The Wenckebach phenomenon between electric pacemaker and ventricle

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Two cases which exhibited a Wenckebach form of exit conduction between an electrical pacemaker and the ventricular myocardium are presented. This manifests with increasing latency (stimulus to QRS intervals) and ultimate stimulus failure (block). The cycle then repeats itself. The QRS duration also increases progressively within each cycle. The phenomenon connotes an adverse prognosis. Several postulates regarding the mechanism are discussed.

The Wenckebach phenomenon has been observed in many locations of cardiac tissue (Decherd and Ruskin, 1946; Anderson, Greenspan, and Bandura, 1970; Wennemark and Bandura, 1974; Friedberg and Schamroth, 1969; Craneffeld, Klein, and Hoffman, 1971; Anderson, Greenspan, and Fisch, 1972; Rosen, 1971), and theoretically can occur in any region which conducts an activating cardiac impulse. This report concerns two instances of the Wenckebach phenomenon which occurred in the

transmission of the impulse between an electrical pacemaker and the ventricular tissue. The phenomenon occurred in two seriously ill patients and presaged death in both cases. It may thus represent an important adverse prognostic sign.

Case reports

Case 1

The electrocardiograms (Fig. 1 and 2) were recorded from a 61-year-old man who was admitted to hospital in shock and cardiac arrest. He had

FIG. 1 Electrocardiograms (standard lead II, recorded at a paper speed of 25 mm/s showing: Strip A. Two paced beats after which the pacemaker is turned off. This is followed by a conducted beat showing prolonged PR interval and widened QRS complex, consistent with a serum potassium of 7 mmol/l. This is followed by six further paced beats. Strip B. Electrical pacing with 2:1 pacemaker-exit block. Strip C. Cardiac arrest with cardiac massage applied during the interval between arrows. This re-establishes 1:1 conduction. Strips D-G. These illustrate decreasing amplitude which results in decreasing QRS amplitude and progressive widening of the QRS complex with late peaking and eventual complete inexcitability.

FIG. 2 Electrocardiograms (standard lead II, strips A and B form a continuous recording; strips C and D form a continuous recording) showing: The Wenckebach form of pacemaker-exit conduction with varying conduction ratios. See text.

chronic lung disease, and had also had an inferior wall myocardial infarction 8 years previously. The electrocardiograms were typical of hyperkalaemia, with wide and bizarre QRS complexes (analogous to the third QRS complex in strip A of Fig. 1). After resuscitation, transvenous ventricular bipolar pacing was performed (Medtronic demand pacemaker model) because of periodic complete AV block. Pacing was initially effective (strip A of Fig. 1) but soon resulted in repetitive sequences of increasing delay and second degree exit block between stimulus artefact and ventricular depolarization. Strip B of Fig. 1 illustrates sequences of 2:1 pacemaker-exit block. The first stimulus of each sequence is followed by a broad QRS complex which is in turn followed by a second deflection of the baseline. This deflection probably represents a late T wave (much less likely, though possible, a second depolarization resulting from re-entry or re-excitation). The second stimulus of the sequence occurs in the midst of this deflection and is ineffective.

Strip C of Fig. 1 illustrates the effect of cardiac massage and intracardiac catecholamine administration. These procedures re-established 1:1 conduction to the ventricle accompanied for a short time by mechanical response, only to be followed by the same electrical alterations and mechanical unresponsiveness (described below). Cardiac massage repeatedly led to the same sequence of events: return of 1:1 electrical activity and effective mechanical response, onset of electrical alterations and loss of mechanical response. Changing the output of the pacemaker conspicuously altered the QRS complex causing a decrease in amplitude and splaying out of the QRS complexes with lower electrical output (strips D and F of Fig. 1). The patient died in electromechanical dissociation in spite of all attempts, including treatment for hyperkalaemia. Strip G of Fig. 1 shows complete electrical unresponsiveness.

Tracings A and B of Fig. 2 are continuous; tracings C and D are also continuous and are from a later part of the same tracing. Strip A of Fig. 2 shows the pacemaker-ventricular Wenckebach phenomenon. The first six stimuli are followed by QRS complexes, the peaks of which (taken as a convenient reference point) are gradually and progressively more delayed. The interval between stimulus artefact and QRS complex is 40 ms and 80 ms, for the first and second stimuli, and increases to 120 ms for the sixth stimulus. The seventh stimulus falls on the T wave deflection noted previously, and fails to depolarize the ventricle. The same sequence is repeatedly observed in strips A-D, with variable conduction ratios 7:6 and 5:4 in A and C; 4:3 in B, 3:2 in D.

Case 2

The electrocardiograms (Fig. 3 and 4) were recorded from a 61-year-old woman who was admitted to hospital with acute anterior wall myocardial infarction. Two days later, and after several episodes of ventricular tachycardia, first degree AV block appeared, with right bundle-branch block and left anterior hemiblock. This was followed shortly thereafter by complete AV block and cardiac arrest. Cardiac resuscitation and intravenous bipolar ventricular pacing were successfully carried out. After initial recovery from the cardiac arrest, the patient nevertheless remained in cardiogenic shock and congestive heart failure. Physical examination indicated a large dyskinetic ventricular area and mitral regurgitation. As early as the second hospital day, at the time of pacemaker insertion, first degree block was noted between the pacemaker and the ventricle, as reflected by a stimulus-QRS interval of 100 ms. The normal value probably does not exceed 40 ms (Brooks *et al.*, 1955; Rogel and Hasin, 1974). The tracings shown in Fig. 3 and 4 were recorded on the next—the third hospital—day. These tracings were selected from a long recording

FIG. 3 Electrocardiogram (tracings of selected segments of a continuous recording) show 2:1, 5:4, 10:9, 11:10 Wenckebach pacemaker ventricular cycles. Numbers represent measurements in ms for the two beats marked by circles. Note that the last RR interval of each series is shorter than the first. See text.

of standard lead II. The first eight stimuli in the top strip of Fig. 3 were conducted with a 2:1 pacemaker-ventricular exit block. The last four stimuli of this strip all depolarized the ventricles with a Wenckebach form of exit block. The SQ intervals increased progressively from 120 ms to 160 ms, to be followed by a blocked stimulus (not shown). The blocked stimulus at the beginning of the second strip was followed by a Wenckebach sequence with a 10:9 conduction ratio. The SQ intervals increased progressively from 120 ms to 180 ms to be followed by a blocked stimulus. The

third strip once again reflected 2:1 pacemaker-ventricular exit block. The last strip reflected a 11:10 Wenckebach conduction cycle. The SQ intervals increased progressively from 120 to 170 ms to be followed by a blocked stimulus. The duration of the QRS complexes also increased slightly from 160 to 180 ms (complexes labelled with a circle in the bottom strip of Fig. 3). The first RR interval of each sequence (strips 2 and 4) is the longest, with a subsequent shortening. Fig. 4 shows the dependence of the SQ delay and QRS duration on rate and current strength. Strips A reflect the effect of

FIG. 4 Electrocardiograms illustrating the effect of (A) changing rate, and (B) varying amperage on latency and QRS width. See text. Measurements in tracings are in ms and apply to the first and last complexes of each strip.

rate: as the rate is increased, both the S-Q intervals and QRS duration increase. Strips B reflect the effect of variable current strength: as amperage was decreased (from top to bottom strips), the SQ interval increased from 160 ms (top strip) to 200 ms (middle strip). The last stimulus in the middle strip is ineffective. A maximum amperage of 20 mA (top strip in B of Fig. 4) resulted in a 1:1 response with first degree exit block. Several attempts were made to modify the electrical behaviour by repositioning the pacemaker electrode tip, but the same phenomenon was observed repeatedly. The patient died in intractable ventricular failure on the 19th day in hospital, two weeks after the onset of the Wenckebach phenomenon. Necropsy showed extensive myocardial necrosis and fibrosis of the anterior and lateral walls, the apex, septum and papillary muscles of the left ventricle, and severe diffuse coronary artery disease.

Patient studies

Attempts at altering latency (SQ interval) and reproducing the Wenckebach phenomenon were carried out in 5 paced patients with chronic AV block and presumably normal myocardium. In all these patients, excitatory threshold was less than 0.5 mA, with bipolar stimulating electrodes similar to those used in the two previously described patients. The current strength was decreased in a stepwise fashion till excitation threshold was reached, while the electrocardiogram was recorded at 50 mm/s speed to obtain accurate measurement of the SQ interval. In none of the patients was there a significant increase in the SQ interval with decreasing current strength. At threshold current strength, occasional stimuli successfully depolarized the ventricles. In none of these was the SQ interval significantly different from those obtained at greater current strength. In no instance was a Wenckebach phenomenon observed. In one patient the configuration of the QRS complex was altered but the SQ interval remained constant.

Discussion

Significance of the SQ duration (latency)

The sequences of gradually increasing stimulus-ventricular delay (increasing SQ interval) followed by a blocked stimulus reflect a Wenckebach form of pacemaker exit block. The classical Wenckebach progressive shortening of the RR intervals (an acceleration of the tissue activation distal to the block) is also evident in these cases.

The SQ interval is here referred to as *latency*.

This comprises (a) the time required for depolarization of the tissue immediately in contact with the stimulating electrodes (true latency) which is dependent on the current strength of the stimulus, (b) the time required for the local electrical response to break out of the vicinity of the stimulating electrodes, and (c) the time required to depolarize sufficient myocardial mass to cause the onset of the QRS complex. Normal values for the SQ interval are difficult to find in published reports. Our observations lead us to believe that it is usually not more than 40 ms. Similar values were seen experimentally by Brooks and associates (1955), and Rogel and Hasin (1974). Increased SQ duration indicates an increase in one or more of the aforementioned time factors, and occurs as a result of severe myocardial depression. This was the result predominantly of severe hyperkalaemia in the first case, and to diffuse myocardial disease in the second.

Dependence of latency on current amplitude is well known (Brooks *et al.*, 1955; Hoffman and Cranefield, 1960), and is clearly documented in both these cases. Threshold stimuli excite surrounding myocardium more slowly than suprathreshold stimuli. Very strong suprathreshold stimuli can also directly stimulate a larger area around the electrodes than threshold stimuli. It is thus the *distance* which the propagated impulse has to travel which is decreased with suprathreshold stimuli.

Several theories have been postulated to explain the mechanisms underlying classic Wenckebach periodicity which has not, as yet, been completely elucidated (Wennemark and Bandura, 1974; Cranefield *et al.*, 1971; Anderson *et al.*, 1972; Rosenblueth, 1958; Watanabe and Dreifus, 1967; Damato *et al.*, 1972). The mechanism of a pacemaker-ventricular Wenckebach phenomenon is also uncertain but could possibly be explained on the basis of one or more of the following factors.

- (1) The phenomenon could be the result of a gradually increasing block around the electrode site with each successive beat, a phenomenon similar to that seen in the classic form of AV nodal Wenckebach periodicity.
- (2) The phenomenon could be the result of a gradual decrease in the effectiveness of each succeeding stimulus to break out of the immediate electrode area into surrounding myocardium. Stimuli are initially suprathreshold, but become successively less effective, and, therefore, threshold and eventually subthreshold. As a result, a progressively smaller area of myocardium is directly excited with each successive stimulus. More and more myocardial

mass is thus dependent on secondary *propagated* excitation and less and less on direct *stimulation*. This difference may be negligible in healthy myocardial tissue. In depressed myocardium, however, in which propagated excitation may proceed in a very non-homogeneous manner, this could result in variable delays with pronounced differences in the SQ interval.

- (3) The phenomenon could possibly result from time-dependent (rather than voltage-dependent) refractoriness, extending well beyond the end of the T wave so that succeeding impulses occur progressively earlier in the refractory period. The Wenckebach phenomenon in Case 1 represents a relatively simple instance in which there is gradual encroachment of the refractory period of the gradually delayed ventricular depolarization upon the time of arrival of the succeeding stimulus as postulated by Rosenblueth (1958).

Significance of varying QRS duration

The duration of the QRS complex also increases within each Wenckebach cycle, as well as with decreased amperage and increased rate of stimulation, as seen in Fig. 1, 3, and 4. This is similar to bundle-branch Wenckebach periodicity. This increase could again result from (a) a time increase in intraventricular conduction time, or (b) a gradual decrease in the size of initial directly depolarized ventricular mass by the electrical stimulus, with greater reliance on non-homogeneous propagated excitation through depressed fibres.

It is thus evident that Wenckebach periodicity of both SQ intervals and QRS duration is the end product of very intricate, interrelated disturbances in refractoriness, responsiveness, latency, conduction characteristics, amplitude of electrotonic spread, and threshold potential in depressed fibres. Depression of the myocardium is undoubtedly a *sine qua non* for an interventricular Wenckebach phenomenon. Our attempts at recording it in patients with chronic complete AV block but normal or relatively normal myocardium have been unsuccessful. It has to our knowledge never been observed with normal or relatively normal myocardium. On the other hand, 'first degree' pacemaker to myocardium exit block has been observed

by us in a patient with extremely severe congestive heart failure and the sick sinus syndrome, and in another two patients with severe hyperkalaemia. Both cases presented here indicate severe myocardial dysfunction, and in one, the appearance of the Wenckebach phenomenon anticipated death by 15 days, suggesting that it may be an additional, useful prognostic sign in the management of acute cardiac emergencies.

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