Case reports

British Heart Journal, 1976, **38**, 201-203.

Wenckebach phenomenon in the exit area from a transvenous pacing electrode

Thomas Peter, Richard Harper, David Hunt, and Graeme Sloman

From the Cardiac Laboratory, Royal Melbourne Hospital, Victoria, Australia

An unusual type of exit block from a transvenous pacing electrode was recorded in a 63-year-old man with an acute inferior infarct and cardiogenic shock. The pacemaker artefact to QRS interval increased gradually till there was loss of capture. A gradual change from 1:1 pacing rhythm to 4:3 and 3:2 Wenckebach cycles was recorded. This was followed by a fixed 2:1 pacemaker artefact to QRS block.

The Wenckebach phenomenon is commonly recognized as a type of second degree atrioventricular block (Schamroth, 1967). Since the advent of His bundle electrograms this has been demonstrated to occur in the various parts of atrioventricular conduction (Narula and Samet, 1970; Castellanos *et al.*, 1972). Examples of the Wenckebach phenomenon have also been recorded in the bundle-branches (Friedberg and Schamroth, 1969; Franchi, Morace, and Fantini, 1973). The occurrence of the phenomenon in the exit area from a pacemaker is well demonstrated by sinoatrial Wenckebach phenomenon (Schamroth and Dove, 1966). The purpose of this paper is to report an unusual example of the Wenckebach phenomenon occurring in the exit area of an impulse from a transvenous pacing electrode.

Case report

A 63-year-old man was admitted to the coronary care unit at the Royal Melbourne Hospital with a history of transient loss of consciousness and severe retrosternal chest pain. Electrocardiogram on admission showed an acute inferior infarction and complete atrioventricular block, with a nodal rate of 48/min. A temporary bipolar pacing electrode was positioned in the right ventricular apex under fluoroscopy and demand pacing started at a rate of 90/min. The patient's clinical condition deteriorated, he became hypotensive, and finally succumbed in cardiogenic shock.

The Figure shows a continuous lead II of the electrocardiogram recorded in this patient 2 hours before death. All the QRS complexes are identical, and preceded by a pacing artefact. The pacing artefact can be

seen in the entire length of the electrocardiogram uninterrupted at a rate of 90/min. The top panel shows regular 1:1 pacing with a first degree of exit block up to R₈. The pacemaker artefact to QRS interval (exit time) gradually increases up to R₈ and the subsequent pacemaker discharge fails to activate the myocardium.

R₁₀, R₁₁, and R₁₂, and R₁₅, R₁₆, and R₁₇ clearly show cycles of 4:3 Wenckebach block occurring between the pacing electrode and the myocardium. The interval between the pacing artefact (P) and the QRS in the beginning of the cycles (PR₁₀ and PR₁₅) is 80 ms and this exit time increases by the end of the cycle (PR₁₂ and PR₁₇) to 120 ms, and is followed by total failure of conduction. R₁₃ and R₁₄, and R₁₈ and R₁₉ are examples of 3:2 Wenckebach cycles in which the exit time increases from 80 ms to 160 ms and the third pacemaker artefact fails to capture the ventricle. Following R₂₀ the electrocardiogram shows a regular pattern of 2:1 exit block.

Discussion

Exit block around a pacing electrode has been well recognized (Preston *et al.*, 1966). To our knowledge, Wenckebach phenomenon at this area has not been demonstrated before. This case, therefore, provides an example to delineate the characteristics of a Wenckebach block around an intrinsic pacemaker focus in the ventricle (Schamroth, 1971). The mechanism of this progressive deterioration of conduction is most probably a form of decremental conduction (Krikler, 1974). The degree of conductivity of the exit area is depressed gradually, the conduction changes from 1:1 to 4:3 and 3:2 Wenckebach cycles, and finally settles as a regular 2:1 response.

FIG. Lead II rhythm strip. Panels A, B, C, and D are continuous. See text for description.

The term 'decremental conduction', however, usually implies a somewhat different phenomenon in electrophysiology (Watanabe and Dreifus, 1968). Both the action potential amplitude and the rate of depolarization are progressively decreased from cell to cell. As a result of this, successive stimuli become weaker and less effective in the course of transmission and finally the impulse fades out when the integrated stimulus strength becomes insufficient to cause a propagated response in more distal and still excitable fibres. Though decremental conduction is commonly seen in the sinoatrial and atrio-ventricular nodes, it has also been described in the Purkinje system (Hoffman, 1966).

Gradual prolongation of exit time from a pacemaker potential was described by Pick; however, cyclical pattern of Wenckebach phenomenon was not recorded in his case (Pick, 1973). It is probable that the prolonged low output state of the patient with consequent myocardial ischaemia and electrolyte imbalance may have contributed to the genesis of this unusual form of exit block. Cellular hypoxia and disturbances of intracellular potassium concentrations have been shown to delay impulse conduction and cause Wenckebach type of block between Purkinje fibres and contractile myocardium (Alanis, Benitez, and Pilar, 1961; Greenspan, Anderson, and Fisch, 1971).

References

- Alanis, J., Benitez, D., and Pilar, G. (1961). A functional discontinuity between the Purkinje and ventricular muscle cells. *Acta Physiologica Latino-Americano*, **11**, 171.
- Castellanos, A., Iyengar, R., Agha, A. S., and Castillo, C. A. (1972). Wenckebach phenomenon within the atria. *British Heart Journal*, **34**, 1121.
- Franchi, F., Morace, G., and Fantini, F. (1973). Wenckebach phenomenon in the bundle-branch system. *British Heart Journal*, **35**, 590.
- Friedberg, H. D., and Schamroth, L. (1969). The Wenckebach phenomenon in left bundle branch block. *American Journal of Cardiology*, **24**, 591.
- Greenspan, K., Anderson, G. J., and Fisch, C. (1971). Electrophysiologic correlate of exit block. *American Journal of Cardiology*, **28**, 197.
- Hoffman, B. F. (1966). The electrophysiology of heart muscle and the genesis of arrhythmias. In *Mechanism and Therapy of Cardiac Arrhythmias*, p. 27. Ed. by L. S. Dreifus and W. Likoff. Grune and Stratton, New York.
- Krikler, D. M. (1974). Cardiac electrophysiology. *Lancet*, **1**, 851.
- Narula, O. S., and Samet, P. (1970). Wenckebach and Mobitz type II AV block due to block within the His bundle and bundle branches. *Circulation*, **41**, 947.
- Pick, A. (1973). Mechanism of cardiac arrhythmias: from hypothesis to physiologic fact. *American Heart Journal*, **86**, 249.
- Preston, T. A., Judge, R. D., Bowers, D. L., and Morris, J. D. (1966). Measurement of pacemaker performance. *American Heart Journal*, **71**, 92.
- Schamroth, L. (1967). The theory and mechanism of the Wenckebach phenomenon. *South African Medical Journal*, **41**, 827.

Schamroth, L. (1971). *The Disorders of Cardiac Rhythm*, p. 462. Blackwell Scientific Publications, Oxford.

Schamroth, L., and Dove, E. (1966). The Wenckebach phenomenon in sinoatrial block. *British Heart Journal*, 28, 350.

Watanabe, Y., and Dreifus, L. S. (1968). Newer concepts in

the genesis of cardiac arrhythmias. *American Heart Journal*, 76, 114.

Requests for reprints to Dr. Graeme Sloman, Cardiac Laboratory, Royal Melbourne Hospital, Victoria, Australia 3050.

Notice

Cardiovascular Nuclear Medicine: Clinical Applications

A symposium will be heard at The Johns Hopkins University School of Medicine on Cardiovascular Nuclear Medicine, on 11 and 12 March 1976. Further particulars can be obtained by writing to Julia C. Burbridge, Office of Continuing Education, Turner Auditorium Building, The Johns Hopkins University, School of Medicine, 720 Rutland Avenue, Baltimore, Maryland 21205, U.S.A.