Retrograde His bundle deflection in bundle-branch re-entry

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Atrial echo beats resulting from a reciprocating mechanism involving the bundle-branches were produced by premature atrial impulses in a patient with an A-V nodal bypass tract. The mechanism of the arrhythmia was suggested by the presence of a retrograde His bundle deflection which appeared 'sandwiched' in between a QRS complex with complete right bundle-branch morphology and a negative P wave. Though at a shorter cycle length the His bundle was still activated retrogradely echo beats were not seen because the retrograde H deflection occurred too early, when both bypass tract and A-V node were still effectively refractory. At the faster driven rate concealed retrograde activation of the right branch (by the premature impulse) was responsible for the right bundle-branch block patterns shown by the post-premature driven beat.

In a recent communication Varghese *et al.* (1974) discussed several tracings showing atrial echo beats which could have resulted from a reciprocating mechanism involving the bundle-branches. However, they could not make the differential diagnosis between intra-His bundle re-entry and bundle-branch re-entry because retrograde His bundle deflections were not seen. The present report deals with a case in which the recording of both antegrade and retrograde His bundle electrograms proved that some atrial echoes indeed result from bundle-branch re-entry.

Case report

A 31-year-old man with a short P-R interval and a history of repetitive supraventricular tachyarrhythmias was referred to the cardiovascular laboratory for electrophysiological evaluation. His bundle studies were performed as previously described in our laboratory after explaining the procedure and obtaining consent (Castillo and Castellanos, 1970, 1971). Fig. 1 was recorded during stimulation of the right atrium in the vicinity of the inferior vena cava. Driving cycle length was 670 ms. The duration of the corresponding intervals of the first (driven) beat was as follows: A1-H1 45 ms; H1-V1 45 ms. The QRS complexes measured 85 ms. The short atrio-His conduction time was attributed to the presence of a total or almost total A-V nodal bypass tract.

Atrial echo beats involving the bundle-branches were produced by a premature impulse (A2) delivered at a coupling interval of 330 ms. Because of the existence of an A-V nodal bypass tract A2 traversed the His bundle

early enough to find the right bundle-branch completely blocked (Fig. 1). However it was conducted to the left ventricle with considerable delay through the left bundle system, thereafter crossing the septum in a left to right direction to activate the right bundle-branch in a retrograde fashion and re-excite the His bundle also retrogradely. Finally, the impulse reached the atria through the A-V node. The corresponding P wave was negative in leads II and III and had a low right atrial-high right atrial activation sequence which was different than that of driven (A2) impulses (Fig. 1). The duration and significance of the pertinent intervals is shown in the Table. To our knowledge, this is the first report in which a retrograde His bundle deflection was recorded in an atrial echo beat resulting from bundle-branch re-entry.

The post-premature driven (A1) impulse (not shown) was again conducted to the ventricles without a complete right bundle-branch block morphology.

In Fig. 2 the driving cycle length was decreased to 420 ms with the specific purpose of evaluating the effects of a faster basic rate on the production of atrial echoes. A2 (delivered at a coupling interval of 290 ms) reached the His bundle (H2), was blocked at the right bundle-branch, and reached the left ventricle with less delay than when driving cycle length was 670 ms. The shorter H2-V2 interval (125 ms) was attributed to the decrease in His-Purkinje refractoriness related to the decrease in driving cycle length. The impulse thereafter crossed the septum in a left to right direction, penetrated the right bundle-branch retrogradely, finally re-exciting the His bundle (H⁻). An atrial echo beat did not occur as in Fig. 1 because H⁻ occurred at a moment in which both bypass tract and A-V node were 'effectively' refractory.

Because the driving cycle length was shorter than in Fig. 1 the post-premature A₁ beat reached the right bundle-branch while this structure was still refractory from the concealed retrograde penetration of the preceding impulse. Hence this driven (A₁) impulse was

FIG. 1 a) Atrial echo beat resulting from reciprocating mechanism involving the bundle-branches. Right atrium was driven from the vicinity of the inferior vena cava at a cycle length of 670 ms while a premature stimulus was delivered at a coupling interval of 330 ms. The first beat is the last of a series of eight consecutively driven (A₁) impulses. HRA = high right atrium. HBE = His bundle electrographic leads with interelectrode distances of 5 mm. LRA = retrograde low right atrial deflection recorded by the His bundle electrographic lead. H⁻ = retrograde His bundle deflection. Remaining abbreviations are conventional. b) Magnification of atrial echo beat. Reciprocating circuit was as follows: A₂→H₂→V₂→H⁻→LRA.

conducted to the ventricles with a complete right bundle-branch block pattern.

Finally, the last driven A₁ in Fig. 2 appeared later in the cycle, when the effective refractory period of the right bundle-branch had expired. It was therefore conducted with the same QRS morphology as the driven impulses.

Discussion

In most cases single atrial echoes result from re-entry within the A-V node, the determining factor being a critical A-H conduction delay (Goldreyer and Bigger, 1971; Goldreyer and Damato, 1971). But Lozano *et al.* (1973) studied one patient and Varghese *et al.* (1974) four patients in whom atrial echo beats appeared after critical His-Purkinje delay. Lozano *et al.* (1973) proposed that in their case a reciprocating mechanism involving the His bundle best explained the genesis of the arrhythmia.

TABLE Duration and significance of conduction interval in atrial echo beat involving the bundle-branches

A ₂ -H ₂	(Atrio-His conduction time through the AV nodal bypass tract) = 80 ms*
H ₂ -V ₂	(His to left ventricular conduction time through the left bundle system) = 175 ms
V ₂ -H ⁻	(left to right transseptal time plus retrograde right bundle conduction time) = 120 ms
H ⁻ -LRA	(retrograde AV nodal conduction time) = 125 ms†

* The slightly longer duration of this interval as compared with A₁-H₁ (45 ms) may be ascribed to conduction delay in the bypass tract. However, the bypass might have been only 'partial', the impulse being delayed in the part of the AV node that the supraventricular impulse had to traverse.

† Note that this interval was longer than both A₁-H₁ (45 ms) and A₂-H₂ (80 ms) intervals.

FIG. 2 Retrograde activation of His bundle without atrial re-excitation. The driven rate was faster than in Fig. 1. Retrograde activation of the right branch (by the premature impulse) was responsible for the right bundle-branch pattern shown by the post-premature driven beat.

On the other hand, Varghese *et al.* (1974) suggested that antegrade conduction of premature atrial beats was delayed in one bundle-branch and blocked in the contralateral branch, the impulse returning to the atria via the previously blocked bundle-branch or because of reciprocation within the conducting bundle.

But for bundle-branch re-entry to result in atrial echoes it is necessary that the His bundle be excited antegradely as well as retrogradely (Moe, Mendez, and Han, 1965). Since they could not detect a retrograde His bundle deflection in their tracings Varghese *et al.* (1974) postulated that it was lost within the ventricular electrogram recorded by the His bundle electrographic lead. The present study corroborates their assumptions. Besides it is also in keeping with the experimental studies of Moe *et al.* (1965), who first demonstrated that premature His bundle impulses could, after reaching the ventricles, activate the right bundle from its ventricular end to re-excite the His bundle.

Moreover, Fig. 2 shows that retrograde invasion of the right bundle-branch could be responsible for the right bundle-branch configuration of the post-premature atrial driven beats (Moe *et al.*, 1965; Cohen *et al.*, 1969). This phenomenon occurred only at the faster driven rates because the oncoming supraventricular impulse could reach the right bundle-branch during its effective refractory period. It is also in keeping with the findings of Spurrell, Krikler, and Sowton (1974), who presented evidence that the perpetuation of a bundle-branch pattern during reciprocating tachycardias could be due to concealed retrograde conduction of the affected bundle-branch.

References

- Castillo, C. A., and Castellanos, A., Jr. (1970). Retrograde activation of the His bundle during intermittent paired ventricular stimulation in the human heart. *Circulation*, **42**, 1079.
- Castillo, C. A., and Castellanos, A., Jr. (1971). Retrograde activation of the His bundle in the human heart. *American Journal of Cardiology*, **27**, 264.
- Cohen, S. I., Lau, S. H., Scherlag, B. J., and Damato, A. N. (1969). Alternate patterns of premature ventricular excitation during induced atrial bigeminy. *Circulation*, **39**, 819.
- Goldreyer, B. N., and Bigger, J. T., Jr. (1971). The site of re-entry in paroxysmal supraventricular tachycardia. *Circulation*, **43**, 15.
- Goldreyer, B. N., and Damato, A. N. (1971). The essential role of atrio-ventricular conduction delay in the initiation of paroxysmal supraventricular tachycardia. *Circulation*, **43**, 679.
- Lozano, J., Mandel, W. J., Hayakawa, H., Shine, K. I., and Eber, L. M. (1973). Re-entrant tachycardia: participation of the distal A-V conduction system. *Chest*, **63**, 23.
- Moe, G. K., Mendez, C., and Han, J. (1965). Aberrant A-V propagation in the dog heart: a study of functional bundle branch block. *Circulation Research*, **16**, 261.
- Spurrell, R. A. J., Krikler, D. M., and Sowton, E. (1974). Retrograde invasion of the bundle branches producing aberration of the QRS complex during supraventricular tachycardia studied by programmed electrical stimulation. *Circulation*, **50**, 487.
- Varghese, P. J., Damato, A. N., Caracta, A. N., Gallagher, J. J., Josephson, M. E., and Lau, S. H. (1974). Intraventricular conduction delay as a determinant of atrial echo beats. *Circulation*, **49**, 805.

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