CARDIOVERSION IN THE TREATMENT OF ATRIAL FLUTTER

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HISTORICAL BACKGROUND

Prevost and Batelli (1899) showed that a beating dog's heart could be stopped by an electric shock, and also that a second shock or countershock could start the heart beating again. Beck, Pritchard and Feil (1947) reported the first case of a human being saved by countershock; this was a boy whose heart stopped after removal of a grossly depressed sternum, and his ventricular fibrillation was terminated by shocks delivered through electrodes placed directly on the surface of the heart. Zoll, Linenthal, Norman, Paul and Gibson (1956) showed that external defibrillation was effective in man.

External countershock was used by Alexander, Kleiger and Lown (1961) for converting ventricular tachycardia to sinus rhythm in a patient with coronary artery disease. The reason that electric shock had not been used earlier for rhythms other then ventricular fibrillation stemmed from Prevost and Batelli's first observation. There was an understandable reluctance on the part of doctors to risk changing a patient's abnormal rhythm for ventricular fibrillation, an immediately life-threatening rhythm. Experiments in animals had indicated the presence of a vulnerable perido somewhere in the cardiac cycle, in which if an electrical shock fell ventricular fibrillation would occur. Lown and his colleagues succeeded in demonstrating that there was such a vulnerable period lasting 30 milliseconds in the early part of the T wave of the electrocardiogram in a number of mammals: Lown, Amarasingham, and Neumann (1962); Lown, Kaid Bey, Perlroth and Abe (1963). They were able to do this because they had built a machine which would deliver a shock lasting only 2.5 milliseconds and synchronised to a chosen part of the E.C.G. This machine is the prototype of machines currently in clinical use, when it is desirable that the electric shock by synchronised to avoid the vulnerable period.

INTRODUCTION

Since 1961 cardioversion (synchronised direct-current countershock) has been used in the termination of both supraventricular and ventricular arrhythmias in man, Lown (1967). Initially only cases which were resistant to or were difficult to control with drugs were submitted to cardioversion; also, the majority of the earlier reports were concerned with the restoration of sinus rhythm in patients with mitral stenosis and atrial fibrillation. As ways of avoiding complications became known, the elective use of cardioversion in the place of drugs in the restoration of sinus rhythm in a variety of arrhythmias became a reasonable therapeutic procedure. Atrial flutter treated by cardioversion was first reported by Lown, Kleiger and Wolff (1964).

Twenty-six occasions when atrial flutter in twelve patients were corrected by cardioversion are reported in this paper.

METHOD

All cases diagnosed as atrial flutter fulfilled the electrocardiographic criteria of having regular "saw-tooth" waves in leads II, III, and avf, at about 300 per min. which abolishes the flatness of the isoelectric line, Katz and Pick (1956). In all cases there was atrio-ventricular block, which was increased in the majority by carotid massage. The ventricular rate was usually between 120-180 per min. The rhythm was recorded again before cardioversion.

Digoxin was stopped for 24 hour before cardioversion. Other drugs were continued as necessary. Anti-coagulants were not used.

Cardioversion was performed in the Intensive Care Ward using a "Cardioverter" (Elliot Automation Company) which delivers a shock lasting 4 milliseconds; circular electrodes 3in. in diameter were used. Lead II recording from the patient was displayed on the cardioverter oscillograph, the R wave identified and suitably spread. All shocks were synchronised to occur just after the peak of the R wave. A small test shock of 5-10 watt-seconds was made through the machine, to see that the shock was in fact delivered when required. The machine was then charged to deliver the shock (50 watt-seconds initially).

Conducting jelly (e.g. E.C.G. jelly) was liberally applied to cover the skin over the upper sternum and the apex, to prevent skin burn from

electrical discharge under the electrodes. Shaving of the chest was unnecessary.

Anaesthesia was induced with "Pentothal" or "Briertal", without premedication; the usual precaution of 5 hours starvation was observed. A mouth-piece was inserted and the patient allowed to breathe oxygen-enriched air; endotracheal intubation was not used. Muscle relaxants were not used; with the shock there was often quivering of the muscles of the chest lasting seconds and a throwing up of the arms.

The electrodes were held firmly on the chest (upper sternum and apex) and the shock given by pressing a button on the machine. If cardioversion was unsuccessful as seen on the oscillograph, further shocks were given at higher energy levels. In all cases, anaesthesia was brief, and efforts to awaken the patient were usually commenced about 5-10 minutes after induction. A 12-lead electrocardiogram was recorded after cardioversion.

Patients were kept overnight in the Intensive Care Ward for observation; on many occasions the patient was discharged the next day.

RESULTS

Details of twelve patients undergoing cardioversion are summarised in the table. Full histories of cases 1 and 2 are given.

Palpitations (19 occasions) and dyspnoea (9) were the commonest symptoms. One patient who had varying atrio-ventricular block presented with giddiness. Anxiety was a common feature. None had angina pectoris. Crepitations in the lungs were common (10 occasions). Raised neck veins were seen in many patients but proved difficult to analyse in the presence of tachycardia; on three occasions raised jugular venous pressures were recorded after cardioversion, and on one occasion was accompanied by sacral oedema found on admission.

After cardioversion, there were no episodes of hypotension, embolism, ventricular fibrillation, tachycardia or asystole; ventricular and supra-ventricular extrasystoles were seen not uncommonly in the first minute or so after cardioversion, but persisted only in case 1. On 21 occasions sinus rhythm was obtained by shocks of 40-90 watt-seconds, and on 1 occasion by a first shock of 100 watt-seconds. On 5 occasions, more than one shock was necessary. On 4 occasions atrial flutter was converted to atrial fibrillation. Case 4 was left in fibrillation and given maintenance doses of digoxin which converted him to sinus rhythm. On the other three occasions, atrial fibrillation was converted to sinus rhythm by cardioversion. In no case were there shifts in the ST segments in the post-cardioversion ECG; in cases where a previous ECG showing sinus rhythm was available for comparison, no changes were noted in the Q waves or the T waves.

Recovery from the short light anaesthesia was uneventful in all cases. On regaining consciousness, those patients who had palpitations before cardioversion noticed the absence of palpitations straight away. Some patients also said that they were less breathless, including those who still had crepitations in their lungs; the pulmonary oedema responded quickly to diuretic treatment. None had chest pain after cardioversion, but some complained of a soreness over their chests for a day or so, which was accompanied by erythema under the electrode areas lasting the same period of time. Case 8 recalled receiving "something like a kick in the chest" at his third cardioversion; on all other occasions, the patients were unaware of what happened during anaesthesia. None found cardioversion unpleasant or showed any reluctance when cardioversion was offered for a recurrence of their atrial flutter.

Case 1: At the age of 34 years, he had a myocardial infarction which left him with permanent Q waves with T inversions in leads I, avl, V3-V6. He remained well over the next 14 years. He was admitted with atrial flutter resistant to the digoxin and quinidine given by his general practitioner. Bilateral basal crepitations with raised neck veins were observed. His drugs were stopped in preparation for cardioversion. The rhythm just before cardioversion turned out to be atrial fibrillation; cardioversion produced sinus rhythm with occasional ventricular ectopics. Serum aspartate transaminase (S.G.O.T.) were normal before and after cardioversion. Three days later he was in fast atrial fibrillation; he was digitalised and discharged on Tab. digoxin 0.25 mg. daily. Four days later he presented with a first attack of gout of his left big toe, (serum uric acid 8.5 mg. per 100 ml.), which responded to colchicine. At the same time he was found to have atrial flutter and raised neck veins; three days later when his arthralgia had subsided and he had been off digoxin he was cardioverted for the second time. He remained well on digoxin and quinidine for two months before atrial flutter recurred. Cardioversion was again successful and he was discharged on

quinidine. A month later he was admitted with marked dyspnoea, having stopped quinidine for three days because of a stomach upset. He had atrial flutter, raised neck veins, crepitations, and a gallop. His fourth cardioversion produced sinus rhythm with occasional ventricular ectopics; his neck veins remained raised. Three hours after cardioversion he developed sudden chest pain and hypotension. He maintained sinus rhythm with ventricular ectopics until the next day when he collapsed and died. At post-mortem, the heart weighed 690 g. with biatrial dilatation and biventricular hypertrophy. The apex was partly calcified. The right coronary artery was almost completely occluded 2 cm. from its origin by a plaque extending 4 cm., the anterior descending branch of the left coronary artery was similarly affected by a 3 cm. plaque starting 1 cm. from its origin, whilst the circumflex branch was severely narrowed.

Case 2: This man presented at the age of 43 with palpitations for two years, at first at intervals of several months, but prior to admission more frequently; these attacks of palpitations lasted from a few minutes to several days. He was in atrial flutter and had cardioversions three times in 8 months, atrial flutter recurring each time despite quinidine. In his fourth attack, intravenous lignocaine was tried with showing of the atrial rate. Digoxin was added to his quinidine without effect. Five days later intravenous lignocaine was again given without improvement. An 80 watt-second shock brought on sinus rhythm which lasted only two hours. The next day the same sequence of events was repeated. Oral propranolol was added, and the dose of digoxin increased. Three days later sinus rhythm returned. The dose of digoxin was reduced because of vomiting. He was, therefore, discharged on digoxin, quinidine, propranolol and phenobarbitone. At follow up two months later he was still in sinus rhythm. He was put on phenobarbitone during his fourth admission because on this occasion, but not before, he gave a history of a first attack of epilepsy at the age of 13, attendance at a neurological hospital, and an EEG that was abnormal. He was taken off phenobarbitone two years before. Six weeks after his last cardioversion he was visited at home by his general practitioner who observed grand mal convulsions. Two weeks after this he had an episode of shaking of his right arm. It was useful to know that the shocks of cardioversion delivered under light anaesthesia did not precipitate convulsions in a convulsive-prone person (Fig. 1).

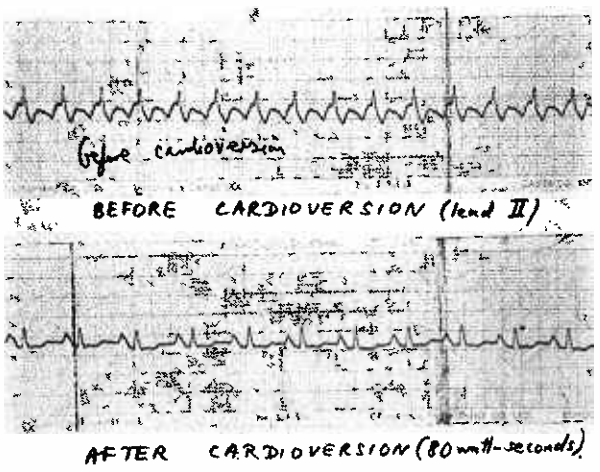

Fig. 1.

DISCUSSION

Over 200 patients with atrial flutter have been treated by cardioversion Killip (1963); Morris, Kong, North, and McIntosh (1964); Towers, Gibson, Burns, and Monro (1965); Szekeley, Batson, and Stark (1966); Castellanos, Lemberg, Gosselin, and Fonseca (1965); Resnekov and McDonald (1967); Lown (1967). A common finding is the uniformly high rate of success of cardioversion with only low energy shocks in terminating atrial flutter and restoring sinus rhythm (97-100%); this was also observed in the present series. This compares favourably with previous reports of conversion of atrial flutter to sinus rhythm with digoxin (50-60%) and with quinidine (30-40%), Belle (1963). An important hazard in the use of the latter drug is that when it is used in the conversion of atrial fibrillation, ventricular fibrillation occurs in 3-5% of cases.

A further notable feature in the above series is the absence of serious complications arising from cardioversion, such as ventricular fibrillation, tachycardia or asystole, and systemic embolism even without the use of anti-coagulants. Castellanos, Lemberg, Gosselin, and Fonseca (1965) had the highest rate of complications, namely, 51% post-cardioversion arrhythmias within 15 minutes of restoration of sinus rhythm, 16% early relapses into atrial flutter in the same period, whilst two patients developed nodal rhythm that gave way to atrial flutter within 24 hours. Their results stand in striking contrast against the low rate of complications encountered by other authors. Thus, Resnekov and McDonald (1967) on reviewing their own cases found an overall complication rate of 14.5% in their cardioversions, but none occurred in their cases of atrial flutter. Possible reasons

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No.	Sex	Age (yrs.)	Diseases	Rhythm before	Drug before	Shock in watt-seconds	Rhythm after	Drugs after	Period in sinus rhythm
1.	M	58	Ischaemic H.D.	Fibrillation	Dig. Quin.	80	Fibrillation		
						160	Sinus R.+V.E.		3 days
				Flutter	Dig.	80	Sinus R.	Dig. Quin.	2 months
				Flutter	Dig. Quin.	50	Sinus R.	Quin.	1 month
				Flutter	Quin	50	Sinus R.+V.E.	Quin.	1 day—Died
2.	M	43	Paroxysmal tachycardia (2 yrs.) Idiopathic epilepsy	Flutter		70	Sinus R.	Quin.	3 months
				Flutter	Quin.	50	Sinus R.	Quin.	5 months
				Flutter	Quin.	50	Sinus R.	Quin.	6 months
				Flutter	Dig. Quin.	80	Sinus R.		2 hrs.
				Flutter		80	Sinus R.		2 hrs.
3.	F	63	Paroxysmal tachycardia (18 yrs.) on Amityptyline for depression	Flutter	Dig. Quin.	80	Sinus R.	Procainamide	2 weeks
				Flutter	Procainamide	60	Sinus R.	Quin.	2 weeks
				Flutter	Quin.	50	Sinus R.	Quin.	3 weeks
				Flutter	Quin.	80	Sinus R.	Quin.	10 months
				Flutter	Quin.	40	Fibrillation		
						80	Sinus R.	Quin. Pheno.	1 month
				Flutter	Quin. Pheno.	90	Sinus R.	Prop. Amytal	3 weeks
				Flutter	Quin. Amytal	80	Sinus R.	Prop. Quin.	3 weeks+
4.	M	62	Paroxysmal tachycardia (6 yrs.)	Flutter	Dig.	50	Flutter		
						80	Fibrillation	Dig.	1 month+
5.	M	81	Ischaemic H.D.	Flutter	Dig.	50	Flutter		
						80	Sinus R.	Dig.	1 month+
6.	F	72	? Ischaemic H.D.	Flutter	Dig.	80	Fibrillation		
						100	Sinus R.	Quin.	1 month +
7.	M	62	? Ischaemic H.D.	Flutter	Dig.	80	Sinus R.	Dig.	
8.	M	54	Paroxysmal tachycardia (20 yrs.)	Flutter	Dig. Quin.	80	Sinus R.	Prop.	1 month
				Flutter	Prop. Pheno.	100	Sinus R.	Prop.	6 months
				Flutter	Prop.	80	Fibrillation		
						150	Fibrillation		
						300	Sinus R.	Prop. Quin.	2 months +
9.	M	58	Paroxysmal tachycardia (1 yr. +)	Flutter		80	Sinus R.	Quin.	2 months +
10.	M	68	Ischaemic H.D. Chronic Bronchitis	Flutter	Dig.	70	Sinus R.	Dig.	2 weeks +
11.	M	59	Ischaemic H.D. Hypertension	Flutter	Dig.	70	Sinus R.	Quin. Dig.	3 weeks +
12.	F	84	Ischaemic H.D.	Flutter	Dig.	80	Sinus R.	Dig.	2 weeks +

V.E.=ventricular ectopics. Sinus R.=sinus rhythm. Dig.=digoxin. Quin.=quinidine. Prop.=propranolol. Pheno.=phenobarbitone.

for the high complication rate encountered by Castellanos, Lemberg, Gosselin and Fonseca (1965) are the use of digoxin in maintenance doses until the time of cardioversion and the use of unnecessarily high energy level shocks, (85% of their cardioversions were with shocks of 100 watt-seconds or more), factors that are avoidable.

Lown (1967) has described a further use of cardioversion in those cases of early relapse into artrial flutter; he advocates the use of cardioversion to induce atrial fibrillation and then digoxin to control the ventricular rate.

The late recurrence of atrial flutter is a feature not disclosed in the above series, with the exception of Szekeley, Batson and Stark (1966) who had three recurrences in two of their patients. Of the 12 patients in the present series, 4 required cardioversion on 17 occasions during the period they were under observation, for 3, 16, 14, 9 months respectively for recurrences of atrial flutter, of which only one recurred within 2 weeks of cardioversion. Three of these patients had a history of paroxysmal palpitations extending over 2, 18 and 20 years. With longer follow-up of these and other patients more recurrences may be expected. The cumulative effect of repeated cardioversion over the years has yet to be assessed. Case 1 died after his fourth cardioversion. The circumstances suggested an acute myocardial infarction occurring three hours after sinus rhythm had been restored by cardioversion, and at post-mortem extensive coronary artery disease was evident. The possibility of cardioversion contributing to his demise seems remote.

The problem of the safety of repeated cardioversions would not arise if there were drugs which effectively suppress paroxysms of cardiac arrhythmias, or which restore sinus rhythm as quickly and as effectively as cardioverison. Quinidine, digoxin, and propranolol, as used in this series have not proved satisfactory in preventing recurrences of atrial flutter. At present, it would seem that the use of drugs in the prevention of paromysmal atrial flutter remains a matter of trying out various drugs or combination of drugs until a suitable regime is evolved for each patient.

SUMMARY

The use of cardioversion in the treatment of patients with atrial flutter is described, and the safety and effectiveness of the procedure discussed.

Four of the patients were cases of paroxysmal atrial flutter and required repeated cardioversions. One of these was a patient with idiopathic epilepsy; repeated cardioversions under light anaesthesia in the absence of administration of anti-convulsive therapy did not bring on epileptic convulsions.

ACKNOWLEDGEMENTS

I am grateful to Dr. J.B. Hollings, M.D., F.R.C.P., and Dr. W.E. Medd, M.D., F.R.C.P., Consultant Physicians to Kingston Hospital, Kingston-on-Thames, Surrey, England, under whose care the patients in this series were admitted for their permission, advice, and encouragement in the preparation of this paper.

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