Transient Bundle-Branch Block


In a recent survey of 167 patients with bundle-branch block 10 instances of transient bundle-branch block were discovered. Since the completion of this study four more patients with this condition have been encountered. These 14 patients represent the subjects of this report.

Transient bundle-branch block is defined as an intraventricular conduction defect that subsequently returns, if only temporarily, to normal conduction. A distinction is made between this condition and intermittent bundle-branch block, which is characterized by the presence, in a single electrocardiographic tracing, of complexes showing bundle-branch block and normally conducted beats, or very rarely pre-excitation complexes. 2 These definitions vary somewhat from those of Holzmann, 3 who also recognized two forms of inconstant bundle-branch block, namely temporary bundle-branch block, in which the conduction defect persists for days or months, and intermittent bundle-branch block, in which the conduction defect lasts for seconds up to several hours.

The condition here described has been referred to in the literature by several other terms including paroxysmal, 4 unstable, 5, 6 and temporary 3 bundle-branch block. It is proposed to review the literature, report 14 new cases and discuss the etiology and prognosis, and to speculate about the mechanism of this conduction disturbance.

Review of the Literature

It is generally agreed that the first case of transient bundle-branch block was reported by Lewis in 1913. 7 His patient was a 32-year-old bookbinder with rheumatic aortic incompetence who, on the third day of a febrile illness, possibly bacterial endocarditis, had an electrocardiographic examination that revealed, according to the then accepted nomenclature, a block in the right bundle of His. An electrocardiogram, repeated on the following day, showed that the conduction defect had disappeared, although, as Herrmann and Ashman 8 pointed out later, the tracing still suggests a slight prolongation of the QRS complexes.

In 1931 Herrmann and Ashman 8 collected 10 cases of transient bundle-branch block from the literature, including the one reported by Lewis. 7 They added to this, five more cases of their own, as well as three cases that they described as most unusual, showing sudden transition from complete bundle-branch block to normal intraventricular conduction for transient periods, as a result, according to the authors, of indirect vagal effects.

One of the most comprehensive papers on this subject was published by Comeau et al. 4 By this time the authors had discovered 58 cases in the literature and were able to add a further 13 cases from the records of the Massachusetts General Hospital, the Boston City Hospital, and the files of private consultant physicians in Boston. They fully discussed the etiology and prognosis of their large material and came to the conclusion that transient bundle-branch block was to be considered as evidence for the presence of organic heart disease.

In the following year Freund and Sokolov 9 reported a follow-up study of 210 patients with bundle-branch block that included nine patients with transient bundle-branch block.

Since then many further contributions have appeared, generally reporting one or two cases, relating some special study or points of unusual interest. Three papers are worthy of separate mention. Sandberg et al. 10 published 12 cases showing at one time or another transient or intermittent bundle-branch block. They included four cases of transient bundle-branch block, two of intermittent bundle-branch block, and four of unstable right bun-

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bundle-branch block of the Wilson type. More recently, Hallhagen reported five patients including two with transient bundle-branch block and one who demonstrated at first transient, then intermittent, and eventually permanent bundle-branch block. Hallhagen concluded that transient bundle-branch block was probably not so rare as previously believed, a view fully supported by this study. Scherlis and Lee recently reported an electrocardiographic and vectorcardiographic study of eight patients with transient right bundle-branch block. They confirm a previous observation by Dodge and Grant that in human right bundle-branch block the initial 0.04 second of the QRS complexes remains unaltered and the main disturbance lies in the terminal part of the ventricular depolarization represented by the broad slurred S wave in lead I and the delayed R waves in V1. These changes indicate late and aberrant activation of portion of the right ventricle and were also seen in the two patients with transient right bundle-branch block in the present series (fig. 1).

Clinical Data

Among 167 patients with bundle-branch block personally examined between October 1952 and July 1961 were 10 with transient bundle-branch block. Since the completion of this study four further patients with this conduction disturbance have come under observation. A summary of the clinical features of these 14 patients is presented in table 1.

The 14 patients with transient bundle-branch block include eight men and six women. Their ages, at the time of onset of the conduction defect, ranged from 33 to 83 years. In 12 instances the conduction defect was of the left bundle-branch block variety, in two right bundle-branch block was found.

All but one patient suffered from hypertensive and ischemic heart disease; the exception had aortic stenosis. These etiologic findings are in general agreement with those found in established bundle-branch block. Over 85 per cent of patients with left bundle-branch block suffer from hypertension or ischemic heart disease and, although either condition alone may be accompanied by this conduction defect, it is the combination of the two which is most consistently complicated by left bundle-branch block. Left ventricular strain with ischemia, absolute or relative, of the dilated and hypertrophied myocardium appears to be the commonest background to the development of left bundle-branch block.

During periods of normal conduction the electrocardiogram was physiologic in three patients, revealed left ventricular hypertrophy in two patients, and demonstrated evidence
of myocardial ischemia or infarction, with or without left ventricular hypertrophy, in the remaining nine patients.

Although the majority of patients with transient bundle-branch block eventually relapsed and developed permanent intraventricular conduction defects, in three of the 14 patients the latest electrocardiogram available showed normal conduction. One patient (case 3) has been in normal conduction for over 4 years following transient left bundle-branch block, another patient (case 8) showed normal QRS complexes for over 5 years but recently has again relapsed into an unstable form of left bundle-branch block. Occasionally, patients revert to normal intraventricular conduction even years after consistently demonstrating bundle-branch block. One such instance is seen in case 12, whose electrocardiogram returned to normal QRS complexes after 6 years of apparently stable left bundle-branch block.

**Representative Case Histories**

**Case 2**

This man was first seen in January 1954 at the age of 71 years, when he complained of palpitation and dyspepsia. The only abnormalities noted in the cardiovascular system were an irregular pulse and a moderately raised blood pressure. The electrocardiogram confirmed the presence of ectopic beats and was otherwise within normal limits. He was seen again on May 17, 1956, because of increasing chest discomfort. An electrocardiogram revealed left bundle-branch block (fig. 2). This pattern remained unchanged until July 24, 1957. On January 15, 1958, without any overt change in his general condition, the electrocardiogram showed normal intraventricular conduction and T-wave changes compatible with anterior and posterior wall ischemia. On May 26, 1958, the electrocardiogram showed again left bundle-branch block and remained so on October 10, 1958. A further tracing on June 24, 1959, once more demonstrated a return to normal conduction with evidence of left ventricular hypertrophy. The T waves in V4 were now isoelectric and the T waves in leads II, III, and aVF upright. On October 12, 1959, and October 5, 1960, left bundle-branch block reappeared. Normal intraventricular conduction returned for a third time, on September 13, 1961, the electrocardiogram was similar to the one taken on June 24, 1959, except for more upright T waves in lead V4. During this repeated appearance and disappearance of left bundle-branch block, the patient, now aged 78 years, has remained in a satisfactory condition.

**Case 8**

This patient first came under medical observation in 1945, aged 34 years, when she suffered from a left retinal artery occlusion followed by
optic atrophy. In 1954, during an attack of vertigo, hypertension was discovered. In August 1957 lower central chest pain on exertion appeared and in October of the same year a central vein thrombosis occurred in the right eye. It was then, on October 3, 1957, that the first electrocardiogram was taken, which revealed left bundle-branch block (fig. 3). Cardiac findings at that time showed a blood pressure of 210/120, slight left ventricular enlargement, but no other relevant findings. She was treated with hypotensive agents and diuretics with easy blood pressure control. The next electrocardiogram, 2 months later on December 4, 1957, showed that the bundle-
branch block had disappeared, the tracing was within normal limits. Her progress has been uneventful, vision in the right eye has nearly fully recovered, ischemic chest pain has subsided, and her blood pressure is well controlled. During the subsequent 5½ years her electrocardiograms showed normal QRS complexes. When recently seen on May 2, 1963, left bundle-branch block had reappeared, but could be voluntarily abolished by slowing the heart rate with deep inspiration and breathholding. This same phenomenon of voluntary control of bundle-branch block has recently been reported in five other patients.14

Case 12

This patient first became aware of hypertension in July 1953, aged 30 years, when he was refused life insurance. He remained symptom-free except for a slight tightness in the throat following severe physical exertion. He was seen on May 12, 1956, for assessment of hypertension. His blood pressure was 210/130, there was left ventricular enlargement, triple heart rhythm, and hypertensive retinal changes with recent hemorrhages. An electrocardiogram (fig. 4) showed intermittent left bundle-branch block, the two tracings A and B being sequentially recorded. The normally conducted complexes revealed evidence of anterior and posterior wall ischemia. Full investigations, including aortography and phentolamine tests, failed to uncover a remediable cause for his hypertension. Follow-up electrocardiograms revealed apparently stable left bundle-branch block. Since November 1960 he has been treated with guanethidine and diuretics.5 On April 26, 1962, his pulse rate had dropped to 60/min., possibly under the influence of guanethidine, his blood pressure was well controlled. The electrocardiogram, for the first time in nearly 6 years, revealed normal intraventricular conduction, with some increase in the height of the R waves in left ventricular surface leads compared with May 1956, suggesting progressive left ventricular enlargement. Subsequently, it was discovered that this patient’s bundle-branch block could be made to appear and disappear by inducing variations in the heart rate.

Cases 10 and 12 have been previously reported among a group of five patients showing voluntary control of bundle-branch block.14

Discussion

The underlying pathology of transient bundle-branch block in all but one of the cases in this series was related to ischemic heart dis-

Figure 3

Case 8. Transient left bundle-branch block.
Case 12. Anterior and posterior wall ischemia, left ventricular hypertrophy, intermittent and transient left bundle-branch block. (By permission of the Editor, British Heart Journal—from Bauer 1964).
ease. This is in agreement with the etiology of bundle-branch block as a whole and appears to correspond with the general experience in the literature. Forty-four of the 71 cases reviewed by Comeau et al.\textsuperscript{4} suffered from coronary or hypertensive heart disease. Transient bundle-branch block may complicate myocardial infarction\textsuperscript{9,10} (cases 1 and 11 of present series) or may be seen during attacks of ischemic chest pain\textsuperscript{4,16} (cases 9 and 13). Bundle-branch block has also been observed during episodes of heart failure, disappearing with improvement of cardiac function\textsuperscript{4,9} (case 7).

Rheumatic heart disease and acute infectious fevers, especially diphtheria, have been the cause of several reported cases of transient bundle-branch block.\textsuperscript{4} Freund and Sokolov\textsuperscript{9} briefly mention a patient in whom bundle-branch block disappeared after appendectomy.

A rare cause of transient bundle-branch block is thyrotoxicosis. Digi\textit{li}o\textsuperscript{17} reported the case of a 29-year-old woman suffering from a toxic goiter and left bundle-branch block. Two days after thyroidectomy intraventricular conduction defect was still present but the QRS complexes were shorter suggesting a diagnosis of incomplete left bundle-branch block. Six weeks later normal conduction was re-established. A similar case was published by Packard and Graybiel,\textsuperscript{18} except that their patient had a right bundle-branch block prior to thyroidectomy.

Transient block, usually of the right bundle, may complicate acute right ventricular stress due to pulmonary embolism.\textsuperscript{19} Transient right bundle-branch block may also complicate right heart catheterization\textsuperscript{20,22} and various open cardiac surgical procedures.\textsuperscript{22}

A number of drugs, especially those with a depressant effect on cardiac conduction, have been responsible for the appearance of transient bundle-branch block. The best known of these are quinidine,\textsuperscript{24,25} procaine amide,\textsuperscript{26} and potassium.\textsuperscript{26}

Psychological stress, especially anxiety, has been reported to induce transient bundle-branch block. Graybiel et al.\textsuperscript{27} recorded the case of an apparently healthy aviator in whom the stimulus of fright, caused by the firing of a pistol, precipitated a transient conduction defect.

It has long been known that alterations in the heart rate are of significance in the appearance of bundle-branch block.\textsuperscript{28} Transition from normal to abnormal intraventricular conduction may be related to alterations of the rate of only one or two beats per minute.\textsuperscript{5,11} In the majority of reported cases retardation of the heart rate favors normal intraventricular conduction while acceleration favors bundle-branch block. The physiologic and pharmacologic stimuli known to induce or abolish bundle-branch block through variations in heart rate have been fully discussed in a report dealing with five cases of voluntary control of bundle-branch block.\textsuperscript{14}

Occasionally, changes in the heart rate have produced paradoxical effects. Dressler\textsuperscript{29} reported two cases of transient bundle-branch block during slowing of the heart rate. Wallace and Laszlo\textsuperscript{26} reported that forceful pressure on the carotid sinus, accompanied by marked bradycardia, not only terminated but also consistently induced bundle-branch block in their well-studied 46-year-old patient with ischemic heart disease.

Transient bundle-branch block is generally regarded as the forerunner of a permanent conduction defect.\textsuperscript{4} The prognosis, however, is probably better than that of permanent bundle-branch block.\textsuperscript{8} This seems to be supported by several patients in the present series who for years after the appearance of transient left bundle-branch block remained in normal intraventricular conduction. Occasionally, bundle-branch block may remit spontaneously after having been established for a considerable length of time. Such cases have been reported.\textsuperscript{10,30,31} Case 2 in the present series reverted to normal conduction on three separate occasions after 1956, after bundle-branch block had been established for over a year. Case 12 returned to normal intraventricular conduction after being in apparently stable left bundle-branch block for a record time of nearly 6 years. Spontaneous reversion
is probably not a rarity and makes it difficult to evaluate therapeutic claims, based on single case reports, for the successful termination of bundle-branch block.

Transient bundle-branch block is of practical importance in the diagnosis of myocardial infarction. It is generally accepted that coronary thrombosis complicated by left bundle-branch block may electrocardiographically be difficult to recognize. However, by observing normally conducted complexes in cases of transient left bundle-branch block a definitive diagnostic pattern may be demonstrated. The incidence of transient bundle-branch block must depend on the diligence with which tracings showing conduction defects are repeated.

The mechanism of transient bundle-branch block remains obscure. Intraventricular conduction defects may probably be produced by a number of disturbances. They may result from anatomic and pathologic interruption of a conducting bundle, from ventricular enlargement and strain with dilatation of the appropriate chamber, and from functional or neurogenic depression, with or without underlying pathologic lesions of the conducting tissues. It may be speculated that a combination of these factors is of importance in the pathogenesis of transient bundle-branch block. Already 40 years ago Carter and Dieuaide\textsuperscript{32} postulated that a few intact fibers of the conducting bundle were able to carry on the normal excitation process, but were liable to fail under less favorable circumstances. These views were supported by animal experiments reported by Baschmakoff\textsuperscript{33} who was able to sever a conducting bundle in such a way that the surviving fibers conveyed impulses satisfactorily as long as the heart rate was below a certain critical level, but were unable to cope with faster rates resulting in the appearance of bundle-branch block patterns.

**Summary**

Fourteen patients with transient bundle-branch block have been personally studied. All but one suffered from ischemic heart disease commonly accompanied by hypertension. The conduction defect was observed to appear during acute infarction, attacks of prolonged chest pain, and episodes of left ventricular failure. Not infrequently, however, the appearance and disappearance of bundle-branch block was unaccompanied by any recognizable change in the patient's physical condition.

Bundle-branch block may revert to normal intraventricular conduction after many years. In one patient bundle-branch block disappeared on three separate occasions after having been present for over 12 months. Another patient is described in whom normal conduction returned after left bundle-branch block had been established for a record duration of 6 years.

The etiology, prognosis, and pathogenesis of transient bundle-branch block have been discussed and the practical importance of this conduction defect in the electrocardiographic diagnosis of myocardial infarction is mentioned.

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