CORONARY ARTERY DISEASE PRESENTING WITH EXERCISE INDUCED COMPLETE HEART BLOCK
A Case Report.

KOH CHAN SING, MBBS (Mal), MRCP (UK)

Ipoh Specialist Centre,
No 26, Jalan Tambun,
30350 Ipoh,
Perak, Malaysia.

INTRODUCTION

An 84 year old man who presented with Stokes Adams syncope with exertion was found to have complete heart block on walking with reversion to sinus rythym on recovery. The resting ECG showed evidence of ischemic heart disease with old anteroseptal and old inferior infarction but there was no recent myocardial infarction as shown by normal cardiac enzymes levels. The probable explanation for the mechanism and causation of the transient A-V block is described.

CASE HISTORY:

An 84 year old Indian male who was previously in good health started to have episodes of black-out lasting several seconds. Over a period of nine months, he had five episodes of syncope brought on while walking around the house and was of a few seconds duration not followed by any convulsion. Fifteen years ago he had a haemorrhoidectomy done and for the past three years he had been treated for hypertension by a private practitioner with Metoprolol 50 mg twice daily. He had no family history of heart disease. He does not smoke or drink alcohol. Physical findings on admission revealed a healthy elderly man with a pulse rate of 80 per minute which is regular and a blood pressure of 120/60 mmHg.

Examination of the cardiovascular system showed cardiomegaly with a prominent left ventricular impulse, an ejection systolic murmur grade 2/6 and a third heart sound over the mitral area. All other systems were normal. He was admitted to the coronary care unit on August 20, 1983 for monitoring which did not show any arrythymias. Investigations showed the following results: Hb 13.2 gm%, Wbc 5.0×10^9/L, blood urea, serum electrolytes, fasting blood sugar and serial cardiac enzymes were normal. Chest radiograph showed moderate cardiomegaly with normal lung fields. Electrocardiogram showed Q waves in lead II, III, AVF, VI, V2 and V3 with T wave inversions in lead VI, V2 and V3 (figure 1) indicative of old inferior and old anteroseptal infarction. He was put on a telemetry monitor and on making him walk around the ward he soon developed faintness and could not walk further. Continuous ECG monitoring showed the development of 2:1 AV block and later complete heart block but with rest, the ECG gradually returned back to normal (figure 2). On the night of August 22, 1983, he developed ventricular fibrillation which reverted back to sinus rythym after defibrillation. The next day he again developed ventricular fibrillation and despite vigorous resuscitation he could not be revived and died. He was not

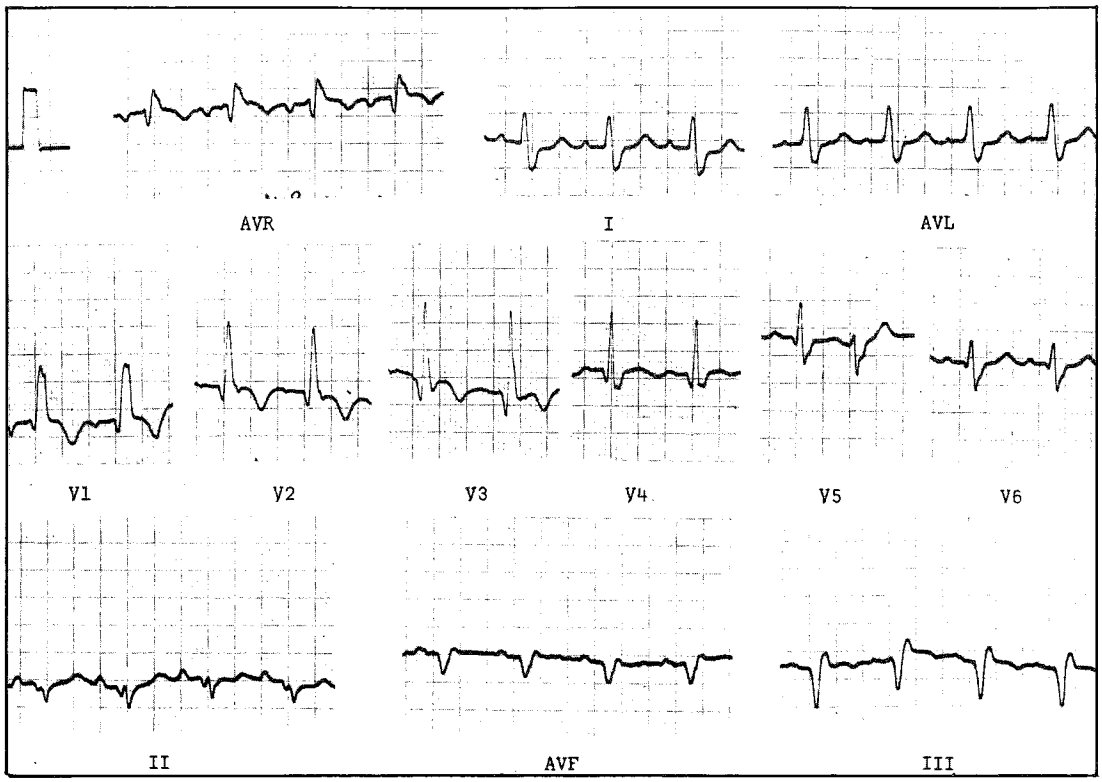

Figure 1

Resting 12 lead ECG of patient. Note the presence of Q waves in lead II, III, AVF, VI, V2, V3 and T inversions in lead VI, V2 and V3.

given any medications which could have induced ventricular fibrillation, his serum electrolytes were all within normal limits and no drugs have been withdrawn prior to development of ventricular fibrillation.

DISCUSSION

Complete or third degree atrioventricular block appears not uncommonly in acute myocardial infarction but this is the first case the author came across whereby complete heart block appeared transiently with exertion and disappeared at rest in a patient with coronary artery disease. The QRS complex of the supraventricular type indicates that the site of A-V block is located in the first portion of the conducting system above the bifurcation of the His bundle. The AV node is supplied by the right coronary artery and this patient had evidence of diseased right coronary artery as shown by the previous old inferior infarct. Development of ischemia to the AV node due to disturbance of blood supply to the A-V node on exercise most probably explains the mechanism of AV block. The transitory ischemic genesis of AV block appearing in this patient is perhaps similar to the mechanism which has been advocated in patients with inferior myocardial infarction and A V block[1]. In these patients the block is usually transient in contrast to those that occur in anteroseptal myocardial infarction in which A-V block is due to extensive damage to the septal myocardium and A-V block tends to be prolonged. On reviewing the literature cases of transient A-V block can occur in patients with Prinzmetal variant angina during pain and revert to sinus rythym after termination of chest pain.[2,3]

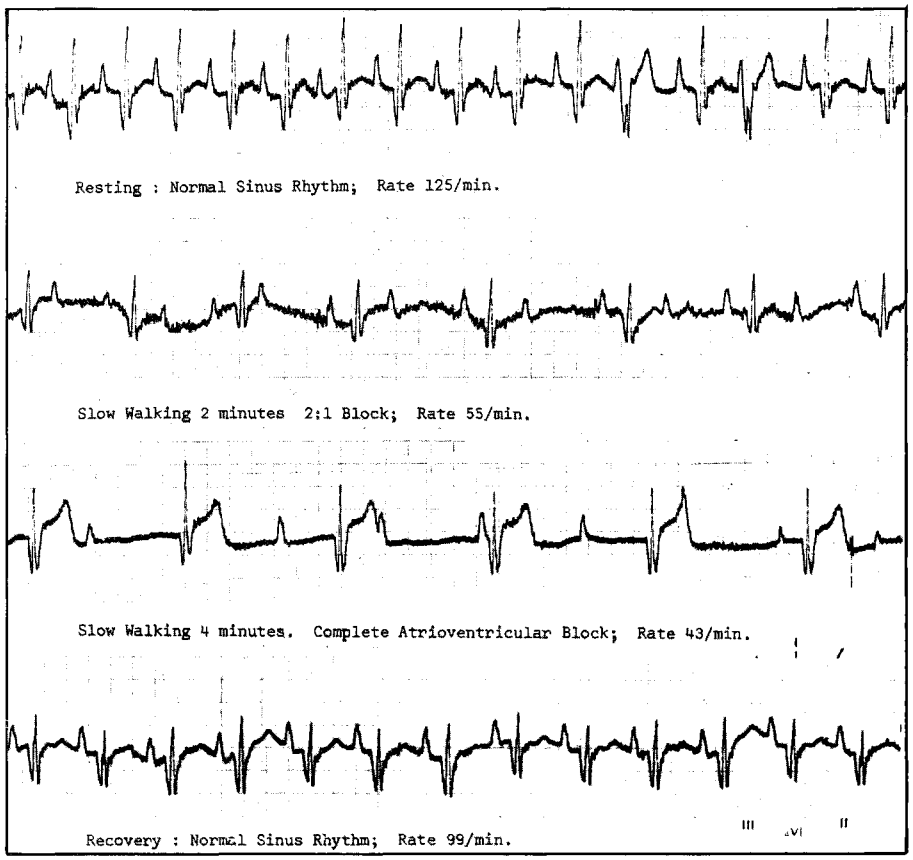

Figure 2

Long lead II at rest, after slow walking at two minutes, four minutes and during recovery. Note progressive changes from sinus rythym to 2:1 AV block, complete AV block and reversion back to sinus rythym.

ACKNOWLEDGEMENT

I would like to thank Ms Stephenie Chin for her secretarial assistance.

REFERENCES

1 Fazzini P.F. and Marchi F.; Atrioventricular Block in acute myocardial infarction; Acta Cardiological; 1970; 25:517 – 527

2 Botti, R.E.; A variant form of angina pectoris with recurrent transient complete heart block; Am. J. Cardiol; 1966; 17:443 – 446

3 Schuatz L.S., Schwedel J.B. and Schuatz S.P.; Adams Stokes Syncope during angina pectoris associated with coronary artery disease; Am. J. Cardiol; 1966; 17:426 – 432