Diagnostic Electrocardiographic Patterns in Bantu Myocardiopathy and Constrictive Pericarditis*

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SUMMARY

The electrocardiogram was analysed in 28 adult Bantu patients with myocardiopathy and 33 with constrictive pericarditis. The over-all pattern was quite distinctive in the two groups. Patients with CP usually had sinus rhythm, notched P waves, low voltage QRS complexes in the standard and precordial leads, a normal QRS duration, no intraventricular conduction defects and a uniform and characteristic pattern of ST-T wave change in most cases. In contrast, the ECG in MCO shows left ventricular hypertrophy, varying degrees of intraventricular conduction disturbance and patterns simulating myocardial infarction.

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Congestive myocardiopathy (MCO) and constrictive pericarditis (CP) are often responsible for cardiac failure in the South African Bantu. Both conditions cause severe heart failure and a clinical distinction based on the physical signs can be difficult, although more complex investigations are diagnostic.[1-3]

It has been our impression that the scalar electrocardiogram (ECG) is quite different in these two diseases. This study was undertaken to examine the ECG in patients with MCO and CP, to define the pattern in each condition and to determine the value of this investigation in differential diagnosis.

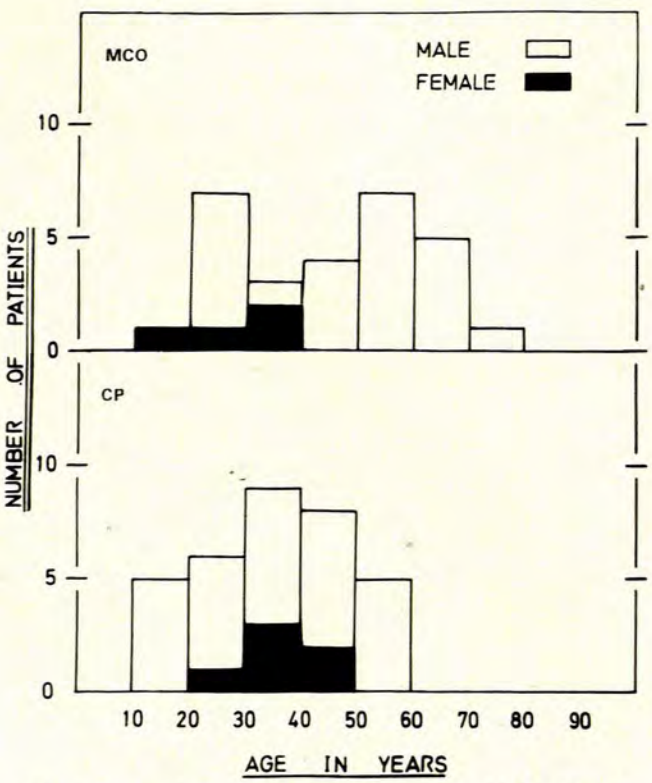

Fig. 1. Age and sex distribution of patients with MCO and CP.

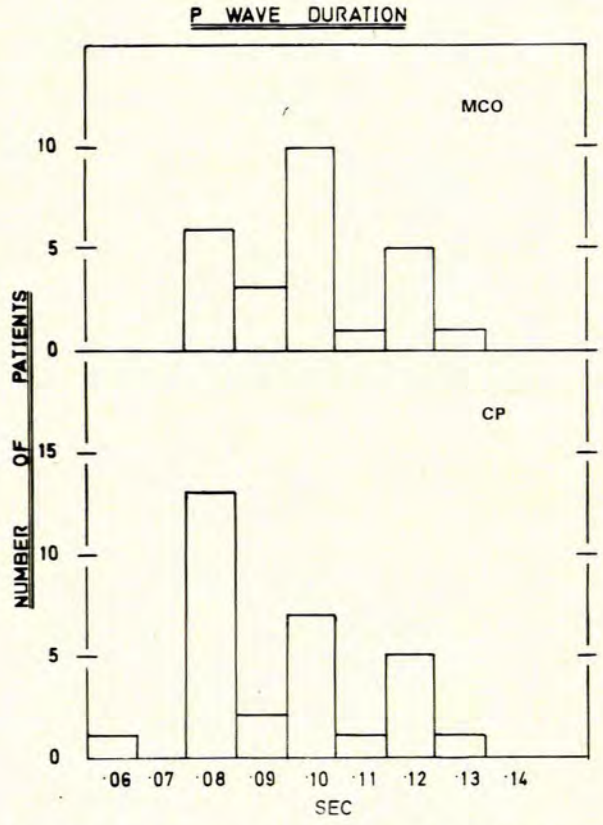

Fig. 2. P wave duration. There was no significant difference between the two groups. CP = constrictive pericarditis. MCO = myocardiopathy.

*Date received: 25 May 1971.

THE PATIENTS

We selected for study 28 consecutive adult Bantu patients with congestive myocardiopathy who underwent cardiac catheterization. These patients had the classical features of Bantu MCO.[4-13] The patients had right and/or left heart failure, an enlarged heart affecting the right and/or left ventricles and a loud apical third heart sound. Cardiac catheterization demonstrated a hypokinetic left ventricle with a low ejection fraction in all patients and the majority had an elevated LVEDP, diminished LV dp/dt, pulmonary venous hypertension, moderate pulmonary arterial hypertension and a low stroke index and cardiac index. Twenty patients with severe disease also had functional mitral incompetence; 4 patients had clinical evidence of pul-

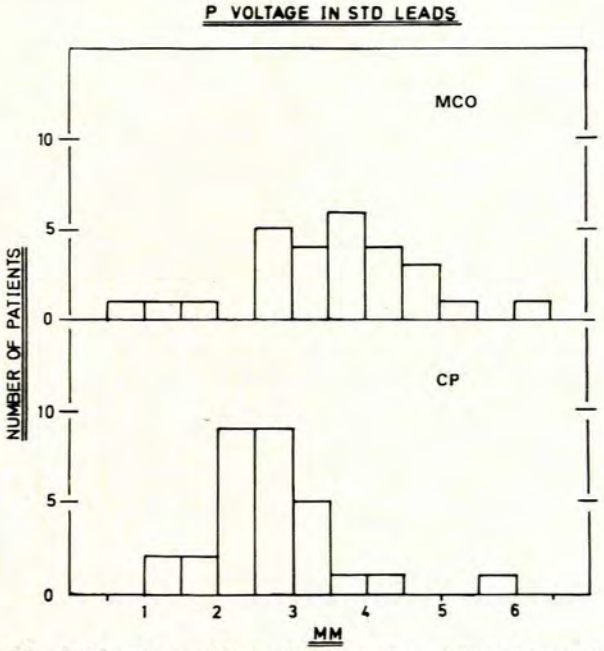

Fig. 3. P wave amplitude. The P wave voltage in leads I, II and III were added together. Patients with CP tended to have smaller complexes.

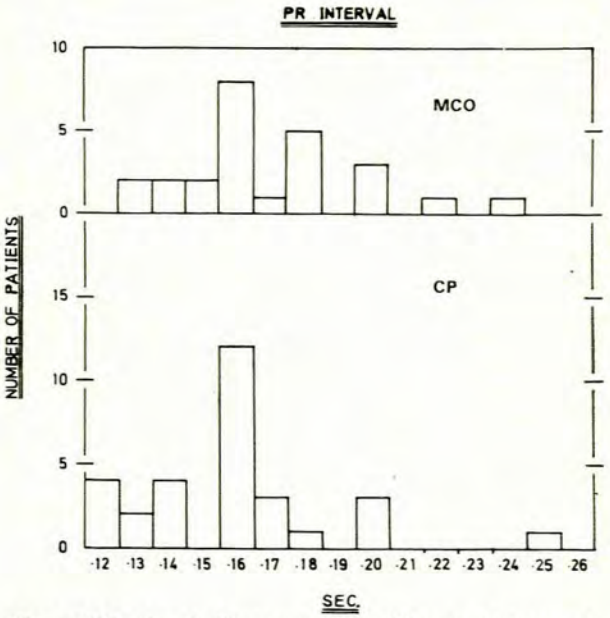

Fig. 5. PR interval. There was little difference between the two groups. Isolated prolongation of the PR interval may have been a consequence of digitalis therapy.

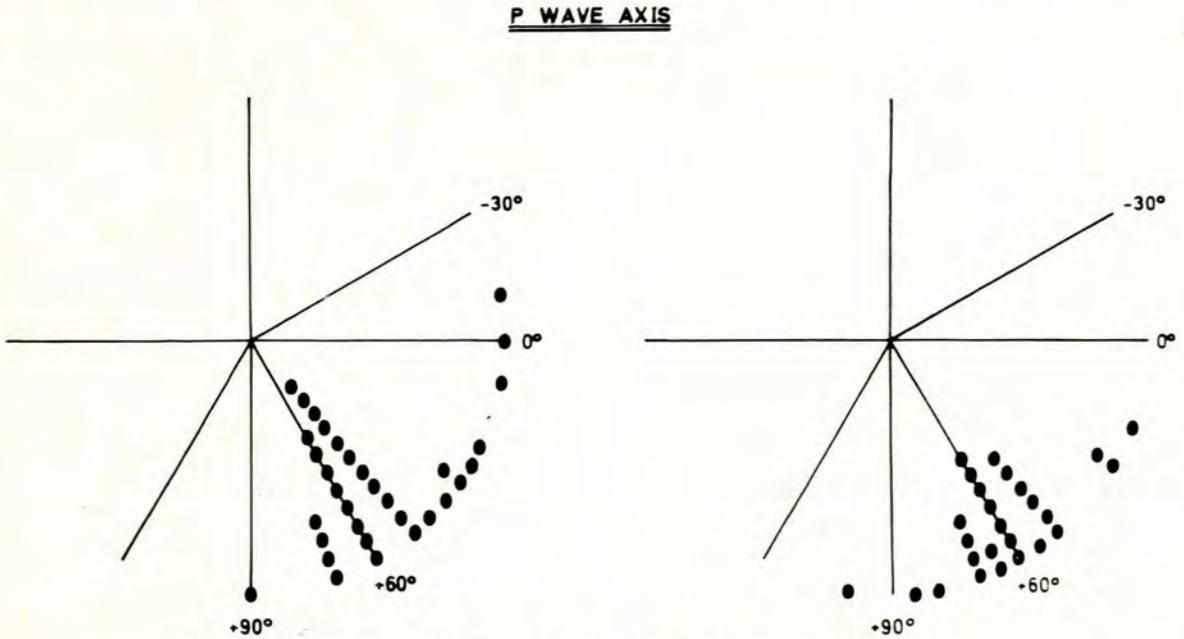

Fig. 4. Frontal P wave axis. There was little difference between the two groups.

monary thrombo-embolism; 1 patient had the haemodynamic features of constrictive MCO.[14,15] Coronary arteriography was performed in 12 patients and their major coronary arteries were shown to be normal. In all the remaining patients, the proximal coronary arteries appeared normal on aortography.

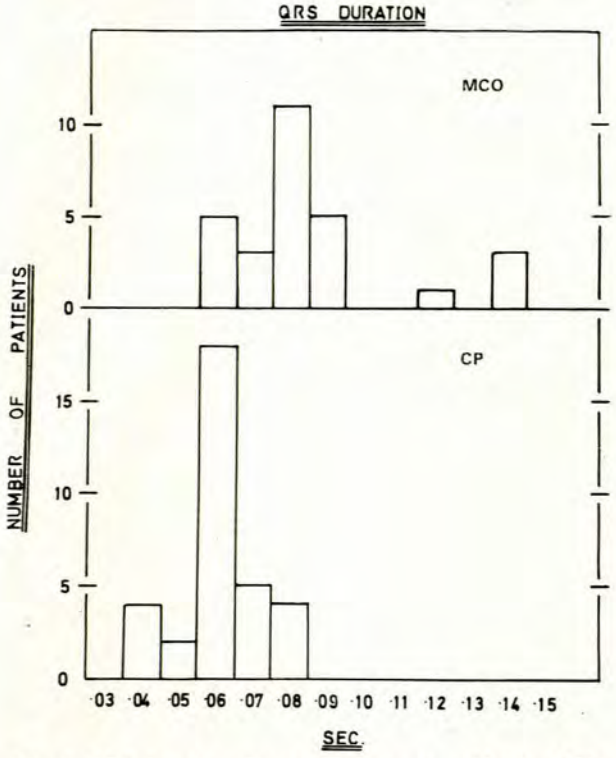

Fig. 6. QRS duration. The complexes were wider in MCO.

Thirty-three consecutive adult patients with constrictive pericarditis were studied. Patients with pericardial effusion and tamponade were excluded. In 32 patients the aetiology was probably tuberculous; 1 patient had amoebic pericarditis. The clinical diagnosis was confirmed by cardiac catheterization and cine-angiography. In 25 patients, the constricting pericardial material was subsequently removed at surgery and in another, the diagnosis was confirmed at autopsy.

METHODS

A standard 12-lead electrocardiograph was recorded on each patient. Only pre-operative tracings were analysed in patients with CP. Each ECG was analysed in detail and the following parameters were examined: heart rate, rhythm, mean frontal plane P wave axis, P wave voltage and duration, mean frontal QRS axis, QRS duration and QRS amplitude in the standard and selected precordial leads. The ST segment and T waves were also studied, although all patients were receiving digitalis.

Standard criteria were used for the assessment of atrial and ventricular enlargement[16,17] and ventricular conduction disturbances.[18-20]

We also assessed the total voltage of the P and QRS complexes—the sum of P wave deflections in leads I, II and III; the sum of the total QRS amplitude in leads I, II and III and the sum of SV1 and RV5. The S wave in lead II was expressed as a fraction of the total R and S amplitude in order to determine the importance of the terminal QRS vector.

RESULTS

The age and sex distribution of the patients in the two groups is shown in Fig. 1. Children were excluded from

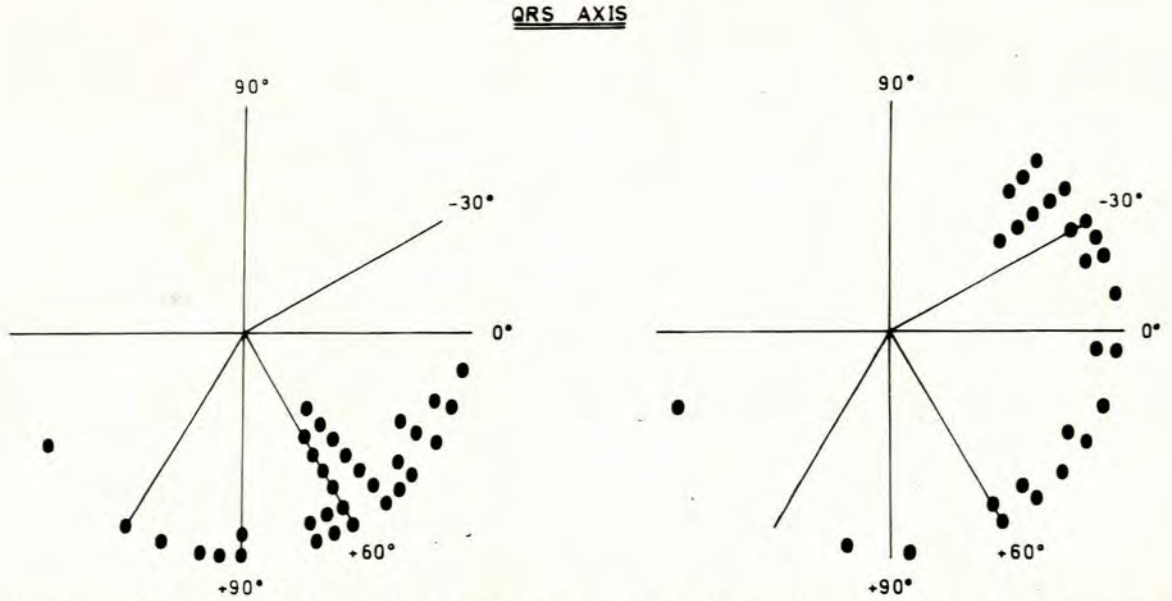

Fig. 7. Mean frontal plane QRS axis. Half the patients with MCO had normal or pathological left axis deviation.

the study. The patients with MCO were slightly older than patients with CP. Most of the patients in each group were males.

Disturbances of rhythm are shown in Table I. Arrhythmias were uncommon and some may have been related to enthusiastic digitalis therapy. Premature beats were more common in the MCO group and when present were numerous, with occasional bigeminy and concealed bigeminy. Atrial fibrillation too was uncommon, in contrast to the high incidence reported in the literature.[3,21-26] This difference may be due to the fact that we studied a younger population who had severe disease with a shorter natural history.

TABLE I. DISTURBANCES OF RHYTHM

	MCO	CP
Sinus rhythm	27 (96%)	30 (91%)
Atrial flutter	0	1 (3%)
Atrial fibrillation	1 (4%)	2 (6%)
Premature beats — PVCs	8 (29%)	2 (6%)
— PACs	2 (7%)	0
Other	1 (PAT on admission)	0

Abbreviations: PVC = premature ventricular contraction; PAC = premature atrial contraction; PAT = paroxysmal atrial tachycardia; LBBB = left bundle branch block; RBBB = right bundle branch block; LAHB = left anterior hemiblock; LPHB = left posterior hemiblock; LAD = left axis deviation; RAD = right axis deviation and LVH = left ventricular hypertrophy.

Abnormalities of the P wave configuration are shown in Table II and in Figs. 2, 3 and 4. Patients with CP tended to have a lower amplitude P wave complex in the standard leads. The presence of any cleft in the P wave was noted (Table III), a bifid P wave often being regarded as a sign of left atrial enlargement or intra-atrial conduction disturbance. P wave notching was far more common in CP and

TABLE II. ATRIAL ENLARGEMENT

	MCO	CP
LA enlargement:		
Definite	13 (48%)	12 (40%)
Probable	10 (37%)	2 (7%)
RA enlargement:		
Definite	2 (7%)	0
Probable	2 (7%)	0

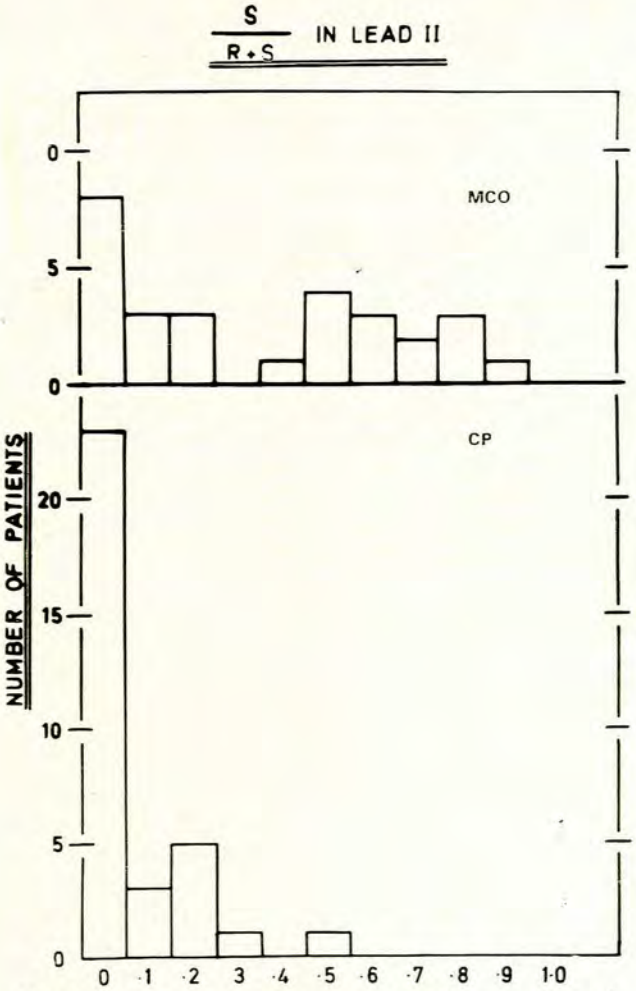

Fig. 8. Ratio of the S wave compared to the total R and S deflection in lead II. Patients with MCO often had a terminal S wave in lead II and there is a significant difference between the two groups.

Fig. 9. S wave in lead III. This was common in MCO and a manifestation of the leftward direction of the terminal vector.

TABLE III. CLEFT IN THE P WAVE

	MCO	CP
P bifid	7 (26%)	18 (60%)

was present in 60% of cases, but only in 26% of MCO patients. Three cases of MCO had a P wave axis of +80° or more (Fig. 4)—in 2 of these pulmonary thromboembolism was important.

The PR interval was not unusual in either group and isolated prolongation was probably a consequence of digitalis therapy (Fig. 5).

The QRS morphology and its aberrations are shown in Table IV and Figs. 6 - 14. The mean QRS duration was prolonged in patients with MCO and was greater than 0·06 seconds in 23 patients (87%); this was seen in only 27% of the CP patients (Fig. 6). There was a significant difference in QRS axis (Fig. 7). Half of the MCO group had normal (0° to −30°) or pathological (−30° to −90°) left axis deviation; this was not seen in the CP patients. Five patients with CP had right axis deviation—the cause of this abnormality was not obvious. In the MCO group, 2 patients had right axis deviation; one with complete right bundle branch block, the other with left posterior hemiblock. The ratio of the S wave compared to the total R and S amplitude in lead II is a measure of the leftward deviation of the terminal vector and a significant difference was observed in the two groups (Fig. 8). A terminal vector abnormality with a dominant S wave in lead II was seen only in MCO. Similarly, an important S wave in lead III occurred mainly in MCO (Fig. 9).

TABLE IV. CONDUCTION DEFECTS

	MCO	CP
Specific:		
AV block (1st °)	1 (4%)	1 (3%)
LBBB	2 (7%)	0
RBBB	4 (1·%)	0
LAHB	17 (61%)	0
LPHB	2 (7%)	1 (3%)
Non-specific:		
QRS notching	5 (18%)	

Patients with constrictive pericarditis had normal or low QRS voltages in the standard and precordial leads; MCO patients tended to have larger voltages in these leads. When the R and S amplitudes were added together in all 3 standard leads, 23 patients (85%) with CP had a total voltage of less than 15 mm in the standard leads; this was present in only 7 patients with MCO (Fig. 10). The depth of the S

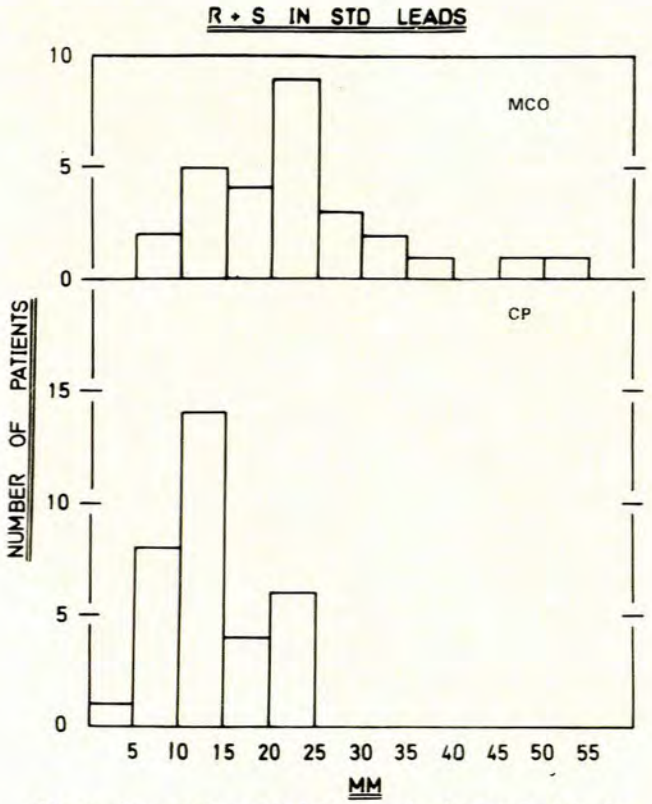

Fig. 10. Sum of R and S deflections in the standard leads. Patients with MCO tended to have larger voltages in these leads, although a low voltage occurred in a few.

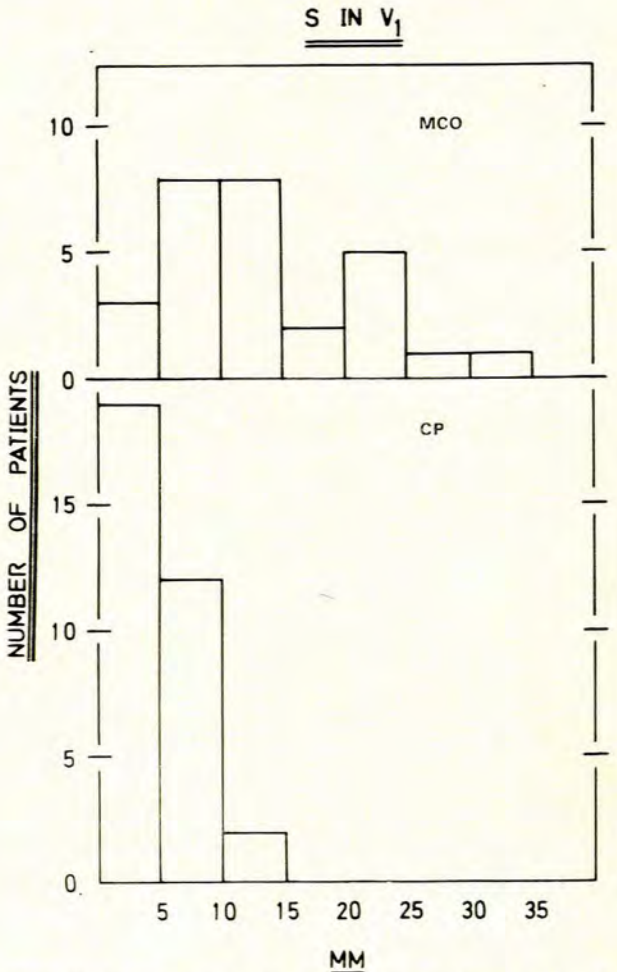

Fig. 11. S wave amplitude in V1. This was low in CP and normal to increased in MCO. (Normal = 8·6 ± 4·3 mm.)

wave in leads V1 and V2 was quite different in the two groups. Patients with CP had deflections of low amplitude, but the values were normal or increased in patients with MCO, particularly in V2 (Figs. 11 and 12). Of the MCO patients studied, 92% showed a deep S wave in V2, and this included the 7 patients with low voltage complexes in the precordial leads (Fig. 13): only 1 patient had an S wave less than the normal mean of 12·7 mm.[27] This represented a narrow posteriorly orientated spatial QRS vector loop, which was not a feature of CP. The R wave amplitude in V5 tended to be larger in patients with MCO, although only 3 fulfilled the voltage criteria for left ventricular hypertrophy (Fig. 14). Fig. 15 shows the sum of SV1 and RV5 and shows that the amplitude is low in CP patients, but normal or increased in MCO patients.

One patient with MCO showed a classical infarct pattern (Fig. 16). Angiography showed akinesis of the left ventricular apex but his coronary arteries were normal. We presumed that he had had coronary embolism with subsequent lysis. Four other patients showed an ECG simulating myocardial infarction (3 anteroseptal and 1 diaphragmatic). In 3 of these cine-angiography was available to show normal major coronary arteries. The 3 with 'anteroseptal infarction' had QS patterns in V1 - V3 which probably represented LV hypertrophy with a narrow posteriorly orientated spatial vector loop.[27,28]

The significant intraventricular conduction disturbances are shown in Table IV. These were common in MCO and rare in CP.

The ST segment and T-wave was abnormal in all cases apart from one patient with MCO. The interpretation of this part of the ECG is complicated by the presence of intraventricular conduction disturbances, ventricular hypertrophy, electrolyte imbalance and the use of digitalis. In 49% of the patients with CP, however, the pattern of the ST segment was uniform and diagnostic (Fig. 17).

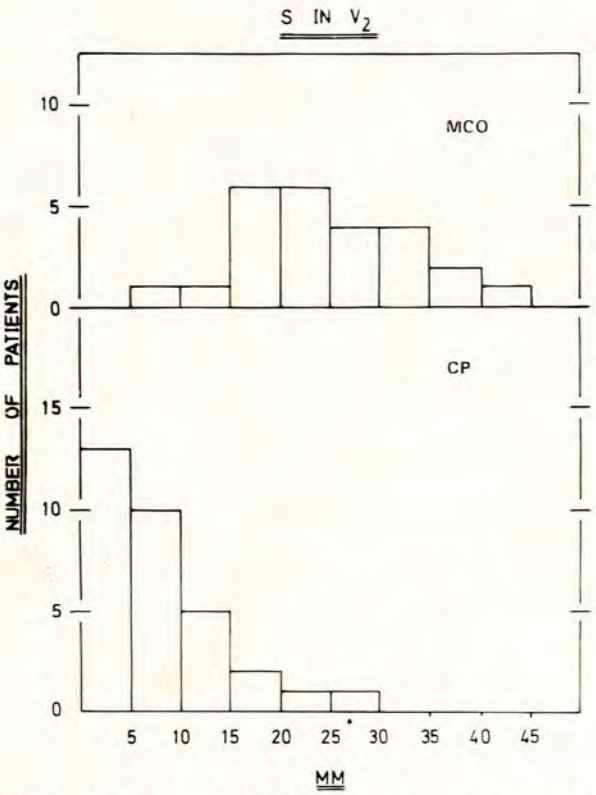

Fig. 12. The S wave in V2. A deep S wave was common in MCO but not in CP and was often the most useful diagnostic feature. (Normal = 12·7 ± 5·3 mm.)

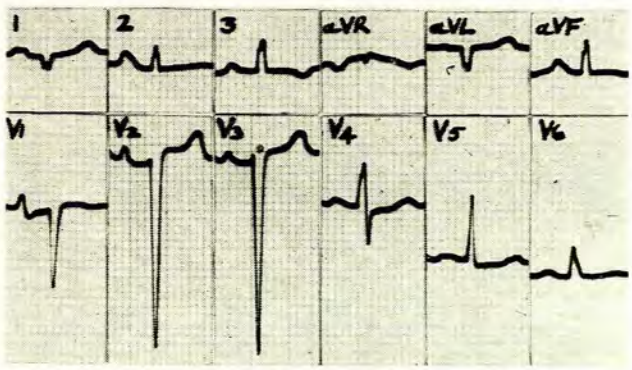

Fig. 13. ECG in MCO shows low voltage in standard leads, but deep S waves in V2 and V3 indicate a long narrow posterior spatial vector loop.

Fig. 14. R wave amplitude in V5. (Normal mean = 12·1 ± 4·4 mm with a range of 4·0 to 26·0 mm.) Patients with CP had low voltage complexes. In MCO this was increased.

DISCUSSION

Numerous authors have commented on the electrocardiographic findings in myocardiopathy and/or constrictive pericarditis (Table V).[3,21-27,29,30] Although certain ECG findings have been considered to be characteristic, the graphs in general have been regarded as non-specific and have not distinguished between constrictive pericarditis and myocardiopathy.

The electrocardiogram in the two conditions is determined by the pathological nature of the disease, the haemodynamic aberrations which are induced, and the conduction of the cardiac potentials to the body surface.

In constrictive pericarditis, the myocardium is normal but compressed, the epicardium is infiltrated by fibrous tissue and the heart is surrounded by an insulating medium of constrictive material. This is reflected in the ECG, which shows that patients with CP usually have sinus rhythm, notched P waves, low voltage QRS complexes in the standard and precordial leads, a normal QRS duration, no intraventricular conduction defects and a uniform and characteristic pattern of ST-T wave change in most cases.

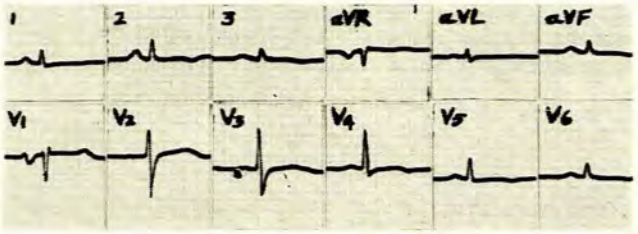

Fig. 17. Classical ECG of CP.

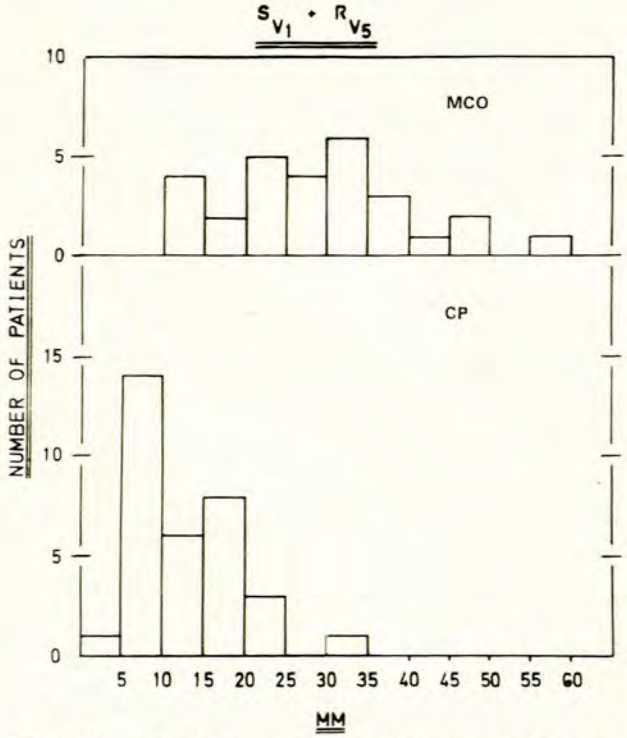

Fig. 15. Sum of the S wave in V1 and R wave in V5. This clearly distinguishes the low voltage of CP.

In contrast, myocardiopathy is a generalized disorder of heart muscle with inappropriate hypertrophy, myocardial fibrosis and intercellular oedema.[32] The ECG features are therefore related to the ventricular hypertrophy and conduction disturbances. Functional mitral incompetence increases the burden on the left atrium while thrombosis on the endocardial surface of the right heart or in the deep venous plexus of the legs may be responsible for pulmonary embolism, pulmonary hypertension and disproportionate right ventricular hypertrophy. Mural thrombus on the surface of the left ventricle may also be dislodged, cause coronary embolism and produce regional myocardial fibrosis.

The spectrum of abnormal electrocardiographic patterns in the patients with myocardiopathy can be summarized. One patient had a *normal* ECG at the time of study; we attributed his previous cardiac failure to acute myocarditis or a myocardiopathy which was resolving. Four patients showed only left *ventricular hypertrophy*, while another 4 showed *left ventricular hypertrophy* with intraventricular conduction disturbances (notched QRS complexes) (Fig. 18). Eight patients showed *pathological left axis deviation*, a manifestation of left anterior hemiblock (Fig. 19); 2 patients showed *left posterior hemiblock* while another 2 had *complete left bundle branch block*. Four patients had *right bundle branch block* and one had associated left anterior hemiblock. Two of these patients had definite evidence of pulmonary emboli while this was an important component of the illness in a third. One patient had a

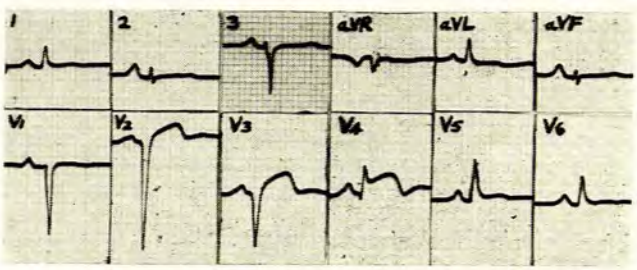

Fig. 16. ECG in MCO showing classical anteroseptal infarction.

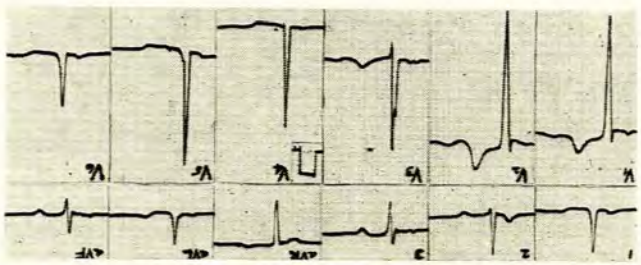

Fig. 18. ECG in MCO shows left ventricular hypertrophy with notched QRS.

	Mycardiopathy					Constrictive pericarditis		Comparison of constrictive pericarditis and myocardiopathy						
Author	**Davies and Evans[29]**	**Schamroth and Blumsohn[30]**	**Marriott[24]**	**Hamby and Raia[27]**	**Stapleton et al.[26]**	**Dalton et al.[21]**	**Hull[22]**	**Wood[3]**		**Hollister and Goodwin[23]**			**Shabetai et al.[25]**	
Date	**1960**	**1961**	**1964**	**1968**	**1970**	**1956**	**1961**	**1961**		**1963**			**1965**	
Subject	**Primary MCO**	**MCO**	**MCO**	**PMD**	**Chronic MCO**	**CP**	**CP**	**CP**	**MCO**	**Congestive MCO**	**Constrictive MCO**	**CP**	**Comparison of pericardial and myocardial disease**	
No. of patients	25	48	Review	60	36	78	Review	40	Review	25	9	10		
Arrhythmias			Frequent	65%		44%		45%						
PVCs		Common		52%	More than	(atrial)		(atrial)						
Atrial flutter or fibrillation		Common		10%	50% 21%	35%	Common	70%	33%	28%	11%	50%	33%	
Abnormal P waves				52%		72%	Low amplitude						Broad P waves	
LA +					Common					24%	33%			
RA +														
Bifid P wave						41%		P mitrale common						
Wide P wave						69%								
Conduction defects		Common	Frequent	Common	Common					Common	Common	Rare		Common
AV block		Common		20%	50% (1st°), 3% (complete)						14% (1st°)			
LBBB				17%	44%			0	Bundle branch block in 25%	24% (incomplete)				
RBBB				10%	8%			0						
QRS axis										Normal	Normal			
LAD normal	64%	23% 69%		27% 63.4%	25%	0								
LAD pathological		42%		42%										
RAD						50%								
Ventricular hypertrophy				LVH in 33%, probable LVH in 13%	LVH occurs			0	LV dominance in 31%	Normal balance	Normal balance	Lone RVH in 50%, normal balance in 50%		LVH occurs
Infarct pattern			Q waves may occur	Q wave in 8%										Q waves occur
Voltages			High or low	Low in standard and limb leads, normal in chest leads		Low in 55%	Low	Low		Normal to low in all leads, SV1 + RV5 = 22.5		Low, SV1 + RV5 = 14.5	Low commonly	
ST segment and T wave						Abnormal in 100%	Shallow T wave inversion	Flat to inverted T waves in 90%.	'Pericardial T wave' in 29%				T wave changes common	
Conclusion/ comment	LAD common in MCO.	LAD distinguishes MCO from pericardial effusion. 48% of 76 consecutive cases of LAD were due to MCO.	ECG 'non-specific' in all cases.	ECG non-specific but combination of abnormal P waves + LAD suggests PMD.	MCO may simulate myocardial infarction. Conduction disturbance common.	All ECGs abnormal but changes varied.	'The ECG is almost never diagnostic.'	90% of CP had classic ECG.		Findings essentially 'non-specific' and it is difficult to distinguish constrictive MCO from CP, except that conduction defects are more common in the former and T wave changes in the latter.			ECG 'fails to distinguish' MCO and CP in many cases.	

TABLE VI. ELECTROCARDIOGRAPHIC DISTINCTION BETWEEN MCO AND CP

		MCO	CP
Normal ECG		1	
Rhythm	Sinus	27 (96%)	30 (91%)
P wave	Voltage	Normal	Low
	Bifid	7 (26%)	18 (60%)
	LA enlarged	23 (85%)	14 (47%)
QRS axis	LAD (0° to −30°)	6 } (50%)	0
	LAD (−30° to −90°)	8 }	0
	RAD	2 (7%)	5 (15%)
Conduction defects	1st° AV block	1	1
	LAHB	17	0
	LPHB	2	1
	LBBB	2	0
	RBBB	4	0
QRS voltage	Standard leads	Normal to low	Low
	Chest leads	Normal to high	Low
	S in V1, V2	Deep	Small
	R in V5	Normal to increased	Low
LVH		4	
LVH + QRS notching		4	
Infarct pattern		1	
Infarct-like pattern		4	
ST segment and T wave		Non-specific	Characteristics in 49%

classical *infarct pattern* with apical akinesis on angiography but normal coronary arteries and 4 others had 'infarct-like patterns' which were attributed to left ventricular hypertrophy with a long narrow posteriorly directed vector.

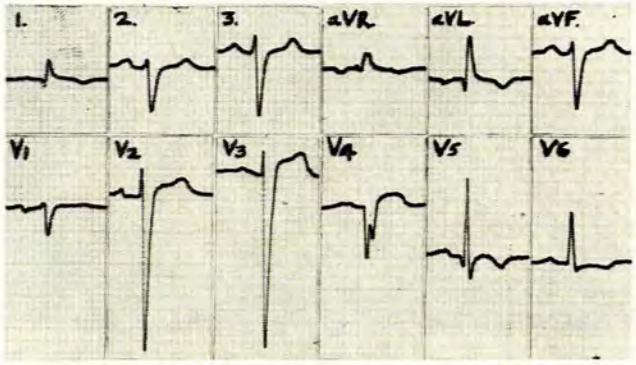

Fig. 19. MCO—left axis deviation due to left anterior hemiblock. There is loss of R wave in V4 simulating localized anterior infarction.

The electrocardiogram in MCO is therefore quite different from the pattern seen in patients with CP (Table VI). The ECG shows left ventricular hypertrophy, varying degrees of intraventricular conduction disturbance and patterns which simulate myocardial infarction. Of great importance is the damage to parts of the left bundle with the resulting terminal vector abnormality of left anterior hemiblock producing left axis deviation and/or an S_2 S_3 pattern in the frontal plane. This pattern is never seen in patients with constrictive pericarditis.

It is important to emphasize that the individual signs of distinction are non-specific, but the over-all ECG pattern is quite different in the two groups and is of great diagnostic value.

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