CASE REPORT

Mechanism of QRS electrical alternans

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Abstract

In patients with latent dual atrioventricular nodal pathways a 2:1 ventriculoatrial block often occurs during ventricular pacing and is generally associated with the concomitant appearance of QRS alternans. This type of QRS alternans is related to retrograde conduction, and a concealed retrograde conduction in the His Purkinje system could explain the QRS alternans. A case that confirms the hypothesis that electrical alternans is secondary to a 2:1 block in the activation of some part of the ventricles is reported.

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Electrical alternans of the QRS complex is an electrocardiographic phenomenon seen in various clinical situations including supraventricular and ventricular tachycardias.¹⁻⁵ The mechanisms of alternans are unclear except in a few clinical circumstances such as the positional changes in the heart that underlie the electrical changes seen in patients with pericardial effusion.^{2,6} Several other potential and

controversial mechanisms have been proposed to explain electrical alternans.

We report a case of alternation of the amplitude and morphology of the QRS complex seen during a paced rhythm that throws light on this electrophysiological mechanism.

Ventricular pacing at incremental rate is routinely performed in our institution during electrophysiological studies and the protocol has been reported elsewhere.⁷ Sinoatrial and atrioventricular conduction are studied by atrial pacing and programmed atrial stimulation with one and two extrastimuli delivered in sinus rhythm and at a paced rhythm (600 ms, 400 ms).⁸ Ventricular pacing is then performed at a progressively incremental rate at the apex of right the ventricle.⁷

Among 950 electrophysiological studies, we found 85 patients with signs of the latent presence of two separate atrioventricular (AV) nodal pathways with fast and slow conduction: there was an increase of > 50 ms in the H1H2 or A2H2 intervals in response to a 10 ms decrement in the A1A2 interval during introduction of premature atrial extrastimuli.⁹⁻¹¹ In 65 of these patients a 2:1 or Möbitz II retrograde ventriculoatrial block occurred during ventricular pacing at an incremental rate. In 25 patients the appearance of the retrograde block was associated with the transient appearance of QRS alternans, which disappeared at a higher rate of ventricular pacing. The same phenomenon was observed at the same rate when pacing was performed at the right ventricular infundibulum. The QRS alternans was not seen in patients with a classic Wenckebach retrograde block.

Case report

An electrophysiological study was performed because of unexplained dizziness/syncope in a 60 year old patient with a prosthetic mitral valve. Sinoatrial and atrioventricular conduction were normal: corrected sinus node recovery time = 300 ms, AH interval = 80 ms, HV interval = 40 ms, and the rate at which second degree AV block occurred during atrial pacing at incremental rate = 150 beats/min. A single premature atrial extrastimulus revealed the existence of a latent double nodal pathway: a 10 ms shortening of the atrial extrastimulus provoked an abrupt increase in the AH inter-

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Figure 1 Ventricular pacing at fixed rates at the apex of the right ventricle showing simultaneous QRS alternans and a 2:1 ventriculoatrial block (25 mm/s).

Figure 2 Hypothesis to explain occurrence of QRS alternans. The diagram on the left shows 1/1 atrial retrograde conduction; that on the right shows the mechanism postulated to explain QRS alternans: concealed anterograde conduction in the left bundle branch and activation of the ventricle only by right bundle branch, producing QRS alternans and an LBBB block on the electrocardiogram.

val (100 ms) followed by an echo beat. Moreover there was increased atrial vulnerability and fragmented atrial activity during shortening of premature atrial extrastimulus and the induction of a sustained (three minutes) atrial fibrillation with a double atrial extrastimulus.

Then ventricular pacing was performed at incremental rate at the apex of the right ventricle. There was an 1/1 atrial retrograde conduction up to the rate of 140 beats/min and then an abrupt 2:1 ventriculoatrial (VA) block (fig 1). Simultaneously the ventricular rhythm paced at constant rate (150 beats/min) exhibited a QRS alternans that was seen in all leads except lead I (fig 1). The QRS alternans was clearly related to VA block: the QRS morphology was similar to the initial paced rhythm only when atrial retrograde conduction was absent. The presence of an atrial retrograde conduction modified the QRS morphology producing a left bundle branch block pattern. The cycle length of the paced rhythm appeared to alternate, particularly in lead V6 but in reality all complexes were paced at a constant rate: the spike was clearly visible just before the QRS complex in lead III. The level of retrograde block was inside the AV node because there was no visible retrograde His bundle activity. When the ventricle was paced at higher rates (170 beats/min) the QRS alternans disappeared.

Discussion

Several mechanisms have been proposed to explain electrical alternans that is not associated with pericardial effusion. An alternating pattern of electrical activation resulting from

fusion of two depolarisation wavefronts and a 2:1 VA conduction which superimposes a P wave on every other QRS complex producing "pseudo" electrical alternans can be excluded; alternatively, 2:1 VA conduction could generate alternating ventricular volumes and produce electrical alternans by the Brody effect.¹² Arguing against a haemodynamic cause of alternans are observations in atrial fibrillation that show no relation between QRS amplitude and stroke volume and the infrequent occurrence of electrical alternans in pulsus alternans.² An alternating conduction delay or block in part of the His Purkinje system or ventricular muscle could lead to an alternating pattern of electrical activation.^{2,3} A relation between QRS alternans and short cycle length was found in some studies.^{4,5}

However, in the present case only the variations in cycle length were apparent. However, the cycle length was constant (paced rhythm) and the apparent changes in cycle length were caused by changes in ventricular activation with a different start to the QRS complex on the surface ECG; there was a correlation between a 2:1 ventriculoatrial retrograde block and QRS alternans. The shift in QRS morphology to an LBBB-QRS pattern after a patent atrial retrograde conduction argues in favour of a concealed anterograde conduction in the left bundle branch after atrial retrograde conduction through the right bundle branch. The phenomenon began only after the second atrial retrograde block. Ventricular activation could depolarise the ventricle only though the right bundle branch block: the left bundle branch could not be retrogradely depolarised because of a previous concealed anterograde conduction (fig 2) and the left ventricle could

be depolarised only from the right bundle branch, causing the QRS complex to have an LBBB pattern as a result.

In conclusion this case report confirms the hypothesis that electrical alternans is secondary to a 2:1 block in the activation of some part of the ventricles.

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