116			2 yr	41	1	LV+	150	-	2 d
117			5 mo	57	1	LV+,RBBB	150	-	2 min
2		Alc. cardiomy.	15 mo	54	2	LV+	350	300mg/8 hr	4 mo
10			4 yr	47	1		250	300mg/8 hr	3 mo
76			14 yr	51	1		400	-	1 hr
92		Cardiomyop.	3 mo	55	1	LV+	150	-	4 mo
100			6 mo	50	1		150	-	1 mo

TABLE 45 (continued)

PATIENT NO.	ARRHYTHMIA	HEART DISEASE	DURATION OF ARRHYTHMIA	CTR%	LEFT ATRIUM N-3	ECG	MAXIMUM ENERGY LEVEL joules	QUINIDINE DOSAGE AFTER DC. mg	SINUS RHYTHM MAINTAINED FOR
3	AF	Lone	3 yr	55	1		200	300mg/8 hr for 7 mo	10 mo
5			6 mo	40	N		350	-	30 min
34			2 wk	50	1		250	300mg/8 hr	1 yr
48			19 mo	50	N		150	-	15 hr
61			4 yr	44	N		250	-	2 d
63			6 wk	50	1		150	-	4 d
91			4 mo	49	N	RBBB	250	300mg/6 hr	1 mo
105			1½ yr	42	N	Inc RBBB	250	-	1 mo
120			5 mo	41	N		150	-	5 min
132			6 mo	52	N		350	-	1½ hr
9		ASD, post op.	3 mo	53	N		150	300mg/6 hr	3 mo
29			7 yr	56	N	Inc RBBB	400	300mg/8 hr	3 wk
93			11 mo	62	1	Inc RBBB	250	-	6 d
142		IHD, chronic	5 yr	48	N	Inch	200	-	6 hr
53		Syst.hyp., MI	1 yr	61	2	LV+	150	300mg/6 hr	15 mo
68		Cor.Transp. "MI"	3 mo	51	1		350	-	4 mo
146		Obstr. cardio	2 wk	54	1	LV+	200	-	3 wk
28	A FLUT	Lone	18 yr	48	1		150	300mg/6 hr	3 mo
18		ASD, post op.	15 mo	50	1		150	300mg/8 hr	1 yr
107			3 yr	54	1	Inc RBBB	150	-	8 mo
47		Syst. hyp., MI	1 yr	54	2	LV+	150	300mg/8 hr	9 mo
50		VSD, Pulm hyp.	8 d	64	3	LV+,RV+	150	-	3 wk
66		Dystr. Myoton.	3 mo	46	N	Inc RBBB	150	100mg/8 hr	9 mo
31	A TACH	MS. post op.	7 mo	64	2		150	300mg/8 hr	14 mo
70		Lone	3 mo	50	N	Inc LBBB	50	300mg/8 hr	1 wk
129		Ac.myoc.inf.	3 d	57	2	Inf.inf.	50	300mg/8 hr	2 d
143		Digitalis	11 d	56	1		50	300mg/6 hr	4 hr
72	VT	IHD, chronic	1 d	50	2		50	-	3 hr

ECG - Only changes other than rhythm or digitalis effect are listed.

Energy levels underlined, delivered using antero-posterior paddles.

Table 45: 70 patients who have reverted to their arrhythmia following successful treatment by synchronised capacitor discharge.

TABLE 46

PATIENT NO.	ARRHYTHMIA	HEART DISEASE	DURATION OF ARRHYTHMIA	QUINIDINE BEFORE DC	ENERGY LEVEL SETTINGS, joules	RESULT
4	AF	MS. Post op.	16 yr	-	100	SR for 2 min
					150	SR for 90 min
5	AF	Lone	6 mo	-	300	SR for 5 beats
					350	SR for 30 min
14	AF	MS. Post op.	6 yr	-	350	SR for 20 sec
					400	Fail
57	AF	MS. Post op.	9 yr	Yes	150	SR for 30 sec to A.Flut.
					250	A. Flut
					350	SR maintained 9 mo
58	AF	MS. Post op.	9 yr	-	400	SR for 2 beats
65	A Tachy	VSD. WPW	9 d	Yes	400	SR for 15 sec
79	AF	MS. Post op.	10 yr	-	150	SR for 90 sec to A.Flut.
					250	do.
					350	do.
					400	SR for 1½ hrs to A. Flut.
97	AF	Lone	10 mo	-	400	SR for 5 beats
140	AF	MS. Post op.	9 mo	-	400	SR for 5 beats

Table 46: 9 Patients in whom sinus rhythm was held only momentarily after synchronised capacitor discharge.

Note: Energy underlined, delivered by antero-posterior paddles.

synchronised capacitor discharge (Table 46). All patients were being treated for atrial fibrillation except for patient number 65 who had atrial tachycardia. Six patients with atrial fibrillation had had a closed mitral valvotomy performed for mitral stenosis and in two the arrhythmia was unassociated with clinical evidence of heart disease. The patient with atrial tachycardia had a haemodynamically unimportant ventricular septal defect and the Wolff-Parkinson-White Syndrome. The average duration of the arrhythmia was 8½ years, excluding the 2 patients with lone atrial fibrillation and the patient with atrial tachycardia.

Quinidine was used in 2 patients (numbers 57 and 65) immediately before synchronised capacitor discharge.

The energy level settings to achieve sinus rhythm varied from 100 to 400 joules and in 6 patients was 300 joules or more.

Patients numbers 57 and 79 were unusual in that sinus rhythm was immediately followed by atrial flutter. This rhythm was followed by atrial fibrillation 1½ hours later in the case of patient number 79. Patient number 57 is the only patient in this group in whom sinus rhythm was maintained for a considerable length of time (9 months) following a further shock at a higher energy level setting.

Illustrative Case Histories:

a) Patient number 65, a man of 25 years, had a ventricular septal defect and the Wolff-Parkinson-White Syndrome. He suffered attacks of supraventricular tachycardia of increasing frequency and severity over three years. The result of treating one attack with synchronised capacitor discharge is shown in Fig. 40.

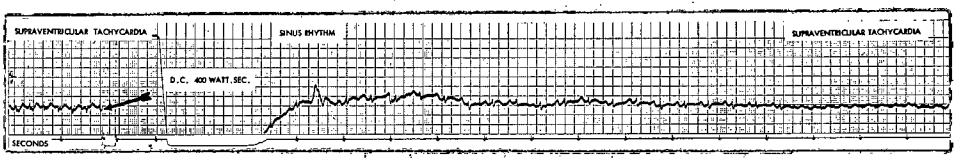


FIG 40: Synchronised capacitor discharge, 400 joules, used in the treatment of supraventricular tachycardia. Following the direct current shock sinus rhythm is present but is replaced by a return of the supraventricular tachycardia.

It became a matter of clinical urgency to control the arrhythmia; hypotension and circulatory collapse were present. Synchronised capacitor discharge was repeated following an intravenous infusion of 600 mg. quinidine given over 38 minutes. (Fig.41). Sinus rhythm was maintained for 5 days only, despite maintenance quinidine. Subsequent attempts to establish sinus rhythm with synchronised capacitor discharge failed despite preceding intravenous quinidine.

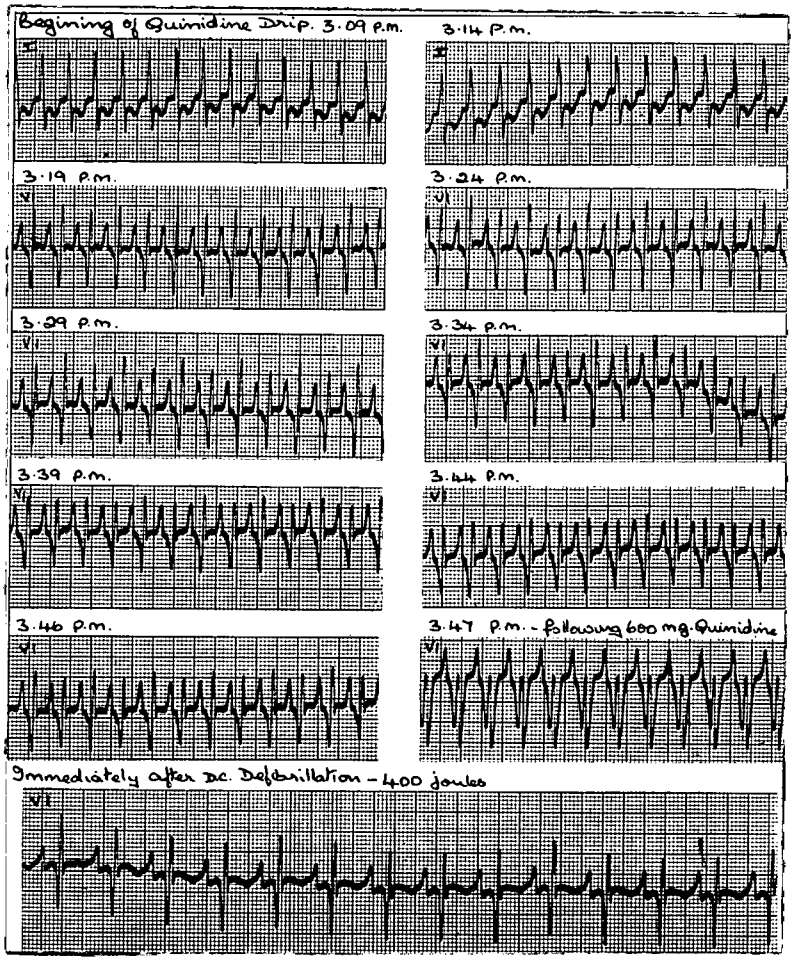


FIG. 41: Synchronised capacitor discharge, 400 joules, preceded by 600 mg. quinidine intravenously in 50 ml. 5% dextrose over 38 minutes. On this occasion sinus rhythm was maintained for 5 days.

b) Patient number 5, a girl of 21 years, had atrial fibrillation unassociated with clinical evidence of heart disease. Synchronised capacitor discharge resulted in sinus rhythm of short duration after shocks of high energy level setting. A short P-R interval (0.10 sec.) but a normal QRS complex were noted in sinus rhythm. An electrocardiogram, standard leads only, which had been recorded at another

hospital at the age of 5, was made available to me thanks to the kindness of Dr. Dynski-Klein. A short P-R interval (0.10 sec.) with normal QRS complex was also present at that age (Fig.42).

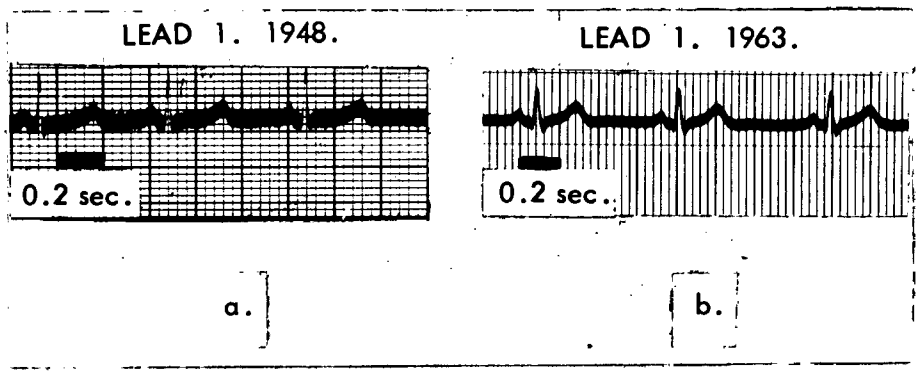


FIG. 42: Electrocardiogram, lead 1

a) At 5 years old.

b) At 21 years old, immediately following synchronised capacitor discharge.

Both records show a short P-R interval (0.10 sec.) and normal QRS.

Sinus rhythm was maintained for only 30 minutes after electrical conversion of atrial fibrillation.

(ii) Within the First Month Following Synchronised Capacitor Discharge: (37).

Reversion to the original arrhythmia was more liable to occur within the first 4 weeks following successful synchronised capacitor discharge (Fig.49). 37 of the

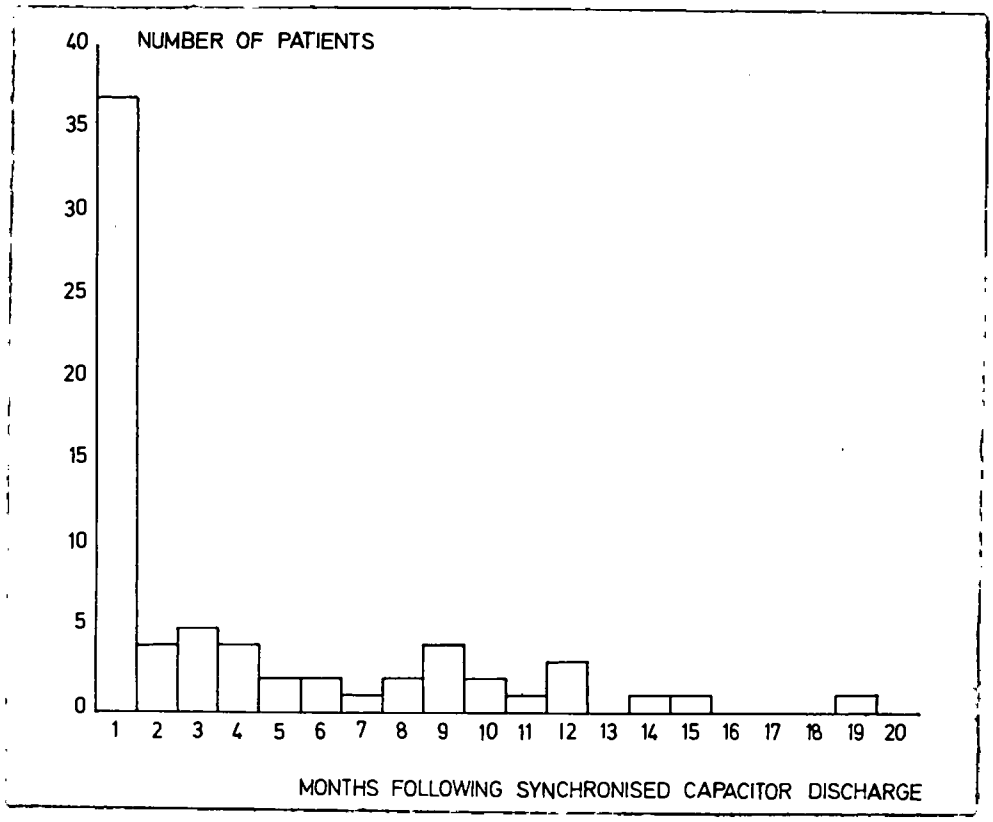


FIG. 49: 70 patients who reverted to their original arrhythmia following successful synchronised capacitor discharge to show the number of reversions per month over 20 months.

70 patients (53%) had reverted to the original arrhythmia by the end of the first month. Furthermore, the highest incidence of reversion occurred within the first day after the electrical treatment (Fig.44); 15 patients (21%) reverted by the end of the first day and a further 8 by the end of the first week.

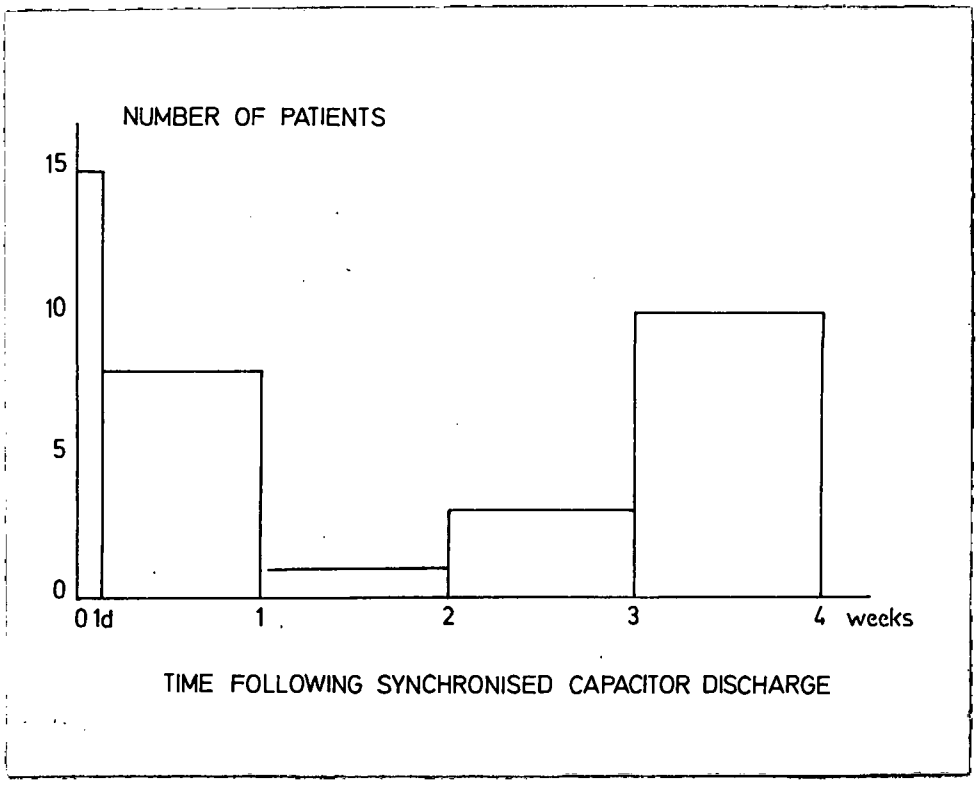


Fig. 44: The recurrence rate of the original arrhythmia during the first month after synchronised capacitor discharge.

(iii) Later Reversions. (33).

From the second month of maintained sinus rhythm a slower reversion rate occurred so that between the beginning of the second and the end of the twelfth month, the reversion rate averaged just less than 3 patients per month. Between the thirteenth and twentieth month of follow up only 3 patients reverted.

The recurrence rate and number of patients remaining in sinus rhythm during each month of the 20 month follow-up is shown graphically (Fig.45). By 9½ months an equal number of patients had reverted to their original arrhythmia as remained in sinus rhythm.

B. THE ARRHYTHMIA

a) ATRIAL FIBRILLATION

59 patients reverted to atrial fibrillation during the follow-up period. One patient (number 90) died several months after reverting back to atrial fibrillation.

(i) Underlying Heart Disease (Table 45).

22 patients had had a closed mitral valvotomy performed for mitral stenosis. 2 patients out of 4 who had had an open operation for aortic valve disease reverted back to atrial fibrillation.

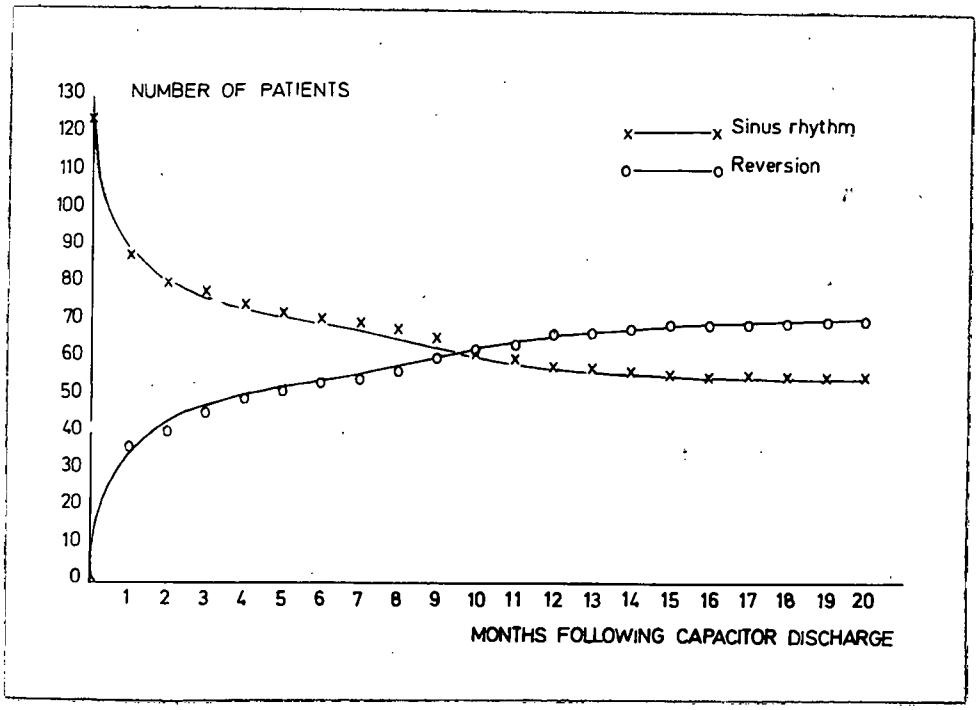


FIG. 45: The number of patients remaining in sinus rhythm and the number in whom the arrhythmia recurred during each month of the follow-up period.

All 13 patients who had post operative mitral and aortic valve disease, unoperated mitral stenosis, mitral regurgitation, mitral stenosis and regurgitation or mitral and aortic valve disease reverted to atrial fibrillation.

3 of the 6 patients who had alcoholic cardiomyopathy reverted to atrial fibrillation.

2 of 4 patients with cardiomyopathy reverted to atrial fibrillation.

10 of 16 patients (63%) with lone atrial fibrillation reverted to the arrhythmia.

All 3 patients who had atrial fibrillation in association with surgical closure of an ostium secundum atrial septal defect reverted to their arrhythmia.

1 patient out of 5, with chronic ischaemic heart disease reverted to atrial fibrillation.

Patient number 53 who had systemic hypertension and mitral regurgitation, patient number 68 with corrected transposition and regurgitation of the systemic atrio-ventricular valve and patient number 146 with obstructive cardiomyopathy of the left ventricle all reverted to atrial fibrillation.

(ii) Duration of the Arrhythmia: Table 47.

30 patients had had atrial fibrillation present for periods in excess of one year, 14 for more than 3 years and 7 for more than 5 years - in 4 of whom atrial fibrillation was present for 10 years or more (Table 47). 21 patients had been in atrial fibrillation for up to 6 months before treatment. The relationship of the percentage of patients who had reverted to the duration of atrial fibrillation before treatment is shown in Fig.46.

(iii) Heart Size: Table 48.

a) Cardiothoracic Ratio:

44 patients in whom atrial fibrillation that was associated with underlying heart disease recurred had a cardiothoracic ratio of more than 50% (Table 48) and 11 had considerable enlargement of the heart, (cardiothoracic ratio in excess of 60%). 15 patients had no radiographic enlargement of the heart 5 of whom had lone atrial fibrillation.

The relationship of the percentage of patients who have reverted to the cardiothoracic ratio before treatment is shown in Fig.47 (excluding patients with lone atrial fibrillation).

TABLE 47

HEART DISEASE	UP TO 1 mo.	1-3 mo	3-6 mo	6 mo-1 yr	1-3 yr	3-5 yr	Over 5 yr
MS. Post op.	1	-	3	3	8	4	3
M.& AOVD Post op.	-	-	-	-	-	-	1
AOVD Post op.	1	1	-	-	-	-	-
MS	-	-	1	1	1	-	-
MI	-	-	1	1	-	-	-
MS. MI	-	-	1	-	1	-	-
M. & AOVD	-	1	2	1	1	-	-
Alc. cardiomy.	-	-	-	-	1	1	1
Cardiomyop.	-	-	1	1	-	-	-
Lone	1	1	3	-	3	2	-
ASD, Post op.	-	-	1	1	-	-	1
IHD, chronic	-	-	-	-	-	-	1
Syst.hyp, MI	-	-	-	-	1	-	-
Cor. Transp, "MI"	-	-	1	-	-	-	-
Obstr. cardiomy.	1	-	-	-	-	-	-
TOTAL	4	3	14	8	16	7	7

Table 47: 59 patients who reverted to atrial fibrillation following the establishment of sinus rhythm by synchronised capacitor discharge to show the duration of atrial fibrillation before treatment.

TABLE 48

HEART DISEASE	CTR%					LEFT ATRIUM			
	40-44	45-49	50-54	55-59	Over 60	N	1	2	3
MS. Post op.	-	4	7	7	4	-	10	12	-
M.&AOVD Post op.	-	-	-	-	1	-	-	-	1
AOVD Post op.	-	-	-	-	2	-	-	2	-
MS	1	2	-	-	-	-	2	1	-
MI	-	-	-	1	1	-	-	2	-
MS. MI	-	-	1	1	-	-	-	2	-
M. & AOVD	1	-	2	1	1	-	2	3	-
Alc. cardiomy.	-	1	2	-	-	-	2	1	-
Cardiomyop.	-	-	1	1	-	-	2	-	-
Lone	4	1	4	1	-	7	3	-	-
ASD, Post op.	-	-	1	1	1	2	1	-	-
IHD, chronic	-	1	-	-	-	1	-	-	-
Syst. hyp, MI	-	-	-	-	1	-	-	1	-
Cor.Transp, "MI"	-	-	1	-	-	-	1	-	-
Obstr. cardiomy.	-	-	1	-	-	-	1	-	-
TOTAL	6	9	20	13	11	10	24	24	1

Table 48: 59 patients who reverted to atrial fibrillation following the establishment of sinus rhythm by synchronised capacitor discharge to show the cardiothoracic ratio and size of the left atrium before treatment.

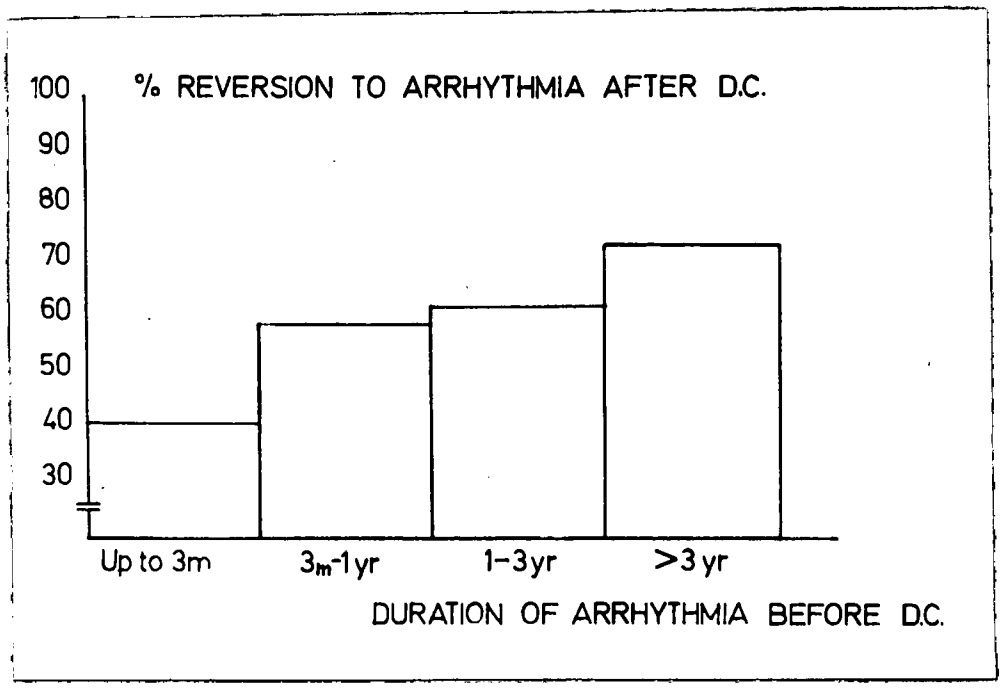


FIG. 46: The percentage of patients who have reverted to atrial fibrillation related to the duration of atrial fibrillation before synchronised capacitor discharge.

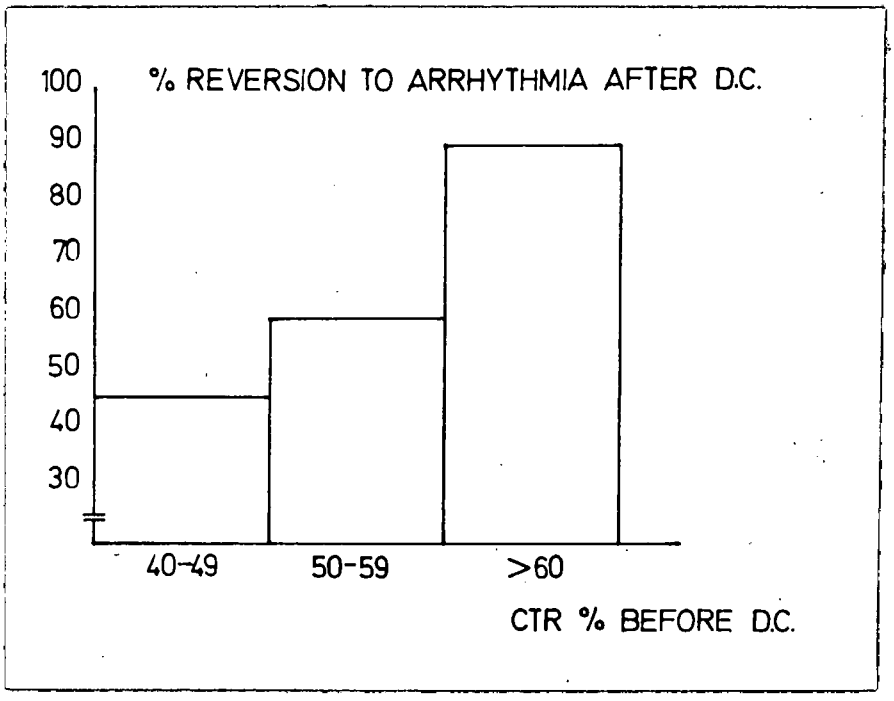


FIG. 47: The percentage of patients who have reverted to atrial fibrillation related to the cardio-thoracic ratio % before synchronised capacitor discharge (excluding patients with lone atrial fibrillation).

b) Size of the Left Atrium

10 patients (Table 48) showed no enlargement of the left atrium radiographically, (0). - of whom 7 had lone atrial fibrillation, slight enlargement, (1+), was present in 24, moderate enlargement, (2+), in 24 and considerable enlargement, (3+), in 1 patient (patient number 69).

(iv) Electrocardiogram:

31 patients showed electrocardiographic changes other than rhythm disturbance or digitalis effect before treatment (Table 45). 20 patients had evidence of hypertrophy of one ventricle and in a further 2, biventricular hypertrophy was present. Evidence of right bundle branch block or delayed activation over the surface of the right ventricle was present in 10.

b) ATRIAL FLUTTER (6) Table 45.

2 patients had had an ostium secundum atrial septal defect surgically closed. One patient had atrial flutter unassociated with clinically detectable heart disease, one had systemic hypertension and mitral regurgitation, one a ventricular septal defect and pulmonary hypertension and one dystrophia myotonica.

Patient number 28 with "lone" atrial flutter had had the arrhythmia present for 18 years before treatment; patient number 107 for 3 years, patient number 18 for 15 months and patient number 47 for 1 year. Patient number 66 had had atrial flutter present for 3 months, and patient number 50 who had a ventricular septal defect and pulmonary hypertension for 8 days.

The cardiothoracic ratio was less than 50% in 2 (patients numbers 28 and 66), was between 50 and 54% in 3 (patients numbers 18, 47 and 107) and was more than 60% in patient number 50.

The radiographic size of the left atrium was normal, (N), in patient number 66 and was slightly enlarged, (1+), in 3 (patients numbers 18, 28 and 107); moderate enlargement, (2+), was present in patient number 47, and severe enlargement, (3+), in patient number 50.

Left ventricular hypertrophy was shown on the electrocardiogram of patient number 47 and biventricular hypertrophy on the electrocardiogram of patient number 50. Delayed activation over the surface of the right ventricle was present in patients numbers 66 and 107.

c) ATRIAL TACHYCARDIA (4) Table 45.

Atrial tachycardia recurred in 4 of 8 patients.

Patient number 31 had had atrial tachycardia for 7 months following mitral valvotomy. Considerable cardiac enlargement was shown radiographically (CTR 64%) and moderate, (2+), enlargement of the left atrium was present.

Patient number 70 had atrial tachycardia which was unassociated with clinically detectable heart disease present for 3 months. The heart was not radiographically enlarged; delayed activation over the surface of the right ventricle was present on the electrocardiogram.

Patient number 29 had atrial tachycardia for 3 days in association with acute inferior myocardial infarction. The heart was enlarged, (CTR 57%) and the left atrium was moderately, (2+), enlarged.

Patient number 143 was the only patient in whom digitalis overdosage was responsible for the arrhythmia. Atrial tachycardia was present for 11 days before synchronised capacitor discharge, and the heart was enlarged (CTR 56%). Slight, (1+), enlargement of the left atrium was present and the electrocardiogram showed only changes of digitalis effect and the arrhythmia.

d) VENTRICULAR TACHYCARDIA (1) Table 45.

Patient number 72 had a recurrence of the arrhythmia. Ventricular tachycardia had been present for one day before treatment and was associated with chronic ischaemic heart disease. Moderate (2+) enlargement of the left atrium was present and the cardiothoracic ratio was 50%.

3. ENERGY LEVEL SETTINGS Tables 42, 45.

(i) Low Energy:

34 of the 55 patients (62%) in whom sinus rhythm was maintained were brought into sinus rhythm with an energy level setting of not more than 150 joules.

40 of the 70 patients (58%) in whom the arrhythmia recurred came into sinus rhythm with an energy level setting of not more than 150 joules.

The difference between the two groups escapes being significant at the 5% level (χ^2 = 2.17, n, 1).

(ii) High Energy: Table 49.

15 patients came into sinus rhythm with energy level settings of between 300 and 400 joules (Table 49). Sinus rhythm has been maintained in 6 (patients numbers 12, 85, 102, 138, 140 and 150).

The remaining 9 patients reverted to atrial fibrillation. In 4 this occurred within 1½ hours after treatment (patients numbers 5, 76, 79 and 132), in patient number 123 by the end of the first day and in patient number 41, after 3 days. Patient number 29 reverted to atrial fibrillation before the end of the first month; patient number 68 reverted at 4 months and patient number 57 at 9 months after treatment.

TABLE 49

PATIENT NO.	HEART DISEASE	DURATION OF ATRIAL FIBR.	QUINIDINE DOSAGE AFTER DC	SINUS RHYTHM MAINTAINED FOR	SINUS RHYTHM STILL PRESENT AFTER
57	MS. Post op.	9 yr	300/6hr	9 mo	-
79		10 yr	-	1½ hr	-
123		1 yr	-	1 d	-
140		9 mo	-	-	4 mo
150	AOVD, Post op.	1 yr	-	-	3 mo
41	MS	4 mo	300/6hr	3 d	-
76	Alc.cardiom.	14 yr	-	1 hr	-
85		6 wk	-	-	14 mo
5	Lone	6 mo	-	30 min	-
12		2 mo	300/6hr	-	19 mo
132		6 mo	-	1½ hr	-
29	ASD, Post. op.	7 yr	300/6hr	3 wk	-
102	IHD, chronic	3½ yr	-	-	11 mo
138		7 mo	300/6hr	-	4 mo
68	Cor.transp. "MI"	3 mo	-	4 mo	-

Table 49: Follow-up on 15 patients, all of whom had atrial fibrillation, and who were brought into sinus rhythm following synchronised capacitor discharge with energy level settings of 300-400 joules.

4. MAINTENANCE QUINIDINE FOLLOWING SYNCHRONISED CAPACITOR DISCHARGE. Tables 50, 51.

55 patients were still in sinus rhythm following the first treatment of whom 20 were taking maintenance quinidine (Table 50). 70 patients had failed to maintain sinus rhythm and of those 33 (Fig.48) had been on quinidine as maintenance (Table 51). 14 patients who reverted by the end of the fourth week were being maintained on quinidine (Fig.49).

The number of patients in sinus rhythm and the number in whom the arrhythmia recurred is shown graphically in Fig.50 with the numbers in each group being maintained on quinidine.

5. FURTHER ATTEMPTS AT SYNCHRONISED CAPACITOR DISCHARGE (Table 52.

A further attempt at treatment by synchronised capacitor discharge was made in 20 patients (Table 52). 17 had reverted to the original arrhythmia following successful treatment but in 3 the first attempt had failed to establish sinus rhythm.

TABLE 50

ARRHYTHMIA	HEART DISEASE	ON QUINIDINE	NO QUINIDINE
A. FIB.	M.S. Post op.	6	16
	AOVD Post op.	-	2
	Alc.cardiomy.	2	1
	Cardiomyop.	-	1
	Lone	3	3
	Post thyrotox.	1	2
	IHD, Chronic	1	2
A. FLUT	M.S. Post op.	-	1
	Cardiomyop.	1	-
	Lone	1	-
	ASD Post op.	3	4
A. TACHY	ASD Post op.	1	-
	Ac.myoc.inf.	1	-
	AOVD. WPW	-	1
VT	Cardiomyop.	-	1
	IHD, chronic	-	1
	TOTAL	20	35

Table 50: 55 patients who remained in sinus rhythm following the first treatment by synchronised capacitor discharge to show the numbers who were taking maintenance quinidine at the end of the follow-up period.

TABLE 51

ARRHYTHMIA	HEART DISEASE	ON QUINIDINE	NO QUINIDINE
A. FIB	M.S. Post op.	9	14
	M. & AOVD, Post op.	1	-
	AOVD Post op.	1	1
	MS	2	1
	MI	2	-
	MS. MI	1	1
	M. & AOVD	3	2
	Ale. cardiomy.	2	1
	Cardiomyop.	-	2
	Lone	2	8
	ASD, Post op.	2	1
	IHD, chronic	0	1
	Syst. hyp. HI	1	-
	Cor. Tremsp. "MI"	-	1
	Obstr. cardiomy.	-	1
A. FLUT	Lone	1	-
	ASD. Post op.	1	1
	Syst. hyp. MI	1	-
	VSD Pulm hyp.	-	1
	Dystroph. myoton	1	-
A. TACH	MS. Post op.	1	-
	Lone	1	-
	Acute myoc. inf.	1	-
	Digitalis	1	-
V.T.	IHD, chronic	-	1
	TOTAL	33	37

Table 51: 70 patients who reverted to their arrhythmia following the establishment of sinus rhythm by synchronised capacitor discharge to show the number who reverted whilst on maintenance quinidine.

TABLE 52

PATIENT NO.	ARRHYTHMIA	HEART DISEASE	SYNCHRONISED CAPACITOR DISCHARGE							
			FIRST TREATMENT			SUBSEQUENT TREATMENT				
			PRECEDED BY QUINIDINE	MAXIMUM ENERGY LEVEL joules	QUINIDINE AFTER DC	DURATION SINUS RHYTHM MAINTAINED	PRECEDED BY QUINIDINE	MAXIMUM ENERGY LEVEL joules	QUINIDINE AFTER DC	DURATION SINUS RHYTHM MAINTAINED
23	AF	MS.Post op.	Yes	150	Yes	11 mo	Yes	150	Yes	Still, 7 mo
37			-	150	For 17 mo	19 mo	Yes	150	Yes	Still, 10 mo
54			Yes	150	For 11 wk	3 mo	-	150	-	Still, 4 mo
110			-	150	-	6 mo	Yes	100	Yes	Still, 4 mo
123			-	350	-	1 d	Yes	350	Yes	2 hr‡
43		MS	Yes	150	Yes	10 mo*	-	150	-	1 mo
61		Lone	-	250	-	2 d	Yes	400	Yes	Still, 11 mo
63			-	150	-	4 d	Yes	250	Yes	1 wk
9		ASD, Post op.	-	150	Yes	3 mo	Yes	150	Yes	Still, 16 mo
142		IHD, chronic	-	200	-	6 hr	Yes	400	Yes	3 wk
68		Cor.Transp."MI"	-	350	-	4 mo	Yes	150	Yes	Still, 9 mo
146		Obstr.cardiomy.	-	200	-	3 wk	Yes	200	Yes	Still, 3 wk
28	AFLUT	Lone	Yes	150	Yes	3 mo	-	150	-	Still, 16 mo
107		ASD, post op.	-	150	-	8 mo	Yes	100	Yes	Still, 3 wk
50		VSD, pulm hyp.	-	150	-	3 wk	-	250	-	1 wk
129	A TACHY	Ao.myoc.inf.	Yes	150	Yes	2 d	Yes	50	Yes	Still, 3 mo
72	V.T.	IHD, chronic	-	50	-	3 hr	Yes	50	Yes	Still, 6 mo
60	AF	MS. Post op.	Yes	400 FAIL	-	-	-	400 FAIL	-	-
39		MI. Post op.	Yes	400 FAIL	-	-	-	400 FAIL	-	-
65	A TACHY	VSD. WPW	Yes	400 FAIL	-	-	Yes	400 FAIL	-	-

Table 52: 20 patients in whom subsequent treatment was undertaken by synchronised capacitor discharge. Energy delivered using antero-posterior paddles is underlined.

* AF recurred at time of mitral valvotomy

‡ Hypotension following DC for 2 hrs.

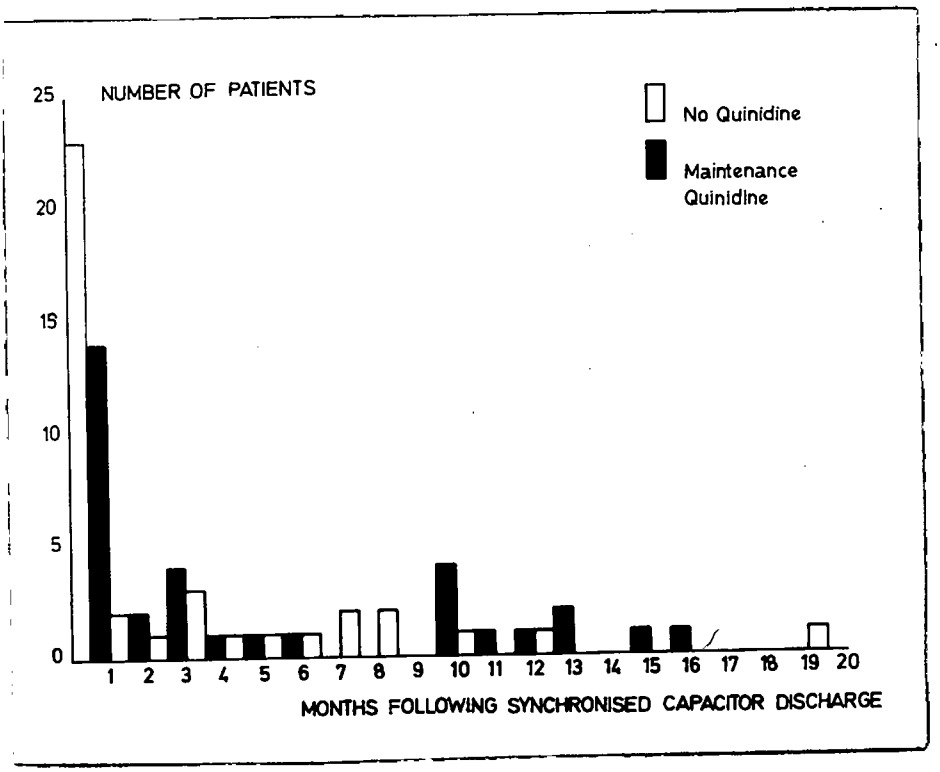


FIG. 48: The number of patients per month who have reverted to the arrhythmia over the follow-up period to show the number being maintained on quinidine.

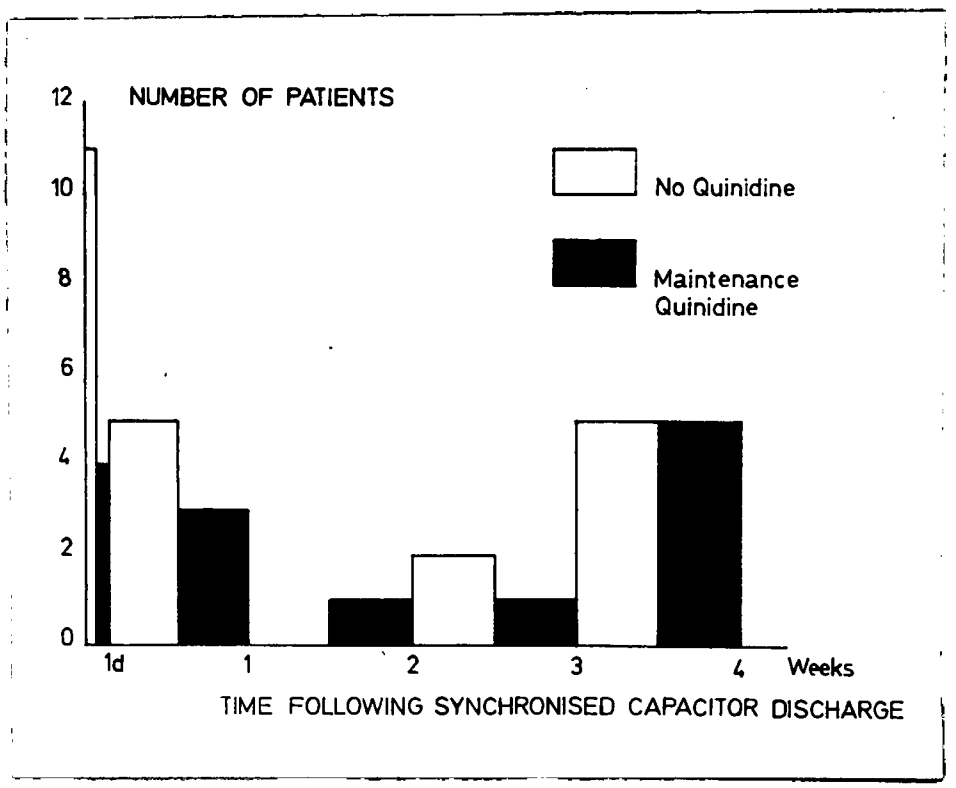


FIG. 49: The recurrence rate of the arrhythmia during the first month following synchronised capacitor discharge to show the number of patients who were being maintained on quinidine.

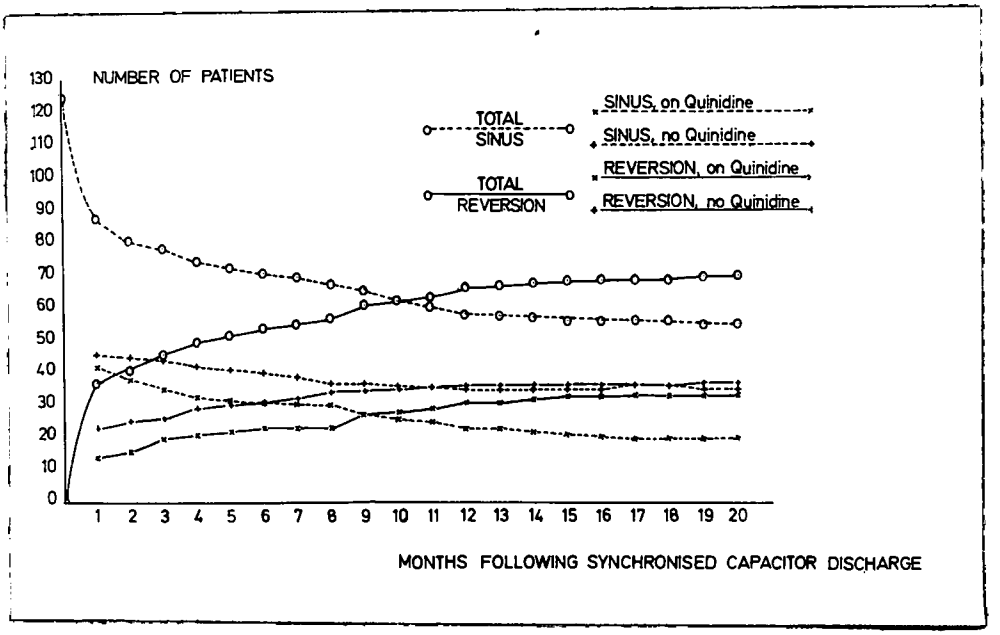


FIG. 50: The number of patients in sinus rhythm and the number in whom the arrhythmia recurred during each month of the follow-up period to show the number being maintained on quinidine.

(i) Patients who had reverted to their arrhythmia: (17).

12 of 17 patients treated for a second time had atrial fibrillation. The recurrence had occurred at time intervals varying from less than 1 day to 19 months after the first treatment (Table 52). In 5 the relapse occurred by the end of the first month (patient numbers 61, 63, 123, 142 and 146).

3 patients had a recurrence of atrial flutter varying from 3 weeks to 8 months after the first treatment.

Patient number 129 developed atrial tachycardia in association with acute myocardial infarction. The tachycardia returned two days after treatment and was not associated with any clinical or biochemical evidence that further cardiac infarction had occurred.

Patient number 72 who had ventricular tachycardia in association with chronic ischaemic heart disease reverted to his arrhythmia three hours after being brought into sinus rhythm.

The second treatment was preceded by quinidine in 13. All 17 patients were once more brought into sinus rhythm and 11 required a similar energy level setting. Of these 11, patients numbers 23 and 129 had both treatments preceded

by quinidine, but in the remaining 9, quinidine not given before the first attempt did precede the second, or if used before the first was withheld before the second.

4 patients required larger energy level settings to achieve sinus rhythm (patients numbers 50, 61, 63 and 142); quinidine preceded the electrical treatment in three. 2 patients were brought into sinus rhythm with energy level settings less than on the previous occasion (patients numbers 68, and 107); quinidine preceded the second but not the first treatment in both.

The follow-up of these 17 patients has of necessity been shorter than on the 131 patients originally brought into sinus rhythm and extends from 3 weeks to 13 months and is over one month in all except one. 12 of the 17 remain in sinus rhythm and of these 9 are taking quinidine as maintenance.

Patient number 54 who reverted to atrial fibrillation three months after the first electrical conversion and one week after stopping quinidine is still in sinus rhythm 13 months after the second treatment; no quinidine was given following the second treatment.

Patient number 123 in whom sinus rhythm persisted for only 1 day after the first treatment remained in sinus rhythm for only 2 hours after the second; no quinidine preceded or followed the first attempt but was used both before and after the second.

Patient number 43 remained in sinus rhythm for 10 months on maintenance quinidine after the first treatment. Atrial fibrillation recurred whilst a mitral valvotomy was being performed. Subsequently sinus rhythm was re-established by synchronised capacitor discharge. Despite the fact that the mitral valvotomy was technically satisfactory, sinus rhythm persisted only for one month; quinidine was not used following the second treatment.

Patient number 61 who had lone atrial fibrillation has remained in sinus rhythm for 11 months after the second treatment (2 days following the first). An energy level setting of 400 joules was needed for the second treatment (250 joules for the first) and on this occasion quinidine is being used as maintenance therapy.

Patient number 68 has remained in sinus rhythm for 9 months after the second treatment, this time on maintenance quinidine, (4 months after the first). Patient number 28, who remained in sinus rhythm and on quinidine for 3 months

after the first attempt is still in sinus rhythm 18 months after the second following which no quinidine was used.

The effect on the duration of sinus rhythm of prescribing or withholding quinidine after the second treatment as compared with giving or withholding quinidine after the first, in 16 of the 17 patients is shown in Table 53. No statistical difference is demonstrated in the small series.

(ii) Patients who failed to achieve sinus rhythm after the first treatment: (3). Table 52.

3 patients (numbers 39, 60 and 65) who failed to come into sinus rhythm after a first treatment attempt were treated on a second occasion (Table 52).

All 3 patients failed to come into sinus rhythm after the second treatment.

(iii) Complications:

Only one complication occurred in the twenty patients who were treated for the second time. Hypotension, (B.P. 80/40 mm. Hg.) followed the final shock of an energy level setting of 350 joules in patient number 123. This persisted for 2 hours and did not require therapy by a sympathomimetic drug.

TABLE 53

QUINIDINE FOLLOWING		PERIOD OF MAINTAINED SINUS RHYTHM FOLLOWING SECOND CONVERSION		
FIRST ATTEMPT	SECOND ATTEMPT	UNCHANGED	LONGER	SHORTER
+	+	2	2	-
-	-	1	-	-
+	-	-	2	1
-	+	4	4	
TOTALS		7	8	1

Table 53: The effect of maintenance quinidine on the duration of sinus rhythm in 16 patients following the second treatment by synchronised capacitor discharge compared to the first. (One patient in whom the follow-up is too short has been excluded).

6. RESULTS OF TOTAL FOLLOW-UP:

67 patients are in sinus rhythm, 30 of whom are being maintained on quinidine.

57 patients have reverted to the arrhythmia, 29 of whom were being maintained on quinidine at the time of reversion.

19 patients could not be brought into sinus rhythm after a first or second attempt at treatment.

6 patients have died during the follow-up period.

The follow-up is not known in 1 patient.

These findings are summarised in Fig.51.

SUMMARY

1. Patients numbers 1 - 50 were shown to have a drug resistant arrhythmia.
2. Toxic reactions to drugs were frequent and often necessitated stopping the drug trial.
3. Synchronised capacitor discharge was successful in re-establishing sinus rhythm in 131 of 150 patients who had atrial fibrillation, atrial flutter, atrial tachycardia or ventricular tachycardia.

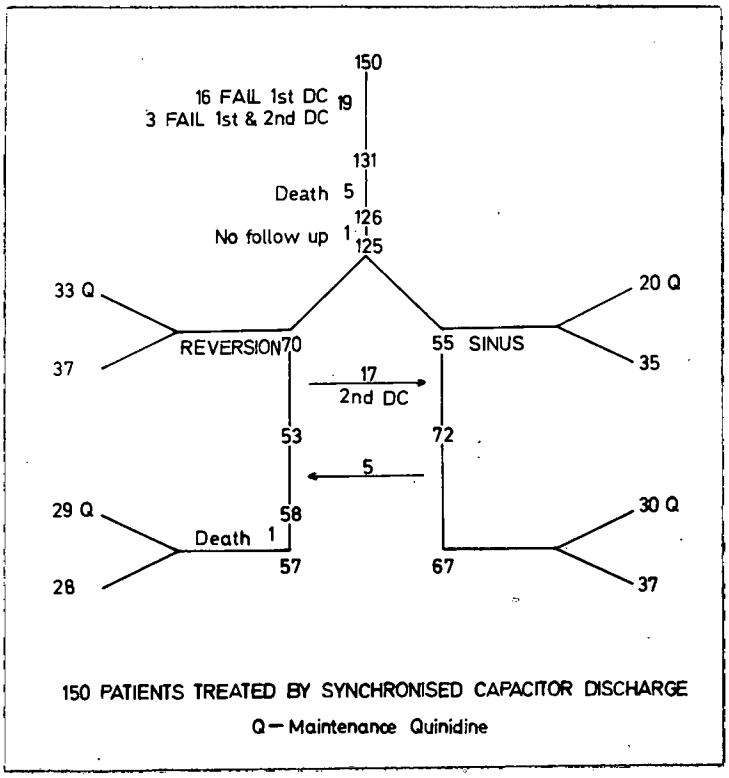


FIG. 51: Results of treatment and follow-up in 150 patients.

4. 43 of 50 patients completely resistant to drug therapy were brought into sinus rhythm with synchronised capacitor discharge.

5. 67 of 131 patients achieved sinus rhythm following the first shock.

6. The chances of successful version diminished with the duration of atrial fibrillation especially when this extended over 5 years, with a cardiothoracic ratio of more than 60% and with more than moderate enlargement of the left atrium.

7. Patients with "lone" atrial fibrillation were exceptional in that their chance of successful version was less than in any other single group, irrespective of the duration of the arrhythmia, the cardiothoracic ratio or size of the left atrium.

8. Atrial flutter, atrial tachycardia and ventricular tachycardia were successfully treated in 98% of cases (29 out of 30 patients) and only lower energy level settings were needed.

9. Energy settings for successful treatment did not relate to the duration of the arrhythmia, the cardiothoracic ratio, the size of the left atrium or the body surface area.

10. Quinidine immediately before synchronised capacitor discharge did not increase the changes of success or lessen the energy level settings needed.

11. Using antero-posterior paddles did not materially lessen energy level settings needed for successful treatment.

12. Synchronised capacitor discharge failed to bring about sinus rhythm in 19 patients. 18 had atrial fibrillation, and one had atrial tachycardia.

13. Lone atrial fibrillation had a particularly high failure rate (27%).

14. Significant valvular heart disease, especially mitral regurgitation, atrial fibrillation of more than 5 years duration, a cardiothoracic ratio of 60% or over, and more than moderate enlargement of the left atrium all increased the chances of failure.

15. Quinidine before synchronised capacitor discharge did not affect the failure rate.

16. Intervening rhythms were frequent, often multiple, but all were transitory. They also occurred in patients not receiving digitalis or diuretics.

17. Ventricular ectopic beats followed more frequently in patients treated for atrial fibrillation with higher energy level settings.

18. 21 patients developed complications other than transient rhythm change. There were 11 episodes of raised levels of serum enzymes, 6 episodes of pulmonary oedema and enlargement of heart size, 7 episodes of hypotension,

3 of embolism, 2 of T wave changes on the electrocardiogram; 2 patients developed gallop rhythms and 1 prolonged multifocal ventricular ectopic beats. Complications were often multiple.

19. 3 deaths were directly or indirectly related to the treatment.

20. Patients with lone atrial fibrillation and ischaemic heart disease were particularly prone to complications.

21. Multiple shocks, high energy levels used in association with the raised levels of serum enzymes which followed, pulmonary oedema and hypotension suggested myocardial damage as the cause of the complications in many.

22. Ventricular fibrillation following synchronised capacitor discharge occurred immediately after treatment or many hours later. If immediate failure of proper synchronisation, overdigitalisation or possibly myocardial damage was the cause. If later, quinidine seemed to be the cause.

23. Circulatory responses at rest and on effort in the steady state were studied in 20 patients before and after the establishment of sinus rhythm by synchronised capacitor discharge. In 6 patients, heart rate and physical work

capacity alone were monitored. These parameters as well as the Fick cardiac output, stroke volume, pulmonary and brachial artery pressures were measured at rest and on effort in a further 14 patients.

24. The heart rate was significantly reduced in sinus rhythm during equivalent work loads.

25. The physical work capacity at a heart rate of 170 beats per minute was increased in sinus rhythm.

26. Oxygen uptake was similar during the arrhythmia and in sinus rhythm

27. Pulmonary artery and brachial artery pressures were similar during the arrhythmia and in sinus rhythm.

28. Mixed venous blood had a higher oxygen content during exercise in sinus rhythm and the arterio-venous oxygen difference was less in sinus rhythm especially on exercise.

29. Cardiac output was only slightly higher at rest in sinus rhythm but the difference increased progressively with the severity of the exercise.

30. Stroke volume as measured sitting on the bicycle was larger in sinus rhythm, especially on exercise.

31. Stroke volumes at equivalent heart rates were significantly increased in sinus rhythm.

32. 55 patients remained in sinus rhythm over a 20 month follow up period.

33. Patients likely to remain in sinus rhythm were those in whom the arrhythmia was present for a shorter length of time, especially less than 1 year; who had a cardio-thoracic ratio of less than 55%. only slight (1+) enlargement of the left atrium and no electrocardio-graphic abnormality other than rhythm change and digitalis effect.

34. Patients who had atrial fibrillation in association with treated thyrotoxicosis had a particularly high incidence of maintained sinus rhythm.

35. Reversion to the arrhythmia was particularly liable shortly after electrical conversion to sinus rhythm; sinus rhythm was maintained only momentarily in 9 patients.

36. 53% who reverted did so by the end of the first month after treatment. This occurred especially in patients with lone fibrillation or those in whom the arrhythmia was of long duration and the heart considerably enlarged.

37. By 9½ months of follow-up an equal number of patients had reverted as remained in sinus rhythm.

38. Patients who had atrial fibrillation in association with chronic rheumatic heart disease or in whom the duration of the arrhythmia exceeded 3 years before treatment were especially liable to revert to the arrhythmia following successful treatment. A cardiothoracic ratio in excess of 55% and moderate enlargement (2+) of the left atrium were also associated with an increased incidence of reversion as were electrocardiographic abnormalities other than rhythm change or digitalis effect.

39. Patients with lone atrial fibrillation were less likely to maintain sinus rhythm even in the absence of prolonged duration of the arrhythmia before treatment, radiographic enlargement of the heart and left atrium and electrocardiographic abnormalities.

40. Maintenance quinidine did not increase the chances of maintaining sinus rhythm over the follow-up period of 20 months, nor did quinidine increase the chances of holding sinus rhythm over the first month.

41. 3 patients who failed to achieve sinus rhythm after a first treatment by synchronised capacitor discharge also failed after the second treatment.

42. 17 patients who reverted to the arrhythmia were brought into sinus rhythm following a second treatment. 6 have maintained sinus rhythm for a significantly longer period of time.

43. 6 of 15 patients in whom sinus rhythm was achieved using higher energy level settings have remained in sinus rhythm. 7 of 9 in this group who reverted to atrial fibrillation did so within the first month of treatment.

44. 67 patients are alive and in sinus rhythm and 57 are alive but have reverted to the arrhythmia following the first and second treatment by synchronised capacitor discharge.

Chapter 5

D I S C U S S I O N

Synchronised capacitor discharge has been an entirely new approach to the management of cardiac arrhythmias. Initial reports of its use in the United States of America (Lown, Amarasingham and Neuman, 1962) demonstrated the feasibility of the method. These workers were in agreement with the findings of Gurvich and Yuniev (1947) who reported results similar to those of Prevost and Battelli (1899, 1900) on the efficacy of capacitor discharge in terminating ventricular fibrillation. The results of the Russian workers, however, were not confirmed by Guyton and Satterfield (1951) or by Kouwenhoven and Milnor (1954).

The first reports on the use of this technique in the treatment of cardiac arrhythmias in the United Kingdom were presented to the Autumn Meeting of the British Cardiac Society, 1963, by O'Brien, Resnekov and McDonald (1964) and by Oram, Davies, Weinbren and Taggart (1964).

Apart from its use in the clinical management of cardiac arrhythmias however, direct current shock is a convenient way of altering the haemodynamic state so that

patients may be studied with and without the benefit of atrial systole and the physiology of atrial arrhythmias investigated.

Harvey (1628) described the action of the atrium as an active contraction, throwing blood into the ventricle. Henderson (1906) however, concluded that the atrium was more important as an elastic reservoir in dogs. This view has recently been put forward once more but in respect of man by Grant, Bunnell and Greene (1964). Lewis (1912) had shown that at a given rapid ventricular rate a regular or irregular ventricular contraction would lead to the same decrease in blood pressure. He felt that the changes in the circulation recorded during atrial fibrillation were entirely rate dependent. Gesell (1911), however, had already shown that an appropriately timed atrial systole would augument blood pressure. Later Gesell (1916) was able to show that the immediate effect of atrial systole was to amplify the cardiac output by 30%. It is interesting to note that an intraventricular pressure tracing from a horse (Chauveau and Marey, 1863) shows preceding atrial contraction, as quoted by Gesell (1911). Ventricular plethysmography was used by Straub (1910), Hirschfelder (1908) and by Wiggers and Katz (1921). All these

investigators concluded that ventricular filling was augmented by atrial systole and that this contribution no longer occurred in atrial fibrillation. Stewart, Crawford and Hastings (1926) demonstrated that the cardiac output in dogs decreased by as much as 60% during atrial fibrillation. It was suggested by Stewart, Crawford and Gilchrist (1928) that this resulted from a marked decrease in the oxygen saturation of mixed venous blood.

A disproportionate increase in the ventricular rate on exercise is found in patients with atrial fibrillation as shown by Blumgart (1924). This is confirmed in the 20 patients studied during the course of this investigation which shows, moreover that even when digitalised to therapeutic levels, the rate response to the stress of exercise is excessive in atrial fibrillation; some rate benefit does occur in the digitalised heart however, as suggested by Modell, Gold and Rothendler (1941).

The concept of the physical work capacity as a measure of the work performed per heart beat, was put forward by Sjöstrand (1947) and by Wahlund (1948). The physical work performed by the patient pedalling a bicycle ergometer at a heart rate of 170/min was obtained in the present investigation from the linear relationship between

heart rate and work performed. The PWC 170 is reduced in the presence of heart disease (Holmgren, Jonsson, Levander, Linderholm, Sjostrand and Strom, 1957), but in addition the present investigation has shown that the combined effects of the loss of atrial systole and a more rapid ventricular rate on exercise during the arrhythmia significantly lowers the work capacity.

Body posture has a profound effect on the circulation both at rest and on exercise (Bevegard, Holmgren and Jonsson, 1960). Cardiac output and stroke volume are both significantly less in the sitting position as compared with figures obtained when supine. These changes occur at rest (McMichael and Sharpey-Shafer, 1944; Donald, Bishop, Cumming and Wade, 1953) and are probably due to a re-distribution of blood from the thorax to the legs. This results in a decrease in the heart volume and consequently a decrease in the stroke volume (Sjostrand, 1953, Holmgren and Ovenfors, 1960). On starting exercise in the sitting position there is a more profound increase in stroke volume probably due to a redistribution of blood from the leg veins to the thorax improving the diastolic filling of the ventricle (Bevegard, Holmgren and Jonsson, 1960).

The effect of position on the circulation is therefore important and might be responsible for some of the conflicting reports in the literature on haemodynamic changes before and after conversion to sinus rhythm. All measurements in this study were made at rest or on effort sitting on the bicycle ergometer (Fig.8, p.34). Furthermore quinidine was not being given to any of the 20 patients investigated. Baer, Weglarz and Killip (1964) demonstrated that the changes in the circulation before and after synchronised capacitor discharge are not due to the electrical treatment alone.

There have been several studies reported on the changes in cardiac output before and after conversion of atrial fibrillation to sinus rhythm with quinidine. These results are often conflicting. Storstein and Tveten (1955) found a significant increase in cardiac output when in sinus rhythm but attributed this in part to the effects of quinidine on the peripheral circulation and on the myocardium; this concept was later challenged by Broch and Müller (1957) who found no evidence that quinidine was beneficial in the way suggested by Storstein and Tveten. Kory and Meneely (1951) failed to demonstrate an improvement in the cardiac output in sinus rhythm, but

larger cardiac outputs in sinus rhythm were shown by Hansen, McClendon and Kinsman (1952).

The results in the 14 patients studied all showed that in sinus rhythm there was a significant increase in the cardiac output. This difference was not necessarily present at rest but could always be provoked by exercise; furthermore the difference was accentuated as the exercise level was increased. These findings are not in agreement with those of Graettinger, Carleton and Muenster (1964) on supine exercise, but are in agreement as regards cardiac output at rest in atrial fibrillation and sinus rhythm. The levels of oxygen consumption on effort, however, are considerably less in the investigations done by these authors and rarely exceeded 300 ml/min/m^2 BSA, whereas oxygen consumption during the maximum exercise load in the present series was usually three times higher. An approximation of the physical activity and oxygen uptake at various work load settings in the study is shown in Table 54. It may well be therefore that more severe exercise would have demonstrated an increased cardiac output in sinus rhythm.

The series reported by Oram, Davies, Weinbren, Taggart and Kitchen (1963) was unusual in that a mean

TABLE 54

WORK LOAD kpm	ACTIVITY	O_2 UPTAKE ml/min
200	walking at moderate pace	900
400	walking very briskly	1350
600	running moderately	1800
800	running hard	2200

Table 54: Approximate physical activity and oxygen uptake at various work load settings.

increase of 70% in the cardiac output measured at rest was found in patients studied in sinus rhythm up to 16 days after electrical conversion. The pre-conversion levels of the cardiac output were excessively low in some of these patients however.

There were 4 patients in whom the arrhythmia (2 with atrial fibrillation, 2 with atrial flutter) was unassociated with clinical evidence of heart disease. All four patients showed significant haemodynamic improvement in sinus rhythm. Indeed in 2 patients (Mr. P.Q. and Mr. M.M.) an extra work load of 200 kpm/min was possible in sinus rhythm and exercise was performed to a total work load of 800 kpm/min. This added work load was achieved with heart rates in sinus rhythm similar to or less than the heart rate of 600 kpm/min during the arrhythmia. Similar beneficial results in sinus rhythm were found in the cardiac output and stroke volume measurements. In contrast, Baer, Weglarz and Killip (1964) could find no improvement once sinus rhythm had been achieved in the "lone" group.

The improvement in the circulation with sinus rhythm was particularly gratifying in Mr. D.H. who had had an operation under cardio-pulmonary by-pass for valvar aortic stenosis. In atrial fibrillation the increase in cardiac

output on exercise was entirely rate dependent and the stroke volume fell progressively as the exercise load increased. Once sinus rhythm had been achieved, however, a more normal response to exercise occurred and the stroke volume was maintained even at a heart rate of 150 beats per minute.

The patient who had obstructive cardiomyopathy of the left ventricle (Mrs. E.K.) demonstrated a significant haemodynamic benefit in sinus rhythm on the very modest exercise load she was capable of performing one hour after electrical conversion of atrial fibrillation. Efficient atrial systole is particularly important in conditions associated with valvar or subvalvar obstruction of the left ventricle and in diseases causing a diminished compliance of that chamber (Braunwald and Frahm, 1961).

The occurrence of haemodynamic benefit in sinus rhythm on exercise was not dependent on the nature of the underlying heart disease in the 14 patients of this series which is in agreement with the findings of McIntosh, Kong and Morris (1964) who reviewed the literature on this subject. The patients who had a virtually normal heart once sinus rhythm had been achieved (Mr. G.E.S., Mr. J.H., Mr. L.L. and Mr. M.M.) however, did show an increased benefit compared with those in whom underlying heart disease still occurred.

The series reported by Kahn, Wilson and Sloan (1964), in which 16 patients were studied at rest and on exercise before and after conversion of atrial fibrillation to sinus rhythm revealed a surprising increase in the cardiac output of 22% at rest and an increase of 27% on exercise in sinus rhythm and reflects the general trend in the literature. As studies by various teams of investigators vary according to method of measuring cardiac output, position of the patient and the amount of exercise performed grouping the results of different investigators is only of limited use in respect of drawing firm conclusions.

The increase in cardiac output in the 14 patients studied resulted from a decrease in the arterio-venous oxygen difference; the oxygen consumption in atrial fibrillation and sinus rhythm was not significantly altered. These findings are in agreement with those of Graettinger, Carleton and Luenster (1964) and with those of Morris, Entman, Thompson, North and McIntosh (1963). In both of these series arterio-venous oxygen difference was also measured directly.

It could be argued that the circulatory benefit in sinus rhythm is entirely rate dependent. Knox (1949) and Wetherbee, Brown and Holzman (1952) both agreed with

earlier workers that the major disability of atrial fibrillation was the rapid ventricular rate especially on exercise. With the establishment of sinus rhythm and slowing of the heart the increased diastolic filling time would result in a larger stroke volume and cardiac output.

The stroke volumes were larger in sinus rhythm than in atrial fibrillation at equivalent heart rates (Fig.38), and increased more in sinus rhythm as the heart rate rose to 120/min. With a further increase in heart rate the stroke volume was held constant in sinus rhythm but began to fall off during the arrhythmia. These figures are deduced from studies in which it is known that the oxygen uptake is similar, and the work load is identical; as heart rate is also similar the beneficial results in sinus rhythm cannot be explained only on the basis of an increased diastolic filling time, but must also be due to the benefit of atrial systole in sinus rhythm. This is in agreement with the conclusions of Skinner, Mitchell, Wallace and Garnoff (1964) who demonstrated in anaesthetised dogs that the lack of atrial systole occurring with the onset of atrial fibrillation when the ventricular rate was held constant caused a fall in stroke volume and cardiac output.

It is perhaps pertinent to consider at this stage what haemodynamic benefits occur as a result of atrial systole. This subject has recently been reviewed by Burchell (1964) and by Braunwald (1964). A properly timed atrial contraction will augment the stroke volume by 5.15% as shown in acute experiments on dogs with denervated hearts by Sellers, Donald and Wood (1962). These were acute experiments, however, and normally after a change in the haemodynamic state a period of re-adjustment occurs in which the cardiac output is reset to a new steady state. When the heart is completely normal in man, this period of readjustment is shorter (Berry, Thompson, Miller, McIntosh, 1959). Where the heart is abnormal the atrial contribution to ventricular filling is more important and this is found even at rest (Braunwald and Frahm, 1961). The 4 patients without underlying heart disease in this present series suggest that on exercise the atrium does function as a booster pump even in the absence of clinically detectable heart disease.

There is little confirmatory evidence in man that an efficient atrial systole prevents atrio-ventricular regurgitation as suggested by Little (1951) and by Sarnoff, Gilmore and Mitchell (1962).

Although the atrium does function as a reservoir (Henderson, 1906; Grant, Bunnell and Greene, 1964) the series of patients presented here demonstrates that with the stress of exercise, or in the presence of a diseased or obstructed left ventricle, an efficient atrial systole becomes functionally important as a booster pump in actively filling the ventricle.

Whilst preliminary reports all dealt with small numbers of patients treated by electrical conversion subsequent reports have justified the initial confidence in the technique. Lown, Perlroth, Kaid Bey, Abe and Harken (1963) reported on the treatment of 50 patients (65 episodes) with atrial fibrillation and named the technique "cardioversion", and Lown, Kaid Bey, Perlroth and Abe (1963a) on a wide variety of cardiac arrhythmias including ventricular tachycardia, atrial flutter, supra-ventricular tachycardia, and atrial fibrillation. Oram and Davies (1964) reported on the use of synchronised capacitor discharge in the management of 100 cases of atrial fibrillation and McDonald and Resnekov (1964) on the treatment of 75 patients who had atrial fibrillation, atrial tachycardia, or atrial flutter. Other large series include reports by Lown (1964a, 1964b) who presented the

results of treatment in 240 patients (305 episodes) including 32 episodes of ventricular tachycardia, 227 episodes of atrial fibrillation, 36 episodes of atrial flutter and 10 episodes of supraventricular tachycardia. Hurst, Faulk, Proctor and Schlant (1964) reported on the treatment of 149 patients (158 episodes) with atrial fibrillation and Pantridge and Halmos (1965) reported the treatment of 83 patients who had atrial fibrillation.

The series of 50 patients who were treated by electrical conversion following the failure of drug therapy to terminate their arrhythmia as presented here has neither been confirmed nor denied in any other report in the literature (McDonald, Resnekov and O'Brien, 1964), although several of the patients presented by Lown, Amarasingham and Neuman (1962) were reported to have been resistant to drug therapy. Despite the fact that the 50 patients reported here were resistant to drugs to maximal tolerance, 43 (86%) were successfully brought into sinus rhythm. Holzman and Brown (1951) reviewed 1,082 cases of atrial fibrillation and reported a conversion rate of 71% using quinidine. Severe toxic reactions using quinidine for the conversion of arrhythmias however, may be as high as 4% (Selzer and Wray 1964), and complications

may also occur following electrical conversion. It is pertinent, therefore to consider the mode of action of quinidine as compared with direct current in the treatment of arrhythmias.

The alkaloid quinidine became available following its preparation from cinchona by Pasteur in 1853 (Conn and Luchi, 1964). Its clinical use in the management of arrhythmias is usually dated from 1912 when a patient of Wenckebach demonstrated he could control his own paroxysmal atrial fibrillation by taking quinine (Wenckebach, 1914). Frey (1918) showed that the antiarrhymic action of quinine was due to quinidine. However, Drake, a physician at Sudbury treated his own cardiac irregularity in 1813, using Cinchona cardifolia, as quoted by Snell (1965), and it seems therefore that 100 years before Wenckebach, a self-prescribing physician was aware of the beneficial action of a crude cinchona extract in treating cardiac irregularities. It is probable that Drake knew of the writing of Senac (1749), as quoted by Willius and Keys (1941), advocating quinine for heart palpitations associated with flatulence. The actions of quinidine may be summarised as follows:- (Conn and Luchi, 1964).

a. Depression of the rate of discharge of the pacemaker. In man, however, the anti-adrenergic action and vagal blockage conflict so that unless hyperthyroidism is present, there is little overall change in rate, (Rowe, Emanuel, Maxwell, Brown, Castillo, Schuster, Murphy and Crumpton, 1957).

b. Depression of atrio- and intraventricular conduction which progresses on the electrocardiogram from prolongation of the P-R interval and widening of the QRS complex to atrio-ventricular dissociation and intraventricular block.

c. Increase in the overall refractory period of the heart but especially involving the relative refractory period (Williams, 1958).

d. Depression of myocardial contractility which may be associated with hypotension and reduction of the cardiac output (Ferrer, Harvey, Werko, Dresdale, Cournand and Richards, 1948).

It has been suggested that under normal circumstances ectopic pacemaker tissue is more sensitive to a given concentration of quinidine than normal pacemaker tissue and that the conduction system of the heart is more affected by quinidine than the muscular tissue (Hoffman, 1957).

Opinion about the use of quinidine in atrial fibrillation before the introduction of electrical methods of conversion could be summarised on the one hand according to the views of Gold (1950) who felt that the use of quinidine had largely been abandoned, and on the other hand according to the views of McMillan and Welfare (1947)

who felt that an attempt at conversion with quinidine should be made in practically all patients with chronic atrial fibrillation.

Toxic reactions to quinidine vary from vascular collapse to hypersensitivity reactions. Although some reports indicate relatively few toxic reactions (Sokolow, 1951) up to 6% of sudden loss of consciousness and circulatory collapse requiring resuscitation has been reported by Rokseth and Storstein (1963). Furthermore, Selzer and Wray (1964) reported the occurrence of syncope due to paroxysmal ventricular fibrillation in 4% of a large series of patients with atrial fibrillation. Experience in the collection of patients 1 - 50 of the present series is in agreement with the potential serious hazards of quinidine therapy.

Estimation of the serum level of quinidine was strongly advocated by Sokolow and Ball (1956) in an attempt to prevent dangerous episodes of quinidine toxicity. Using the fluorometric measurement of Linenthal, Ulick and Patterson (1947) which was based on the quinine and quinidine estimation of Brodie and Udenfriend (1943), they demonstrated that serious toxicity was unusual with serum concentrations less than 6 µg./ml. Toxic reactions did occur, however, with serum concentration levels lower

than this possibly due to the fact that the estimation also gives the absorption peak of less active or inactive quinidine metabolites (Brodie, Baer and Craig, 1951).

Several long-acting preparations have been introduced in an attempt to maintain a constant serum level of quinidine and prevent dangerous peaking related to the variable absorption rates. These compounds have usually proved disappointing in their claims (Modell, 1962 - 63). More recently a tablet in which quinidine bisulphate has been embedded in a porous insoluble plastic compound has been described by Sannerstedt (1960). When serum quinidine levels were measured according to the method of Balatre, Lefèvre and Merlen, (1960) a sustained level of quinidine over 24 hours could be achieved by a tablet every 12 hours (Cramér, Varnauskas and Werkö, 1963). It is disappointing therefore, that loss of consciousness occurred in 11 of 200 patients being maintained on this tablet despite satisfactory blood levels of quinidine (Rokseth, 1963).

It seems therefore that even with the added safeguard of slow-release preparations of quinidine controlled by the estimation of the serum levels of quinidine dangerous toxic reactions may still occur.

The results in patients numbers 1 - 50 indicate that synchronised capacitor discharge can establish sinus rhythm even when quinidine has failed. It has been stated:

> "One can predict with some certainty that the major complication following cardioversion will result not from the procedure, but from the drugs utilized to sustain sinus rhythm" (Lown, 1964b).

21 patients in the present series (14%) were considered to have complications directly related to the electrical treatment.

Capacitor discharge can result in myocardial damage under certain circumstances. Peleška (1960) related damage of the heart muscle to an unmodified capacitor impulse i.e. without an iron-cored choke in circuit; to higher voltage and energy levels used and to uneven distribution of the electrical energy over the myocardium - the paddles must be large enough to distribute the energy evenly. Nevertheless, Kong and Proudfit (1964) reported 140 direct current shocks given to a patient over a 69-hour period with no evidence of myocardial injury; all the shocks however were delivered at an energy level setting of between 180 and 220 joules.

Episodes of pulmonary oedema and increase in size of the heart following synchronised capacitor discharge have not been reported prior to the communication of Resnekov and McDonald (1965) but have subsequently been confirmed by Honey, Nicholls and Towers (1965). It could be argued that in the presence of atrial fibrillation and mitral valve obstruction a sudden increase in cardiac output with the establishment of sinus rhythm might result in an increase in left atrial and pulmonary venous pressure and so be responsible for the pulmonary oedema and increase in heart size. It has been shown, however, that there is little change in the cardiac output in patients in atrial fibrillation and following version to sinus rhythm measured at rest.

There were six patients with pulmonary oedema and an increase in size of the heart, of whom three showed significant changes in the serum levels of enzymes and three were also hypotensive for up to three hours following synchronised capacitor discharge. Four of the six patients were given shocks with energy level settings of 350 of 400 joules. Three of the six patients had chronic ischaemic heart disease and one cardiomyopathy, so that a disease primarily affecting the left ventricle was present

in four. Furthermore in the case of patient number 68 who had corrected transposition, a morphological right ventricle was subjected to the work and pressure of the left ventricle. Acute breathlessness occurred only in one patient so that it is possible to be unaware of this particular complication unless chest radiographs are routinely performed one day following treatment.

Five of 22 patients with lone atrial fibrillation developed complications after treatment (23%) and five of 12 patients with acute myocardial infarction or chronic ischaemic heart disease (42%). Five of 51 patients who had had a closed mitral valvotomy and were being treated for atrial fibrillation developed complications (10%).

A raised serum level of glutamic oxaloacetic transaminase will reflect myocardial damage (Ladue, Wroblewski, and Karmen, 1954) but could also indicate damage to skeletal muscle. A raised serum level of lactic dehydrogenase however is even more specific of myocardial damage (MacDonald, Simpson and Nossal, 1957).

Significant changes in the serum enzyme levels occurred in 11 patients (13.5%). Of these, 9 received shocks with an energy level setting of 350 or 400 joules and 4 were hypotensive for from 1 - 3 hours after treatment;

2 developed loud left ventricular third heart sounds.

There were no episodes of ST segment elevation following synchronised capacitor discharge as reported by Sussman, Woldenberg and Cohen (1964), and by Killip (1963). Two episodes of flat or inverted T waves were recorded on the day following synchronised capacitor discharge and in one patient this was associated with significant elevation of the serum enzyme levels and with the development of a left ventricular third heart sound.

Only one episode of ventricular fibrillation occurred immediately following synchronised capacitor discharge. This was in a patient already in extremis and in "cardiac arrest" although the electrocardiogram revealed that ventricular tachycardia was still present. Failure of synchronisation, the shock falling during the vulnerable phase of ventricular repolarisation has been implicated as the cause by Killip (1963), Lemberg, Castellanos, Swenson and Gosselin (1964), Oram and Davies (1964), Morris, Kong, North and McIntosh (1964), and by Towers, Gibson, Burn and Monro (1965). However, in a second episode of ventricular fibrillation following direct current shock in the same patient Killip (1963) was unable

to implicate failure of synchronisation as the cause. Apart from electronic failure of synchronisation particular attention must be paid to stability of the base-line of the electrocardiogram to prevent the capacitor being discharged by an artefact in a vulnerable phase of the cardiac cycle. Over digitalisation was thought to be responsible for three episodes of ventricular fibrillation coming on 1 - 1½ minutes after a properly synchronised shock as reported by Rabbino, Likoff and Dreifus (1964). Neither a failure of synchronisation nor digitalis was thought to be the cause in the case reported by Willis (1964). Digitalis might well have been an associatiated factor in the patient who developed ventricular fibrillation after a synchronised shock of 100 joules which had been preceded by the intravenous injection of 1 mg. Lanatoside C (Ross, 1964).

The late onset of ventricular fibrillation as occurred in patient number 51 (36 hours after treatment) is unusual although a similar case is reported by Semple, Murdoch and Sinclair (1965) in whom ventricular fibrillation also occurred 36 hours after synchronised capacitor discharge. There was no evidence of damage to the myocardium or to the conducting system on post-mortem examination of patient number 51. As both patients were receiving quinidine

following electrical conversion it is possible that both result from quinidine arrest.

Ventricular tachycardia following direct current shock has been reported by Graf and Etkins (1964) who also quote similar complications reported to them by Wyman (1964) and by Hoffman (1964). In the opinion of Graf and Etkins (1964) low energy shocks are more liable to cause ventricular arrhythmias (Kouwenhoven and Milnor, 1954) but this is contrary to the work of Peleška (1963) and against the opinion of Lown (1964b). Three patients developed ventricular tachycardia following direct current shock thought to be due to noradrenaline in one patient, and to potassium depletion in a second, (Lown 1964b).

Three patients developed emboli (2%). Two embolic episodes occurred amongst 158 episodes of treatment in the series reported by Hurst, Faulk, Proctor and Schlant (1964) and three patients developed emboli amongst 70 (94 attempts at conversion) treated by Morris, Kong, North and McIntosh (1964). One cerebral embolus and one pulmonary infarct occurred amongst the 100 patients treated for atrial fibrillation by Oram and Davies (1964); one cerebral embolus and one pulmonary embolus in 65 patients treated were reported by Rabbino, Likoff and Dreifus (1964).

The patient who developed ventricular tachycardia following direct current shock (Graf and Etkins, 1964) developed a popliteal embolus three days later. From the reports in the literature and from the analysis of the 150 patients of this series, it appears that the incidence of emboli following version by synchronised capacitor discharge is the same as that reported following version by quinidine (Goldman 1959 - 60). Where there is a risk of embolus or thrombosis, for example following recent myocardial infarction, or in mitral stenosis unrelieved or partially relieved by surgery or with a history of previous embolism, anticoagulant therapy using a coumadin derivative should be given and satisfactory control maintained for at least three weeks before electrical conversion unless treatment of the rhythm is urgently needed when heparin should be substituted. In contrast, following successful mitral valvotomy or closure of an atrial septal defect, anti-coagulant therapy need not be given. Patient number 90 in whom a cerebral embolus followed changing rhythms six weeks after synchronised capacitor discharge underlines the fact that this form of treatment is best restricted to patients in whom prolonged sinus rhythm can reasonably be expected. As severe valvular heart disease was still present and considerable enlargement of the heart shown,

atrial fibrillation with a ventricular rate controlled by digitalis would in retrospect have been preferable to the few short weeks of sinus rhythm followed by changing rhythms and then a cerebral embolus.

There were three deaths directly or indirectly related to synchronised capacitor discharge. 2 deaths were reported by Towers, Gibson, Burn and Monro (1965). The first occurred in a patient in extremis who had cardiomyopathy and atrial fibrillation; the second was in a patient with atrial tachycardia and acute myocardial infarction. Fatal cardiac arrest during induction of anaesthesia for synchronised capacitor discharge is reported by Semple, Murdoch and Sinclair (1964). Ventricular fibrillation following direct current shock leading to death is reported by Ross (1964) and by Rabbino, Likoff and Dreifus (1964). Three deaths occurred amongst 100 patients treated by Oram and Davies (1964) although in one patient it is doubtful whether direct-current shock was responsible.

The mode of action of electrical reversion is to cause instantaneous depolarisation of all the myocardial fibres and so allow the sino-atrial node to resume its function as pacemaker. It is of fundamental importance that the shock does not occur during the so-called vulnerable

period. An electrical stimulus of sufficient strength may cause ventricular fibrillation during a short period of repolarisation of the ventricle which approximates to the apex of the T wave. A diagrammatic representation of this phase is shown in Fig. 52. Repolarisation, however,

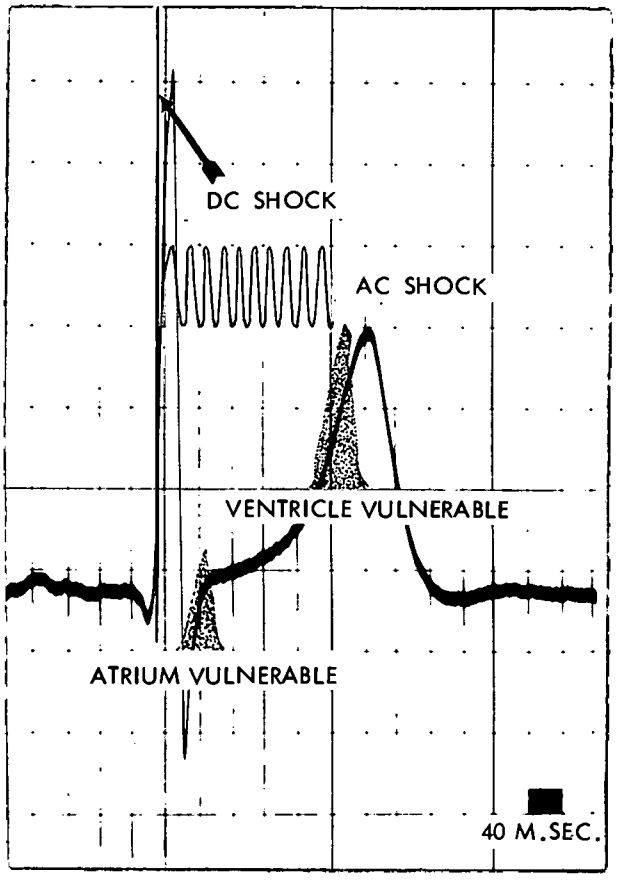


FIG. 52: Diagrammatic representation of the vulnerable atrial and ventricular phases of the cardiac cycle and their temporal relationship to an alternating and direct current shock synchronised with the R-wave of the electrocardiogram.

does not occur at the same fixed interval after depolarisation at all points in the ventricle (Hoffman and Cranefield, 1964). These authors are of the opinion that only if a stimulus is made to occur after repolarisation is complete for the entire heart, for example between the T-wave and the beginning of the P-wave, is there little danger of fibrillation of the atrium or ventricle. A further point of some importance is that the threshold to fibrillation varies during the vulnerable phase, being lower in the middle than at either end of this period (Hoffman, Suckling and Brooks, 1955). The threshold may drop precipitously shortly after the administration of adrenaline or noradrenaline (Siebens, Hoffman, Enson, Farrell, Brooks, 1953), a point of some importance in patients being treated for circulatory collapse with sympathomimetic drugs whilst awaiting treatment of the arrhythmia by synchronised capacitor discharge. These facts as outlined above might help to explain the occurrence of ventricular fibrillation following electrical reversion when a failure of synchronisation had not been responsible as typified by the experience of Killip (1963).

Simultaneous depolarisation of all the fibres of the heart muscle requires a current of between 1 - 5 amperes

(Hooker, Kouwenhoven and Langworthy, 1933). The voltage needed to pass this current through the heart depends on the resistance of the heart muscle and varies with the nature, duration and magnitude of the current (Mackay, Mooslin and Leeds, 1951) and with the size of the electrodes used (Peleška, 1960). As shown by Guyton and Satterfield (1951), the resistance of the heart lies between 20 - 50 ohms.

The waveform of a capacitor discharge can be varied widely as regards magnitude and shape. The waveform shown in Fig.53 was obtained by discharging a capacitor without

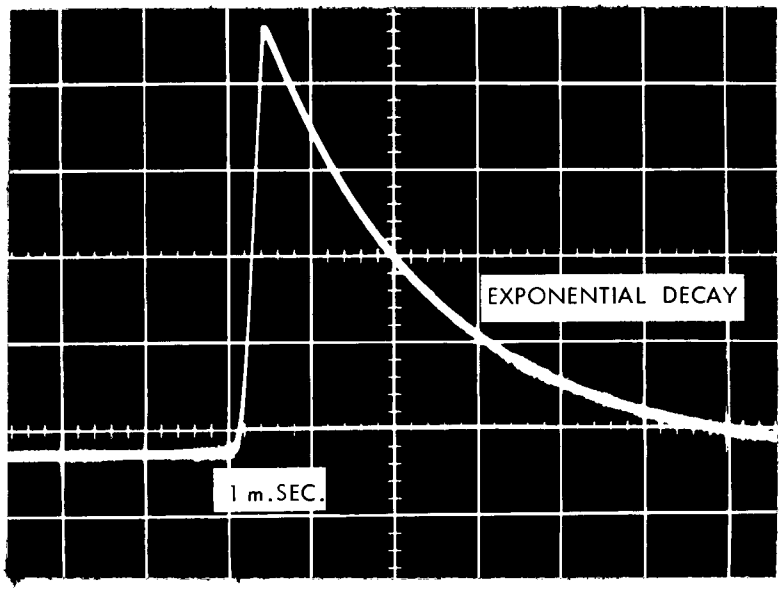


FIG. 53: Unmodified capacitor discharge.

additional inductance in the circuit. Its characteristics are an almost instantaneous rise, and an abrupt peak which is followed by an exponential decay. The use of such an unmodified capacitor discharge transthoracically in dogs resulted in ventricular arrhythmias (Lown, Neuman, Amarasingham and Berkovits, 1962). By the introduction of an inductance coil into the circuit, however, the waveform is modified from being critically damped using a small inductance to being overdamped with larger values of inductance. An overdamped waveform is characterised by a slower rate of rise and a more gradual peak; the pulse is broader and ends in a small flat peak of opposite direction (ringing). The voltage and current waveform of the apparatus used throughout the study on the 150 patients is shown in Fig.54 obtained by discharging the capacitor across a 50-ohm resistance. This circuit which incorporates a capacitor of 16-microfarads and an inductance coil of 100-millihenrys was found to produce a clinically safe underdamped waveform and to be more effective than commercially available alternating current defibrillators in the management of ventricular fibrillation in dogs (Lown, Neuman, Amarasingham, Berkovits, 1962). The energy delivered by discharging a capacitor is measured in joules and is equal to

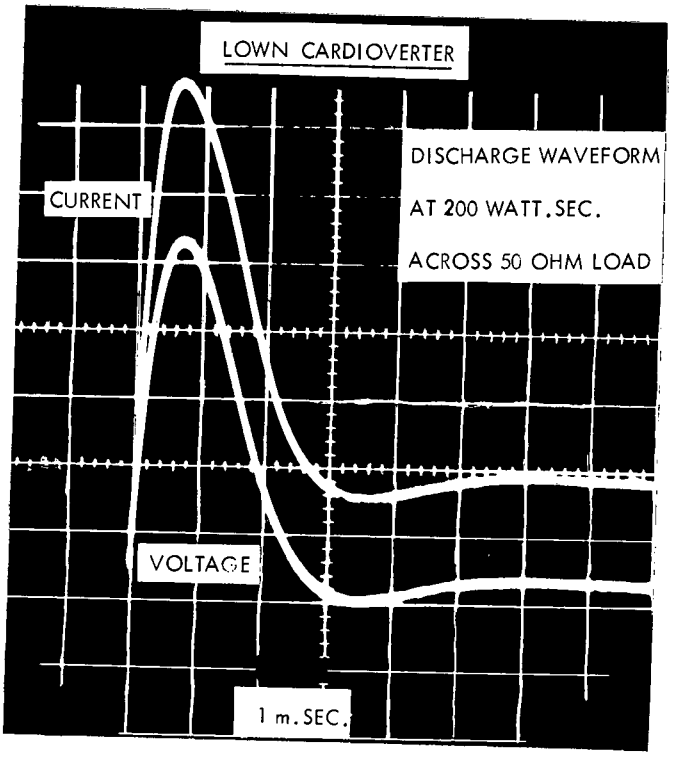


FIG. 54: Underdamped voltage and current waveform. 16 microfarad capacitor with an inductance coil of 100 millihenrys in circuit (Lown Cardioverter).

$\frac{1}{2} CV^2$, where C = charge in farads
V = voltage.

In contrast the energy delivered by alternating current is equivalent to

(volts x amperes) x duration i.e. wattage x time

measured in joules.

An example of the waveform obtained from a commercially available alternating current defibrillator is shown in Fig.55. The energy needed for defibrillation with a capacitor discharge is less than with alternating current (Lown, Neuman, Amarasingham and Berkovits, 1962) and the heat generated in a patient as a result is also less (Tesdeschi and White, 1954). Furthermore ventricular function is less disturbed in dogs with ventricular fibrillation treated by direct current shock (Yarbrough, Ussery and Whitley, 1964). Energy is not the only factor in the effectiveness of a capacitor discharge, however, so that details of the circuit used with particular reference to inductance and the resultant waveform should always be stated (Mapleson, 1965).

Up to 7,000 volts is needed to charge a capacitor of 16 microfarads to produce an energy level of 400 joules.

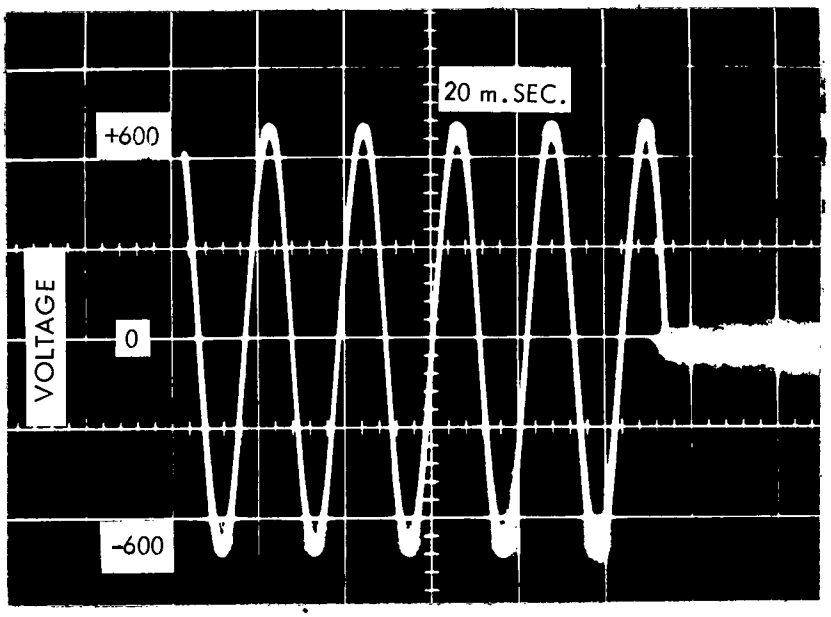


FIG. 55: Waveform recorded from an alternating current defibrillator. 600 volts setting. (Beeley).

A solid dielectric capacitor capable of storing 400 joules weighs about 25 kg. and apparatus using this form of capacitor tends to be bulky. The alternative electrolytic capacitor is more efficient but less reliable and usually cannot be charged to more than 600 volts (Norman, 1965). By using an electrolytic capacitor of about 3,000 microfarads charged to a few hundred volts in association with a pulse transformer, Peleška, Ponanka and Blazek (1962), were able

to design a more portable machine and also one in which earthing problems were eliminated.

From the clinical point of view desirable features of apparatus for synchronised capacitor discharge may be summarised as follows:-

(i) Safety to the patient and operator -

Both paddles should be isolated from the earth potential. If one is earthed, the other will be live and the operator could receive a dangerous shock by touching the electrode or the patient during defibrillation. Furthermore, the patient could receive a burn at the earth electrode and, part of the energy delivered would be dissipated through the earthed electrocardiograph electrode or to any conducting material on which the patient was lying (Towers, Gibson, Burn, Monro, 1965).

(ii) Avoidance of the vulnerable phases of the cardiac cycle -

The shock is usually synchronised with the R or S wave of the electrocardiogram. Provision for changing the ECG lead to one in which an unequivocal R or S wave is present is highly desirable. It would further be preferable for the signal for synchronisation to be the rate of rise of the R wave rather than its amplitude, to prevent synchronisation with a base-line artefact or sudden movement of the patient.

(iii) Provision for immediate recording of the electrocardiogram -

Oscilloscopic monitoring of the ECG is frequently unsatisfactory in determining the rhythms after synchronised capacitor discharge. To be sure whether sinus rhythm is present a V lead, usually V_1 or V_2 is often needed. A one-channel

direct-writing pen recorded incorporated in the apparatus with provision for lead selection would be desirable.

(iv) Provision for discharging the capacitor -

It is frequently necessary to discharge the capacitor other than across a patient. Allowing the charge to "leak" away could still leave the paddles live when next used unless sufficient time had elapsed.

(v) A switch for re-charging the capacitor -

It should be necessary to set the capacitor to re-charge rather than allowing automatic re-charging to follow the discharge. Unless this is done the paddles are always live from the time of being charged and could constitute a source of danger.

It might be considered that the need for an anaesthetic is a disadvantage of the technique of electrical conversion. Stock (1963), and Lown (1963), have questioned whether an anaesthetic is really necessary. Pantridge and Malmos (1965) use no anaesthetic with energy level settings of 100 joules or less. Patient number 145 admitted in severe circulatory collapse with ventricular tachycardia and ischaemic heart disease was treated without an anaesthetic and had no recollection of the event. Many patients are apprehensive, however, and where more than one shock is needed, or when higher energy level settings are required a brief general anaesthetic seems preferable (Gilston, Fordham and Resnekov, 1965). The combination of pethidine

as pre-medication, sodium thiopentone (Shephard and Vandam 1965) or sodium methohexitone for induction and thereafter nitrous oxide and oxygen by inhalation to which is added 0.5% halothane if needed, is recommended. The administration of suxamethonium to any patient receiving intravenous quinidine should be avoided as the interaction of these two drugs may result in prolonged muscle paralysis and apnoea (Grogono, 1963).

The present series of 150 patients who had atrial fibrillation, atrial flutter, atrial tachycardia and ventricular tachycardia with a success rate of 87% compares favourably with other reports. In the four series reported by Lown (1964), Hurst, Paulk, Proctor and Schlant (1964), Oram and Davies (1964) and Pantridge and Halmos (1965) there were 654 episodes of arrhythmias treated with an overall immediate success rate of 87.3%. In the series of 158 episodes of atrial fibrillation reported by Hurst, Paulk, Proctor and Schlant (1964) a 95% success rate is reported; the extremely high success rate is probably due to the fact that there were only 40 patients who had underlying rheumatic heart disease in contrast to 71 of the 120 patients with atrial fibrillation in the present series.

Atrial fibrillation in the absence of underlying heart disease was a less rewarding group for treatment by synchronised capacitor discharge; 73% of 22 patients were brought into sinus rhythm. Oram and Davies (1964) reported successful immediate results in 12 of 14 patients with lone atrial fibrillation.

Despite the reports of Lown (1964b) and of Oram and Davies (1964) the duration of atrial fibrillation was an important factor in the success or failure of the electrical treatment as confirmed by Pantridge and Halmos (1965). In any patient in whom atrial fibrillation had been present for more than 5 years, the immediate success rate fell progressively to about 50% when the arrhythmia had been present for 10 years.

Similarly, contrary to earlier reports (Oram and Davies, 1964), a cardiothoracic ratio in excess of 60% was found to lessen the chances of success (other than in patients with lone atrial fibrillation). Selective enlargement of the left atrium also mitigated against successful treatment (lone atrial fibrillation excepted).

The presence of coarse f waves in lead CR was found by Aber (1962) to be a good prognostic sign for the ease of

the conversion of atrial fibrillation to sinus rhythm by quinidine. Oram and Davies (1964) who analysed the height of f waves in lead V_1, found no correlation at the 5% level between higher fibrillation waves and the ease of conversion by synchronised capacitor discharge.

As with most other series patients with atrial flutter were particularly gratifying to treat; all were brought into sinus rhythm. In contrast to Lown (1964b) however, not every patient was brought into sinus rhythm on the first shock. The youngest patient in the series was a boy of 2½ years in whom sinus rhythm was achieved with the minimal energy level setting used, namely 10 joules. Atrial flutter following surgical closure of an atrial septal defect is an arrhythmia which in general is quite resistant to drug therapy. There were 9 patients in this group, all 9 were successfully treated and no patient required a large energy level setting.

Seven of eight patients with atrial tachycardia were brought successfully into sinus rhythm which compares with 7 successful treatments in 10 episodes (8 patients) reported by Lown (1946b). Lower energy level settings were required for successful treatment than in atrial fibrillation.

All 5 patients with ventricular tachycardia were successfully treated, only one patient requiring more than one shock. Energy level settings were low throughout. 31 successful treatments of ventricular tachycardia are reported by Lown (1964b) in 32 episodes (15 patients) although an average of 2 shocks per treatment had to be used.

Details of energy levels needed for the successful re-establishment of sinus rhythm analysed according to the duration of the arrhythmia and the radiographic size of the heart or selective enlargement of the left atrium are not given in the literature. No correlation was found in the present series. Most investigators are in agreement with the findings presented here that 67% patients who are successfully treated achieve sinus rhythm following the first shock. All 15 patients who required a maximum energy level setting of 400 joules had atrial fibrillation. Great care was always taken to adopt a uniform method of preparing the skin before application of the paddles so that the skin resistance was probably uniform in all the 150 patients. The considerable difference in energy levels for successful treatment might well depend on the dissipation of energy across the skin and tissues and on differences in the tissue depth between the heart and the paddles and was shown not to be related to the body surface area.

Although Killip (1963), Lown (1964b) and Hurst, Paulk, Proctor and Schlant (1964) all advocated the use of quinidine before electrical conversion quinidine in this series did not affect either the immediate success rate or the energy levels needed for successful treatment.

Using a posterior paddle in conjunction with one anteriorly placed was reported to diminish energy level settings for successful re-establishment of sinus rhythm (Lown, Kleiger and Wolff, 1964), the pathway between the paddles being shortened and the intensity of the electrical field across the heart being augmented. Similar findings were reported by Morris, Kong, North and McIntosh (1964) and by Hurst, Paulk, Proctor and Schlant (1964).

Conspicuously less energy settings were not found in the patients of this series successfully treated using the antero-posterior paddles compared with those in whom the anterior paddles were used.

The immediate failure rate was 12.6% (19 out of 150 patients). 18 out of 120 patients treated for atrial fibrillation (15%) failed to come into sinus rhythm. Lown (1964b), failed to establish sinus rhythm on 19 occasions (8%). An analysis of the causes of atrial fibrillation was only given for 150 of the patients. 63 episodes of atrial

fibrillation, treated by Morris, Kong, North and McIntosh (1964) were unsuccessful in 14 (22%). Towers, Gibson, Burn and Monro (1965) were unsuccessful in 7%. There was a 22% failure rate amongst the patients treated by Pantridge and Halmos (1965). Oram and Davies (1964) reported a 16% failure rate in 100 cases (129 episodes) of atrial fibrillation. The series of 149 patients (158 episodes) of atrial fibrillation reported by Hurst, Faulk, Proctor and Schlant (1964) had a failure rate of 4.2%. The failure rate was 9% in the series of 86 patients (101 episodes) reported by Lemberg, Castellanos, Swenson and Gosselin (1964).

There were no failures amongst patients with atrial flutter and experience in the literature bears out that failures are unusual when treating this arrhythmia (Lown 1964b, Morris, Kong and McIntosh 1964).

Only one patient with atrial tachycardia failed to come into sinus rhythm. 3 out of 10 patients reported by Lown (1964) with atrial or supraventricular tachycardia failed to come into sinus rhythm.

There were no failures amongst the 5 patients with ventricular tachycardia which compares with a 3% failure rate amongst 32 episodes (15 patients) treated by Lown (1964b).

Patients in whom atrial fibrillation was unassociated with underlying disease of the heart had the highest failure rate of any group (27%) even when the heart was not enlarged. Oram and Davies (1964), however, reported a failure rate of only 14% in this group.

The association of haemodynamically significant underlying heart disease with atrial fibrillation increased the likelihood of failure especially when considerable enlargement of the heart and of the left atrium were present, and similar findings are commented on by Lown (1964b) and by Killip (1963).

Age of the patient did not adversely effect the chances of success unlike the findings of Oram and Davies (1964).

Intervening rhythms were frequent and occurred both in patients treated successfully and unsuccessfully. There were 107 episodes following the treatment of 150 patients. Atrial and ventricular ectopic beats were the commonest noted but frequently more than one arrhythmia occurred. Lown (1964b) suggested that these rhythms could relate to overdigitalisation and a lowered potassium content of the myocardial cell produced by prolonged diuretic therapy. Killip (1963) who reported a slow nodal rhythm following direct current shock considered a low myocardial intracellular

potassium content as the cause. In this series intervening rhythms also occurred in patients who had not received diuretic therapy or digitalis. All patients had digitalis stopped for at least 24 hours before synchronised capacitor discharge. Another explanation for transient arrhythmias could be myocardial damage produced by the passage of the electrical current across the heart. Energy level settings of more than 150 joules were used in 23 of 36 patients treated for atrial fibrillation who developed ventricular ectopic beats and in 10 of 31 patients who developed atrial premature contractions. This difference is statistically significant,

$$\chi^2 = 6.7(n,1;\ p > 0.01)$$

and suggests that higher energies are associated with an increased incidence of ventricular ectopic beats.

The occurrence of arrhythmias following capacitor discharge depend on the voltage used rather than the total energy delivered (Peleška, 1963) and are related to myocardial damage. The capacitor is charged to 7,000 volts when the highest energy levels are used.

40 patients (39%) who had atrial fibrillation remained in sinus rhythm following the first treatment. In the series reported by Hurst, Faulk, Proctor and Schlant (1964)

58% of 121 patients with atrial fibrillation have remained in sinus rhythm following synchronised capacitor discharge, but the follow-up period is shorter. 92% of 65 patients have persisted in sinus rhythm over a 3½ month follow-up (Pantridge and Halmos, 1965). Similarly, 79% of 56 patients (49 were treated for atrial fibrillation and 7 for atrial flutter) have remained in sinus rhythm over a follow-up period also much shorter, extending from 1 to 9 months (Morris, Kong, North and McIntosh, 1964). 67% of maintained sinus rhythm in 53 successful episodes of treatment for atrial fibrillation amongst 46 patients is reported by Killip (1963) over 8 months of follow-up. Miller (1964), however, reported that over a 3 month period of follow-up 50% of 28 patients remained in sinus rhythm. 42% of 100 patients remained in sinus rhythm up to 3 months following treatment, (Towers, Gibson, Burn and Monro, 1965) and less than one third of the patients reported by Rabbino, Likoff and Dreifus (1964) have remained in sinus rhythm. 50% of patients with longstanding atrial fibrillation will remain in sinus rhythm (Lown, 1964b) following synchronised capacitor discharge and this is in accordance with the findings of Oram and Davies (1964) who reported that 47.5% of 84 patients remained in sinus rhythm. In this series, too, however the follow-up is relatively short.

24 patients in whom atrial fibrillation was associated with chronic rheumatic heart disease have remained in sinus rhythm. These findings are similar to those of Lown (1946b) and it is significant that in the series of Hurst, Paulk, Proctor and Schlant (1964) in which 58% have remained in sinus rhythm up to 9 months after treatment, rheumatic heart disease was associated in only 40%.

It appears therefore that persisting underlying heart disease decreases the likelihood of sinus rhythm being maintained after version and this is especially true of rheumatic heart disease. Paradoxically only 38% of the patients with "lone" atrial fibrillation remain in sinus rhythm. Post thyrotoxic atrial fibrillation as a group maintained sinus rhythm well following synchronised capacitor discharge.

Patients in whom atrial fibrillation was of short rather than long duration and those in whom radiographic enlargement of the heart was least (cardiothoracic ratio and size of the left atrium) were more likely to maintain sinus rhythm. Furthermore, electrocardiographic abnormalities were present in only 9 patients in whom sinus rhythm was maintained (changes other than rhythm or digitalis effect).

The numbers of patients followed up who had been treated for atrial flutter, atrial tachycardia and ventricular tachycardia is small. Nevertheless sinus rhythm has been maintained in 70% of patients treated successfully for atrial flutter. 78% in whom atrial flutter was associated with post-operative ostium secundum atrial septal defect maintained sinus rhythm.

Only 3 of 8 patients who were treated for atrial tachycardia have remained in sinus rhythm. One of the 8 patients has died. Similarly only 2 of 5 patients who had ventricular tachycardia were in sinus rhythm at the end of the follow-up period, but 2 patients have died.

Sinus rhythm was maintained only for seconds or minutes in 9 patients. In 2, accessory pathways of conduction were responsible. Patient number 65 suffered from the usual form of the syndrome described by Wolff, Parkinson and White (1930). Patient number 5 who was shown to have a short P-R interval but a normal QRS is an example of the syndrome described by Lown, Ganong and Levine (1952) who were able to show that only 15% of patients prone to attacks of paroxysmal tachycardia and in whom the P-R interval is short when in sinus rhythm, suffer from the Wolff-Parkinson-White Syndrome. The mechanism of the

arrhythmia in the group with a normal QRS is similar to the mechanism in the Wolff-Parkinson-White syndrome.

This mechanism, however, is not responsible for sinus rhythm being maintained only temporarily in the other patients. Nevertheless it does underline the fact that the mode of action of synchronised capacitor discharge is to cause instantaneous depolarisation of all the fibres of the heart and so allow the sino-atrial node to take over once more as the pacemaker of the heart. For sinus rhythm to be maintained, however, the pacemaker of the heart must not be diseased or atrophied and alternative pathways of conduction must be absent. There is evidence that the sino-atrial node atrophies when atrial fibrillation has been present for several years (Hudson, 1960). Although an atrial wave of depolarisation may return following electrical conversion, the pressure tracing in such patients frequently shows only a small 'a' in both atria which may be of little functional significance in filling the ventricle (Braunwald, 1964).

Arrhythmias occurring immediately after the establishment of sinus rhythm following direct current shock, especially atrial premature contractions acting as a trigger mechanism to initiate atrial fibrillation or flutter were thought to

be responsible for sinus rhythm being maintained only temporarily in 9 patients (Lemberg, Castellanos, Swenson and Gosselin, 1954). These investigators feel that the routine use of quinidine preceding electrical conversion would reduce the incidence of ectopic beats and would therefore help to maintain sinus rhythm, although the number of patients analysed is too small on which to base firm conclusions.

Incomplete depolarisation of the atria, a result of the direct current energy level setting being too small, was considered to be responsible for atrial fibrillation being converted to atrial flutter, or atrial flutter to atrial tachycardia with a slower atrial rate (Lemberg, Castellanos, Swenson and Gosselin, 1964).

Recurrence of the arrhythmia is particularly liable to occur within the first month after treatment and especially during the first day. 15 of 70 patients in whom the arrhythmia recurred, relapsed within the first 24 hours (excluding 9 in whom sinus rhythm was only temporary). 10% of patients with atrial fibrillation are liable to revert within hours or days of successful treatment by direct current (Lown 1964b) and the largest number of reversions occurred within the first 2 weeks of treatment,

(Rabbino, Likoff, and Dreifus, 1964). 88% of the reversions reported by Towers, Gibson, Burn and Monro (1965) occurred within the first month following treatment.

The patients most likely to revert to atrial fibrillation within the first day of successful synchronised capacitor discharge were those in whom atrial fibrillation was associated with chronic rheumatic heart disease and especially where fibrillation has been present for 3 years or more or where considerable radiographic enlargement of the heart was associated. Prolonged atrial fibrillation and cardiac enlargement were bad prognostic signs for maintaining sinus rhythm and reversion within the first day occurred when one or both were present in patients with cardiomyopathy and ischaemic heart disease. The single patient in the series in whom atrial tachycardia was due to digitalis overdosage not unexpectedly reverted within a few hours of treatment. This is contrary to the experience of Corwin, Klein and Friedberg (1963) who reported the successful electrical treatment of a patient with paroxysmal atrial tachycardia with block thought to be digitalis induced and resistant to drug therapy. As was shown by Oram, Resnekov and Davies (1960) the mechanism of this arrhythmia is usually a lowered potassium content in the myocardial cell so that treatment

should be directed first at correcting the metabolic abnormality. 4 of the 10 patients with lone atrial fibrillation reverted within the first day; unlike the other groups these recurrences were not restricted to patients with long standing atrial fibrillation or severe enlargement of the heart.

Once the first month of maintained sinus rhythm had passed there was a much higher chance of sinus rhythm being maintained in all the groups. Nevertheless by 9½ months after treatment one half of the patients being followed up had reverted to the arrhythmia.

53.5% of patients who reverted to atrial fibrillation showed electrocardiographic changes other than rhythm or digitalis effect.

The incidence of complications rose with higher energy level settings. 6 of 15 patients brought into sinus rhythm with energy level settings of between 300-400 joules are still in sinus rhythm (40%). 6 reverted by the end of the third day and a further patient reverted by the end of the first month so that a decision to use the maximum energy level settings must be taken by weighing the possibility of the benefits of sinus rhythm likely to be of short duration against the risk of increased complications resulting from the treatment.

5% of patients with established atrial fibrillation have no clinically detectable heart disease (Evans and Swann, 1954). 15% of patients with paroxysmal atrial fibrillation have no evidence of underlying heart disease (Parkinson and Campbell, 1930). There is usually no overall cardiac enlargement in both groups unless cardiac failure has occurred and the left atrium is only enlarged to a degree consistent with atrial fibrillation being present over a prolonged period. It might be thought, therefore that these patients would prove an ideal group for treatment by synchronised capacitor discharge.

Lone atrial fibrillation is frequently unassociated with any limitation of function (Orgain, Wolff and White, 1936) a view confirmed by Phillips and Levine (1949). The haemodynamic studies performed in the present series demonstrates that sinus rhythm is beneficial in this group as well. Congestive cardiac failure may occur in patients with lone atrial fibrillation (Brill, 1937). The group of patients in whom atrial fibrillation is familial (Wolff, 1943) is of considerable interest; the male members of the family are particularly affected. Gould (1957) who studied 113 members of a family during the course of 36 years, detected 22 with atrial fibrillation. Atrial fibrillation

was familial in patient number 99 who also had had thyrotoxicosis; two male relatives had developed atrial fibrillation at an early age but neither had thyrotoxicosis.

The immediate results of attempts to establish sinus rhythm with quinidine has been reported to be as high as 85% (Phillips and Levine, 1949), and up to 68% in the series reported by Parkinson and Campbell (1928 - 29). Furthermore, these last investigators felt that the prognosis for maintaining sinus rhythm was particularly good in the group of patients with lone atrial fibrillation. Nevertheless Prinzmetal, Corday, Brill, Oblath and Kruger (1952) felt that patients with lone atrial fibrillation in whom the ventricular rate was satisfactory are preferably left with no attempt made at conversion.

The results of treatment by synchronised capacitor discharge in the present series summarised in Table 55, was far from encouraging and was associated with a high incidence of complications. Furthermore, the relapse rate once sinus rhythm was established was particularly high. These findings are in agreement with Oram and Davies (1964) who attempted electrical conversion in 16 patients with lone atrial fibrillation.

TABLE 55

NUMBER OF PATIENTS	FAILED DC		SINUS RHYTHM ESTABLISHED				SINUS RHYTHM MAINTAINED < 1 month	SINUS RHYTHM STILL PRESENT
	NUMBER	COMPLICATIONS FOLLOWING DC	NUMBER	ENERGY LEVEL SETTING, joules <250	>250	COMPLICATIONS FOLLOWING DC		
22	6	3	16	13	3	2	8	6

Table 55: The short and long-term results of treatment by synchronised capacitor discharge in 22 patients with lone atrial fibrillation.

There is considerable disagreement on the need for maintenance quinidine following direct current shock. In this series patients selected at random were maintained on an average of 1.0 G quinidine per day as experience with patients numbers 1 - 50 suggested that serious side-effects might result with bigger dosages. Oram and Davies (1964) abandoned a controlled trial using 1.5 G quinidine per day after direct current shock because of episodes of asystole and ventricular fibrillation amongst 32 patients. 26 of 186 patients prescribed quinidine in a dose of 800 mg. per day developed toxic symptoms severe enough to demand withdrawal of the drug (Hurst, Paulk, Proctor and Schlant, 1964) and in a further 10 patients milder symptoms occurred. Nevertheless, the need for maintenance quinidine following successful version to sinus rhythm is stressed by Sokolow and Perloff (1960-61) and by Goldman (1959-60). Both these groups of investigators indicate that 80 - 90% of patients can be maintained in sinus rhythm following version of atrial fibrillation to sinus rhythm by quinidine, with a maintenance dose of 1.6 G per day. Quinidine is advocated after successful electrical version by Lown (1946b) and by Hurst, Paulk, Proctor and Schlant (1964). Miller (1964) reported that all patients in his series not receiving quinidine after direct current shock reverted to the

arrhythmia, but patients requiring more than 2.4 G per day of maintenance quinidine were less likely to remain in sinus rhythm.

In the present series, 35 patients of the 55 who were still in sinus rhythm had been maintained on quinidine and 37 patients of 70 who relapsed, were being maintained on quinidine. A chi-square test on these figures gives the result

$$\chi^2 = 3.25 \quad (n = 1,\ p \leq 0.05)$$

indicating that prescribing quinidine does not appear to be of overwhelming benefit in maintaining sinus rhythm. Furthermore if the chi-square test is applied to the groups following the second treatment (67 in sinus rhythm, 37 of whom are on maintenance quinidine; 58 reversions, 29 of whom were on maintenance quinidine) the result is

$$\chi^2 = 1.17 \quad (n = 1,\ p > 0.30)$$

indicating that maintenance quinidine is even less statistically significant.

The majority of patients who revert to the arrhythmia following the establishment of sinus rhythm do so within the first month. At the end of the first month after treatment 88 patients were in sinus rhythm of whom 42 were on maintenance

quinidine; 37 patients had reverted of whom 14 were on maintenance quinidine. The chi-square test applied to these groups gives the result

$$\chi^2 = 2.35 \quad (n = 1,\ p < 0.10)$$

suggesting that once more quinidine is not statistically important in maintaining sinus rhythm. A pilot trial to determine whether propranolol would maintain sinus rhythm following synchronised capacitor discharge was undertaken by Tsolakas, Davies and Oram (1964) which gave negative results. Furthermore this drug which removes the sympathetic drive of the heart should be used with caution on a long term basis as it is a myocardial depressant (Hamer, 1965) and reduces the cardiac output both at rest and on effort (Robinson, Kahler, Epstein, Braunwald, 1965).

A further attempt to establish sinus rhythm was made in 20 patients, 17 of whom had reverted to sinus rhythm and in 3 of whom the initial treatment had failed. None of the 3 initial failures was successfully brought into sinus rhythm. The follow up on these 17 patients is shorter than on the other patients and the numbers are small. The giving or withholding of quinidine following the second treatment attempt, whereas the opposite was done at the first treatment attempt, did not significantly alter

the duration of maintained sinus rhythm in the patients who reverted within the first month of treatment. 6 patients however, have been in sinus rhythm for considerably longer periods after the second attempt than after the first.

Chapter 6

C O N C L U S I O N S

The aim of this work has been to evaluate the technique of synchronised capacitor discharge in the management of cardiac arrhythmias and to study the haemodynamic significance of atrial systole by investigating a group of patients before and after conversion to sinus rhythm.

It has been shown that in order to appreciate the haemodynamic changes produced by atrial fibrillation and flutter patients have to be studied during severe exercise as well as at rest and that the position of the patient during these measurements is of considerable importance. There is little functional change at rest in a heart normal other than for the arrhythmia, but atrial systole may be important even at rest when the ventricle is diseased. On exercise, however, the beneficial effects of sinus rhythm are clearly demonstrated both with a normal and an abnormal heart; the larger the exercise load the more definite is the demonstration. A disproportionate increase in heart rate occurs when patients

are exercised during atrial fibrillation and flutter which is not entirely abolished by giving digitalis. This leads in turn to a reduction of the physical work capacity. The cardiac output and stroke volume are less for equivalent work loads during the arrhythmia. In contrast once sinus rhythm has been achieved equivalent exercise loads are performed at a slower heart rate, the physical work capacity is higher and cardiac output and stroke volume are significantly increased due to a narrower arterio-venous oxygen difference. Whilst some of the disability of the atrial arrhythmia relates to the faster heart rate and therefore to the decreased diastolic filling time of the ventricle, it was also found possible to compare the stroke volume at rest and on exercise during the arrhythmia and in sinus rhythm but at equivalent heart rates. In sinus rhythm the stroke volume is larger and is held more constant as the heart rate increases under the stress of exercise even when the diastolic filling time is identical. These studies demonstrated, therefore that with the stress of exercise or in the presence of a diseased ventricle the booster pump action of atrial systole actively filling the ventricle is of considerable haemodynamic benefit.

150 patients with atrial and ventricular arrhythmias were treated by synchronised capacitor discharge and the overall success rate was 87%. 50 of these patients were shown to have an arrhythmia which was resistant to drugs to maximum tolerance yet electrical conversion was successful in 85%. The chances of successful conversion diminish when atrial fibrillation has been present for more than 3 years especially when associated with significant mitral regurgitation, when considerable radiographic enlargement of the heart is shown, when more than moderate enlargement of the left atrium occurs and electrocardiographic changes of ventricular hypertrophy are present. Patients with atrial flutter, atrial tachycardia and ventricular tachycardia have a 98% chance of being brought into sinus rhythm irrespective of underlying heart disease or duration of the arrhythmia.

Intervening rhythms were frequent and occurred immediately after the direct current shock both in patients treated successfully and unsuccessfully. All were transitory and occurred also in patients who had not been treated with digitalis or diuretics. It was possible to demonstrate that the development of ventricular ectopic beats related to higher energy level settings.

Complications were not infrequent and occurred in 14% of the patients treated. They included raised serum enzyme levels, emboli, pulmonary oedema and enlargement of the heart, systemic hypotension and persisting multifocal ventricular ectopic beats. Complications were frequently multiple and were often related to higher energy level settings. Myocardial damage was postulated as the cause in many.

3 deaths occurred which were directly or indirectly related to the electrical treatment and there were 3 further deaths during the follow-up period.

Synchronised capacitor discharge causes instantaneous depolarisation of all the myocardial fibres and so allows the sino-atrial node to resume as the natural pacemaker of the heart. Whilst very effective in allowing sinus rhythm to resume it will not cause sinus rhythm to be maintained. Recurrence of the arrhythmia was frequent and took place in 6% within seconds of being successfully brought into sinus rhythm. For sinus rhythm to be maintained the sino-atrial node must not be diseased or atrophied and accessory pathways of conduction must be absent. Atrial premature contractions occurring after the direct current shock could act as a trigger mechanism and initiate atrial arrhythmias shortly after the establishment of sinus rhythm.

21% of patients who reverted to the arrhythmia did so by the end of the first day and 53% by the end of the first month. Those most likely to revert had an arrhythmia present for 3 years or more associated with chronic rheumatic heart disease; the heart was enlarged radiographically and ventricular hypertrophy shown on the electrocardiogram. Conversely those most likely to remain in sinus rhythm had an arrhythmia present for less than one year, the heart was only slightly enlarged and the electrocardiogram was normal other than for rhythm change and digitalis effect. Prescribing quinidine as maintenance did not statistically increase the chance of sinus rhythm being maintained. 17 patients who reverted to their arrhythmia were brought into sinus rhythm for a second time of whom 6 have maintained this rhythm. No patient who failed to achieve sinus rhythm following the first attempt, achieved sinus rhythm on a subsequent attempt.

Patients with lone atrial fibrillation were shown to be relatively resistant to electrical conversion despite the absence of underlying heart disease or enlargement of the heart. Complications in this group were frequent and the relapse rate was high especially over the first month.

A comparison was made of the mode of action of quinidine and synchronised capacitor discharge in the management of cardiac arrhythmias. Present apparatus for electrical conversion frequently falls short of the ideal and suggestions are made to increase the safety both to the operator and to the patient.

Under ideal circumstances synchronised capacitor discharge is safer and more effective than quinidine. It has a very real place in the management of acute arrhythmias of whatever cause associated with circulatory collapse. These desperately ill patients usually respond to small energy level settings, anaesthesia is not mandatory and the sudden improvement in the circulation with the establishment of sinus rhythm is gratifying both to the patient and to the doctor. Conversely patients with a chronic arrhythmia who require maximum energy level settings to achieve sinus rhythm usually maintain this rhythm only for a disappointingly short space of time. Although these patients do achieve significant haemodynamic benefit in sinus rhythm, complications following electrical conversion occur and relate to the higher energy level settings. It is doubtful therefore whether an energy level setting of more than 300 joules is justified in this group. Each

patient, however, requires individual assessment to decide whether the haemodynamic benefit of sinus rhythm likely to be for a brief period is justified.

Synchronised capacitor discharge on the whole is an easy method of treatment, both for the patient and the physician. Therein, however, lies its particular danger. Complications are by no means rare and myocardial damage may occur. Its use should be restricted therefore to those patients with a chronic arrhythmia in whom maintained sinus rhythm can reasonably be expected.

APPENDIX A

Heart Rate and Physical Work Capacity.
6 Patients.

A DURING ARRHYTHMIA.

Patient sits on bicycle ergometer.

ECG leads attached (lead CR_5)

	min	sec		
Control	0	30	ECG	
	1	0	ECG	
	1	30	ECG	
	2	0	ECG	
	2	30	ECG	
	3	0	ECG	
	3	30	ECG	Insert mouthpiece, attach nose-clip
	4	0	ECG	Begin expired air collection
	4	30	ECG	
	5	0	ECG	
	5	30	ECG	
Begin 1st work load	6	0	ECG	End air collection; remove mouthpiece and nose-clip

Repeat 0 min 30 sec to 6 min 0 sec

Begin 2nd work load

Repeat 0 min 30 sec to 6 min 0 sec.

Begin 3rd work load

Repeat 0 min 30 sec to 6 min 0 sec

End of investigation.

B REPEAT A IN SINUS RHYTHM.

APPENDIX B

Cardiac Output Study
14 patients

A DURING ARRHYTHMIA

Patient lies on catheter room table.

ECG leads attached (lead CR_5)

Portex tubing passed percutaneously from arm vein to pulmonary artery

Pressure recorded from pulmonary artery (PAP)

Teflon tubing passed into brachial artery by Seldinger technique.

Pressure recorded from brachial artery, (BAP)

Patient moves to sit on bicycle ergometer

PA pressure, BA pressure and ECG recorded

(continued on next page)

Control	min.	sec.				
	0	30	ECG	PAP	BAP	
	1	0	ECG	PAP	BAP	
	1	30	ECG	PAP	BAP	
	2	0	ECG	PAP	BAP	
	2	30	ECG	PAP	BAP	
	3	0	ECG	PAP	BAP	
	3	30	ECG	PAP	BAP	Insert mouth-piece, attach nose-clip.
	4	0	ECG	)		Begin expired air collection
	4	30	ECG	)	PA sample	
	5	0	ECG	)	BA sample	
	5	30	ECG	)		
Begin 1st work load	6	0		PAP	BAP	End air collection. Remove mouth-piece and nose-clip

Repeat 0 min. 30 sec. to 6 min. 0 sec.

Begin 2nd work load

Repeat 0 min. 30 sec. to 6 min. 0 sec.

Begin 3rd work load

Repeat 0 min. 30 sec. to 6 min. 0 sec.

End of Investigation.

B REPEAT A IN SINUS RHYTHM

APPENDIX C

Cardiac Output Study and Synchronised Capacitor Discharge — 1 patient.

A DURING ARRHYTHMIA

Patient lies on catheter-room table

ECG leads attached

Right saphenous vein cut down

Cournand cardiac catheter advanced to pulmonary artery under X-Ray control

Pressure recorded from pulmonary artery (PAP)

Teflon tubing passed into femoral artery by Seldinger technique and advanced to ascending aorta

Pressure recorded from aorta (AP)

Patient moves to sit on bicycle ergometer

PA pressure, aortic pressure and ECG recorded

(continued on next page)

	min.	sec.				
Control	0	30	ECG	PAP	AP	
	1	0	ECG	PAP	AP	
	1	30	ECG	PAP	AP	
	2	0	ECG	PAP	AP	
	2	30	ECG	PAP	AP	
	3	0	ECG	PAP	AP	
	3	30	ECG	PAP	AP	Insert mouthpiece, attach nose-clip
	4	0	ECG			Begin expired air collection
	4	30	ECG	PA sample		
	5	0		Aortic sample		
	5	30	ECG			
Begin work at 100 kpm/min.	6	0	ECG	PAP	AP	End air collection. Remove mouthpiece and nose-clip

Repeat 0 min. 30 sec. to 6 min. 0 sec.

Patient moves back to catheter room table.

B SYNCHRONISED CAPACITOR DISCHARGE

General anaesthetic - I.V. Sodium methohexitone, 100 mg.

Energy level for version, 200 joules (antero-posterior paddles, pulmonary artery and aortic pressure recorded with ECG throughout.

Recovery from anaesthetic - 60 minutes.

C IN SINUS RHYTHM

Patient moves from table to sit on bicycle ergometer

PA Pressure, aortic pressure and ECG recorded

Repeat control and work load at 100 kpm/min. as under A

Patient moves back to catheter room table

Catheter and tubing removed.

End of investigation.

APPENDIX D

Abbreviations used in Tables

A	- After
AC	- Alternating current
AF	- Atrial fibrillation
A Flut	- Atrial flutter
AI	- Aortic regurgitation
Alc	- Alcoholic
Ant	- Anterior
Anticoag	- Anticoagulant
AOVD	- Aortic valve disease
ASD	- Atrial septal defect
A Tachy	- Atrial tachycardia
Atrial pc	- Atrial premature contraction
AVO_2 diff	- Arterio -venous oxygen difference
B	- Before
BA	- Brachial artery
Bradycard	- Bradycardia
BSA	- Body surface area
Cer. emb.	- Cerebral embolus
Chr.	- Chronic
C.O.	- Cardiac output

Cor. Transp.	- Corrected transposition
CTR%	- Cardiothoracic ratio %
d	- Day
DC	- Direct current
Dig.	- Digitalis effect
Dystrophia myoton	- Dystrophia myotonica
ECG	- Electrocardiogram
ESR	- Erythrocyte sedimentation rate
F	- Female
G. and Gr	- Gram
HD	- Heart disease
hr	- Hour
HS	- Heart sound
Hyp	- Hypertension
IHD	- Ischaemic heart disease
Inc.	- Incomplete
Inf.	- Inferior
inf.	- Infarction
inf. resec.	- Infundibular resection
Isch. seg.	- Ischaemic segment
kpm	- Kilopond metre
L	- Litre
LBBB.	- Left bundle branch block

LA	- Left atrium
LDH	- Lactic dehydrogenase
LVH	- Left ventricular hypertrophy
M	- Male
m^2	- square metre
MI	- Mitral regurgitation
mg	- milligramme
min	- minute
ml	- millilitre
mm Hg	- millimetre of mercury
mo	- month
MS	- mitral stenosis
Myoc. inf.	- myocardial infarction
N	- Normal
No	- Number
Obstr. cardiomyop	- Obstructive cardiomyopathy
PA	- Pulmonary artery
PHT	- Pulmonary hypertension
Post	- Posterior
post op	- Post operative
Proc. amide	- Procaine amide
Pulm	- Pulmonary
PVR	- Pulmonary vascular resistance

PWC	- Physical work capacity
Q	- Quinidine
RBBB	- Right bundle branch block
Rh. HD	- Rheumatic heart disease
RVH	- Right ventricular hypertrophy
SBE	- Subacute bacterial endocarditis
SGOT	- Serum glutamic oxaloacetic transaminase
Sten	- Stenosis
S.V.	- Stroke volume
Thyrotox.	- Thyrotoxicosis
Valvot.	- Valvotomy
VE	- Ventricular ectopic beat
VF	- Ventricular fibrillation
VSD	- Ventricular septal defect
VT	- Ventricular tachycardia
Wand. pacem	- Wandering pacemaker
Wk	- Week
WPW	- Wolff-Parkinson-White Syndrome
yr	- Year

Severity of symptoms and signs was graded:

Gr 1 - Slight

Gr 2 - Moderate

Gr 3 - Severe

APPENDIX E

ACKNOWLEDGMENTS

This work was undertaken whilst holding the position of Senior Registrar at the National Heart Hospital and was done under the general supervision of Dr. Lawson McDonald to whom I am most grateful for constant encouragement.

I would also like to thank the Physicians and Surgeons of the National Heart Hospital who referred patients for study and treatment.

It is a pleasure to acknowledge the unstinted technical assistance of Mr. John Norman, Chief Technician, the Institute of Cardiology and his staff in obtaining the electrical waveforms, the pressure and ECG records, the analyses of expired air and the Van Slyke blood gas analyses.

I am grateful to the nursing staff of the Wards of the National Heart Hospital and to the Theatre Staff who willingly allowed patients to be treated at times that were often inconvenient.

Dr. Reginald Hudson, Dr. Eunice Lockey and the staff of the Pathology Department, the National Heart Hospital, measured the serum levels of lactic dehydrogenase

and glutamic oxaloacetic transaminase and the blood sedimentation rates, and performed the thrombotests for the anticoagulant control.

Finally I would like to record my gratitude to my wife who willingly undertook more than her share of family duties to enable me to prepare this thesis.

APPENDIX F

DECLARATION

The clinical examination of all the patients both before and after treatment, the interpretation of the chest radiographs and the electrocardiograms, the electrical conversions, the haemodynamic studies, the statistical analyses and the design of the figures and tables were all done personally.

APPENDIX G

REFERENCES

ABER, C.P., (1962). - Quinidine therapy after mitral valvotomy. Thorax, 17, 274.

ANDRUS, E.C., CARTER, E.P., WHEELER, H.A., (1930). - Refractory period of the normally-beating dog's auricle, with a note on the occurrence of auricular fibrillation following a single stimulus. J. Exp. Med., 51, 357.

ALDINI, G., (1819). - General views on the application of Galvanism to medical purposes. J. Callow. London.

BAER, R., WEGLARZ, S., KILLIP, T., (1964). - Cardiac function in atrial fibrillation and sinus rhythm. (Abstr.) Clin. Res., 12, 175.

BALATRE, P.H., LEFEVRE, Cl., MERLEN, J.F., (1960). - Dosage fluorescimétrique de la Quinine et de la Quinidine dans le sérum sanguin et dans les urines. Ann. biol. Clin. 18, 228.

BECK, C.S., PRITCHARD, W.H., FEIL, H.S., (1947). - Ventricular fibrillation of long duration abolished by electric shock. J. Amer. Med. Ass. 135, 985.

BECKMAN, H., In: Treatment in general practice. Philadelphia, 1934, W.B. Saunders Company.

BERGER, L., BROIDA, D., (1957, 1960). - Colorimetric determination of lactic dehydrogenase. Technical Bulletin no. 500. Sigma Chemical Company, St. Louis.

BERRY, J.N., THOMPSON, H.K. Jnr., MILLER, D.E., McINTOSH, H.D., (1959). - Changes in cardiac output, stroke volume and central venous pressure induced by atropine in man. Amer. Heart J., 58, 204.

BEVEGÅRD, S., HOLMGREN, A., JONSSON, B., (1960). - The effect of body position on the circulation at rest and during exercise, with special reference to the influence on the stroke volume. Acta. physiol. scand., 49, 279.

BLUMGART, H., (1924). - The reaction to exercise of the heart arfected by auricular fibrillation. Heart, 11, 49.

BRAUNWALD, E., (1964). - Symposium on Cardiac Arrhythmias. Introduction. With comments on the hemodynamic significance of atrial systole. Amer. J. Med., 37, 665.

BRAUNWALD, E., FRAHM, C.J., (1961). - Studies on Starling's law of the heart. IV. Observations on the hemodynamic functions of the left atrium in man. Circulation, 24, 633.

BRILL, I.C., (1937). - Auricular fibrillation with congestive failure and no other evidence of organic heart disease. Amer. Heart J., 13, 175.

BROCH, O.J., MULLER, O., (1957). - Haemodynamic studies during auricular fibrillation and after restoration of sinus rhythm. Brit. Heart J., 19, 222.

BRODIE, B.B., BAER, J.E., CRAIG., L.C., (1951). - Metabolic products of the cinchona alkaloids in human urine. J. biol. Chem. 188, 567.

BRODIE, B.B., UDENFRIEND, S., (1943). - Estimation of quinine in human plasma, with a note on estimation of quinidine. J. Pharmacol. & Exp. Therap., 78, 154.

BURCHELL, H.B., (1964). - A clinical appraisal of atrial transport function. Lancet 1, 775.

CHAVEAU, A., and MAREY, (1863). - Appareils et expériences cardiographiques. Démonstration nouvelle du mécanisme des mouvements du coeur par l'emploi des instruments enregistreurs à indications continues. Mem. Acad. Méd. (Paris) 26, 268.

COMEAU, W.J., and WHITE, P.D., (1942). - Critical analysis of standard methods of estimating heart size from roentgen measurements. Amer. J. Roentgenol., 47, 665.

CONN, H.L., LUCHI, R.J., (1964). - Some cellular and metabolic considerations relating to the action of quinidine as a prototype anti-arrhythmic agent. Amer. J. Med., 37, 685.

CORWIN, N.D., KLEIN, E.J., FRIEDBERG, C.K. (1963). - Countershock conversion of digitalis associated paroxysmal atrial tachycardia with block. Amer. Heart J., 66, 804.

CRAMÉR, G., VARNAUSKAS, E., WERKÖ, L. (1963). - A new quinidine preparation with sustained release. Acta med. Scand., 173, 511.

de BOER, S., (1921). - On the fibrillation of the heart. J. Physiol., 54, 400.

DANZER, C.S., (1919). - Cardiothoracic ratio, an index of cardiac enlargement. Amer. J. med. Sci. 157, 513.

DAWSON, J.B., (1960). - The E.S.R. in a new dress. Brit. med. J., 1, 1697.

DONALD, K.W., BISHOP, J.K., CUTTING, G., WADE, O.L., (1953). - The effect of nursing positions on the cardiac output in man. Clin. Sci., 12, 199.

DUBOIS, D., DUBOIS, E.F., (1916). - A formula to estimate the approximate surface area if height and weight be known. Clinical calorimetry. Tenth paper. Arch. intern. Med., 17, 863.

DUCHOSAL, F., (1931). - Etude électrocardiographique des effets de l'electrocution sur le coeur. Comp. Rendu. Soc. de Biol., 106, 12.

EVANS, W., SHANN, F., (1954). - Lone auricular fibrillation. Brit. Heart J., 16, 189.

FERRER, M.I., HARVEY, R.M., WERKÖ, L., DRESDALE, D.T., COURNAND, A., RICHARDS, D.W. Jnr., (1948). - Some effects of quinidine sulfate on the heart and circulation in man. Amer. Heart J., 36, 816.

FERRIS, L.P., KING, B.G., SPENCER, P.W., WILLIAMS, H.B., (1936). Effects of electrical shock on the heart. Electrical Engineering, 55, 498.

FICK, A., (1870). - Ueber die Messung des Blutquantums in den Herzventrikeln. xiv. Sitzung am 9 Juli 1870., (In Verh. phys-med. Ges. Wurzb (1869-1872) n.s. 2 heft 1 and 2, p. xvi).

FREY, W. (1918). - Ueber Vorhofflimmern beim Menschen und seine Beseitigung durch Chinidin. Berlin Klin. Wchnschr. 55, 450.

GALVANI, (1737-1798). - As quoted by KOUWENHOVEN, W.B. (1964). - The effects of electricity on the human body. Bull. Johns Hopkins Hosp. 115, 425.

GESELL, R.A., (1911). - Auricular systole and its relation to ventricular output. Amer. J. Physiol., 29, 32.

GESELL, R.A., (1916). - Cardiodynamics in heart block as affected by auricular systole, auricular fibrillation and stimulation of the vagus nerve. Amer. J. Physiol. 40, 267.

GILSTON, A., FORDHAM, R., RESNEKOV, L. (1969). - Anaesthesia for direct current shock in the treatment of cardiac arrhythmias. Brit. J. Anaesth., in the press.

GLENN, W.W.L., MAURO, A., LONGO, E., LAVIETES, P.H., LACKAY, S.J., (1959). - Remote stimulation of the heart by radio-frequency transmission. Clinical application to a patient with Stokes-Adams Syndrome. New Engl. J. Med., 261, 948.

GOLD, H., (1950). - Quinidine in Disorders of the Heart. New York. Hoeber, 1950.

GOLDMAN, M.J., (1959-60). - The management of chronic atrial fibrillation: indications for and methods of conversion to sinus rhythm. Progr. cardiovasc. Dis., 2, 465.

GOLDMAN, M.J., Principles of Clinical Electrocardiography. 4th Edition, Los Altos, 1962. Lange.

GORDON, A., (1745). - Cited by P. Benjamin, "The Intellectual Rise in Electricity", Longmans & Co., London & New York, 1895.

GOULD, W.L., (1957). - Auricular fibrillation. Report on a study of a familial tendency 1920-1956. Arch. Int. Med., 100, 916.

GRAETTINGER, J.S., CARLETON, R.A., MUENSTER, J.J., (1964). - Circulatory consequences of changes in cardiac rhythm produced in patients by transthoracic direct-current shock. J. clin. Invest., 43, 2290.

GRAF, W.S., ETKINS, P., (1964). - Ventricular tachycardia after synchronised direct current countershock. J. Amer. med. Ass., 190, 470.

GRANT, C., BUNNELL, I.L., GREENE, D.G., (1964). - The reservoir function of the left atrium during ventricular systole. An angiocardiographic study of atrial stroke volume and work. Amer. J. Med., 37, 36.

GROGONO, A.W., (1963). - Anaesthesia for atrial defibrillation. Lancet, 2, 1039.

GURVICH, N.L., (1952). - Vosstanovleniye zhiznennikh funktsiy organizma posle smertelnoy elektrotravmy. Klin. med. (Mosk.), 30, 60. Translated by P. Gilbert.

GURVICH, N.L., YUNIEV, G.S., (1947). - Restoration of heart rhythm during fibrillation by a condenser discharge. Amer. Rev. Soviet Med., 4, 252.

GUYTON, A.C., SATTERFIELD, J., (1951). - Factors concerned in electrical defibrillation of the heart, particularly through the unopened chest. Amer. J. Physiol., 167, 81.

HAMER, J., (1965). - Personal communication.

HANSEN, W.R., McCLENDON, R.L., KINSMAN, J.M., (1952). - Auricular fibrillation: hemodynamic studies before and after conversion with quinidine. Amer. Heart J. 44, 499.

HARVEY, W., (1628). - De Motu Cordis. Keynes Edition, 1928.

HENDERSON, Y., (1906). - The volume curve of the ventricles of the mammalian heart and the significance of this curve in respect to the mechanics of the heart beat and the filling of the ventricles. Amer. J. Physiol., 16, 325.

HIRSCHFELDER, A.D., (1908). - The volume curve of the ventricles in experimental mitral stenosis and its relation to physical signs. Bull. Johns Hopkins Hosp., 19, 319.

HOFFA, M., LUDWIG., C., (1850). - Einige neue Versuche uber Herzbewegung. Ztschr. f. rationelle Medizin, 9, 107.

HOFFMAN, B.F., (1957). - The action of quinidine and procaine amide on single fibres of dog ventricles and specialised conducting systems. An. Acad. brasil. Cien, 29, 365.

HOFFMAN, B.F., CRANEFIELD, P.F., (1964). - The physiological basis of cardiac arrhythmias. Amer. J. Med., 37, 670.

HOFFMAN, B.F., SUCKLING, E.E., BROOKS, C.McC., (1955). - Vulnerability of the dog ventricle and effects of defibrillation. Circulat. Res., 3, 147.

HOFFMAN, I., (1964). - Personal communication to Graf, W.S., and Etkins, P. (1964).

HOLMGREN, A., JONSSON, B., LEVANDER, M., LINDERHOLM, H., SJOSTRAND, T., STROM, G., (1957). - Low physical working capacity in suspected heart cases due to inadequate adjustment of peripheral blood flow (vasoregulatory asthenia). Acta med. scand., 158, 413.

HOLMGREN, A., MATTSSON, K.H., (1954). - a new ergometer with constant work load at varying pedalling rate. Scand. J. clin. Lab. Invest., 6, 137.

HOLMGREN, A., OVERFORS, C.O., (1960). - Heart volume at rest and during muscular work in the supine and in the sitting position. Acta med. scand., 167, 267.

HOLZMAN, D., BROWN, M.C., (1951). - The use of quinidine in established auricular fibrillation and flutter. Amer. J. Med. Sci., 222, 644.

HONEY, M., NICHOLLS, T.T., TOWERS, R.K., (1965). - Pulmonary oedema following direct current defibrillation (letter). Lancet, 1, 765.

HOOKER, D.R., KOUWENHOVEN, W.B., LANGWORTHY, O.R., (1933). - The effect of alternating electrical currents on the heart. Amer. J. Physiol. 103, 444.

HUDSON, R., (1960). - The human pacemaker and its pathology. Brit. Heart J., 22, 153.

HUDSON, R., (1964). - Personal communication.

HURST, J.W., FAULK, E.A. Jnr., PROCTOR, H.D., SCHLANT, R.C., (1964). - Management of patients with atrial fibrillation. Amer. J. Med., 37, 728.

JOHNSTONE, M., NISBET, H.I.A., (1961). - Ventricular arrhythmia during halothane anaesthesia. Brit. J. Anaesth., 33, 9.

KAHN, D.R. WILSON, W.J., WEBER, W., SLOAN, H., (1964). - Hemodynamic studies before and after cardioversion. J. thor. cardiovasc. Surg. 48, 898.

KERLEY, P., (1933). - Radiology in heart disease. Brit. med. J., 2, 594.

KILLIP, T., (1963). - Synchronised DC precordial shock for arrhythmias. J. Amer. med. Ass., 186, 1.

KING, B.G., (1954). - The effect of electric shock on heart action with special reference to varying susceptibility in different parts of the cardiac cycle. Ph.D. Thesis, Columbia University, New York, (1954), Aberdeen Press.

KNOX, J.A.C., (1949). - The heart rate with exercise in patients with auricular fibrillation. Brit. Heart J., 11, 119.

KONG, T.Q., PROUDFIT, W.L., (1964). - Repeated direct current shock without myocardial injury. J. Amer. med. Ass., 187, 60.

KORY, R.C., MENELY, G.R., (1951). - Cardiac output in auricular fibrillation with observations on the effects of conversion to normal sinus rhythm. J. Clin. Invest., 30, 653.

KOUWENHOVEN, W.B., (1964). - The effects of electricity on the human body. Bull. John Hopkins Hosp., 115, 425.

KOUWENHOVEN, W.B., MILNOR, W.R., (1954). - Treatment of ventricular fibrillation using a capacitor discharge. J. Appl. Physiol., 7, 253.

LADUE, J.S., WRÓBLEWSKI, F., KARMEN, A., (1954). - Serum glutamic oxaloacetic transaminase activity in human acute transmural myocardial infarction. Science, 120, 497.

LANGWORTHY, O.R., KOUWENHOVEN, W.B., (1930). - An experimental study of abnormalities produced in the organism by electricity. J. Industr. Hyg. 12, 31.

LEMBERG, L., CASTELLANOS, A., SWENSON, J., GOSSELIN, A., (1964). - Arrhythmias related to cardioversion. Circulation, 30, 163.

LEWIS, T., (1912). - Fibrillation of the auricles: its effects upon the circulation. J. exp. Med., 16, 395.

LINENTHAL, A.J., ULICK, S., PATTERSON, L.A., (1947). - Fluorometric measurement of plasma quinidine and its correlation with cardiac effects in man. (Abstr.). J. Clin. Invest., 26, 1188.

LITTLE, R.C., (1951). - Effect of atrial systole on ventricular pressure and closure of the A - V valves. Amer. J. Physiol., 166, 289.

LOWN, B., (1963). - Cardioversion without anesthesia. New Engl. J. Med., 269, 535. (letter).

LOWN, B., (1964a). - "Cardioversion" of arrhythmias I. Mod. Conc. cardiov. Dis. 33, 865.

LOWN, B., (1964b). - "Cardioversion" of arrhythmias, II. Idem., 33, 869.

LOWN, B., AMARASINGHAM, R., NEUMAN, J., (1962). - New method for terminating cardiac arrhythmias. Use of synchronised capacitor discharge. J. Amer. med. Ass., 182, 548.

LOWN, B., GANONG, W.F., LEVINE, S.A., (1952). - The syndrome of short P - R interval, normal QRS complex and paroxysmal rapid heart action. Circulation, 5, 693.

LOWN, B., KAID BEY, S., FERROTH, K.G., ABE, T., (1963a). - Cardioversion of ectopic tachycardias. Amer. J. Med. Sci., 246, 257.

LOWN, B. KAID BEY, S., FERROTH, K., ABE, T. (1963b). - Comparative studies of ventricular vulnerability to fibrillation (abstr.). J. Clin. Invest., 42, 953.

LOWN, B., KLEIGER, R., WOLFF, G., (1964). - The technique of cardioversion. Amer. Heart J., 67, 282.

LOWN, B., NEUMAN, J., AMARASINGHAM, R., BERKOVITS, B.V., (1962). - Comparison of alternating current with direct current electroshock across the closed chest. Amer. J. Cardiol., 10, 223.

LOWN, B., FERROTH, K.G., KAID BEY, S., ABE, T., HARKEN, D.E., (1963). - "Cardioversion" of atrial fibrillation; a report on the treatment of 65 episodes in 50 patients. New Engl. J. Med., 269, 325.

MACDONALD, R.P., SIMPSON, J.R., MOSSAL, E., (1957). - Serum lactic dehydrogenase - a diagnostic aid in myocardial infarction. J. Amer. med. Ass., 165, 35.

MACKAY, R.S., MCCOLLIN, K.E., LEEDS, S.E., (1951). - The effects of electric currents on the canine heart with particular reference to ventricular fibrillation. Ann. Surg., 134, 173.

MALLESON, ... - The design of defibrillating apparatus. In: Resuscitation and cardiac pacing, Ed. Shaw, G. Smith, G., Thomson, T.J. London, 1969. Cassell, p. 114.

MILLER, H.S. Jnr., (1964). - Synchronised precordial electroshock for control of cardiac arrhythmias. J. Amer. med. Ass., 189, 549.

MILNOR, W.R., KNICKERBOCKER, C.G., KOUWENHOVEN, W.B., (1958). - Cardiac responses to transthoracic capacitor discharges in the dog. Circulat. Res., 6, 60.

MODELL, W., (1962-63). - In: Drugs of choice 1962-63. The C.V. Mosby Co., Saint Louis, U.S.A., 1962, p. 395.

MODELL, W., GOLD, H., ROTHENDLER, H.H., (1941). - Use of digitalis to prevent exaggerated acceleration of the heart during physical exercise in patients with auricular fibrillation. J. Amer. med. Ass., 116, 2241.

MORRIS, J.J. Jnr., ESTES, E.L., THOMPSON, H.K. Jnr., KORTH, W.C. and McINTOSH, H.D., (1963). - Cardiac output in atrial fibrillation and ~~/~~ sinus rhythm (abstr.). Circulation, 28, 772.

MORRIS, J.J. Jnr., KONG, Y., KORTH, W.C. McINTOSH, H.D., (1964). - Experience with "cardioversion" of atrial fibrillation and flutter. Amer. J. Cardiol., 14, 94.

McDONALD, E.L., RESHEKOV, L., (1964). - Atrial defibrillation. In "Resuscitation and Cardiac Pacing", ed., Shaw, G., Smith, J., Thomson, T.J., London 1965. Cassell, p. 140.

McDONALD, L., RESHEKOV, L., O'BRIEN, K., (1964). - Direct current shock in treatment of drug resistant cardiac arrhythmias. Brit. med. J., 1, 1468.

McDONALD, L., RESHEKOV, L., ROSS, D., (1963). - Resistant ventricular tachycardia a year after surgical correction of Fallot's Tetralogy, treated by external electrical countershock. Lancet, 2, 708.

McINTOSH, H.D., KONG, Y., MORRIS, J.J. Jnr., (1964). - Hemodynamic effects of supraventricular arrhythmias. Amer. J. Med., 37, 712.

McMICHAEL, J., SHARPEY-SCHAFER, E.P., (1944). - Cardiac output in man by a direct Fick method. Effects of posture, venous pressure change, atropine and adrenaline. Brit. Heart J., 6, 33.

McMILLAN, R.L., SELLERS, C.R., (1947). - Chronic auricular fibrillation. J. Amer. med. Ass., 135, 1132.

NORMAN, J., (1965). - Personal communication.

O'BRIEN, K., RESNEKOV, L., McDONALD, L., (1964). - Direct current countershock in the treatment of drug-resistant arrhythmias. Brit. Heart J., 26, 137.

ORAM, S., DAVIES, J.P.H., (1964). - Further Experience of electrical conversion of atrial fibrillation to sinus rhythm. Analysis of 100 patients. Lancet, 1, 1294.

ORAM, S., DAVIES, J.P.H., WEINBREN, I., TAGGART, P., (1964). - Conversion of atrial fibrillation to sinus rhythm by direct-current shock. Brit. Heart J., 26, 137.

ORAM, S., DAVIES, J.P.H., WEINBREN, I., TAGGART, P., KITCHEN, L.D., (1963). - Conversion of atrial fibrillation to sinus rhythm by direct-current shock. Lancet, 2, 159.

ORAM, S., RESNEKOV, L., DAVIES, J.P.H., (1960). - Digitalis as a cause of paroxysmal tachycardia with atrio-ventricular block. Brit. Med. J., 2, 1402.

OROAIN, E.S., WOLFF, L., WHITE, P.D., (1936). - Uncomplicated auricular fibrillation and auricular flutter. Frequent occurrence and good prognosis in patients without other evidence of cardiac disease. Arch. Int. Med., 57, 493.

OWREN, P.A., (1959). - Thromboteet, a new method for controlling anticoagulant therapy. Lancet, 2, 754.

PARTRIDGE, J.F., HAIMOS, P.B., (1965). - Conversion of atrial fibrillation by direct current countershock. Brit. Heart J., 27, 128.

PARKINSON, J., CAMPBELL, M., (1928-29). - The quinidine treatment of auricular fibrillation. Quart. J. Med., 22, 281.

PARKINSON, J., CAMPBELL, M., (1930). - Paroxysmal auricular fibrillation: a record of 200 patients. Quart. J. Med., 24, 67.

PELEŠKA, B., (1957). - Transthorakální primá defibrilace. Rozhl. Chir., 36, 731. Translated by the author.

PELEŠKA, B., (1958). - La défibrillation transthoracique et directe à haute tension. Anesthesie Analgesie, 15, 238.

PELEŠKA, B., (1959). - Der "Universal defibrillator Prema", ein Gerät zur Beseitigung des Herzkammerglimmerns bei geöffnetem oder geschlossenem Brestkorb. KOVO - Tschechoslow Exportzeitschrift, 9, 5.

PELEŠKA, B., (1960). - A high voltage defibrillator and the theory of high voltage defibrillation. Proceedings, Third International Conference on medical electronics. London 1960, p. 265.

PELEŠKA, B., (1963). - Cardiac arrhythmias following condenser discharges and their dependence upon strength of current and phase of cardiac cycle. Circulat., Res., 13, 21.

PELEŠKA, B., POHANKA, J., BLAZEK, Z., (1962). - Un défibrillateur portatif destiné à la défibrillation transthoracique ou directe, indépendant du réseau et pourvu d'un convertisseur transistorisé. Anesth, Analg. Reanim., 19, 837.

PHILLIPS, E., LEVINE, S.A., (1949). - Auricular fibrillation without other evidence of heart disease. Amer. J. Med., 7, 478.

PREVOST, J.L., BATTELLI, F., (1899). - Sur quelques effets des décharges électriques sur le coeur des mammifères. C.R. Acad. Sci. (Paris), 129, 1267.

PREVOST, J.L., BATTELLI, F., (1900). - Quelques effets des décharges electriques sur le coeur des mammifères. J. Physiol., gen., 2, 40. J. Physiol. Path., gen., 2, 40.

PRINZMETAL, M., CORDAY, E., BRILL, I.C., OBLATH, R.W., KRUGER, H.E. In: The auricular Arrhythmias. Springfield, 1952. Charles C. Thomas, p. 328.

RABBINO, M.D., LIKOFF, W., DREIFUS, L.S., (1964. - Complications and limitations of direct-current counter shock. J. Amer. med. Ass., 190, 417.

RESNEKOV, L., McDONALD, L., (1965). - Pulmonary oedema following treatment of arrhythmias by direct current shock. Lancet, 1, 506.

ROBINSON, B.F., KAHLER, R.L., EPSTEIN, S.E., BRAUNWALD, E., (1965). - Effects of β -adrenergic blockade in man on the hemodynamic response to maximal exercise. Fed. Proc., 24, 590 (Abstr.).

ROKSETH, R., (1963). - Clinical considerations in quinidine therapy of chronic auricular fibrillation. Acta med. scand., 174, 171.

ROKSETH, R., STORSTEIN, O., (1963). - Quinidine therapy of chronic auricular fibrillation: the occurrence and mechanism of syncope. Arch. int. Med., 111, 184.

ROSS, E.M., (1964). - Cardioversion causing ventricular fibrillation. Arch. intern. Med., 114, 811.

ROWE, G.G., EMANUEL, D.A., MAXWELL, G.M., BROWN, J.F., CASTILLO, C., SCHUSTER, B., MURPHY, J.R., CRUMPTON, C.W., (1957). - Hemodynamic effects of quinidine: including studies of cardiac work and coronary blood flow. J. Clin. Invest., 36, 844.

SANNERSTEDT, R., (1960). - Clinical and experimental investigation of a new type of oral prolonged-action tablet (Duretter). Acta med. scand., 167, 245.

SARNOFF, S.J., GILMORE, J.P., MITCHELL, J.H., (1962). - Influence of atrial contraction and relaxation on closure of the mitral valve. Circulat. Res., 11, 26.

SCHÖLANDER, P.F., (1947). - Analyzer for accurate estimation of respiratory gases in one-half cubic centimeter samples. J. Biol. Chem., 167, 235.

SELDINGER, S.I., (1953). - Catheter replacement of the needle in percutaneous arteriography. A new technique. Acta radiol. (Stockh.), 39, 368.

SELLERS, F.J., DONALD, D.E., WOOD, E.H., (1952). - Atrial contribution to stroke volume in dogs with chronic cardiac denervation. Physiologist, 5, 211.

SELZER, A., WRAY, H.W., (1964). - Quinidine syncope. Paroxysmal ventricular fibrillation occurring during treatment of chronic atrial arrhythmias. Circulation, 30, 17.

SEMPLE, T., MURDOCH, W.R., SINCLAIR, R.N. - Practical results of cardiac resuscitation and of cardioversion in the treatment of arrhythmias. In: "Resuscitation and Cardiac Pacing", Ed. Shaw, G., Smith, G., Thomson, T.J., Cassell, London, 1965, p. 152.

SENAC, M. - Traité de la structure du coeur, de son action et de ses maladies. Vol. II, Paris 1749, p. 524, translated by N. Walsh.

SHEPHARD, D.A.E., VANDAM, L.D., (1965). - Anesthesia for cardioversion. Amer. J. Cardiol., 15, 55.

SIEBENS, A.A., HOFFMAN, B.F., ENSON, Y., FARRELL, J.E., BROOKS, C. McC., (1953). - Effects of l-epinephrine and l-nor-epinephrine on cardiac excitability. Amer. J. Physiol. 175, 1.

SIGMA-FRANKEL Method (1956, 1961). - A simplified method for the colorimetric determination of glutamic oxaloacetic and glutamic pyruvic transaminases. Sigma technical Bulletin No. 505, Sigma Chemical Company, St. Louis.

SJÖSTRAND, T., (1947). - Changes in the respiratory organs of workmen at an ore smelting works. Acta. med. scand., Suppl. 196, 687.

SJÖSTRAND, T., (1953). - Volume and distribution of blood and their significance in regulating circulation. Physiol. Rev., 33, 202.

SKINNER, N.S. Jnr., MITCHELL, J.H., WALLACE, A.G., SARNOFF, S.J., (1964). - Hemodynamic consequences of atrial fibrillation at constant ventricular rates. Amer. J. Med., 36, 342.

SMIRK, F.H. and PALMER, D.G., (1960). - A myocardial syndrome: with particular reference to the occurrence of sudden death and of premature systoles interrupting antecedent T waves. Amer. J. Cardiol. 6, 620.

SNELL, E.E., (1965). - Nathan Drake, M.D.: A literary practitioner and his illness. Proc. roy. Soc. Med., 58, 263.

SOKOLOW, M., (1951). - The present status of therapy of the cardiac arrhythmias with quinidine. Amer. Heart J., 42, 771.

SOKOLOW, M., BALL, R.E., (1956). - Factors influencing conversion of chronic atrial fibrillation with special reference to serum quinidine concentration. Circulation, 14, 568.

SOKOLOW, M., PERLOFF, D.B., (1960-61). - The clinical pharmacology and use of quinidine in heart disease. Progr. cardiovasc. Dis. 3, 316.

STEWART, H.J., CRAWFORD, J.H., GILCHRIST, A.R., (1928). - Studies on the effect of cardiac irregularity on the circulation. 1. The relation of pulse deficit to rate of blood flow in dogs subject to artificial auricular fibrillation and to regular tachycardia. J. Clin. Invest., 5, 317.

STEWART, H.J., CRAWFORD, J.H., HASTINGS, A.B., (1926). - The effect of tachycardia on the blood flow in dogs. 1. The effect of rapid irregular rhythms as seen in auricular fibrillation. J. Clin. Invest., 3, 435.

STOCK, R.J., (1963). - Cardioversion without anesthesia (letter). New Engl. J. Med., 269, 534.

STORSTEIN, O., TVETEN, H., (1955). - The hemodynamic effects of restoring normal sinus rhythm in patients with auricular fibrillation. Scand. J. Clin. Lab. Invest., 7, 167.

STRAUB, H., (1910). - The diastolic filling of the mammalian heart. J. Physiol. 40, 378.

SUSSMAN, R.M., WOLDENBERG, D.H., COHEN, M., (1964). - Myocardial changes after direct current electroshock. J. Amer. med. Ass., 189, 739.

TEDESCHI., C.G., WHITE, C.W. Jnr., (1954) . - A morphologic study of canine hearts subjected to fibrillation, electrical defibrillation and manual compression. Circulation, 9, 916.

TOWERS, M.K., GIBSON, R.V., BURN, J.M.B., MONRO, J.A., (1965). - The termination of cardiac arrhythmias by direct current shock. Postgrad. Med. J., 41, 120.

TSOLAKAS, T.C., DAVIES, J.P.H., ORAM, S., (1964). - Propranolol in attempted maintenance of sinus rhythm after electrical defibrillation (letter). Lancet, 2, 1064.

VAN SLYKE, D.D. and NEILL, J.M., (1924). - Determination of gases in blood and other solutions by vacuum extraction and manometric measurements. J. Biol. Chem., 61, 523.

VASSALLI, (1802). - Quoted by GLENN, W.W.L., MAURO, A., LONGO, E., LAVIETES, P.H., MACKAY, F.J., (1959). - New Engl. J. Med., 261, 948.

WAHLUND, H., (1948). - Determination of the physical working capacity. Acta med. scand. Suppl. 215.

WENCKEBACH, K.F. In: die unregelmässige Herztätigkeit und ihre Klinische Bedeutung. W. Engelmann, Leipzig and Berlin, 1914, p. 173.

WETHERBEE, D.G., BROWN, E.G., HOLZMAN, D., (1952). - Ventricular rate response following exercise during auricular fibrillation and after conversion to normal sinus rhythm. Amer. J. med. Sci., 223, 667.

WIGGERS, C.J., KATZ, L.N., (1921). - The contour of the ventricular volume curves under different conditions. Amer. J. Physiol., 58, 439.

WIGGERS, C.J., WEGRIA, R., (1940). - Ventricular fibrillation due to single, localized induction and condenser shocks applied during vulnerable phase of ventricular systole. Amer. J. Physiol., 128, 500.

WILLIAMS, E.J.V., (1958). - The mode of action of quinidine on isolated rabbit atria interpreted from intracellular potential records. Brit. J. Pharmacol., 13, 276.

WILLIS, W.H., (1964). - Letter to the Editor. Amer. Heart J., 67, 575.

WILLIUS, F.A., KEYS, T.E., - In: Cardiac Classics, London 1941. Henry Kimpton, p. 162.

WOLFF, L., (1943). - Familial auricular fibrillation. New Engl. J. Med., 229, 396.

WOLFF, L., PARKINSON, J., WHITE, P.D., (1930). - Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. Amer. Heart J., 5, 685.

WOOD, P., (1958). - Pulmonary hypertension with special reference to the vasoconstrictive factor. Brit. Heart J., 20, 557.

WYMAN, M., (1964). - Personal communication to Graf., W.S., Etkins, P., (1964).

YARBROUGH, R., USSERY, G., WHITLEY, J., (1964). - A comparison of the effects of A.C. and D.C. countershock on ventricular function in thoracotomized dogs. Amer. J. Cardiol, 14, 504.

ZOLL, P.M., (1952). - Resuscitation of the heart in ventricular standstill by external electric stimulation. New Engl. J. Med., 247, 768.

ZOLL, P.M. LINENTHAL, A.J., (1962). - Termination of refractory tachycardia by external countershock. Circulation, 25, 596.

ZOLL, P.M., LINENTHAL, A.J., GIBSON, W., PAUL, M.H., NORMAN, L.R., (1956). - Termination of ventricular fibrillation in man by externally applied electric countershock. New Engl. J. Med., 254, 727.