Case reports

British Heart Journal, 1977, **39**, 1386-1389

Bradycardia-dependent peri-infarction block

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A case is reported in which electrocardiographic signs appeared during the acute phase of myocardial infarction, which we have interpreted as indicating bradycardia-dependent peri-infarction block.

Bradycardia-dependent intraventricular block of the bundle-branch type (paradoxical bundle-branch block) is a recognised electrocardiographic finding (Massumi, 1968; Sarachek, 1970). Similarly, focal (Rosenbaum *et al.*, 1968) or intramyocardial (Hecht and Kossmann, 1973) block, during the acute phase of myocardial infarction (peri-infarction block), unrelated to rate, has been previously documented (First *et al.*, 1950; Shadaksharappa *et al.*, 1968). Tachycardia-related focal block has also been reported (Gambetta and Childers, 1973). However, to our knowledge a bradycardia-dependent peri-infarction block has not yet been reported, and it constitutes the subject of this paper.

Case report

A 60-year-old male patient was first seen in June 1970 with typical antero-septal myocardial infarction. He made an uneventful recovery but remained in clinical class II, with physical signs of ventricular asynergy. For over a year the ST segment was still displaced (Fig. 1A) in leads I, aVL, and V2 to V6, though he was still in sinus rhythm at 70 bpm, with a PR of 0.16 s, QRS of 0.09 s, and a frontal plane AQRS of 90°. In October 1971 he had an episode of weakness and diaphoresis, without chest pain, lasting 2 hours. Three days later he presented with pulmonary oedema and the electrocardiogram showed sinus tachycardia at 102 bpm, PR 0.21 s, left atrial enlargement, widening of the QRS to 0.11 s, and no changes in either the frontal plane AQRS or the initial vectors (Fig. 1B). However, the late vectors were changed in III, aVF and aVL, producing a W-shaped complex in the last lead. Left ventricular failure persisted and 6 days after the episode of weakness the electrocardiogram showed an atrial rate of 120 bpm and a ventricular rate of 60 bpm, with a 2:1 atrioventricular block and a bizarre QRS configuration (Fig. 1C).

The latter showed a slowing of the ventricular depolarisation process after the first 0.05 to 0.06 s, lasting 0.08 to 0.09 s, and resulting in a wide QRS of 0.18 s. The erythrocyte sedimentation rate and white blood cells were raised on the same day but no increase in enzyme level was noted. On the following day both the 2:1 atrioventricular block and the peri-infarction block disappeared (Fig. 1D).

An electrocardiogram obtained on the afternoon of the same day (14 October 1971) showed a sinus rate of 109 bpm (PP interval of 0.55 s), a 3:2 atrioventricular block of the Wenckebach type (4:3 in V6), and a ventricular response with the following characteristics (Fig. 2): alternating cycle lengths of 1.02 and 0.63 s; the long pauses being terminated by wide QRS of the type described above, and short pauses terminated by normal duration QRS. It can be seen from this tracing that the early vectors are identical for both types of complexes, mainly in right praecordial leads, while there is an obvious delay in activation after those early vectors.

Discussion

The clinical picture, laboratory findings, and electrocardiographic abnormalities are suggestive of a new infarct in the lateral wall of the left ventricle, probably in an area near the previous infarction. On the sixth day a transient second degree atrioventricular block associated with intraventricular conduction delay was noted. This conduction defect shows a different morphology than the usual defect found in either total or partial bundle-branch block, but retains the characteristic features of peri-infarction block (First *et al.*, 1950). This case also presents several clinical similarities with the cases reported by Shadaksharappa *et al.* (1968): onset in the first week of a myocardial infarction, being transient and more frequent during a second infarction.

Fig. 1 (A) Electrocardiogram a year after the first infarction. (B) Acute phase of second infarction before appearance of 2:1 atrioventricular block. (C) Acute phase of second infarction: appearance of 2:1 atrioventricular block and peri-infarction block. (D) Acute phase of the second infarction after atrioventricular block.

Fig. 2 Bradycardia-dependent peri-infarction block recorded during 3:2 atrioventricular block.

A change from 2:1 to 3:2 atrioventricular block revealed the most interesting finding: the intra-ventricular conduction delay became intermittent, appearing always after the long pauses and disappearing after the short ones, best seen during the 4:3 block recorded in V6. This rate-dependent peri-infarction block behaves like the paradoxical bundle-branch block which sometimes accompanies a variable atrioventricular block during the course of a myocardial infarction (Puech and Grolleau, 1972).

It might be speculated whether the block is located in the distal Purkinje system or in the myocardial fibres. The former will most probably alter the initial vector forces, like the fascicular or the focal septal block described by Gambetta and Childers (1973). Its occurrence in the latter part of the QRS (0.05 s after the onset) and its duration (0.08 to 0.09 s) suggest a conduction delay within the myocardial fibres affected by the ischaemic insult. Its appearance only during the prolonged RR intervals explains the name of paradoxical or bradycardia-dependent peri-infarction block.

The early work of Singer *et al.* (1967) was followed by numerous reports of slow rate-related arrhythmias, i.e. paradoxical bundle-branch block (Massumi, 1968; Sarachek, 1970), aberrant late nodal beats (Sarachek, 1970), normalisation of WPW syndrome with slow rhythms (Massumi and Vera, 1971), paroxysmal atrioventricular blocks (Coulmel *et al.*, 1971; Rosenbaum *et al.*, 1973), and the underlying mechanism of other types of arrhythmias like intermittent parasystole (Cohen *et al.*, 1973), and intermittent bigeminy (Levy *et al.*, 1975).

According to these reports, several factors might increase the slope of phase 4 of the Purkinje system and, consequently, a late stimulus could find the diastolic potential less negative, producing an action potential of diminished amplitude with decreased dV/dT of phase 0, resulting in conduction disturbance. A similar situation is produced when a premature stimulus falls on the early part of phase 3, before complete repolarisation is reached. Both disturbances are voltage dependent (Singer *et al.*, 1967; Singer and TenEick, 1971), and the conduction delays associated with rapid or slow rhythms have been called phase-3 and phase-4 blocks (Rosenbaum *et al.*, 1973), though some authors prefer to call them tachycardia- or bradycardia-dependent blocks (El-Sherif, 1972).

It appears that the critical factor is an increase in extracellular potassium following the cell necrosis during the course of a myocardial infarction (Harris, 1966; Sutton and Davies, 1968; Lazzara and Sherlag, 1972). It is difficult to explain a tachy-

cardia-dependent block, in a disease state, only on the basis of voltage changes of phase 3; other factors, such as the time relation and the sodium pump, a decrease of the resting potential, or cell membrane responsiveness, are probably involved (Fisch *et al.*, 1973; El-Sherif *et al.*, 1974; Neuss *et al.*, 1974). In bradycardia-dependent block, besides the slope of phase 4, other factors such as hypopolarisation, increase in threshold potential, and alterations of cell membrane responsiveness might play a role (Singer *et al.*, 1967; El-Sherif *et al.*, 1974; Neuss *et al.*, 1974; Rosenbaum *et al.*, 1974; Kretz *et al.*, 1975).

Under normal conditions myocardial cells do not have automaticity, but during an ischaemic insult they may suffer electrophysiological changes, such as spontaneous diastolic depolarisation during phase 4 (Solberg *et al.*, 1972), which, if associated with other factors, will cause the block; on the other hand, if we accept these changes at the myocardial cell level, the case reported here would be an instance of pacemaker activity of these cells.

The combination of a variable atrioventricular block and intramyocardial block make it possible to speculate on a common aetiological factor, such as the increase in extracellular potassium.

Lastly, why is this a rare phenomenon? If we consider the frequency of peri-infarction block (39%—Shadaksharappa *et al.*, 1968) and also of atrioventricular nodal block during infarction (3%—Stock and Macken, 1968), at least 1 per cent of the infarction cases would meet the necessary conditions of heart rate and cell necrosis for its appearance. The fact that paradoxical block is less frequently seen as peri-infarction block than as bundle-branch block, makes us think that the conditions necessary for the occurrence of the first are more difficult to meet. In our case all these rare circumstances were present: old infarction, acute infarction in a nearby area, variable atrioventricular block, and patient survival.

I thank Dr. R. Uceda for the referral of the patient and Dr. F. Duclos and Dr. C. Piñero for their assistance in writing this paper.

References

- Cohen, H., Langendorf, R., and Pick, A. (1973). Intermittent parasystole—Mechanism of protection. *Circulation*, **48**, 761–774.
- Coulmel, P., Fabiato, A., Waynberger, M., Motte, G., Slama, R., and Bouvrain, Y. (1971). Bradycardia-dependent atrioventricular block. Report of two cases of AV block elicited by premature beats. *Journal of Electrocardiology*, **4**, 168–177.
- El-Sherif, N. (1972). Tachycardia-dependent versus bradycardia-dependent intermittent bundle-branch block. *British Heart Journal*, **34**, 167–176.

- El-Sherif, N., Scherlag, B. J., Lazzara, R., and Samet, Ph. (1974). Pathophysiology of tachycardia- and bradycardia-dependent block in the canine proximal His-Purkinje system after acute myocardial ischemia. *American Journal of Cardiology*, **33**, 529-540.
- First, S. R., Bayley, R. H., and Bedford, D. R. (1950). Peri-infarction block; Electrocardiographic abnormality occasionally resembling bundle-branch block and local ventricular block of other types. *Circulation*, **2**, 31-36.
- Fisch, Ch., Zipes, D. P., and McHenry, P. L. (1973). Rate dependent aberrancy. *Circulation*, **48**, 714-724.
- Gambetta, M., and Childers, R. W. (1973). Rate-dependent right precordial Q-waves: 'Septal focal block'. *American Journal of Cardiology*, **32**, 196-201.
- Harris, A. S. (1966). Potassium and experimental coronary occlusion. *American Heart Journal*, **71**, 797-802.
- Hecht, H. H., and Kossmann, C. E. (1973). Atrioventricular and intraventricular conduction. Revised nomenclature and concepts. *American Journal of Cardiology*, **31**, 232-244.
- Kretz, A., Da Ruos, H. O., and Leguizamon Palumbo, J. R. (1975). Delay and block of cardiac impulse caused by enhanced phase-4 depolarization in the His-Purkinje system. *British Heart Journal*, **37**, 136-149.
- Lazzara, R., and Scherlag, B. J. (1972). Washout of arrhythmogenic substance liberated after coronary occlusion. *Circulation*, **45-46**, Suppl. II, 116.
- Levy, M. N., Adler, D. S., and Levy, J. R. (1975). Three variants of concealed bigeminy. *Circulation*, **51**, 646-655.
- Massumi, R. A. (1968). Bradycardia-dependent bundle-branch block. A critique and proposed criteria. *Circulation*, **38**, 1066-1073.
- Massumi, R. A., and Vera, Z. (1971). Patterns and mechanisms of QRS normalization in patients with Wolff-Parkinson-White syndrome. *American Journal of Cardiology*, **28**, 541-554.
- Neuss, H., Thormann, J., and Schlepper, M. (1974). Electrophysiological findings in frequency-dependent left bundle-branch block. *British Heart Journal*, **36**, 888-898.
- Puech, P., and Grolleau, R. (1972). *L'Activité du Faisceau de His Normale et Pathologique*. Editions Sandoz, Paris.
- Rosenbaum, M. B., Elizari, M. V., Chiale, P., Levi, R. J., Nau, G. J., Halpern, M. S., Lazzari, J. O., and Novakovsky, A. (1974). Relationships between increased automaticity and depressed conduction in the main intraventricular conducting fascicles of the human and canine heart. *Circulation*, **49**, 818-828.
- Rosenbaum, M. B., Elizari, M. V., and Lazzari, J. O. (1968). *Los Hemibloques*. Paidós, Buenos Aires.
- Rosenbaum, M. B., Elizari, M. V., Lazzari, J. O., Halpern, M. S., Nau, G. J., and Levi, R. I. (1973). The mechanism of intermittent bundle-branch block: relationship to prolonged recovery, hypopolarization and spontaneous diastolic depolarization. *Chest*, **63**, 666-677.
- Sarachek, N. S. (1970). Bradycardia-dependent bundle branch block: Relation to supernormal conduction and phase 4 depolarization. *American Journal of Cardiology*, **25**, 727-729.
- Shadaksharappa, K. S., Kalbfleisch, J. M., Conrad, L. L., and Sarkar, N. K. (1968). Recognition and significance of intraventricular block due to myocardial infarction (peri-infarction block). *Circulation*, **37**, 20-26.
- Singer, D. H., Lazzara, R., and Hoffman, B. F. (1967). Interrelationships between automaticity and conduction in Purkinje fibers. *Circulation Research*, **21**, 537-558.
- Singer, D. H., and TenEick, R. E. (1971). Aberrancy: electrophysiologic aspects. *American Journal of Cardiology*, **28**, 381-401.
- Solberg, L., TenEick, R., and Singer, D. (1972). Electrophysiological basis of arrhythmia in infarcted ventricle. *Circulation*, **45-46**, Suppl II, 116.
- Stock, R. J., and Macken, D. L. (1968). Observations on heart block during continuous electrocardiographic monitoring in myocardial infarction. *Circulation*, **38**, 993-1005.
- Sutton, R., and Davies, M. (1968). The conduction system in acute myocardial infarction complicated by heart block. *Circulation*, **38**, 987-992.

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