CASE REPORTS

Demonstration of Intra-atrial Conduction Delay, Block, Gap and Reentry: A Report of Two Cases

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SUMMARY Intra-atrial reentry, although often suspected to be one of the mechanisms for atrial arrhythmias, has been difficult to document in the human heart. The phenomena of intra-atrial conduction delay, block and reentry were observed in two patients and led to sustained atrial fibrillation in one of the cases. In both cases, the area of conduction delay appeared to be localized in the region of the high right atrium. The electrophysiological findings and the possible mechanisms for the observed phenomena are discussed.

REENTRY HAS BEEN FREQUENTLY proposed as a mechanism for a variety of cardiac arrhythmias. For certain types of arrhythmias, the role of reentry appears well established.1-10 While intra-atrial reentry has long been suspected as a mechanism of some form of atrial arrhythmias, the phenomena of inter- or intra-atrial reentry in man have rarely been documented.8,11 The inability to demonstrate inter- or intra-atrial reentry in part relates to the limited number of intra-atrial recordings that can be used during routine electrophysiological studies.8,11,12 In this report we present observations in two patients showing the phenomena of intra-atrial conduction delay, block, gap and reentry.

Case 1

The patient, a 56-year-old male, had a clinical diagnosis of aortic regurgitation of rheumatic origin. A 12-lead surface ECG showed notched P waves and left ventricular hypertrophy by voltage criteria. Chest x-ray was normal. A diagnostic right and left heart catheterization revealed mild aortic regurgitation, the value having three cusps. All intracardiac pressures were within normal limits.

Electrophysiological Studies

This patient underwent two electrophysiological studies in accordance with methods previously described.13 Informed consent was obtained for both studies. The initial study was performed as a part of an unrelated research protocol for which the patient volunteered. After analyzing data from the first study, permission was obtained for restudy in order to determine the consistency of the findings and to rule out artifact.

For the initial study, three quadrupolar electrode catheters (interelectrode distance 1 cm) were percutaneously introduced into peripheral veins and fluoroscopically positioned in the region of the tricuspid valve, high right atrium (near its junction with superior vena cava) and right ventricular apex. The proximal and distal pairs of the quadrupolar catheter in the high right atrial (HRA) region were used for local recording and pacing, respectively. At the time of study the patient was not taking any cardioactive medications. The spontaneous rhythm appeared to be sinus at a cycle length of 800-900 msec, with upright P waves in leads I-III. The A-H and H-V intervals measured 90 and 40 msec, respectively. During both sinus and paced atrial rhythms (fig. 1), the HRA electrogram was composed of two deflections, a small amplitude deflection followed by a more rapid deflection of larger amplitude. This latter larger deflection coincided with the major rapid deflection of the low right atrial (LRA) electrogram, as seen on the His bundle electrogram (HBE) tracing. The small amplitude HRA deflection preceded the large amplitude deflection during both the basic driven and sinus beats (last beat in each panel) by 20-40 msec (fig. 1). The figure illustrates the findings during atrial premature stimulation (S2) at a basic atrial paced cycle length (S1S1) of 900 msec. At an S1S2 interval of 440 msec (panel A), the A1A2 interval (large amplitude deflections) on HRA measures 480 msec due to prolongation of the interval between the small and large amplitude deflections (intra-atrial conduction delay). No conduction delay is noted between S2 and small amplitude deflection or A2 at the low right atrium, i.e., HBE recording. At a closer S1S2 interval of 400 msec (panel B), no large amplitude A2 response is recorded at the high right atrium (intra-atrial block) whereas A2 on HBE occurs on time. A further decrease in S1S2 interval to 380 msec (panel C) is accompanied by reappearance of large amplitude A2 deflection on HRA (a form of intra-atrial gap). The

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FIGURE 1. Tracings in each panel from top to bottom are ECG leads I, II, III and V1, high right atrial (HRA) electrogram. His bundle electrogram (HBE) and time lines (T). The basic paced atrial cycle length (S1S1 or A1A1) during high right atrial pacing is the same in all panels and measures 900 msec. Designations S1, A1, H1 and V1 are used for the basic drive beats and S2, A2, H2 and V2 for premature beats. On HRA recording, the small amplitude deflection for both the basic drive beats and premature beats is marked by an asterisk, whereas the large amplitude deflection is labeled A. S denotes stimulus artifact. All measurements are in msec.

FIGURE 2. (opposite page) The basic paced ventricular cycle length (S1S1 or V1V1) measures 700 msec, panels A–C. The sequence of ventricular premature stimulation is shown. Unless otherwise indicated, abbreviations in this and subsequent figures are the same as in figure 1.
interval between the small and large amplitude deflections on HRA measures 190 msec, and the A2 on HRA is followed by another small deflection (*) 85 msec later. The A2 response on HRA in panel C is also followed by atrial reexcitation labeled A3 on HBE. The A2 (HRA) to A3 (HBE) measures 85 msec (not labeled). In panel D, the effective refractory period of the right atrium is encountered at an S1S2 interval of 370 msec. Regardless of the time of occurrence of the large amplitude A2 deflection on the HRA, the small deflection marked by asterisks has a fairly constant temporal relationship to both the stimulus artifact and the A2 deflection on the LRA. In addition, the shortest A1A2 interval recorded on HRA measures 480 msec which is significantly longer than the shortest A1A2 of 385 msec on LRA (panel C). Similar findings were also noted at paced atrial cycle lengths (CL) of 800, 700 and 600 msec. The S1S2 intervals resulting in intra-atrial block and reentry were found to be shorter at shorter cycle lengths.

Figure 2 shows essentially similar findings when the atria were retrogradely activated during right ventricular (RV) apical pacing. Retrograde activation as seen on the HRA, during paced ventricular drive (S,S, or V1V1) is again composed of two deflections, a small amplitude and large amplitude deflection. At a V1V2
coupling interval of 410 msec (panel A), there is no conduction delay between A₂ of the HBE tracing and the small amplitude (*) deflection on the HRA. However, a conduction delay of 185 msec occurs between LRA and large amplitude deflection of the HRA. The interval between small and large amplitude deflections on HRA measures 135 msec (not labeled). In panel B, only the small amplitude deflection appears at a V₁V₂ of 400 msec, suggesting a retrograde intra-atrial block. At closer coupling interval (panel C), the large amplitude deflection on the HRA reappears 185 msec after the small amplitude deflection. The interval between the large amplitude deflections on the HBE and HRA tracings is 235 msec. The delayed A₂ on HRA tracing is followed by a small amplitude deflection 90 msec later and is also accompanied by atrial reexitation (A₃) on the HBE tracing. The phenomena of intra-atrial conduction delay and block were also seen during incremental ventricular pacing as depicted in figure 3. Panel A shows 1:1 retrograde conduction to the high right atrium at a paced ventricular cycle length of 400 msec, while panel B displays the onset of 2:1 retrograde intra-atrial block at a shorter paced ventricular CL of 380 msec. Findings of intra-atrial conduction delay, block and reentry were also noted during incremental atrial pacing (not shown).

Because of the unusual findings in this case, the patient was asked and consented to undergo a second study, which was performed 27 days after the initial study. During the second study, a quadripolar catheter was positioned in the midcoronary sinus (CS) to obtain a left atrial electrogram (LAE) as well as to stimulate from this region. Observations made during premature stimulation from the coronary sinus are shown in figure 4. Since the electrode catheters could obviously not be placed in identical positions compared to the first study, the local electrograms appear slightly different. The initial small amplitude (*) and the subsequent large amplitude deflections are, however, clearly identifiable on HRA. Intra-atrial conduction delay and reentry were again registered by the catheters in the HRA region. No intra-atrial conduction delay was recorded in the regions of the left and low right atrium.

Case 2

The patient is a 25-year-old female with Wolff-Parkinson-White syndrome who was referred for electrophysiological evaluation because of disabling supraventricular arrhythmias. Routine studies were carried out with electrode catheters positioned in the region of the HRA, midcoronary sinus (LAE) and atrioventricular junction (HBE). The orientation of delta vector in V₁ was almost isoelectric and the type
of ventricular preexcitation could not be clearly defined. Reentrant supraventricular tachycardia (SVT) used the accessory pathway retrogradely and could be repeatedly induced with single atrial premature stimulation during sinus rhythm. The phenomena of intra-atrial conduction delay and block were observed both during premature atrial stimulation and sustained episodes of SVT. During the latter, varying degrees of intra-atrial conduction delay and block subsequently led to the development of atrial fibrillation (figs. 5 and 6).

As seen in figure 5, the HRA electrogram during sinus or driven atrial beats is composed of a small amplitude atrial deflection (•) which precedes a larger amplitude deflection. In panel A, premature stimulation at the midcoronary sinus (LAE) results in conduction delay to the large amplitude deflection but not to the small amplitude deflection. In panels B and C, a progressive decrease in coupling interval (300 and 200 msec, respectively) results in no change in the interval between the site of stimulation and small amplitude deflection, but absence of large amplitude deflection indicates intra-atrial block. The atrium is effectively refractory at an S3S2 interval of 260 msec (panel D).

Panels A and B of figure 6 depict an episode of supraventricular tachycardia in which the first few beats are of aberrant configuration. Activation of the atria is in a retrograde direction and for the first four beats there is slight conduction delay between the small and large amplitude deflections. Thereafter, a 2:1 block occurs between the two deflections. In panel B, starting with the third QRS complex, only the large amplitude atrial deflection is seen on the HRA tracing after which atrial fibrillation ensues.

Discussion

The data presented from these two cases demonstrate the occurrence of localized intra-atrial conduction delay and block in the human heart. Case 1 appears to have, in addition, intra-atrial gap and reentry occurring as a single beat, whereas in case 2 intra-atrial conduction delay and block gradually transformed into a sustained chaotic atrial rhythm. In
Figure 5. Sequence of atrial premature stimulation from coronary sinus at a basic cycle length of 700 msec (case 2) is shown. The A-H and H-V intervals during sinus beats measured 85 and 25 msec, respectively. Ventricular preexcitation during sinus and paced beats is apparent. See text for details. Abbreviations are the same as in figures 1 and 4.

Both cases the phenomena were localized to the HRA region and were demonstrable irrespective of the site of stimulation (i.e., HRA, CS or RV pacing) and in case 1 at different paced cycle lengths. This, plus the fact that phenomenon could be demonstrated on repeat study several months later in case 1, indicate that the phenomena was artifactual.

While no evidence of conduction delay and block
were observed in other parts of the atria, we must acknowledge that only three intra-atrial electrogram recordings were made. The anatomic substrate responsible for intra-atrial conduction delay and block occurring in the HRA region is not known in these patients and the role of sinus node, if any, can only be conjectural. Neither of the patients have ever had any symptoms or ECG evidence of spontaneous sinus node dysfunction. Furthermore, the phenomena herein reported have not been noted in most reported cases of sinus node reentry. Despite these negative associations, the sinus node and its immediate environs could be the site of delay, block and reentry.

In the absence of elaborate intra-atrial mapping, the precise areas and mechanisms for the observed phenomena cannot be delineated. However, the most likely explanation for the results obtained in case 1 are schematically presented in figure 7. From the site of stimulation (S) in the HRA region, the S impulse approached the recording electrodes of HRA and LRA catheters at more or less the same time, activating the area represented by the small amplitude deflection (HRA) before that of the large amplitude deflection (panel A). At progressively shorter S-S intervals, increasing intra-atrial conduction delay, block and low atrial reexcitation occurred and are depicted in panels B through D of figure 7; for correlation with actual intracardiac tracings, this schema may be compared to panels A through C of figure 1. Figure 7 is representative of findings when HRA was the site of stimulation and similar schemas can be drawn for other sites of stimulation, i.e., the RV and CS. In a strict sense, reentry, which generally implies return of an impulse to or toward its site of origin, may not be the underlying mechanism of A (figs. 1, 2 and 4) at all times. During RV premature stimulation the sequence of atrial activation from LRA → HRA (small deflection) → HRA (large deflection) → HRA (small deflection) → LRA (A) indicates that intra-atrial reentry was the most likely explanation for the phenomena observed. However, a somewhat different mechanism might have been operative during HRA stimulation, as shown in figure 7. The S impulse from stimulation site (HRA) traveled rapidly to the LRA and to the area represented by small amplitude deflection in HRA, whereas the impulse conducted slowly to the area represented by the large amplitude deflection on the HRA. Following activation of the latter, the area of small amplitude HRA and LRA could be reexcited if recovery of excitability had taken place (panel C of fig. 1, and panel D of fig. 7). Alternately, S impulse while blocked in the HRA (between the area of small and large amplitude deflections) could proceed to activate LRA → HRA (large amplitude) → HRA (small amplitude) → HRA (small amplitude).
amplitude) → LRA. This latter sequence of events, which is more like reentry, appears unlikely, since the area of small amplitude deflection on the HRA was not activated following LRA, as occurred during RV stimulation. With the limited number of recordings available, the precise location of impulse reciprocation within the area of HRA cannot be ascertained. The sequence of atrial activation and reexcitation during coronary sinus pacing is probably similar to what was observed during HRA pacing rather than RV pacing.

Diagnostic right and left heart catheterization was performed in case 1; the results, however, did not provide any insight into the etiologic basis of the electrophysiologic findings observed. Similarly, the anatomic basis for the localized intra-atrial conduction delay, block and reentry in case 2 remains uncertain, although conversion of reciprocating supraventricular tachycardia into atrial fibrillation in patients with the Wolff-Parkinson-White syndrome has been previously published.

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Fibrolipoma of the Mitral Valve in A Child

Clinical and Echocardiographic Features

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SUMMARY Echocardiographic examination, performed in a 12-year-old boy who had signs of mitral regurgitation, showed the presence of an abnormal mass of echoes in the left atrium and mitral orifice. These were initially interpreted as representing an atrial myxoma. Surgical exploration showed that the tumor originated from the posterior leaflet of the mitral valve, and, microscopically, the lesion was diagnosed as a fibrolipoma.

The clinical and echocardiographic features of this unique type of cardiac tumor are discussed.

PRIMARY TUMORS OF THE HEART are rare in infancy. The most frequent type is probably the rhabdomyoma which, in most cases, is associated with tuberous sclerosis.1–3 Few reports have appeared in the literature regarding primary tumors of heart valves in children.4–5

This report describes the clinical, echocardiographic and anatomical-pathological features of a fibrolipoma of the posterior leaflet of the mitral valve (PML) in an asymptomatic 12-year-old boy.

Case Report

The patient is a 12-year-old male who was referred to the pediatric cardiology department in October 1977 for evaluation of a heart murmur discovered at the time of an emergency room visit for a fracture of the right wrist. The child was asymptomatic with respect to the cardiovascular system and there was no history of rheumatic heart disease.

On physical examination, the patient was well-developed and healthy looking. The blood pressure was 90/60. Peripheral pulses were normal and equal in all four limbs. On palpation of the precordium, the apex was displaced to the sixth left intercostal space at the anterior axillary line. On auscultation, the first heart sound was of normal intensity and the second heart sound was normally split. At the apex there was a grade 3/6 soft pansystolic murmur radiating to the axilla. Also at the apex, there was an S₂ followed by a low frequency grade 2/6 diastolic rumble without presystolic accentuation. There was no opening snap. The lungs were clear to auscultation. The liver and the spleen were not enlarged.

The ECG showed signs of left atrial enlargement. On the chest x-ray, the heart size was normal, with a slight prominence of the left ventricle and dilatation of the left atrium. There was prominence of the pulmonary veins in the upper third of the lungs, suggesting the presence of pulmonary venous congestion. Complete blood count, erythrocyte sedimentation rate and protein electrophoresis were all normal.

Echocardiography (fig. 1)

Standard M-Mode echocardiography was obtained with an Ekoline 20A ultrasonoscope interfaced with a Honeywell strip chart recorder #1856. At the level of the aortic root, an abnormal mass of echoes was recorded during systole in the left atrium. As the transducer was swept toward the left ventricular outflow tract (LVOT) and the mitral valve, abnormal
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