Electrophysiological properties of right heart and atrioventricular conducting system in patients with alcoholic cardiomyopathy

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SUMMARY In 20 men with alcoholic cardiomyopathy, right atrial and right ventricular monophasic action potential, relative repolarisation rate of phase 3, and effective refractory period were studied, as well as atrioventricular conduction properties. The duration of right atrial monophasic action potential and effective refractory period recorded in the middle of the right atrium was shorter in comparison with control patients while the right atrial relative repolarisation rate phase 3 was unchanged. In 5 patients with atrial fibrillation, after conversion to sinus rhythm, great variations were found between right atrial monophasic action potential duration at 90 per cent and 50 per cent repolarisation, atrial effective refractory period, and right atrial relative repolarisation rate of phase 3 recorded in 3 different sites of the right atrium. The right ventricular monophasic action potential duration at 90 per cent repolarisation was longer, while right ventricular monophasic action potential duration at 50 per cent, and right ventricular relative repolarisation rate of phase 3 were shorter in alcoholics compared with the control group. The greatest differences between right ventricular monophasic action potential duration and ventricular effective refractory period obtained in 3 different sites of the right ventricle was observed in alcoholics with frequent ventricular premature beats.

The alcoholic patients showed PR prolongation in 35 per cent of cases and intraventricular conduction defects in 50 per cent of cases. In 11 patients His bundle electrograms and atrial pacing showed latent conduction disturbances proximal and distal to the bundle of His.

Chronic ethanol ingestion has been found to have deleterious effects on cardiac rhythm and conduction, quite apart from leading to nutritional deficiency (Regan, 1971). Though arrhythmias and conduction disturbances are frequently seen in alcoholics (Evans, 1959; Sereny, 1971), to the best of our knowledge there are only limited electrophysiological data in these patients.

The purpose of this paper is to evaluate the electrophysiological properties of the right heart and the atrioventricular conducting system in patients with alcoholic cardiomyopathy. The patients were investigated with the aid of monophasic action potential recordings, atrial and ventricular pacing, and His bundle electrograms.

Subjects and methods

Twenty men aged between 24 and 49 years were studied. All had drunk spirits for more than 10 years and showed criteria for alcoholic cardiomyopathy (Burch and Giles, 1976). The patients had no clinical features of nutritional heart disease, were on an adequate diet, and had no coronary heart diseases or other primary or secondary heart muscle disease. The laboratory data were non-contributory. None of the patients had been taking cardioactive drugs for at least 4 days before the investigation, nor was premedication given. After informed consent, monophasic action potential recordings were obtained with a bipolar suction electrode catheter passed percutaneously to the right chambers of the heart (Gavrilescu *et al.*, 1972; Puech *et al.*, 1974). His bundle electrograms were recorded using a tripolar electrode, introduced via the right femoral vein (Scherlag *et al.*, 1969). A bipolar electrode was introduced via the brachial or femoral vein into the high right atrium. The atrium was paced up to the rate at which type I (Wenckebach) atrioventricular block occurred. Atrial, ventricular, and atrioventricular nodal refractory

periods were assessed using the extrastimulus method (Wit *et al.*, 1970) with a basic cycle length of 600 ms and the extrastimulus of twice diastolic threshold delivered after every 8th beat. His bundle intervals were measured in sinus rhythm and during atrial pacing. The refractoriness of the right atrium and ventricle was measured during pacing of the heart by the monophasic action potential electrode catheter (Brorson, 1975).

Fifteen subjects aged between 27 and 45 years with non-specific heart or thoracic disorders in whom electrophysiological studies were performed to rule out organic heart disease acted as a control group. The monophasic action potentials were recorded from the mid-right atrium and from the lateral right ventricular wall in all patients. In 3 subgroups of these subjects the monophasic action potentials and investigation of refractoriness were performed from the high, middle, and low right atrium and from the apex, the lateral wall, and the base of the right ventricle:

Group 1 included 5 patients with alcoholic cardiomyopathy who had atrial fibrillation converted to sinus rhythm, and no more than 6 ventricular extrasystoles a minute.

Group 2 consisted of 3 patients with alcoholic cardiomyopathy in stable sinus rhythm with less than 6 ventricular extrasystoles a minute.

Group 3 included 4 of the controls. The monophasic action potentials were analysed according to Brorson (1975) and Cabasson *et al.* (1975). All calculations were performed from 10 complexes and the measurements were expressed as mean values.

Results

In the 20 patients with alcoholic cardiomyopathy, 7 demonstrated ST-T segment changes previously regarded as specific (Evans, 1959), and 4 exhibited non-specific ST-T abnormalities. Significant changes in the electrophysiological properties of the atrioventricular conduction system were found. Arrhythmias and conduction disturbances were common in these patients (Table 1). In 3 patients with chronic atrial fibrillation DC shock was successful, but in 2 cases it was not followed by sinus rhythm. In the case with complete atrioventricular block associated with atrial fibrillation and syncopal attacks, the conduction was interrupted proximal to the bundle of His; 2 patients with normal PR interval had prolonged His-Purkinje conduction times. In 6 patients the HV prolongation was associated with bundle-branch block or fascicular blocks. During atrial pacing at increasing rates, a first group of 7 patients with prolonged PR interval developed the Wenckebach phenomenon proximal to the bundle of

Table 1 Arrhythmias and conduction disturbances found in 20 patients with alcoholic cardiomyopathy

Chronic atrial fibrillation	6	
Paroxysmal atrial fibrillation	2	
Paroxysmal atrial flutter	1	
Ventricular premature beats > 6/min	10	
First degree atrioventricular block	7	
Intra-atrial + infra-Hisian		1
Atrionodal		3
Atrionodal + infra-Hisian		1
Infra-Hisian		2
Complete heart block with atrial fibrillation	1	
Left bundle-branch block	5	
Left anterior hemiblock	3	
Left posterior hemiblock	1	
Right bundle-branch block	1	

His at relatively slow rates (between 112 and 138 beats a minute), showing altered intra-atrial or atrio-nodal conduction. During spontaneous sinus rhythm the AH interval was within normal limits in 3 cases in this group. A second group of 6 patients with normal PR interval and normal atrionodal conduction time developed a similar Wenckebach phenomenon at a similar pacing rate (between 129 and 135 beats a minute), suggesting latent conduction disturbances in the atrioventricular nodal area. In a third group of 5 patients with normal PR and AH intervals atrial pacing could be increased above 145 beats a minute. The mean effective refractory period of the atrioventricular node in the first group was $387 \text{ ms} \pm 20$, in the second group $353 \text{ ms} \pm 7$, and in the third group $330 \text{ ms} \pm 98$. These values were significantly prolonged compared with a control group (mean $294 \text{ ms} \pm 7$).

The following terms were used in order to evaluate the electrophysiological properties of the heart:

(1) The duration of the right atrial and ventricular monophasic action potential complexes measured at 90 per cent and 50 per cent of repolarisation.

(2) The relative repolarisation rate of phase 3 of the right atrial and right ventricular monophasic action potential complexes. This represents the difference in time between 50 per cent and 90 per cent repolarisation (e) expressed as per cent/second using the formula: relative repolarisation rate phase 3 = $40 \times 1000/e$. This index is valuable in assessing phase 3 (Olsson, 1971; Brorson, 1975).

(3) Effective refractory period of the atrium is the longest S_1 - S_2 interval at which the premature atrial extrastimulus fails to depolarise the atria.

(4) Effective refractory period of the atrio-ventricular node is the longest A_1 - A_2 interval at which the premature extrastimulus response (A_2) fails to conduct to the His bundle (no H_2).

(5) Effective refractory period of the ventricle is the longest S_1 - S_2 interval at which the premature

ventricular extrastimulus fails to depolarise the ventricles.

The schematic illustration of the analyses of a single atrial or ventricular monophasic action potential complex is shown in Fig. 1. Fig. 2 and 3 show examples of the determination of the effective refractory periods in the high, middle, and low right atrium, and in the apex, lateral wall, and base of the right ventricle.

Fig. 1 Schematic illustration of the analyses of a single monophasic action potential complex (MAP). The amplitude of the MAP was measured and expressed in millivolts (A). The duration of the MAP was measured at amplitude of 50 per cent and 90 per cent repolarisation (B and C) and expressed in milliseconds (b and c); 0, 1, 2, 3, and 4 represents the phases of MAP complex. IC, intracavitary electrogram.

Comparison of the monophasic action potential complexes recorded from the mid-right atrium between patients with alcoholic cardiomyopathy and the control group showed that the right atrial monophasic action potential duration at 90 per cent and 50 per cent repolarisation and the atrial effective refractory periods were shorter in the patients with cardiomyopathy. No difference was found in the right atrial relative repolarisation rate of phase 3 between these groups (see Table 2). From the right ventricular studies, right ventricular monophasic action potential duration at 50 per cent repolarisation, right ventricular effective refractory period and right ventricular relative repolarisation rate of phase 3 were found to be shorter in the alcoholic cardiomyopathy group. However, right ventricular monophasic action potential duration at 90 per cent repolarisation was longer than in the control group (see Table 2).

Table 3 lists the values of monophasic action potential recordings and effective refractory period determinations obtained from multiple sites from

alcoholic patients with atrial and ventricular arrhythmias, as well as from patients with stable sinus rhythm and from control subjects. The first group showed the greatest differences in the mean monophasic action potential duration at 90 per cent and 50 per cent repolarisation, right atrial relative repolarisation rate phase 3 per cent/s, and the effective refractory period between the 3 different sites.

Fig. 2 Case 15. The right atrial monophasic action potential (RAMAP) obtained in the high (H), middle (M), and low (L) right atrium and the determination of the effective refractory period. S_1 , driving stimulus; S_2 testing stimulus; CL, cycle length.

Discussion

In animals alcohol ingestion produces metabolic disturbances and structural damage as well as depression of myocardial function (Maines and Aldinger, 1967; Opie, 1969; Burch *et al.*, 1971). Alcohol administration in man has been shown to reduce mechanical efficiency of the heart (Mitchell and Cohen, 1970) and to alter the electrocardiogram (Evans, 1959; Gould *et al.*, 1974). Atrial fibrillation,

Fig. 3 Case 5. The right ventricular monophasic action potential (RVMAP) obtained in the apex (A), lateral wall (L), and base (B) of the right ventricle and the determination of the effective refractory period. S₁, driving stimulus; S₂, testing stimulus; CL, cycle length.

paroxysmal atrial tachycardia, ventricular extrasystoles, sometimes in the presence of increased sinus rate, and atrioventricular or bundle-branch block were frequently observed in alcoholic patients with no clinically detectable myocardial disease as well as in those with manifest cardiomyopathy (Regan, 1971; Burch and Giles, 1975). Dimpled, bifid, peaked, or narrowly inverted T waves were thought to be appropriate electrocardiographic ST-T changes but in some cases non-specific ST-T changes could also be seen (Evans, 1959; Sereny, 1971; Regan, 1971).

Our patients represent a series of men between the third and fifth decades of life, chronically consuming large quantities of alcohol and with clinically detectable heart muscle disease. In 6 patients in the advanced stage of the disease, chronic atrial fibrillation has been observed; 3 patients with early cardiomyopathy had paroxysmal atrial fibrillation or flutter. In case 1 atrial fibrillation was associ-

ated with complete atrioventricular block, and pacemaker implantation was necessary to improve his haemodynamic state. Most of the patients with advanced stage of the disease and chronic atrial fibrillation also had ventricular extrasystoles.

Monophasic action potential recordings together with the determination of the effective refractory periods enhanced an understanding of the underlying electrophysiological mechanism in our patients. The right atrial monophasic action potential duration at 90 per cent and 50 per cent repolarisation and atrial effective refractory period recorded in the mid-right atrium were significantly shorter in alcoholics than in the control group, while no differences could be observed for the right atrial relative repolarisation rate of phase 3 per cent/s in these 2 groups. These changes probably suggest the shortening of phase 1 and 2 of the action potential, while phase 3 remains unchanged (Hoffman *et al.*, 1959), and may account for the frequency of the atrial arrhythmias in these patients (Hoffman and Crane-field, 1964; Crane-field *et al.*, 1973). Monophasic action potential duration in man is shortened under the influence of hyperthyroidism, catecholamines, digitalis, and in patients in whom atrial fibrillation tends to recur after successful conversion (Olsson, 1971; Cotoi *et al.*, 1972a, b). Thyrotoxicosis and digitalis effects were not present in our case but excessive sympathetic stimulation and catecholamine release could not be excluded. Though we have not assessed the tendency to relapse of atrial fibrillation in our patients and have no data on right atrial monophasic action potential duration before chronic atrial arrhythmias developed, it is plausible that there is a correlation between arrhythmias and shortening of right atrial monophasic action potential duration.

In the group of patients in whom atrial fibrillation had been converted to sinus rhythm, the results of multiple recordings of right atrial monophasic action potential and multiple determinations of the atrial effective refractory period showed non-uniformity of the refractoriness as compared to the patients without atrial arrhythmias and the controls. In this group we found the greatest differences between the values obtained at the three sites in the right atrium. The right atrial relative repolarisation rate of phase 3 per cent/s recorded in the mid-right atrium was similar in alcoholics and in controls. However, greater differences could be seen between values obtained at the 3 sites of the right atrium in alcoholics with atrial arrhythmias in comparison with the others. The lack of homogeneity in the rate of repolarisation favours the occurrence of arrhythmias, particularly atrial fibrillation and ventricular extrasystoles (Wit *et al.*,

Table 2 Main clinical and electrophysiological data obtained in 20 patients with alcoholic cardiomyopathy and mean electrophysiological values obtained in 15 control subjects

Case no.	Sex	Age (y)	Cardio-megaly	Surface ECG	PR interval (ms)	His bundle intervals (ms)			Paced Wenckebach AV block (rate/min)
						PA	AH	HV	
1	2	3	4	5	6	7	8	9	10
1	M	49	+++	AF, complete AV block LBBB, PVB	—	—	—	65	—
2	M	45	++	AF, LAH, NST, VES	215	20	150	45	118
3	M	42	++	AF, RBBB, NST	225	20	155	50	125
4	M	47	+++	AF, LBBB, VES	—	—	—	65	—
5	M	40	++	AF, LBBB, VES	250	30	150	70	115
6	M	24	++	LAH	190	30	110	50	155
7	M	32	+	SST	210	35	115	60	130
8	M	38	+	SST	195	25	125	45	135
9	M	41	+++	LBBB, VES	220	20	130	70	138
10	M	35	+	SST, VES	180	30	100	50	145
11	M	28	++	LAH, NST, VES	150	40	70	40	160
12	M	39	++	SST, VES	240	35	155	50	120
13	M	34	++	LPH, PAFI	190	40	90	60	132
14	M	44	+++	LBBB, VES	210	60	90	60	112
15	M	31	++	AF, NST, VES	190	40	90	60	135
16	M	30	+	PAF, SST	190	30	110	50	132
17	M	33	+	Normal	175	25	105	50	158
18	M	29	+	PAF	190	30	100	60	130
19	M	37	+	SST	185	25	120	40	129
20	M	39	+	SST	170	30	95	45	160
Patients									
Mean		37			199	31	114	54	135
SD		2			7	3	17	3	7
SE		2			6	2	6	2	4
No.		20			18	18	18	20	18
Control									
Mean		37			157	19	113	44	185
SD		2			7	3	17	3	7
SE		1			5	1	16	1	6
No.		15			15	15	15	15	15
Significance									
5%		NS			S	S	NS	S	S

+, ++, and +++, minimal, moderate, and gross; AF, atrial fibrillation; LBBB, left bundle-branch block; LAH, left anterior hemiblock; LPH, extrasystoles; SST, specific ST-T changes; NS, not significant; S, significant. Symbols can be deduced from text.

1974). Possible mechanisms include alterations of cell membrane permeability and stimulation of catecholamine secretion from the adrenal medulla by ethanol (Sereny, 1971).

The right ventricular monophasic action potential duration at 90 per cent repolarisation was longer while the right ventricular monophasic action potential duration at 50 per cent repolarisation, and the ventricular effective refractory period, were shorter in alcoholics than in controls. The right ventricular relative repolarisation rates of phase 3 per cent/s were lower than in controls. Such changes suggest that phase 1 and 2 of the action potential in the right ventricle are shortened while phase 3 is prolonged. The modification in phase 3 of the myocardial action potential could be responsible for the T wave abnormality (Sereny, 1971), but the technique of recording the monophasic action potential does not permit analysis of this phenom-

enon. The patients with ventricular arrhythmias showed the greatest differences in the right ventricular monophasic action potential duration at 90 per cent and 50 per cent repolarisation and of the ventricular effective refractory period recorded at multiple sites, though such differences were not found for the right ventricular relative repolarisation rate of phase 3 per cent/s. These local differences in conduction properties and refractoriness in right atrium and ventricle fit the experimental data and may be a factor favouring re-entry (Hoffman and Cranefield, 1960). The changes could be explained by non-uniform histopathological damage of the myocardium and are probably non-specific (Burch and Giles, 1976).

Studies in dogs receiving ethanol, without protein or vitamin deficiencies, have shown significant prolongation of the His-Purkinje conduction time compared with that in controls (Ettinger *et al.*,

Paced cycle length (600 ms)

RAMAP 90% (ms)	RAMAP 50% (ms)	RARRR phase 3 %s	RVMAP 90% (ms)	RVMAP 50% (ms)	RVRRR phase 3 %s	AERP (ms)	AVERP (ms)	VERP (ms)
11	12	13	14	15	16	17	18	19
—	—	—	340	255	471	—	—	310
215	105	364	330	240	444	180	360	300
210	100	364	335	240	421	185	390	335
—	—	—	340	260	500	—	—	320
215	110	381	350	250	400	180	410	330
210	105	381	345	265	444	195	335	300
230	120	364	330	260	571	200	390	315
205	110	381	340	250	444	190	365	330
220	120	400	355	220	296	200	415	345
245	125	333	360	270	444	220	350	320
230	130	400	335	275	667	205	315	305
220	115	364	330	240	444	180	385	290
220	140	500	325	235	444	185	350	285
230	110	333	340	245	421	200	365	325
220	115	381	350	260	444	205	350	340
225	125	400	345	265	500	180	345	310
240	130	361	330	275	727	210	330	305
220	135	471	335	285	800	185	350	320
220	120	400	320	265	727	205	360	285
230	130	400	310	245	615	215	320	280
222	119	388	337	255	515	196	360	312
4	5	15	6	6	35	4	8	5
2	2	10	3	4	25	3	7	4
18	18	18	20	20	20	18	18	20
241	135	387	323	270	753	230	295	324
4	5	15	6	6	35	4	8	5
4	5	11	5	5	25	3	4	3
15	15	15	15	15	15	15	15	15
S	S	NS	S	S	S	S	S	S

left posterior hemiblock; NST, non specific ST-T changes; PAF, paroxysmal atrial fibrillation; PAFI, paroxysmal atrial flutter; VES, ventricular

1970). Conduction disturbances are common electrocardiographic findings in about one-third of alcoholics and include varying degrees of atrioventricular block and left or right bundle-branch block (Regan, 1971). In cardiomyopathy the conduction defects are believed to be diffuse and the lesions fibrotic (Stapleton *et al.*, 1970). In a series of 70 patients, 90 per cent of whom had alcoholic cardiomyopathy, Bashour *et al.* (1976) found first degree atrioventricular block in 40 per cent, left bundle-branch block in 6 per cent, left posterior fascicular block in 2 per cent, and 'bi-fascicular block' in 13 per cent. His bundle electrograms were performed in only a few of these cases. The incidence of the PR prolongation in our patients was 35 per cent, while intraventricular conduction defects were found in 50 per cent. On the surface electrocardiogram 83 per cent of patients with moderate and gross cardiomegaly showed

different types of atrioventricular block, while only 25 per cent of patients with minimal cardiomegaly had such conduction disturbances. During electrophysiological studies each alcoholic patient had longer atrioventricular effective refractory periods than the controls, though some patients showed normal atrioventricular nodal conduction times on His bundle electrograms.

Our data show that in alcoholic patients conduction disturbances are frequent and localised at multiple sites in the atrioventricular conduction system. Electrophysiological studies sometimes showed latent conduction disturbances which were not apparent on standard electrocardiographic tracings.

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Table 3 MAP and ERP durations obtained in 3 sites of right atrium and right ventricle and maximum differences (%) between mean values in same chamber in 8 alcoholic patients and in 4 normal subjects

Case no.	Paced cycle length (600 ms)											
	RAMAP 90% (ms)			RAMAP 50% (ms)			RARRR phase 3%/s			AERP (ms)		
	H	M	L	H	M	L	H	M	L	H	M	L
1	2	3	4	5	6	7	8	9	10	11	12	13
Group 1 patients												
2	180	215	255	90	105	180	444	364	533	170	180	230
3	180	210	230	85	100	150	471	364	571	165	185	210
5	190	215	240	100	110	165	444	381	533	180	180	235
15	210	220	165	180	115	135	1333	381	1333	200	205	150
18	205	220	250	110	135	175	421	471	533	190	185	230
Mean	193	216	228	113	113	161	623	392	701	181	187	211
MD (%)	15			30			44			14		
Group 2 patients												
6	200	210	235	130	105	125	571	381	364	200	195	215
7	215	230	230	140	120	175	533	364	727	220	200	235
8	210	205	225	140	110	145	571	381	500	215	190	200
Mean	209	215	230	137	112	148	559	375	530	212	195	217
MD (%)	9			25			33			10		
Group 3 controls												
	220	240	250	130	130	155	444	364	421	215	235	240
	240	260	270	125	145	165	348	348	381	230	250	250
	230	245	255	145	135	170	471	364	471	220	235	240
	235	250	250	170	160	150	615	444	400	225	235	240
Mean	231	249	256	142	142	160	383	380	418	222	239	242
MD (%)	10			11			9			8		

H, M, and L are high, middle, and low right atrium; A, L, and B are apex, lateral wall, and base of the right ventricle; MD is maximum differences between values obtained in the 3 sites. The other symbols are similar to Table 2.

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RVMAP 90% (ms)			RVMAP 50% (ms)			RVRRR phase 3 %/s			VERP (ms)		
A	L	B	A	L	B	A	L	B	A	L	B
14	15	16	17	18	19	20	21	22	23	24	25
350	330	300	250	240	210	400	444	444	320	300	280
360	335	330	270	240	250	444	421	500	350	335	310
365	350	310	255	250	220	364	400	444	345	330	300
355	350	320	250	260	230	381	444	444	350	340	320
330	335	310	260	285	250	444	800	667	315	320	300
352	340	314	357	255	232	407	502	500	336	325	302
	10			10			19			10	
320	345	320	250	265	250	571	444	571	300	300	310
330	330	325	260	260	245	571	571	571	305	315	310
325	340	320	230	250	250	471	444	571	315	330	320
325	338	322	247	258	248	538	487	571	307	315	313
	5			5			15			3	
340	320	330	260	260	240	500	667	444	330	340	315
355	360	350	290	305	280	615	727	571	350	345	340
330	315	325	270	265	260	500	800	615	320	325	210
300	295	300	240	245	250	666	800	800	290	305	300
331	322	326	265	268	257	571	748	608	323	329	316
	3			4			24			4	

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