Permanent right bundle-branch block heralded by intermittent block and ventricular extrasystoles

A 13-year observation

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SUMMARY A patient is described in whom the occurrence of frequent ventricular extrasystoles of right ventricular origin and intermittent right bundle-branch block caused by interpolation of these extrasystoles heralded the slow development of permanent right bundle-branch block over a period of observation of 13 years. Such a clinical course suggested gradual progression of an organic lesion in the intraventricular conducting system. The differential diagnosis of intermittent bundle-branch block and aberrant intraventricular conduction, and possible mechanisms of ectopic impulse formation in the presence of bundle-branch block are discussed.

The dependence of intermittent and transient bundle-branch block on heart rate changes is well known,¹⁻³ with the tachycardia-dependent variety more commonly seen than the bradycardia-dependent variety. The former is caused usually by sinus acceleration and rarely by other mechanisms. In contrast to aberrant conduction of supraventricular premature systoles, intermittent and transient bundle-branch blocks are thought to reflect pathological changes in the intraventricular conducting system,¹⁻³ but few reports have documented their transition to permanent bundle-branch block. In this communication, we report a case of intermittent right bundle-branch block caused by interpolated ventricular extrasystoles, which slowly progressed to right bundle-branch block over 13 years of observation.

Case report

A 70-year-old man was admitted to the Izu-Nirayama Hot Spring Hospital on 25 October 1966, for rehabilitation of a left hemiplegia resulting from an attack of cerebral thrombosis seven months earlier. He had no speech disturbance, was able to stand up using crutches, but was unable to walk. Cardiological examination was unremarkable except for a prominent aortic knuckle on the chest x-ray film. He was discharged in April 1967, but re-entered the hospital in August 1967 for further rehabilitation where he remained until May 1980, when he was transferred to

another hospital for the treatment of a bladder tumour. While in hospital his blood pressure was 140 to 180/70 to 90 mmHg. Serum electrolytes were normal and blood urea nitrogen fluctuated between 5.7 and 10.7 mmol/l (16 and 30 mg/100 ml). His cholesterol level was 6.5 to 7.8 mmol/l (250 to 300 mg/100 ml) in the early years in hospital, with subsequent drop to 4.7 to 6.2 mmol/l (180 to 240 mg/100 ml). He never experienced anginal pain or congestive failure.

ELECTROCARDIOGRAPHIC FINDINGS

The electrocardiogram on his first admission showed normal sinus rhythm at a rate of 65/min, with a PR interval of 0.16 s and a QRS duration of 0.06 s. The QRS complex in lead I showed an R pattern, and the frontal QRS axis was +45°. No signs of ventricular hypertrophy or ischaemia were noted. In April 1967, an Rs pattern appeared in lead I, with slight QRS widening (0.08 s) and a change in the axis to +20°. The QRS morphology in lead I changed back to R in March 1968, to Rs in November 1968, and again to R in December 1968. An rsR' pattern was never observed in lead V1 during this period. ST-T abnormalities suggesting slight anterolateral wall ischaemia gradually developed in late 1968. On 11 December 1968, he developed a fever of 39.3°C, with symptoms of acute bronchitis. An associated sinus tachycardia of 130/min did not significantly alter intraventricular conduction. After January 1969, sinus bradycardia prevailed. When his rate rose transiently to 80/min on

18 November 1969, QRS duration was increased to 0.10 s, and incomplete right bundle-branch block was diagnosed for the first time, with an rsR' pattern in V1. Thereafter, sinus bradycardia again persisted with normal intraventricular conduction.

During October 1972, several atrial extrasystoles appeared with aberration of right bundle-branch block type. In July 1974, an interpolated ventricular extrasystole was first observed, its left bundle-branch block configuration suggesting a right ventricular origin.^{4 5} The following sinus beat had a prolonged PR interval, but a normal QRS. Electrocardiograms obtained in September and October showed similar ventricular extrasystoles with full compensatory pauses. When another interpolated extrasystole occurred in November 1974, the next sinus beat showed a right bundle-branch block configuration

(Fig. 1A). From this month to May 1975, repeated electrocardiograms almost always showed ventricular extrasystoles. Whenever they were interpolated, the following sinus beat showed right bundle-branch block, whereas no QRS changes occurred after extrasystoles with compensatory pauses. On 20 June 1975, for the first time right bundle-branch block was present during sinus rhythm (60/min), with only the sinus beat following a compensatory pause showing a normal QRS (Fig. 1B). During the next two years, sinus beats showed either a normal QRS or right bundle-branch block—transient right bundle-branch block. When intraventricular conduction was normal during this period, an interpolated ventricular extrasystole was followed by one to seven sinus beats showing right bundle-branch block. On 27 February 1976, the sinus beats in leads I, II, III, aVR, aVL,

A 14/11/74

B 20/6/75

C 27/2/76

Fig. 1 In (A) (14/11/74), an interpolated ventricular extrasystole (beat 3 in both leads I and V1) caused the appearance of a right bundle-branch block configuration in the following sinus beat. In (B) (20/6/75), a right bundle-branch block pattern persisted during sinus rhythm, whereas the sinus beat terminating the compensatory pause (beat 3 in V1) after a ventricular extrasystole showed restoration of a normal QRS. In (C) (27/2/76), most sinus beats showed normal intraventricular conduction in lead I, where an interpolated ventricular extrasystole (beat 3) caused the appearance of right bundle-branch block in two subsequent sinus beats. Extrasystoles with compensatory pauses (beats 7 and 11) did not affect the following sinus QRS. In lead V1, right bundle-branch block persisted, and a ventricular extrasystole with a compensatory pause (beat 8) returned the QRS of the following sinus beat to normal. The sinus rate was slightly faster in V1 as compared with I. Note the almost identical QRS configuration of all the extrasystoles, with a left bundle-branch block pattern.

Fig. 2 Heart rate and intraventricular conduction in all the electrocardiograms recorded during the 13 years. *mth*, month; *y*, year; *VE*, ventricular extrasystoles. A solid triangle under an arrow indicates the appearance of right bundle-branch block in sinus beat(s) following an interpolated extrasystole. An open circle under an arrow shows restoration of a normal QRS pattern in sinus beats following a ventricular extrasystole with a compensatory pause. Those arrows unaccompanied by symbols indicate either extrasystoles with a compensatory pause in the presence of normal intraventricular conduction or interpolated extrasystoles in the presence of right bundle-branch block in sinus rhythm, so that no alterations in QRS of the following sinus beats occurred. Note the continuous extrasystolic activity from July 1974 to June 1976. The increased heart rates between October 1977 and June 1978 were caused by a slightly higher body temperature after a fracture of the femur. Persistence of right bundle-branch block after re-establishment of sinus bradycardia suggests permanent blockage of the bundle.

aVF, and V4, V5, V6 had a normal QRS, and interpolated ventricular extrasystoles produced right bundle-branch block in several following sinus beats, whereas an extrasystole with a compensatory pause in the presence of right bundle-branch block in leads V1, V2, and V3 restored the QRS to normal in one sinus beat (Fig. 1C).

From July 1976, these ventricular extrasystoles almost completely disappeared, and sinus beats occasionally showed transient right bundle-branch block. Finally, right bundle-branch block became permanent in June 1977, with all tracings in the subsequent three years showing this morphology. Fig. 2 summarises the entire course of these electrocardiographic abnormalities.

Discussion

The first noteworthy observation in this case is that

the intermittent right bundle-branch block was caused by interpolated ventricular extrasystoles and not by the usual acceleration of sinus rhythm.¹⁻³ To the authors' knowledge, only one similar case has previously been reported.³ When the QRS of a supraventricular beat becomes abnormal upon a significant shortening of the cycle length, however, aberrant conduction must be differentiated. Based on earlier observations on aberrant conduction of atrial extrasystoles,⁶ Watanabe and Nishimura⁷ proposed that the appearance of a bundle-branch block pattern in a supraventricular beat occurring later than the preceding U wave would favour the diagnosis of intermittent bundle-branch block. This is based on the premise that aberrant conduction results from physiological refractoriness of the intraventricular conducting system, whereas tachycardia-dependent bundle-branch block is caused by an abnormally prolonged refractory period in a damaged bundle-branch.⁸ Though the

above criterion is thus arbitrary, its application to Fig. 1 suggests intermittent right bundle-branch block as the likely diagnosis.

Second, in the present case, a slow progressive development of right bundle-branch block was documented by continuous observation. The various stages included, first, the appearance of a right bundle-branch block pattern in one (to seven) sinus beat(s) following an interpolated ventricular extrasystole, secondly the restoration of a normal sinus QRS after an extrasystole with a compensatory pause in the presence of right bundle-branch block, and thirdly persistence of right bundle-branch block in all sinus beats regardless of the heart rate. Such a clinical course suggests progression of an organic lesion involving the right bundle-branch system. It is interesting to ask how long it took for the conduction disturbance to mature into permanent block. If the appearance of the right bundle-branch block pattern in the sinus beat after an interpolated ventricular extrasystole in November 1974 is considered the first sign of abnormal intraventricular conduction, two and a half years elapsed before the right bundle-branch block became permanent in June 1977. Conversely, if we assume that certain pathological changes in the right bundle-branch were already present in November 1969, when acceleration of sinus rhythm was associated with transient, incomplete right bundle-branch block, the process took more than seven and a half years. Since repeated electrocardiographic recordings during the intervening five years never showed sinus rates faster than 62/min, and since the first interpolated extrasystole in July 1974 did not alter the following sinus QRS, it is difficult to determine which of the above two calculations is more accurate. Furthermore, it may be questioned whether the waxing and waning of the small s wave in lead I, beginning in April 1967, indicated a mild conduction disturbance in the right bundle-branch.

The third peculiarity in the present case is that extrasystoles of right ventricular origin occurred frequently when the right bundle-branch block was intermittent or transient, whereas they disappeared entirely after the block became permanent. Watanabe *et al.*⁵ earlier reported that, in the presence of intraventricular conduction disorders, the incidence of ventricular extrasystoles was significantly increased and that those extrasystoles tended to arise from the areas of the blocked bundle or fascicle. These observations suggested a close relation between the conduction disturbance and ectopic impulse formation, and hence, the role of re-entry in the genesis of extrasystoles. One may speculate, therefore, that in our present case, areas of unidirectional block and slow conduction were present in the Purkinje system at the time conduction in the right bundle-branch was still

unstable.

With reference to the occurrence of intermittent bundle-branch block associated with ventricular extrasystoles, Castellanos *et al.*⁹ reported a case of pseudo-left bundle-branch block produced by a concealed premature beat. In their case, an ectopic impulse arising in the left bundle-branch apparently depolarised only part of this bundle, left refractory tissue, and blocked the conduction of the next sinus impulse therein. Conversely, as the ectopic impulse of right ventricular origin in our case produced a manifest QRS, the impulse must have depolarised the entire ventricular mass, and probably the left bundle-branch system as well (perhaps in a retrograde fashion). These two cases would thus carry different implications. The case reported by Tokunaga *et al.*³ is also different from ours, as the interpolated ventricular extrasystole had a right bundle-branch configuration (suggesting a left ventricular origin), which caused the appearance of right bundle-branch block in the following sinus beat. Here, the right bundle-branch may be the last tissue to be depolarised by, and to repolarise after, the extrasystole, thus readily blocking the sinus impulse which closely follows the extrasystole even when the refractory period in this bundle is not abnormally prolonged.

Addendum

After this paper was submitted for publication, it was learned that the patient died of renal failure resulting from a cancer of the urinary bladder, and associated left heart failure, in June 1981. All his electrocardiograms during the last 13 months showed persistence of right bundle-branch block.

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