TRANSIENT LEFT BUNDLE BRANCH BLOCK— A CASE REPORT

By L.S. Chew
(Medical Unit III, General Hospital, Singapore)

INTRODUCTION

It is generally believed that bundle branch block once established tends to remain permanent. In most of the cases of bundle branch block, in which the aetiological factors have been established, the patients were found to have hypertension or arteriosclerotic coronary artery disease. In such cases it is believed that arteriosclerotic narrowing of a coronary artery branch supplying the bundle results in anoxia with eventual fibrosis. There are, however, exceptions, in which the block is temporary, and have been described as intermittent, paroxysmal or transient bundle branch block. This paper reports a patient with transient left bundle branch block.

CLINICAL RECORD

The patient, CCF, a 48 year old female housewife was first admitted to hospital on 28.12.1967. She was well until about three weeks prior to admission, when she began to experience breathlessness on exertion which progressively became severe. At the time of admission she had several occasions of paroxysmal nocturnal dyspnoea. She had only some central chest discomfort associated with the breathlessness but had not experienced any occasion of severe central chest pain of myocardial infarction.

On examination, the patient was seen to be obese, afebrile and in a generally satisfactory condition. She was not dyspnoeic and there was no evidence of cyanosis or ankle oedema. The jugular venous pulse was normal. She had a blood pressure of 130/80 mm. Hg. The apex of the heart was not palpable. The heart rate was dual, regular and there was no evidence of triple rhythm. There were no crepitations in the lung bases.

Investigations showed a haemoglobin of 12.3 gms. with a total white cell count of 7,400/c. mm. with a normal differential distribution of the white cells. The basal sedimentation rate was 21 mm./hr. The serum glutamic oxaloacetic transaminase was 125 units (normal 120 units). The chest radiogram showed enlargement of the cardiac configuration with pulmonary oedema. The electrocardiogram showed left bundle branch block.

She had an uneventful stay and was discharged after one month's hospitalization.

She was admitted again on 10.2.68, a month later, with the history of progressive breathlessness and oedema of the legs. For the three days prior to admission, she had experienced central chest discomfort off and on. On the day of admission, she had one episode of central chest pain lasting one hour. There was radiation of this pain up the neck and down the left arm.

On admission, she was seen to be breathless and in cardiac failure, with ankle oedema and raised jugular venous pressure. Her blood pressure was recorded at 140/100 mm. Hg. The apex beat was not felt and the heart showed evidence of triple rhythm with a marked presystolic gallop. There was evidence of crepitations in the lung bases. The liver was enlarged two finger-breadths below the costal margin.

Investigations showed a haemoglobin of 14.3 gms., and a total white cell of 7,700/c.mm. with a normal differential. The serum glutamic oxaloacetic transaminase was 92 units, the blood uric acid was 3.6 mgm.% and the fasting blood sugars on two occasions were 118 mgm.% and 105 mgm.% respectively.

The electrocardiogram showed left bundle branch block with raised *ST* segments over V_3 and V_4. There was the notching of the *S* wave of the avF lead.

In view of her history of chest pain which simulated a recent myocardial infarct and her previous admissions for possible myocardial infarction on 29.12.67, she was given anticoagulant therapy in addition to digitalis for cardiac failure.

She made an uneventful recovery after the second admission and showed general improvement. There was never any occasion of shock or deterioration of cardiac failure during the stay in the ward. Her blood pressure was always in the region of 120/80 mm. Hg.

During the follow-up period of about one and a half years, she was seen at regular monthly intervals. Except for the occasions when she

complained of palpitations on exertion, there were no other significant symptoms of cardiac decompensation. There was never any recurrence of chest pain. The blood pressure fluctuated around 120/80 mm. Hg. and on occasions it was recorded as high as 185/80 mm. Hg.

All through the follow-up period, she was maintained on pentanitrol, chlordiazepoxide and phenindione 60 mgm./day. The coagulation activity was monitored by the thrombo-test (Owren's) and was always in the range of 10-20%.

The serial electrocardiograms of the patient over the period of one and a half years are shown in Fig. 1. When she first presented for admission, the accompanying electrocardiogram of 29.12.1967 showed a left bundle branch block pattern, which remained the same in the weekly recordings till she was discharged after one month's hospitalization.

During the second admission, the patient presented with more definite clinical evidence of myocardial infarction with central chest pain and its radiation up the neck and down the left arm. The electrocardiogram recorded on this admission showed again the presence of left bundle branch block and raised *ST* segments. The essential feature to note is the presence of notching of the *S* wave in avF lead. This notching of the *S* wave in the avF lead is present in all the serial electrocardiogram recordings.

The reversion of the electrocardiogram to a normal pattern was first recorded on 23.5.69. There was an absence of the left bundle branch block pattern and also the loss of the *S* wave

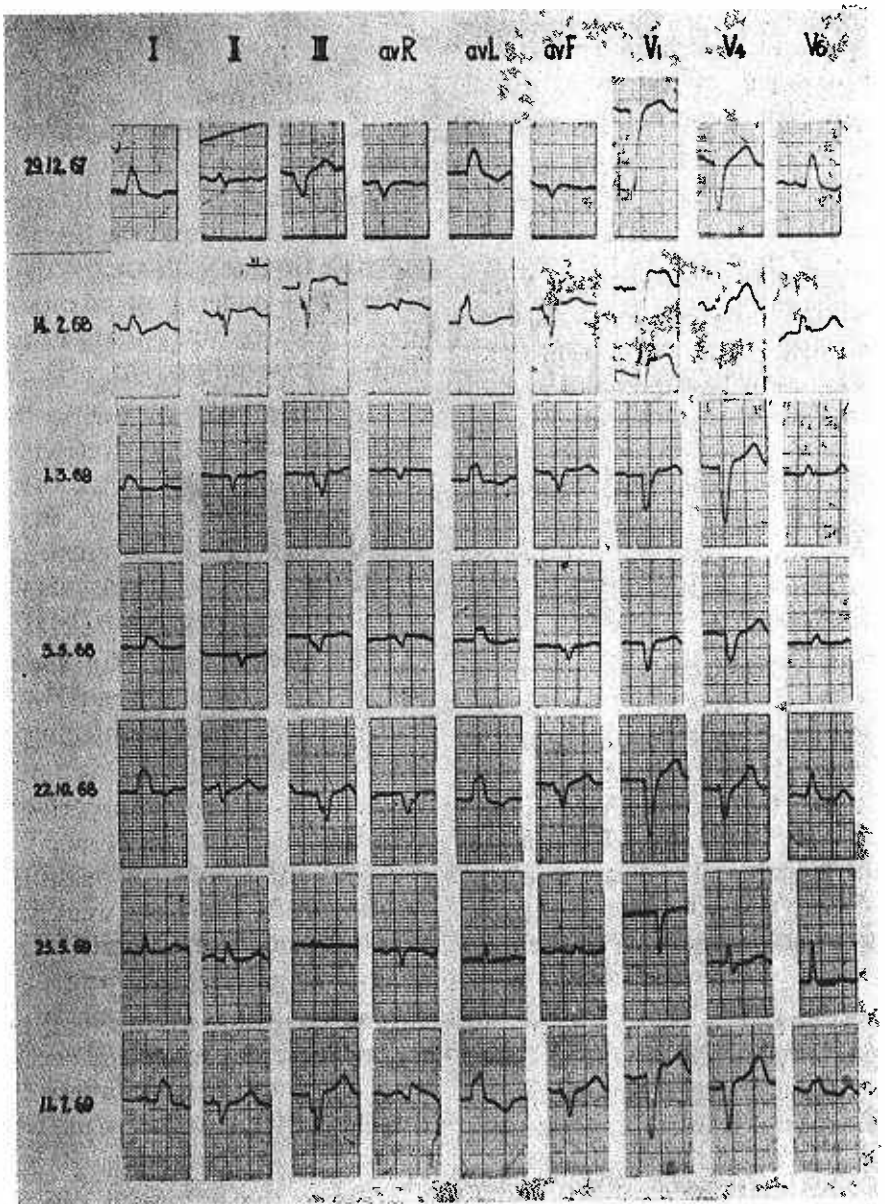


Fig. 1. Serial electrocardiogram showing left bundle branch block and notching of *S* wave in lead avF. Reversion to normal conduction in electrocardiogram of 23.5.69.

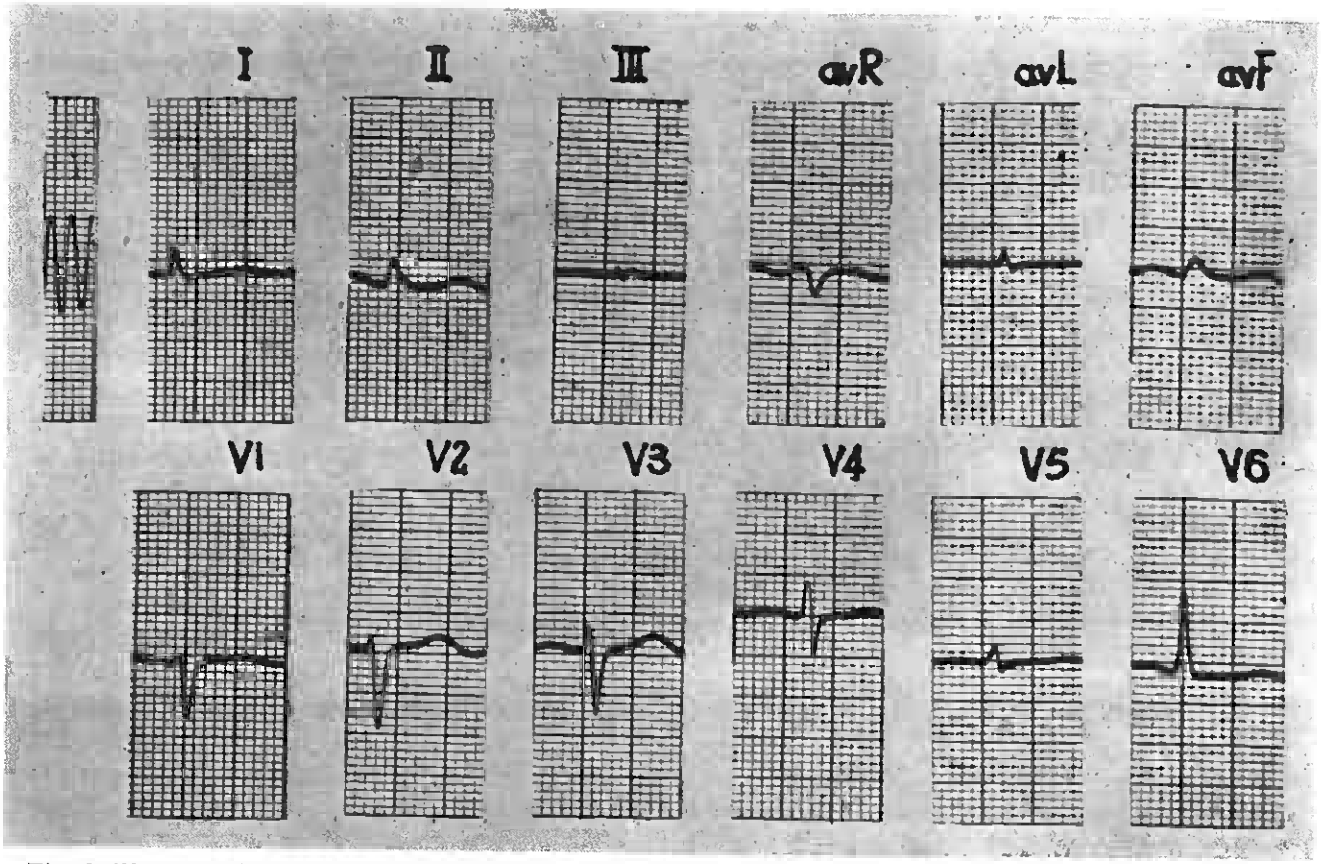


Fig. 2. Electrocardiogram of 22.4.69 shows normal bundle branch conduction without evidence of myocardial infarction.

in lead III and avF. A similar recording was made one month earlier (Fig. 2), and from this electrocardiogram it was not possible to obtain any information as to the site of the previous myocardial infarction.

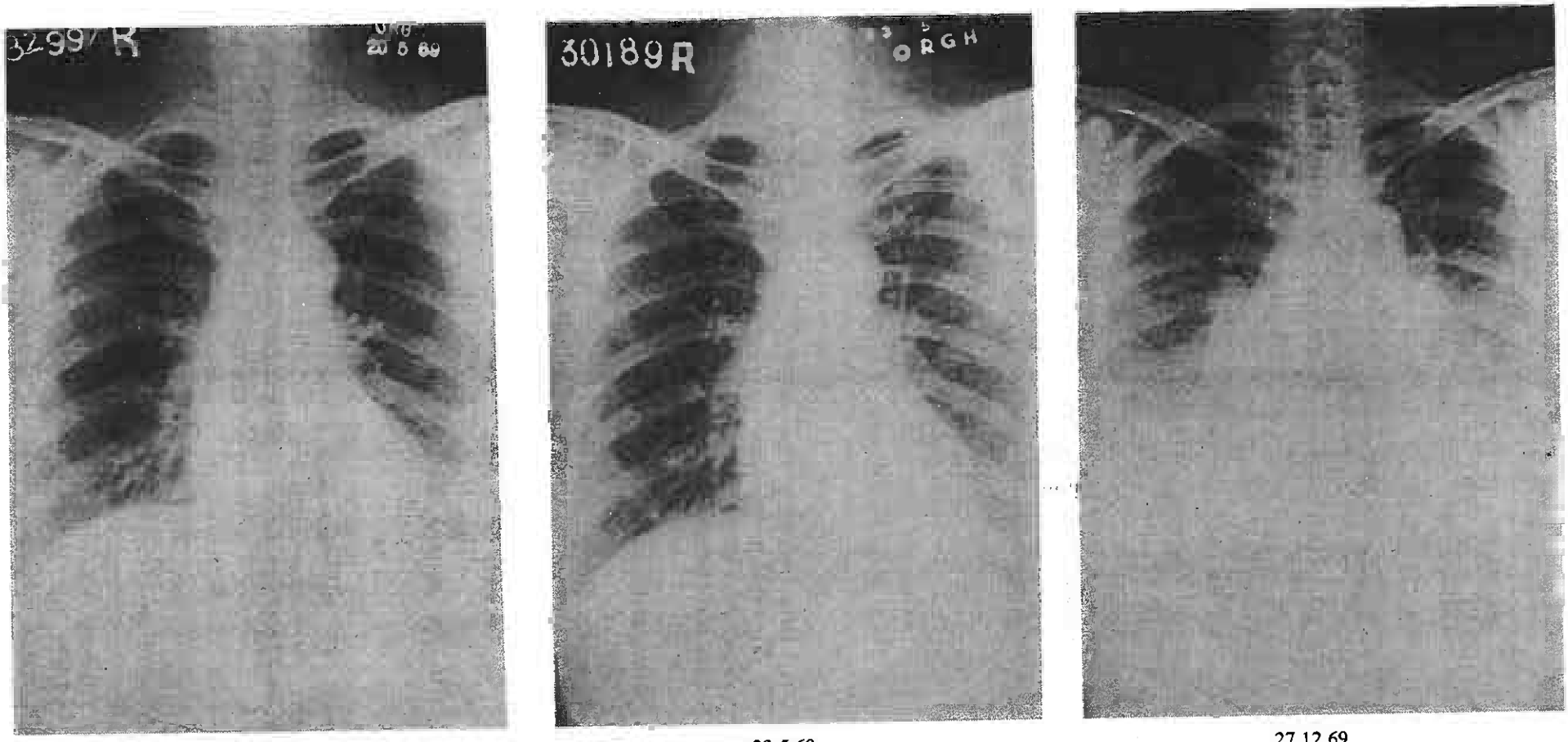
Subsequent electrocardiogram recordings at monthly outpatient follow-up showed a reversion to the left bundle branch block pattern with the feature of the notching of the *S* wave in avF lead.

The initial chest X-ray of 27.12.67 showed a large heart with pulmonary oedema of the interstitial type (Fig. 3). The chest X-ray of 23.5.68 showed the absence of pulmonary oedema. The heart size had also returned to fairly normal limits except for possible left ventricular enlargement. The last chest X-ray of 20.5.69, a year later, showed essentially the same features as that of 23.5.68.

DISCUSSION

Bundle branch block is no longer regarded as a static condition always associated with an organic disease process interrupting the main bundles of the ventricular conduction system. The frequency of transient and intermittent appearances of bundle branch block complexes and the demonstration that intraventricular conduction defect may occasionally be made to appear and disappear at will—with increase in vagal tone (Dressler, 1959) or tachycardia (Eichert, 1946)—suggest the need for a more dynamic concept of this condition (Bauer, 1964). There appears to be at least three mechanisms by which bundle branch patterns may be produced. (1) Anatomical severance of a conducting bundle, (2) ventricular hypertrophy and ischaemia of the appropriate chamber and (3) functional and neurogenic depression with or without underlying pathological lesion of the conducting tissue.

Transient bundle branch block is defined as an intraventricular conduction defect that subsequently returns, if only temporarily to normal conduction (Bauer, 1964). It is difficult to conceive of time interval involved in transient bundle branch block, which in the patient described, the left bundle branch block had persisted for a year. Furthermore it is likely that the infarcted area involving the left bundle branch would have undergone fibrosis and hence resulting in loss of normal conduction. However, Myre and Fuller (1951) described a patient with left bundle branch block remitting spontaneously after having been established for a period of three years. The majority of patients



20.5.69 23.5.69 27.12.69

Fig. 3. Serial chest radiograms show no enlargement of cardiac configuration except for prominence of left cardiac border. The chest radiogram on admission 27.12.69 shows gross pulmonary oedema.

described by Bauer (1964) with transient bundle branch block eventually relapsed and developed permanent intraventricular conduction defects. In three of the fourteen patients the last electrocardiogram available showed normal conduction.

In the patient described, however, the normal conducting pattern of the bundle branches was recorded only on two occasions separated by an interval of one month. There was unfortunately no electrocardiographic recordings made in the months prior to the appearance of the normal conduction (23.5.69) and therefore it cannot be surmised if the pattern had not reverted to a normal one at an earlier date.

It is generally accepted that coronary thrombosis complicated by left bundle branch block may electrocardiographically be difficult to recognize. Chapman and Pearce (1957) have shown that five out of six patients who had infarctions that predominantly involved the septum, each of these had initial notching of the *S* wave in lead avF. In each of these there was a large *S* wave or *QS* deflection in lead avF with a small upward deflection during the early part of the *S* wave almost reaching the base line. The electrocardiogram of the patient described, shows a persistent notching of the *S* wave in lead avF. This involvement of the septum probably resulted from an extension of the myocardial infarct which had initially brought her to hospital on 28.12.67. Unfortunately the restoration of the transient left bundle branch block to normal conduction did not allow for the observation of a definitive diagnostic pattern of the site of infarction as represented electrocardiographically to be made.

The co-existence of coronary artery disease and hypertension provide the usual background for pathologic changes leading to left ventricular conduction defect. The pathogenesis of left bundle branch block in this patient can be inferred to have resulted from ischaemic heart disease. However, there is no cardiac enlargement that is commonly present with left bundle branch block of ischaemic origin. The cardiac configurations in the chest radiograms show only prominence of the left ventricle. There is in addition no evidence of hypertension in the blood pressure recorded throughout the follow-up period.

From the clinical and electrocardiographic evidences, the left bundle branch block in this patient is the result of coronary artery disease and myocardial infarction of the septum.

ACKNOWLEDGEMENT

I am grateful to Dr. A.L. Gwee for permission to publish the case.

REFERENCES

1. Bauer, G.E. (1964): "Development of Bundle branch block." Amer. J. Cardiology 14, 346.
2. Bauer, G.E. (1964): "Transient bundle branch block." Circulation 29, 730.
3. Chapman M.G. and Pearce M.L. (1957): "Electrocardiographic diagnosis of myocardial infarct in the presence of left bundle branch block." Circulation 16, 558.
4. Dressler, W. (1959): "Transient bundle branch block occurring during slowing of the heart beat and following sagging." Amer. Heart J. 58, 760.
5. Eichert, H. (1946): "Transient bundle branch block associated with tachycardia." Amer. Heart J. 31, 511.
6. Myre, S.L., Fuller, B.F. (1951): "Left bundle branch block with spontaneous remission after at least three years." Arch. Int. Med. 34, 1497.