THE TERMINATION OF CARDIAC ARRHYTHMIAS BY DIRECT CURRENT SHOCK

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THE use of DC shock in the treatment of cardiac arrhythmias has been pioneered by Lown and his associates (1962). We are reporting our first 100 patients treated by this method. For those unfamiliar with this technique a short historical and technical introduction is appended.

Introduction

In 1899 Prevost and Batelli showed that the passage of a small current through the heart muscle (shock) provoked ventricular fibrillation and that the flow of a very strong current over a short period stopped fibrillation (counter-shock). They used both Alternating Current (AC) and Capacitor Discharge (Direct Current—DC) for defibrillating the heart. This discovery remained unexploited until the advent of cardiac surgery.

Ventricular fibrillation in the human heart was first successfully terminated with AC shock by Beck, Pritchard and Feil (1947) in the exposed heart (internal defibrillation) and by Zoll, Lunenthal, Gibson, Paul and Norman (1956) through the intact chest (external defibrillation). DC shock (capacitor discharge) was compared with AC in the treatment of ventricular fibrillation by Kouwenhoven and Milnor (1956) who considered it inferior, but Lown, Neuman, Amarasingham and Bertovitz who restudied the problem in 1962, convincingly demonstrated the superiority of DC.

There are two differences between AC and DC shocks (Fig. 1). The former consists of alternating positive and negative waves of electricity, the latter of a very brief unidirectional pulse. An AC shock lasts 100-200 milliseconds, a DC shock approximately 4 milliseconds. Both the peak voltage and current of a DC shock are very high (about 4,000 volts and 80 amps) so that a very large amount of energy is released in a very short time. An AC shock of similar defibrillating efficiency would have a much smaller current and voltage, but because of very

much longer duration the total energy would be rather larger. The effectiveness of a DC shock can be improved by altering the waveform from the exponential pattern of simple capacitor discharge to a more rounded form by means of an inductance in the circuit.

Zoll and others (1956) showed that AC shock would terminate supraventricular arrhythmias including atrial flutter and fibrillation, but in a proportion of cases ventricular fibrillation was produced. Lown, Kaidbey, Perlroth and Abe (1963) and Lown, Perlroth, Kaidbey, Abe and Harken (1963) explored the mammalian cardiac cycle in a number of species using a DC shock of 2.5 milliseconds duration and found that there was a 'vulnerable period' of 20-40 milliseconds duration just before the peak of the T wave (Fig. 2). A shock delivered during this period would initiate ventricular fibrillation. The risk of hitting this 'vulnerable period' in a cardiac cycle of average length with a DC shock of 4 milliseconds duration is very small and the risk of producing ventricular fibrillation in the human in this way is considered to be about 2%. Obviously, the very much longer AC shock carries a much higher risk of producing ventricular fibrillation. Zoll and Linenthal (1962) reported that AC shock, though effective in the treatment of supraventricular tachycardias, had to be reserved for those cases which were desperate and resisted drug therapy.

It is possible to arrange by electronic means for the DC shock to be delivered at any desired interval after the QRS complex ("synchronised") and well away from the 'vulnerable period'. In practice the shock is delivered about 20 milliseconds after the R wave and the risk of producing ventricular fibrillation appears to be nil. The safety of synchronised DC shock "cardioversion" (Lown and others, 1963) has opened up the whole field of the atrial arrhythmias to electrical defibrillation.

FIG. 1.—Comparison of DC pulse and alternating current AC.

Method

We have used three different machines* for defibrillation, but most of our experience is with a machine which has a pulse transformer output circuit†, thereby eliminating earthing problems. Fig. 3 shows basic circuits of AC and DC defibrillators. The pulse transformer in Fig. 3(3) incorporates the inductance L in Fig. 3(2). It is important that both defibrillating electrodes should be isolated from earth potential. If one electrode is earthed the other will be live and this could be dangerous both to the operator and the patient. The operator could receive a dangerous shock by touching the electrode or the patient during defibrillation. The patient could receive an electrical burn at the earth electrocardiograph electrode, especially if a needle electrode were used. Part of the defibrillating current would pass not through the heart but to the earthed electrocardiograph electrode and to any conducting material on which the patient was lying and would, therefore, be wasted.

Though we have treated patients with other arrhythmias, most of our patients had atrial fibrillation. It quickly became apparent that DC shock was safe, simple and highly effective in restoring sinus rhythm. Patients of any age, with atrial fibrillation of any duration and aetiology, were therefore accepted, but patients in heart failure or with very large hearts seemed certain to revert to atrial fibrillation and were excluded.

FIG. 2.—The 'Vulnerable Period' on the ascending portion of the T wave. Duration of AC and DC shocks.

The embolic risk has been assessed carefully in each case. Where there is considered to be an embolic risk, i.e. with a previous history of embolism, a low cardiac output, or in those few cases where we have suspected more than trivial mitral stenosis, patients have been kept under good anticoagulant control for a month before cardioversion.

Lead II of the electrocardiogram is recorded during the procedure and the record is lost for only two or three seconds immediately after the shock. If P waves are difficult to see after the shock, the left leg electrode is laid on the chest (and lead CR1 recorded). The defibrillating electrodes are placed over the upper right border of the sternum and in the left mid-axillary line. The synchroniser is set to deliver

* Made by the G.U. Manufacturing Co. Ltd., Cardiac Recorders Ltd. and Corbin Farnsworth Inc.† (Oxygenaire).

①

T = Transformer
AC Defibrillator

②

Charging Circuit

R = Rectifier

T = Transformer

DC Pulse Circuit

C = Capacitor

L = Inductance

Conventional DC defibrillator. Capacitor C is charged by transformer T and rectifier R on the left. The inductor L in series with patient gives the shock a more rounded waveform.

③

DC defibrillator using pulse transformer P, which shapes the waveform and steps up the voltage so that an electrolytic capacitor can be used for C.

FIG. 3.

the shock approximately 20 milliseconds after the peak of the R wave. A shock of 80 watt-seconds (joules) is first delivered, and is normally successful. If not, the power is increased in fixed steps to 100, 200, 300 and 400 watt-seconds.

Quinidine, 330 mg., or procaine amide 500 mg. is given by mouth 2-3 hours before the procedure to facilitate conversion. One of our early patients had a bradycardia with frequent ventricular ectopic beats after conver-

sion, and we attributed this to the large dose of digitoxin (0.1 m.g. t.d.s.) which she was receiving. Since then we have omitted digitalis on the day of the procedure, and reduced it one day or two beforehand in those who were on large doses or where the digitalis effect was potentiated by oral diuretics.

The patient is prepared for a general anaesthetic. A combination of hyoscine and either a morphine derivative or phenothiazine has been given as premedication. The choice of drugs is not critical but atropine is probably better avoided because of its tendency to produce a tachycardia.

While the electrocardiograph electrodes are being attached, the patient is given oxygen by face-mask and then anaesthesia is induced with intravenous thiopentone. The dose of thiopentone (300-500 mg.) must be given slowly in view of the patient's cardiac condition; but it must also be sufficient to obtund reflex movement when the defibrillating electrodes are applied, lest extraneous movement should trigger the synchroniser. Suxamethonium (30 mg.) permits smaller doses of thiopentone to be used, for it ensures absolute stillness, but the possibility of causing muscle pains is a distinct disadvantage and is hardly justified for so small a procedure, especially since the muscle contraction of skeletal muscle following DC shock does not necessitate its use. Moreover, suxamethonium itself is capable of producing bizarre ECG changes, and these could confuse the post-defibrillation picture. In our experience use of suxamethonium has been attended by a relatively prolonged apnoea (10 min.) in these cases, though the reason for this has not been elucidated. The use of thiopentone alone is followed by the patient's recovery of consciousness in three or four minutes after the shock; should more than three shocks be necessary for conversion, then either more thiopentone or supplementation with nitrous oxide and halothane may be required. In the desperately ill patient, conversion can be carried out without anaesthesia; the unpleasant sensation thus produced is probably more desirable than the hazards attendant upon general anaesthesia.

After conversion, quinidine has been continued in doses of 200 mg. t.d.s. or occasionally 330 mg. t.d.s. for periods of up to three months. Digitalis has been continued in normal or reduced dosage to try to keep the heart rate fairly slow if atrial flutter or fibrillation should supervene.

TABLE 1

RESULTS OF DEFIBRILLATION IN 100 PATIENTS

Condition associated with atrial fibrillation	No. of patients	No. of defibrillations	Initial failures	No. of patients in sinus rhythm after one month	No. of patients in sinus rhythm after three months
Mitral stenosis (Post operative)	37	41	2	16	12
Mitral valve disease	15	15	0	7	6
Mitral incompetence (None severe)	4	4	0	2	2
Tricuspid stenosis	1	1	0	1	1
"Lone" A.F.	15	15	0	7	7
Cardiomyopathy	5	5	2	0*	0
Cardiac ischaemia	4	8	0	0*	0
Atrial septal defect after closure	9	10	0	7	6
Ventricular septal defect (post operative)	1	1	0	1	1
Coarctation of aorta	1	1	0	0	0
Replacement of aortic valve with Starr valve (post operative)	2	7	0	2	2
Constrictive pericarditis after operation	2	2	0	2	2
Cor pulmonale	2	2	0	1	1
Carcinoma of lung	2	3	2	0	0
TOTAL	100	115	8	48	42

*One patient in each of the two groups died.

Results

Because the procedure was repeated on a number of different occasions in some patients, 115 defibrillations have been carried out in 100 patients. The arrhythmias treated were:—atrial flutter, six times; supraventricular tachycardia, six times; ventricular tachycardia, once; ventricular fibrillation once, the remainder being atrial fibrillation. The patients, whose ages ranged from 18 to 77, are classified by diagnosis in the accompanying table. (Table 1). Atrial fibrillation had been present for periods up to 11 years before defibrillation. Thirty-seven patients were on anticoagulant therapy, most of them as a matter of post-operative routine.

The DC shock acts by depolarising all myocardial fibres simultaneously and after the refractory period the heart starts again in sinus rhythm. The shock cannot maintain sinus rhythm. The procedure has, therefore, been successful if sinus rhythm is recorded after defibrillation, however briefly. Some patients after conversion to sinus rhythm revert to atrial fibrillation while the electrocardiograph is still

recording. Often, and especially if a small shock is successful, the transition from atrial fibrillation to sinus rhythm is quite smooth but intervening rhythms such as ectopic beats of atrial or ventricular origin or nodal rhythm are common. Sometimes nodal rhythm may persist for 24 hours or more before sinus rhythm becomes established.

All but 8 of the 115 defibrillations were successful. Two groups seemed to be especially unfavourable:

1. Patients with cardiomyopathy (i.e., with large hearts without demonstrable cause and without evidence of ischaemic heart disease) where two of five defibrillations failed, and the remaining three patients were back in atrial fibrillation within a month.

2. Two cases of carcinoma of lung—cardioversion was unsuccessful in both though one had been initially defibrillated for a short period.

Thus half the total failures occurred in the seven patients in these two groups.

FIG. 4.—Follow up of 92 patients 3 months after successful defibrillation. On the left patients who had been in atrial fibrillation for less than six months. On the right patients who had been in atrial fibrillation for more than six months. Five patients with cardiomyopathy, two with carcinoma of lung, and one patient with ischaemic heart disease who died have been excluded.

On 79 of the 107 occasions on which defibrillation was successful an output of 80 watt-seconds (the lowest fixed setting on the machine) was sufficient. One hundred watt-seconds was required on 14 occasions, 200 watt-seconds on 11 occasions and 300 watt-seconds on three occasions.

The technique has been remarkably free of complications except in one patient. This man had cor pulmonale and was in atrial fibrillation. Although the synchroniser was set up correctly it would not work when everything was ready and the gain on the electrocardiograph had to be increased. The synchroniser may have been triggered by an artefact, and the electrocardiogram shows that a shock of 80 watt-seconds was delivered on the ascending portion of the T wave. Ventricular fibrillation supervened at once, and though a shock of 100 watt-seconds was ineffective, a shock of 200 watt-seconds restored sinus rhythm. The ventricular fibrillation lasted 15 seconds and the patient was none the worse for it. The synchroniser unit was defective and was replaced.

Two patients died after defibrillation. The first, a man of 64, was moribund from heart failure due to cardiomyopathy with atrial fibrillation. Cardioversion restored sinus rhythm but he deteriorated steadily and died 36 hours later. The second, a man of 47 who had had a large anterior myocardial infarct six weeks previously, became very ill when he developed a supraventricular tachycardia resistant to drug treatment. Cardioversion restored sinus rhythm which persisted for two weeks but thereafter

supraventricular and ventricular tachycardia recurred at shorter and shorter intervals. Drugs would not control the situation and the patient was defibrillated five times. Death occurred 1½ hours after an episode of ventricular tachycardia had been terminated by defibrillation. The patient had regained consciousness after his anaesthetic but then went into ventricular fibrillation. It was felt that efforts to resuscitate him would be pointless. Defibrillation was not thought to have contributed to the fatal outcome in these two cases; indeed it prolonged life in the second case.

There have been no instances of embolism and no hypotensive episodes after conversion. After shocks of 300 watt-seconds transient electrocardiographic changes have been seen. In one patient there was elevation of the S-segment lasting about two minutes. None had clinical or electrocardiographic sequelae. Erythema at the site of the defibrillating electrodes is usually gone in 24 hours.

Some patients have mild side effects (nausea, vomiting, mild visual disturbance) on quinidine 330 mg. t.d.s. With a dose of 200 mg. t.d.s. intolerance was seen in only three patients.

Many patients revert to atrial fibrillation during the first month after defibrillation, most of them during the first week. A few more revert during the next two months. The duration of the atrial fibrillation is the most important factor in determining relapse (Fig. 4). If atrial fibrillation has been present for less than six months, two patients out of three can be expected to be in sinus rhythm three months

after cardioversion; when atrial fibrillation has been present for more than six months less than one patient out of three will be in sinus rhythm three months after cardioversion.

Two patients were transferred to us as emergencies, having developed supraventricular tachycardia some weeks after large cardiac infarcts. Both were very ill, had hypotension and had received large doses of digitalis and procaine amide. A DC shock was given after intravenous opiates. One patient was immediately restored to sinus rhythm and within three minutes his blood pressure rose from 90/70 mm. Hg. to 110/80 mm. Hg. He felt much better and looked much better. The second patient, after a few sinus beats, went into a slow nodal rhythm without any improvement in his condition, but within half an hour sinus rhythm returned spontaneously and he improved dramatically. The shock was felt as a thump in the chest and though painful, the pain did not last long.

Two patients had recurrent supraventricular arrhythmias after replacement of the aortic valve with a Starr prosthesis. One patient was defibrillated four times, the other three times. In these circumstances the decision to try to keep the patient in sinus rhythm with drugs and by repeated defibrillation if necessary, or to control the ventricular rate with drugs and accept a reduced cardiac output, can be very difficult. It is important to avoid toxic doses of drugs which may make defibrillation more difficult, or less effective, e.g., the patient may be converted to nodal rather than sinus rhythm.

In 20 patients with mitral stenosis and atrial fibrillation cardioversion was attempted at the time of valvotomy (using internal electrodes). In five patients cardioversion failed and four patients reverted to atrial fibrillation before the end of the operation. A further 10 patients reverted subsequently usually about the third post-operative day. Only one patient left hospital in sinus rhythm as a result of this procedure. Nine of the 19 patients with atrial fibrillation underwent elective cardioversion before leaving hospital, usually about the 14th post-operative day. It was successful in all but one patient. Four were in sinus rhythm three months later.

Thus the results of cardioversion of the exposed heart in 20 patients undergoing mitral valvotomy were disappointing, and where defibrillation is not readily achieved, there must be a temptation to use larger shocks with the risk of cardiac burns (Rivkin, 1963). Elective cardioversion during the post-operative period

seems to be the method of choice. When an arrhythmia arises at operation, however, DC defibrillation may well be the best treatment.

Discussion

There are three reasons for wanting to restore sinus rhythm in patients with atrial fibrillation.

1. The maximum cardiac output is almost certainly greater in sinus rhythm, even though the resting output may be little changed.

2. Nearly all patients notice that the heart's action is quieter in sinus rhythm.

3. The risk of embolism is less in sinus rhythm.

The effect of controlled atrial fibrillation on the resting cardiac output has been studied by several observers. The results are controversial and are reviewed by Oram and Davies (1964). Immediately after successful defibrillation there is a slowing in ventricular rate suggesting that the stroke volume increases, presumably due to the supercharging action of atrial contraction. The dramatic clinical improvement that results when patients who develop arrhythmias shortly after large myocardial infarcts or major heart surgery are restored to sinus rhythm, can only be due to a rise in resting cardiac output. In this situation, the cardiac output is compromised not only by myocardial injury and lack of atrial boost, but often by functional disturbances in ventricular conduction and hence by poorly co-ordinated ventricular contraction as well.

Embolism is the greatest hazard of conversion to sinus rhythm and the incidence ranges in reported series between 0 and 3.3% (Lown, 1964; Morris, Kong, Ivorth and McIntosh, 1964). Although we have made every effort to avoid it by the use of anticoagulants when they seemed to be indicated, we attributed its absence in this series and a further 43 patients (whose follow-up is too short for inclusion) to good fortune.

Large shocks can cause myocardial damage, may give rise to ectopic beats after defibrillation, and are probably the cause of hypotension after defibrillation. We have rarely had to give shocks of 300 watt-seconds or greater and this may be due to the favourable output circuit of the machine used.

A method which restores sinus rhythm in 92% of a wide variety of patients with cardiac arrhythmias and is free of all but trivial complications, leaves little to be desired. Experience, the use of antero-posterior electrodes described by Lown (1964) and the elimination of unfavourable groups of patients could improve

this figure still further. Maintaining the patient in sinus rhythm is the difficult problem.

We have used small doses of quinidine to try to maintain sinus rhythm. The demonstration by Oram and Davies (1964) that quinidine is a relatively ineffective drug for this purpose, and that large doses are dangerous and toxic confirms two propositions accepted by us from the beginning. It is to be hoped that the effectiveness of electrical defibrillation will lead to the development of a better antiarrhythmic drug than quinidine.

All workers report a high relapse rate after cardioversion of patients with atrial fibrillation. The duration of atrial fibrillation seems to be the most important factor determining relapse. Morris and others (1964) reached the same conclusion. Even after successful mitral valvotomy the duration of atrial fibrillation seems to determine the relapse rate. Nevertheless small numbers of patients with atrial fibrillation of long duration do remain in sinus rhythm for long periods after cardioversion.

Lown (1964) has shown that the onset of atrial fibrillation is presaged by a gradual lengthening of the PR interval over the years. This, and probably other changes in the electrophysiological properties of atrial muscle must set the stage for the positive feedback oscillatory mechanism of atrial fibrillation. It is tempting to speculate that, as the threshold for atrial fibrillation is approached, this oscillation might be triggered by some temporary event and could be terminated by a shock. If the threshold has been considerably exceeded (i.e., if atrial fibrillation has been present for some time) the prospect of maintaining sinus rhythm would be poor.

Summary

External DC shock is now the method of choice for the conversion of patients with atrial fibrillation to sinus rhythm. Large doses of quinidine for this purpose are dangerous and very much less effective. The effectiveness of cardioversion highlights the urgent need for a better antiarrhythmic drug.

The small number of patients with large hearts and heart failure of some weeks' duration in this series were difficult to convert to, or did not remain in, sinus rhythm. Cardioversion is probably not indicated in this group unless the underlying lesion can be treated.

After the cardioversion of patients in atrial fibrillation a relapse rate of approximately 60% is to be expected over a 3 month period. The duration of atrial fibrillation is the most important factor in determining the incidence

of relapse. Nevertheless, a small number of patients with atrial fibrillation of long duration have remained in sinus rhythm for long periods after cardioversion, and in view of the safety of the method this group is still probably worth treating.

Where an arrhythmia occurring shortly after a large myocardial infarct or major cardiac surgery results in a catastrophic fall in cardiac output, and does not respond at once to simple medical treatment, DC defibrillation should be used, without a general anaesthetic if necessary. This can be life saving.

We would like to thank our colleagues at the Brompton and other hospitals who have sent us patients. We are grateful to the surgeons at the Brompton Hospital for their help, and especially to Mr. M. Paneth, F.R.C.S., for his support and encouragement during the early part of this work.

Addendum

1. Our 146th patient, a man of 63, had disabling right hemiplegia after cardioversion. Atrial fibrillation of five years duration was due to thyrotoxicosis which had been controlled medically. The heart size was small, and as the risk of embolism seemed slight, he was not given anticoagulants. Cardioversion was uneventful but two days later he reverted to atrial fibrillation and was found to have hemiplegia.

2. We have demonstrated a high relapse rate in patients who have had atrial fibrillation of long duration before cardioversion. This is certainly true of patients with mitral valve disease but it may not be true of patients in whom arrhythmias are associated with other conditions. Further experience suggests that, in particular, patients who have atrial arrhythmias soon or later after closure of atrial septal defects can remain in sinus rhythm for long periods after cardioversion, even though the arrhythmia had been present for several years.

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