NORMAL ELECTROCARDIOGRAM AFTER MYOCARDIAL INFARCTION

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INTRODUCTION

THE PRESENCE of pathological Q waves on the surface electrocardiogram is generally recognised to indicate a transmural infarction. With healing, the Q wave often persists because a dead core of electrically inert scar tissue remains. Horan *et al.* (1971) demonstrated a correlation between surface electrocardiographic evidence of scarring with corresponding zones in the left ventricle at autopsy. Other workers have shown pathological Q waves associated with severe degrees of left ventricular asynergy in up to 78% of cases (Bodenheimer *et al.* 1975, Banka *et al.*, 1974). It is also recognised that in some instances there is complete restitution of the electrocardiogram after acute myocardial infarction. This observation is generally stated in various textbooks of cardiology, but a search in the literature provided scanty discussion on this aspect of myocardial infarction. Early reports by various authors found incidences of 6-14% in studies varying from one to six years (Table 1).

This report is of 4 cases of acute myocardial infarction which showed complete electrocardiographic restitution on follow up. They comprise 4% of 96 patients with definite myocardial infarction admitted to the University Kebangsaan Division of the Coronary Care Unit, General Hospital, Kuala Lumpur, followed up from 3 to 12 months.

Table 1

Incidence of normal electrocardiograms following myocardial infarction

	AUTHOR	NO. OF PATIENTS	NORMAL ECG		PERIOD OF STUDY
1.	Kaplan & Berkson (1964)	211	2	1	8 weeks
			12	6	3½ years
2.	Master *et al.* (1942)	202	28	14	4 years
3.	Mills *et al.* (1949)	100	9	9	6 years
4.	Gittler *et al.* (1956)	51	1	8	1 year

CASE 1

K.A., a 29 years old Malay meter-reader was admitted to the Coronary Care Unit on 14-12-1977, 4 hours after he developed severe chest pains while driving home from work. Electrocardiogram on admission showed acute inferior infarction. Serum aspartate transaminase on consecutive days were raised with a peak valve of 198 I.U./L. Clinical examination also revealed symptoms and signs of thyrotoxicosis. This was subsequently confirmed by thyroid functions tests — Serum T_4, 21μg/dl., I_{131} uptake at 4 hours 61%, uptake at 24 hours 76%.

The post infarction clinical course was uneventful. Propranolol (Inderal) was started on the third day and adequate blockade was achieved with 240 mg/day. Neomercazole was given when diagnosis of thyrotoxicosis was confirmed. It was discontinued when an extensive rash developed, and methylthiouracil 100mg 8 hourly substituted. He was discharged 7 weeks after admission.

On the fifth follow up visit (14-6-1978), he had resumed normal work and was clinically enthyroid. Electrocardiogram by this visit had returned to normal.

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CASE 2

A.G., a 48 years old Malay electrician had been on regular treatment for hypertension for 2 years. Two weeks prior to admission he developed angina pectoris. On 18-1-1978 he experienced severe retrosternal pains lasting 20 minutes associated with sweating. electrocardiogram on admission showed a fresh anteroseptal infarction (Fig. 1). Serial serum aspartate transaminase values were raised with a peak of 90 I.U./L.

Clinical course in the coronary care and rehabilitation ward was uneventful. Propranolol was commenced on the third day and maintained on 160 mgm/day. Isosorbide dinitrate 10mg 4 hourly was added later. He was discharged seventeen days after admission.

The last review on 19-7-1978, six months after infarction, he remained well and was back to full time work. Electrocardiogram on this visit showed a complete reversal to normal (Fig. 2)

CASE 3

E.S., a 42 years old Indian clerk, experienced severe retrosternal pains radiating to the left shoulder while resting after dinner. Electrocardiogram on admission (1-6-1978) showed a fresh inferior infarction. Serial serum aspartate transaminase on three consecutive days were 38, 126 and 86 I.U./L. respectively. His post infarction clinical course was unremarkable. He was discharged on the 15th day on propranolol 160 mgm./day.

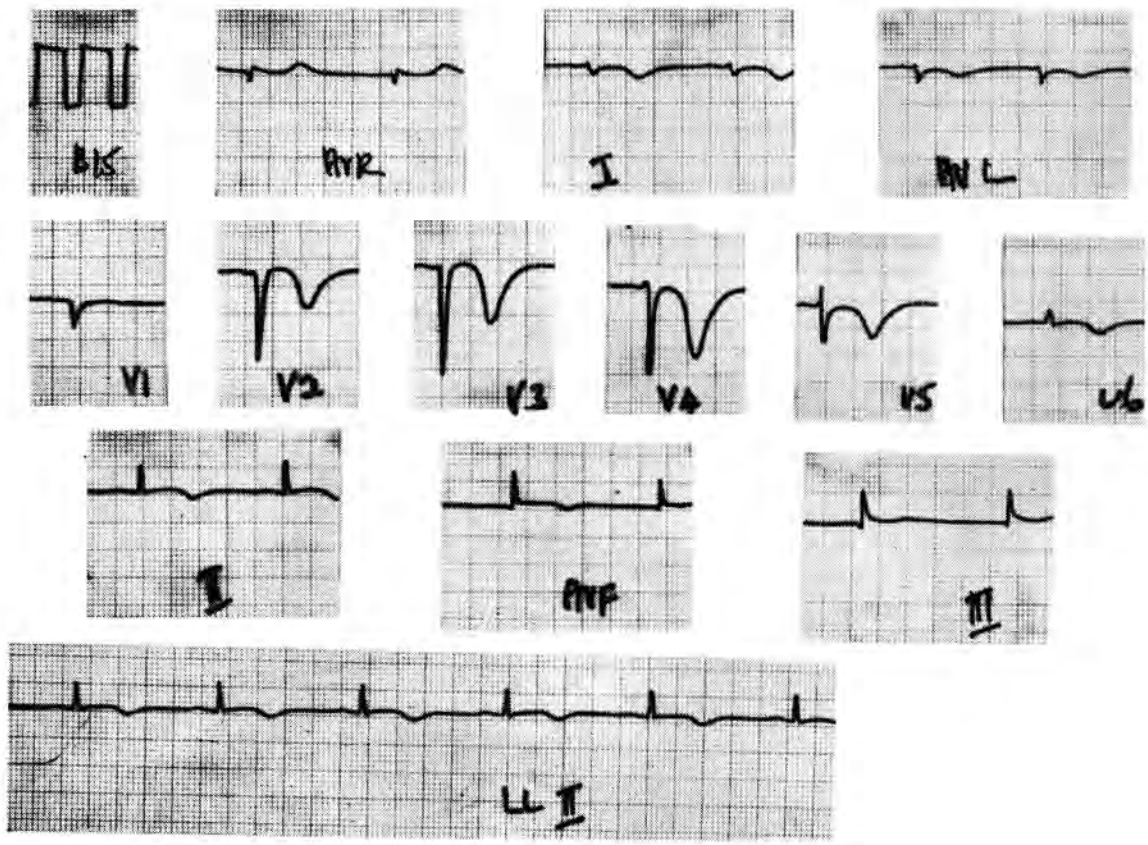

Fig. 1 Electrocardiogram on admission (18-1-78) showing acute antero-septal infarction

On the second follow up visit on 25-7-1978, 2 months post infarction, he remained well and electrocardiogram on this visit was normal.

CASE 4

K.S., a 35 years old Indian security guard, developed severe chest pains while taking his evening bath on 25-1-1978. Admission electrocardiogram showed a fresh antero-septal infarction Confirmatory serum aspartate transaminase values were 33,182 and 142 I.U./L. His stay in the Coronary Care Unit and rehabilitation ward was uneventful and was discharged on the 22nd post infarction day on metroprolol (Betaloc) 300 mg/day.'

On the third review on 2-5-1978 he was well. Electrocardiogram on this visit was normal and has remained normal on the next two visits.

DISCUSSION

Abnormal Q waves have been regarded as pathognomonic of myocardial infarction. A widely held concept of the origin of the Q waves is that it is produced by the failure of electrically inert muscle tissue in the vicinity of the electrode to contribute a positive vector force in the initial 0.04 seconds of the depolarization process. Cook *et al.* (1958) demonstrated that for Q waves to be produced, between 50-70% of the thickness of the left ventricular wall must be ischaemic. The small r or R wave that follows represents depolarization of normal sub-epicardial tissue adjacent to the infarct. Gross *et al.* (1964), in experimental studies involving ligation of coronary arteries of dogs, showed transitory Q waves in multiple leads. The Q waves appeared within 3 minutes of ligation and disappeared within 5 minutes of ligature release. Similar transitory Q waves have

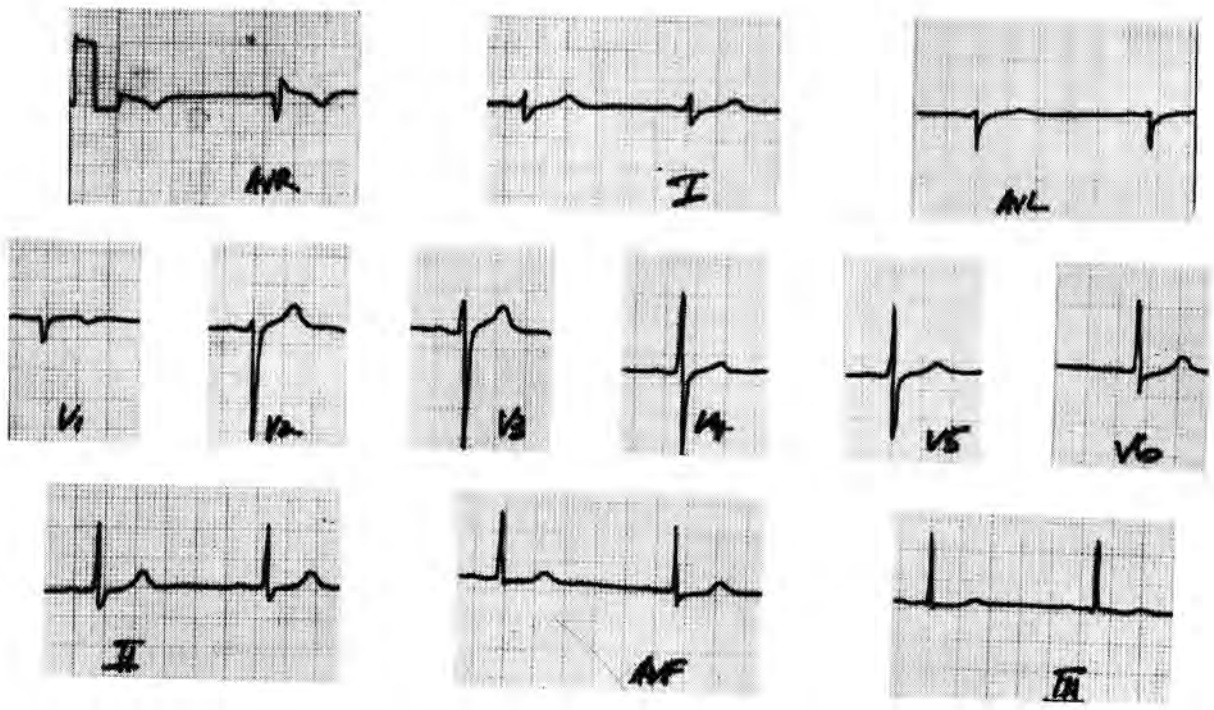

Fig. 2 Normal electrocardiogram on 19-7-78

also been reported with electrolyte imbalance (Nora and Ritz, 1959), shock and hypoglycaemia (Goldman *et al.*, 1960). The Q waves may persist up to 5 days but disappeared with correction of the metabolic disturbances. However, no mention was made of the associated cardiac enzyme changes. It is reasonable to assume that a period of time exists after the interruption of blood flow to an area of myocardium during which the QRS abnormalities that appear would be reversible. A lessening of the severity of the metabolic stress in the peripheral areas of the infarcted core enables islands of myocardium around to recover and resume electrical activity. The period of reversibility would be related to the serverity of the resultant metabolic disturbance of the myocardium. The disappearance of the Q wave with electrocardiographic restitution as seen with these 4 cases would suggest that as significant amount of myocardium was effected to give rise to the early electrocardiographic and enzymatic changes. A sufficient area of myocardium was present which, although severely affected by ischaemia, was reversible.

Thyrotoxicosis with cardiac manifestations has been reported by De Groot (1972). The increased adrenergic activity accounts for many of the classical symptoms and circulatory changes. Chest pains of angina pectoris or myocardial infarction may be produced especially on a compromised coronary circulation. Conduction defects also occur with thyrotoxicosis. Campus *et al.* (1975) described two patients with heart block, both of whom reverted to normal when rendered enthyroid. Resnekov and Falicov (1977) reported 3 cases of thyrotoxicosis in young female patients presenting with severe angina and myocardial infarction. Normal coronaries were found at angiography. An abnormal lactate response occured with pacing-induced stress suggesting myocardial cellular hypoxia due to increased oxygen demand as the reason for the chest pain. After treatment and when enthyroid, one patient showed persistent Q wave with ST elevation but no mention was made of the other 2 cases described. Case 1 demonstrates normalization of the electrocardiogram when enthyroid after treatment. Selective coronary angiography was not performed to demonstrate any major occlusion of his coronary arteries. It would be unusual to find severe coronary artery disease in his age in the absence of other risk factors. A satisfactory symptomatic response with electrocardiographic restitution seen after adequate treatment of his thyrotoxic state would suggest a metabolic basis for the infarction.

Beta-blockers are used extensively in post infarction angina and arrythmias with good results. Its use in acute myocardial infarction was first reported by Snow (1966), who showed a substantial reduction in mortality of patients who had received propranolol. Moroko *et al.* (1971), and Shell and Sobel (1973), showed that propranolol reduced infarct size in experimental infarction. This has revived recent interest in the use of beta-blockers in the early post infarction phase in the attempt to preserve ischaemic myocardium; delay evolution of infarction and enhance reversibility of ischaemic area. In all the 4 cases beta-blockade was used in the early post infarction period. This could have contributed to the electrocardiographic restitution seen.

Selective coronary angiography was not performed on any of the 4 cases studied. No literature is available on the relationship between patients who show electrocardiographic restitution after myocardial infarction and the state of their coronary circulation. This radiological procedure would provide invaluable information regarding long-term prognosis as electrocardiographic changes alone cannot prognosticate survival of the patients.

SUMMARY

This report is a retrospective study of the electrocardiograms that revert to normal after myocardial infarction. 4 patients (4%) showed complete electrocardiographic restitution on 3-12 months follow up. The incidence is comparable to other reported series. Possible pathophysiologic mechanisms are discussed.

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