High Resolution Mapping of Koch’s Triangle Using Sixty Electrodes in Humans With Atrioventricular Junctional (AV Nodal) Reentrant Tachycardia

Mark A. McGuire, MB, PhD, FRACP; John P. Bourke, MB, MRCP; Monica C. Robotin, MB, BS; David C. Johnson, MB, BS, FRACS; William Meldrum-Hanna, MB, BS, FRACS; Graham R. Nunn, MB, BS, FRACS; John B. Uther, MD, FRACP; David L. Ross, MB, BS, FRACP

Background. Recent evidence suggests that atrioventricular junctional reentrant tachycardia (AVJRT) uses a reentrant circuit that involves the atrioventricular (AV) node, the atrinodal connections, and perinodal atrial tissue. Electrogram morphology has been used to target the delivery of radiofrequency energy to the site of the “slow pathway,” a component of this reentrant circuit. The aim of this study was to localize precisely the sites of atrinodal connections involved in AVJRT and to examine atrial electrogram morphologies and their spatial distribution over Koch’s triangle.

Methods and Results. Electrical activation of Koch’s triangle and the proximal coronary sinus was examined in 13 patients using a 60-point plaque electrode and computerized mapping system. Recordings were made during sinus rhythm (n=12), left atrial pacing (n=8), ventricular pacing (n=12), and AVJRT (n=12). During sinus rhythm electrical activation approached Koch’s triangle and the AV node from the direction of the anterior limbus, activating the anterior part of the triangle before the posterior part. A zone of slow conduction during sinus rhythm was found within Koch’s triangle in 64% of patients. The pattern of atrial activation in Koch’s triangle during anterograde fast pathway conduction was similar to that seen during anterograde slow pathway conduction. Retrograde fast pathway conduction during ventricular pacing and during anterior (typical) AVJRT caused earliest atrial activation at the apex of Koch’s triangle near the AV node–His bundle junction. In individual patients the site of earliest atrial activation was similar for both anterior AVJRT and retrograde fast pathway conduction during ventricular pacing. Retrograde slow pathway conduction during ventricular pacing and during posterior (uncommon or atypical) AVJRT caused earliest atrial activation posterior to the AV node near the orifice of the coronary sinus. This posterior or “slow pathway” exit site was 15±4 mm from the His bundle. In individual patients the site of earliest atrial activation was similar for both posterior AVJRT and retrograde slow pathway conduction during ventricular pacing. In one patient anterograde and retrograde conduction occurred via separate slow pathways during AVJRT. Complex atrial electrograms with two or more components were observed near the coronary sinus orifice and in the posterior part of Koch’s triangle in all cases. These were categorized as either low or high frequency potentials according to the rapidity of the second component of the electrogram. Low frequency potentials were present at the site of earliest atrial excitation during retrograde slow pathway conduction in 5 of 5 cases (100%) and high frequency potentials in 4 of 5 cases (80%). However, both slow and high frequency potentials could be found at sites up to 16 mm from the site of earliest atrial excitation.

Conclusions. At least two distinct groups of atrinodal connections exist. The site of earliest atrial activation during anterior AVJRT is similar to that of fast pathway conduction during ventricular pacing. This site is close to the His bundle–AV node junction. The site of earliest atrial activation during posterior AVJRT is similar to that of slow pathway conduction during ventricular pacing. This site is near the coronary sinus orifice, approximately 15 mm from the His bundle. The anterograde slow pathway appears to be different from the retrograde slow pathway in some patients. Double atrial electrograms are an imprecise guide to the site of earliest atrial excitation during retrograde slow pathway conduction. (Circulation. 1993;88[part 1]:2315-2328.)

Key Words • atrioventricular node • tachycardia • electrophysiology

Atrioventricular junctional reentrant tachycardia (AVJRT) is a supraventricular tachycardia caused by reentry in the region of the atrioventricular (AV) node.1-3 The exact site of reentry and the component tissues of the reentrant circuit are controversial, but recent evidence suggests that the reentrant circuit involves both the AV node and perinodal atrium.4-11 A number of surgical and catheter ablation techniques have been developed for the cure of...
AVJRT 4,5,9-16 These techniques have been designed with the aim of destroying part of the reentrant circuit, leaving AV conduction intact. Nevertheless, in a proportion of cases the AV conduction system is damaged, causing permanent complete heart block. 12,15,17 Thus, precise localization of the pathways used by this arrhythmia may enable the development of improved techniques for its cure. In addition, recent reports suggest that the morphology of the atrial electrogram during sinus rhythm may help to identify the site of the “slow pathway,” an essential part of the reentrant circuit. 18,19

Although electrode-tipped catheters introduced percutaneously under fluoroscopic guidance may be used to map electrical activity in this region, the use of a plaque electrode introduced at surgery has several advantages. 20 The latter technique allows a large number of signals to be acquired simultaneously from a defined region and confers precise knowledge of the spatial relationships of the electrodes. Furthermore, the plaque can be positioned accurately under direct vision. The aim of this study was to map the electrical activation of Koch’s triangle and proximal coronary sinus in patients with AVJRT using a 60-point plaque electrode and computerized mapping system to precisely localize the sites of the atrionodal connections and to examine the atrial electrogram morphology.

Methods

Patients

These were 13 patients undergoing routine surgery for attempted cure of AVJRT. The mean age of patients was 44±13 years (range, 16 to 73), and 9 patients (69%) were female. All patients gave informed consent for this procedure.

Preoperative Electrophysiological Study

AVJRT was defined as tachycardia using only AV nodal tissue or atrial and AV nodal tissue as components of the reentrant circuit. In all patients a comprehensive electrophysiological study was performed before surgery using methods described previously. 21 Anterior AVJRT (typical AV nodal reentrant tachycardia) was defined as AVJRT in which earliest atrial activation was recorded anterior to the AV node by the His bundle recording catheter. 4,7 Posterior AVJRT (uncommon or atypical AV nodal reentrant tachycardia) was defined as AVJRT in which earliest atrial activation was recorded posterior to the AV node near the coronary sinus orifice. 4,7 In all cases the ventricular extra-stimulus test was used to distinguish AVJRT from supraventricular tachycardia using concealed septal accessory pathways. 21,22 Tachycardias caused by intratral reentry were excluded using methods described previously. 7,21 At the preoperative electrophysiological study, sustained anterior AVJRT alone was induced in 9 patients. In one patient with concomitant Wolff-Parkinson-White syndrome, both sustained anterior AVJRT and orthodromic tachycardia using a posterior septal accessory pathway were induced. In 3 patients sustained posterior AVJRT alone was induced.

The Mapping Plaque

The plaque consisted of 60 electrodes of fine Teflon-insulated stainless steel wire (0.125-mm diameter, Mediwire Corporation, Mt Vernon, NY) embedded in a polyorganosiloxone matrix (President Light Body, Coltene AG, Alstatten, Switzerland). The wire electrodes were trimmed flush with the surface of the plaque, and only the extreme tips of the wires were bared of insulation. The electrodes were arranged in a polygonal array with a 3-mm interelectrode distance (see Fig 1). Fifty-six electrodes were apposed to the right atrial endocardial surface overlying Koch’s triangle and the surrounding perinodal atrium. Four electrodes were positioned along a process that extended from the surface of the plaque. When the process was inserted in the coronary sinus, these latter four electrodes were positioned at distances of 3 mm, 6 mm, 9 mm, and 12 mm from the orifice of the coronary sinus. Leads I, aVF, and V1 (modified) were used to record the surface ECG.

In one patient (case 6) a different, rectangular electrode array was used. In the latter case, signals were not recorded from the coronary sinus.

Positioning the Mapping Plaque

Normothermic cardiopulmonary bypass was instituted using separate venous cannulae for the superior and inferior venae cavae. The mapping plaque was introduced via an incision placed in the anterolateral wall of the right atrium. The process extending from the plaque was inserted in the coronary sinus, and the plaque was then rotated until His bundle signals were recorded. The position of the plaque was then adjusted so that adequate atrial signals rather than ventricular signals were recorded from the lower rows of the plaque. The approximate position of the plaque during mapping is shown in Fig 2.

Recording of Electrical Activity

Unipolar signals were recorded from the endocardium. An “alligator” clip placed on the sternal retractor served as the indifferent electrode. Attempts were made to record signals during sinus rhythm, atrial pacing, ventricular pacing, and during AVJRT, but because it was necessary to limit the time spent under normothermic cardiopulmonary bypass, it was not possible to make recordings during all of these rhythms in all patients. A button electrode was sutured to the right ventricular outflow tract and a clip electrode was fixed to the left atrial appendage to pace these chambers.

Signal Processing

Signals recorded from the right atrial endocardium and the surface ECG were recorded simultaneously using a customized 64-channel mapping system. Signals recorded from the endocardium were amplified with a gain of 500 to 1000. An analog low-pass filter (–3 dB point, 500 Hz) was used for all endocardial recordings. The lower corner frequency of the amplifiers was 0.2 Hz, but an optional high-pass analog filter (–3 dB point 35 Hz) was used for most of the recordings. Signals were digitized at 1000 Hz with 12-bit accuracy and stored in the memory buffer of a Masscomp model 5402 computer (Massachusetts Computer Corporation, W教授d, Mass). After the signals had been examined and found to be satisfactory, the contents of the memory buffer were stored on a hard disk.
Tachycardia Induction

Tachycardia was induced using rapid atrial or ventricular pacing. If tachycardia could not be induced in the baseline state, isoproterenol ( aliquots of 5 to 10 μg) was administered intravenously. If tachycardia could still not be induced atropine 1 mg was administered intravenously.

Distinction of Atrial Activity From Ventricular Activity During AVJRT

In five cases, atrial activation occurred almost simultaneously with ventricular activation during AVJRT so that it was difficult to distinguish atrial from ventricular activity. In these cases we attempted to deliver single or paired premature extrastimuli to the right ventricular apex during tachycardia so that ventricular activation was completed before the onset of atrial activation. The coupling intervals of these extrastimuli were such that the atrial cycle length during tachycardia was not altered. In one patient ( case 6) the spontaneous occurrence of 2:1 AV conduction during AVJRT allowed direct visualization of atrial activation.

Reproducibility of Signals

As a measure of reproducibility, the standard deviation of the activation times of atrial activity recorded by the 60 endocardial electrodes was measured over 10 consecutive beats of AVJRT in 3 patients selected at random. The standard deviations of activation times were 0.7 milliseconds, 1.2 milliseconds, and 1.4 milliseconds, respectively. Variations in atrial activation time occurred predominantly in channels recording fractionated electrograms.

Analysis of Electrical Activation

The computer was used to calculate dV/dt each millisecond for each channel, and the point of maximum negative dV/dt was assumed to represent the time of local activation. For each point, the dV/dt was calculated over a 3-millisecond interval. Isochron maps were constructed by joining points activated simultaneously. A manual editing function could be used to alter the computer-designated timing of local activation in channels recording noisy or fragmented electrograms. In channels recording fragmented electrograms, the plot of dV/dt usually revealed several troughs of almost equally negative value. In these cases the timing of local activation was assigned to the point that was most consistent with the time of local activation recorded by surrounding electrodes.

Atrial Electrogram Morphology

"Double" atrial electrograms were defined as those exhibiting two or more discrete deflections during sinus rhythm or atrial pacing. Electrograms were categorized
according to the morphology of the second deflection. “Low frequency” potentials, similar to those described by Haissaguerre et al., were defined as atrial electrograms in which the first deflection had a rapid intrinsic deflection and the second deflection was less rapid and of lower amplitude. High frequency potentials, similar to those described by Jackman et al., are defined as those in which the first deflection was of relatively low amplitude and frequency and the second deflection was rapid and of greater amplitude.

**Results**

**Sinus Rhythm**

Recordings were made during sinus rhythm in 12 patients (92%). In the remaining patient (case 4), atrial rhythms occurred spontaneously during cardiopulmonary bypass and sinus rhythm did not occur. During sinus rhythm electrical activation approached Koch’s triangle from the anterior limbus in 11 patients (92%), then swept posteriorly so that the apex of the triangle was activated before its base (Fig 3A). In one patient electrical activation approached Koch’s triangle via the posterior limbus. Activation of the coronary sinus commenced near the orifice then spread leftward in all cases. An area of slow conduction within the triangle of Koch was observed in 7 patients (64%) (see Fig 3A).

**Atrial Pacing**

Recordings were made during left atrial pacing in 8 patients (62%). The activation of the lower atrial septum was almost identical to that during sinus rhythm in 4 patients (50%). In 3 patients the pattern of activation was very similar to that during sinus rhythm, but the wave front approached the mapping plaque from a slightly more anterior direction (2 patients) or a slightly more posterior direction (1 patient) (see Fig 3B). In all patients, the activation pattern of atrial premature beats was similar to that of drive cycle beats.

In 1 patient (case 3), however, the pattern of activation was markedly different from that during sinus rhythm. In this patient, parts of the triangle of Koch were activated before more superior parts of the atrial septum and anterior limbus. We cannot identify with certainty the route of septal activation in this patient, but we speculate that activation proceeded in a superoinferior direction along the left side of the septum before breaking through to the endocardium of the right atrial side of the septum. The activation wave front did not appear to travel along the coronary sinus since all electrodes in the proximal coronary sinus were activated almost simultaneously. In this patient, the patterns of activation during ventricular pacing and AVJRT were also different from those of the other patients (see below).
Fig 3. Activation maps recorded by the plaque electrode. The plaque was positioned as shown in Fig 2. Isochrons are drawn at 3-millisecond intervals. Arrows indicate the direction of activation. The positions of the coronary sinus orifice (CS) and His bundle (HB) are marked. Activation time is indicated by the large horizontal bar at the bottom of the figure. The small horizontal bar below each map indicates the activation of the electrodes within the coronary sinus. The electrode nearest the orifice of the coronary sinus is represented by the rectangle to the left of each "coronary sinus" bar. A, Sinus rhythm (case 5). Note the area of bunched isochrons near the coronary sinus orifice indicating a zone of slow conduction. B, Left atrial pacing (case 5). The direction of the wave front is slightly different from that in sinus rhythm and the direction of activation in the coronary sinus is reversed compared with sinus rhythm. C, Ventricular pacing (case 4). Conduction to the atrium is via the anterior exit or so-called fast pathway. The anterior site of exit was in a similar position in all patients. D, Ventricular pacing with conduction via a posterior exit or "slow pathway" (case 4). E, AVJRT of the anterior or "typical" type (case 4). The site of earliest atrial activation is very similar to that seen in this patient during ventricular pacing (see panel C). F, AVJRT of the posterior or atypical type (case 13). The site of earliest atrial activity is posterior to the AV node. The site of earliest atrial activation in this patient during ventricular pacing was similar (not shown).

**Dual Anterograde Pathways**

Dual anterograde AV nodal pathways were demonstrated in 6 patients during the intraoperative mapping study. The pattern of activation of the lower atrial septum and Koch's triangle during fast pathway conduction was similar to that observed during slow pathway conduction.
Ventricular Pacing: Patients With Anterior (Typical) AVJRT Induced at the Preoperative Electrophysiological Study

Recordings of atrial activation during ventricular pacing were obtained in 9 of the 10 patients (90%). In all patients, an anterior exit from the AV node was present. This site was at the apex of the triangle of Koch close to the junction of the AV node and His bundle (Fig 3C) except in one patient (case 3) in whom almost the entire triangle of Koch was activated simultaneously. Apart from case 3, the pattern of atrial activation in all patients was similar to that shown in Fig 3C.

In 7 patients a single site of earliest atrial activation was observed. In 2 patients (cases 4 and 10), dual sites of earliest atrial activation were observed. In one of the latter (case 4), during ventricular pacing at longer cycle lengths, the site of earliest atrial activation was at the apex of the triangle of Koch as described above. With closely coupled ventricular extrastimuli, however, the VA interval lengthened and the site of earliest atrial activation was posterior to the AV node inferior to the mouth of the coronary sinus (see Figs 3C, 3D, and 4). In another patient (case 10), simultaneous activation of both anterior and posterior sites was observed during rapid ventricular pacing. The posterior sites of endocardial activation were up to 21 mm from the His bundle and varied slightly from patient to patient as shown in Fig 5.

Fig 4. Electrograms recorded by the plaque electrode during ventricular pacing (case 4). Two paced beats are shown. The first paced beat (S1) is conducted to the atrium via an anterior AV nodal exit or fast pathway (see Fig 3C). Earliest atrial activation is seen in electrode 46, near the His bundle. The second beat with a coupling interval of 200 milliseconds is conducted via a posterior or slow pathway (see Fig 3D). Earliest atrial activation of this beat is recorded by electrode 60. Note the QS pattern of the atrial electrograms recorded by electrodes close to the site of earliest activation. I, aVF, and V2 (modified) are surface ECG leads. The positions of electrodes are indicated in the panel at upper right. Electrodes 32, 54, 63, and 64 were positioned in the coronary sinus at distances of 3, 6, 9, and 12 mm, respectively, from the orifice. The vertical lines may be used as a time reference to align each panel. CS indicates coronary sinus orifice; CFB, central fibrous body; TT, tendon of Todaro; and TVA, tricuspid valve orifice.

Fig 5. The approximate positions of earliest atrial activation during posterior nodal ("slow pathway") conduction in 5 patients (A through E) and in 1 patient during conduction via a posterior septal accessory pathway (F). The area enclosed by the isochron line drawn 3 milliseconds after the onset of atrial activation is shown (hatched areas). Each panel was recorded from a different patient: B and C were recorded during posterior atrioventricular junctional reentrant tachycardia; A, D, and E were recorded during ventricular pacing. The site of earliest atrial activation during conduction via the septal accessory pathway was not bracketed. CFB indicates central fibrous body; CS, coronary sinus orifice; TT, tendon of Todaro; and TVA, tricuspid valve annulus.
Ventricular Pacing: Patients With Posterior (Atypical) AVJRT Induced at the Preoperative Electrophysiological Study

Recordings of atrial activation during ventricular pacing were obtained in all 3 patients. In 2 patients, only the posterior site of atrial activation (as described above) was observed at all pacing rates. In 1 patient dual exits from the AV node (as described above) were observed.

AVJRT: Patients With Anterior (Typical) AVJRT Induced at the Preoperative Electrophysiological Study

Recordings of atrial activation during anterior AVJRT were obtained in all 10 patients. The site of earliest atrial activation in these cases was similar to the anterior exit found during ventricular pacing, ie, in 9 patients this was at the apex of the triangle of Koch (Figs 3E, 6, 7, and 8). Earliest atrial activation in these patients was recorded by electrodes overlying or adjacent to the His bundle. In 1 patient (case 10), almost the entire triangle was activated simultaneously. The apex of Koch’s triangle was activated on average 15±10 milliseconds (range, 0 to 32) before the proximal coronary sinus.

AVJRT: Patients With Posterior (Atypical) AVJRT Induced at the Preoperative Electrophysiological Study

Posterior AVJRT was induced in 2 of the 3 patients in whom it was induced preoperatively. In addition, non-

Fig 6. Recordings during anterior atrioventricular junctional reentrant tachycardia (case 4). Electrode positions and abbreviations are identical to Fig 4. The position of the local atrial electrogram is indicated by arrows. Earliest atrial activation is recorded by channel 46. Vertical line indicates onset of QRS in surface ECG.

Fig 7. Recordings in case 4. Left panel: Spontaneous atrial rhythm. This panel has been included to show pure ventricular electrograms for comparison with the fused atrial and ventricular electrograms recorded during atrioventricular junctional reentrant tachycardia (AVJRT). Middle panel: AVJRT. The positions of the local atrial electrograms during AVJRT are marked with arrows. This panel is identical to the lower right panel of Fig 5. Some of the atrial electrograms overlie the ventricular electrograms during AVJRT. Right panel: Ventricular pacing, fast pathway conduction. This panel is identical to the lower right panel of Fig 4. Note that during AVJRT earliest atrial activity is recorded by the electrodes nearest the His bundle. The atrial activation sequences during AVJRT and ventricular pacing are similar but not identical (see Fig 3).
sustained posterior AVJRT was induced in 1 patient in whom only anterior AVJRT was induced preoperatively (case 4). The sites of earliest atrial activation in cases of posterior AVJRT were near the orifice of the coronary sinus (Figs 3F, 5, and 9). In individual patients, the site of earliest atrial activation during posterior AVJRT was similar to that of the posterior AV nodal exit observed during ventricular pacing. The center of earliest atrial activation during conduction via the posterior exit lay approximately 15 ± 4 mm (range, 12 to 21) from the His bundle.

Additional Observations

In two cases (cases 4 and 10), rapid atrial pacing was performed during AVJRT. In both of these cases it was possible to advance atrial activity in all channels by 20 to 30 milliseconds without altering the timing of ventricular activity.

In one case (case 4), spontaneous cessation of anterior AVJRT was observed on two occasions. In both instances, anterograde conduction occurred via the “slow pathway,” as indicated by the AH interval of approximately 220 milliseconds. In the final beat of tachycardia, however, retrograde conduction also occurred via a slow pathway (as evidenced by the posterior site of atrial activation and the relatively long HA interval of 155 milliseconds), indicating that the anterograde slow pathway was different from the retrograde slow pathway (see Fig 10). This beat did not appear to be an atrial ectopic beat because the phenomenon was reproducible and the sequence of atrial activation was similar to that demonstrated previously during slow pathway activation.

Accessory Pathway–Mediated Tachycardia

In the single patient with orthodromic supraventricular tachycardia using a posterior septal accessory pathway for retrograde conduction, the site of earliest atrial activation was posterior to the coronary sinus orifice (see Fig 5F). The site of earliest atrial activation was not “bracketed” by the plaque in this case, but this site was
Fig 10. Recordings in case 4. Plaque has been rotated slightly compared with Figs 4, 5, and 6. A, Spontaneous termination of anterior ativoventricular junctional reentrant tachycardia (AVJRT). B, Detailed view of above. Channels 25 through 34 are recorded by a row of electrodes approximately 9 mm above the AV ring. Channel 54 is recorded by an electrode in the coronary sinus approximately 6 mm from the orifice. The AVJRT is using a slow pathway for anterograde conduction and a fast pathway for retrograde conduction. Mapping of sustained tachycardia, including the penultimate beat (marked with diamond), revealed an activation pattern similar to that seen in Fig 3E. After the penultimate beat, anterograde conduction is via the slow pathway as indicated by the AH interval of 220 milliseconds. Note that retrograde conduction after this beat is also via a slow pathway (marked with asterisk). Mapping of the beat marked with the asterisk revealed an activation pattern similar to that during slow pathway conduction as shown in Fig 3D. This observation indicates that the anterograde slow pathway must be different from the retrograde slow pathway in this patient. C, Ladder diagram of panel B. Dashed lines indicate slow pathway conduction and solid lines indicate fast pathway conduction. A indicates atrium; AVJ, AV junction; H, His bundle electrogram; and V, ventricular electrogram.

Electrogram Morphology

During sinus rhythm and atrial pacing, electrodes positioned over the anterior half of Koch's triangle and those positioned near the fossa ovalis typically recorded atrial electrograms having an RS pattern or an rSr pattern (see Fig 11). During ventricular pacing and AVJRT, the electrograms were similar except that electrodes lying over the site of earliest atrial activation typically recorded electrograms with a QS or QSr pattern. As distance increased from the site of earliest atrial activation, small r waves appeared, increasing in size, producing an rS or RS pattern.

Double electrograms were recorded in the posterior half of Koch's triangle and near the coronary sinus orifice in all patients. The area in which double potentials were found during sinus rhythm coincided approximately with the endocardium overlying the posterior septal space. Low frequency potentials, similar to those described by Haisaguerre et al.18 were found in all patients and high frequency potentials, similar to those described by Jackman et al.19 were observed in 12 of 13 patients (92%).

When the direction of the atrial wave front was altered (eg, atrial pacing versus sinus rhythm), small differences in the temporal relationship between the two components of the atrial electrogram were sometimes observed (see channel 53, Fig 12). During ventricular pacing, when retrograde conduction occurred via the fast pathway, Koch's triangle was activated in an anteroposterior direction (similar to sinus rhythm), and the sequence of the two components of the electrogram was unchanged. In contrast, when retrograde conduction occurred via the slow pathway, the sequence of components was reversed (see Fig 13).

The zone of the low frequency potentials was contiguous with the zone of high frequency potentials, but the former was anterior to the latter. The border between the two zones was not distinct, and in this transition zone the morphology of the double potentials gradually changed from one morphology to the other (see Figs 11 and 14). In this transition zone the double potentials were of intermediate form, with both components being of approximately equal size and equally rapid.

The approximate area of the right atrial endocardium covered by double potentials was 133±84 mm² (low frequency potentials, 71±52 mm²; high frequency potentials, 49±32 mm²). The area covered by high frequency potentials was underestimated because the plaque did not "bracket" the area of high frequency potentials in most cases. Retrograde slow pathway conduction was demonstrated in 5 cases (see above), and in all cases the first 3 milliseconds of atrial excitation was within the zone of double potentials (Fig 14). The area enclosed by the isochron marking the first 3 milliseconds of atrial excitation contained low frequency potentials in all cases and high frequency potentials in 4 cases (80%). The area enclosed by the 6-millisecond isochron contained low frequency potentials and high frequency potentials in all cases. The area covered by double potentials, however, was larger than the area enclosed by the 3-millisecond isochron (133±84 mm² versus 40±21 mm²). The largest low frequency potential was found within the 3-millisecond isochron in all cases, but the largest high frequency potential was found outside the 6-millisecond isochron in 3 cases (60%). High frequency potentials could be found at distances as great as 16 mm from the 3-millisecond isochron and could be found at sites activated as late as 24 milliseconds after initial atrial excitation during retrograde slow pathway conduction. Low frequency potentials could be found at distances as great as 17 mm from the 3-milli-
second isochron and could be found at sites activated as late as 24 milliseconds after initial atrial excitation.

Discussion

Two previous studies have examined the sequence of electrical activation of the atrial septum in patients with AVJRT. Ross and coworkers used the single exploring electrode technique, and Chang et al used a plaque electrode and computerized mapping system. The latter authors were primarily concerned with mapping the whole of the atrial septum during several different types of supraventricular tachycardia and thus used more

Fig 11. Simultaneous recordings during atrial pacing (slow pathway conduction). The electrograms are shown in the approximate position of recording. For the sake of clarity only alternate channels are shown. A paced beat rather than a sinus beat has been shown in order to aid differentiation of atrial and His potentials. The dashed circle shows the position of the coronary sinus orifice. The dashed line shows the position of the AV groove. Electrograms recorded from the coronary sinus are shown in the rectangle at right (channel 32 was 3 mm from orifice; channel 64 was 12 mm from orifice). Note the double potentials recorded in the posterior part of Koch's triangle, particularly those in channels 31, 42, 49, 51, 53, and 60. Examples of low frequency potentials are marked with heavy arrows, high frequency potentials with an asterisk. CS indicates coronary sinus orifice and H, His bundle electrogram.

Fig 12. Selected recordings from same patient as Fig 11 showing effect of atrial and ventricular pacing on double potentials. The left panel shows a paced atrial beat (S1, 340 milliseconds) conducted via the fast pathway followed by a second paced beat (S2, 190 milliseconds) conducted via the slow pathway, followed by a sinus beat (asterisk). The right panel shows a ventricular paced beat (S1, 340 milliseconds) followed by a second paced beat (S2, 180 milliseconds), both conducted via the fast pathway. The low frequency "hump" (arrow) connecting atrial and Hisian activity in channel 47 probably is due to ventricular repolarization rather than AV node depolarization as it coincides with the T wave of the surface ECG.
widely spaced electrodes (5-mm interelectrode distance) to cover the larger area. In the current study the use of more closely spaced electrodes in the region immediately surrounding the AV node has allowed more precise localization of the atrial exits from the AV node and provided several new insights into the mechanism of AVJRT.

Sinus Rhythm and Atrial Pacing

Our observations of the sequence of atrial activation during sinus rhythm are similar to those made by Chang and colleagues. These authors reported that in 8 of their 10 patients, activation spread from the sinoatrial node along the anterior limbus toward Koch's triangle but that in two patients the route of activation was along the posterior limbus. As far as we are aware, however, the presence of a zone of slow conduction within Koch's triangle has not been reported previously. An anatomic cause of this slow conduction was not apparent. It is possible that slow conduction resulted from a change in the orientation of atrial muscle fibers. It seems doubtful that the zone of slow conduction contributes significantly to AV conduction delay during normal sinus rhythm because our observations suggest that the anterior inputs to the AV node are activated before the zone of slow conduction is activated. It is possible, however, that if the anterior inputs became refractory during rapid atrial pacing, an atrial impulse would then have to traverse the slow conduction zone before entering the AV node via the posterior inputs. In this situation it is conceivable that atrial conduction delay would contribute to delay of AV transmission.

Ventricular Pacing and AVJRT

Our findings confirm that two or more groups of atrionodal connections exist in patients with AVJRT.
The site of the posterior exit was less consistent among patients (see Fig 5). In most cases this site was located in the area between the coronary sinus orifice and the tricuspid annulus. In individual patients this site was similar during posterior AVJRT and during ventricular pacing with conduction via the slow pathway. Although the site of the atrial termination of the single posterior septal accessory pathway reported in this study was distinct from the sites of the posterior exits from the AV node, we do not believe that the site of atrial activation alone is useful for discriminating between concealed septal accessory pathways and posterior AV nodal exits. We have previously used a plaque electrode to map the sites of septal accessory VA connections and have observed numerous cases in which the atrial termination of an accessory pathway was found in a position similar to that of the posterior AV nodal exits reported in this study.

Although we noted only two main groups of atrio-nodal connections, it is likely that more than two atrio-nodal connections exist. The current study suggests that the anterograde slow pathway is different from the retrograde slow pathway in some patients, and we have previously presented other evidence for this assertion. Others have demonstrated that the anterograde fast pathway is different from the retrograde fast pathway, and a number of studies have documented three or more AV nodal pathways. Thus, if "AV nodal pathways" are equivalent to atrio-nodal connections, it is likely that three or more atrio-nodal connections exist. Previous work has demonstrated that the compact AV node is connected to atrial tissue via a mass of transitional cells. Clearly, there is a myriad of potential atrio-nodal connections, and it is likely that any two of these have the potential to sustain reentry provided that they possess the appropriate properties of conduction velocity and refractoriness. The two main sites of atrial activation during retrograde conduction are consistent with the anterior and posterior groups of atrio-nodal connections demonstrated histologically.

We also note that the sites of the major atrio-nodal connections correspond to the sites of lesions created in attempts to cure AVJRT. We have demonstrated cure of anterior AVJRT by selective surgical dissection of the anterior AV nodal connections and cure of posterior AVJRT by selective dissection of the posterior atrio-nodal connections. A preliminary report has confirmed the site of these lesions in an animal model. Others have successfully cured AVJRT using catheter ablation techniques directed at either the anterior or posterior AV nodal connections.

The reason for the atypical activation patterns recorded in case 3 is unclear. Rudert et al have reported a similar case in which atrial activation during AVJRT was almost equally early over the entire triangle of Koch. These authors attributed this unusual pattern of atrial activation to an atypical muscle bundle that traversed Koch's triangle. In our case, the atrial musculature appeared normal. It is possible that during AVJRT the site of exit from the AV node in this patient was more leftward (ie, deeper in the atrial septum). Such an exit site would allow activation to spread transversely before reaching the right atrial endocardium.
**Electrogram Morphology and Slow Pathway Potentials**

The finding that electrograms at the site of earliest atrial activation during ventricular pacing or AVJRT have a QS pattern is consistent with theoretical considerations and with previous observations. This information may be useful when attempting to identify the sites of the atrionodal connections during percutaneous electrophysiological studies.

We acknowledge that the “double potentials” reported in this study may not be identical to those described by Haissaguerre et al or those described by Jackman et al since we used unipolar rather than bipolar electrodes. Moreover, unipolar electrodes are more susceptible to “far-field” effects, and thus it is possible that the area covered by double potentials would have been smaller if bipolar electrodes had been used. Nevertheless, the double potentials of the present study were found in a similar position to that described by these authors. Jackman and colleagues have proposed that high frequency potentials similar to that demonstrated in Fig 12 represent depolarization of the slow AV nodal pathway or the atrial termination of this pathway. Furthermore, both Haissaguerre et al and Jackman et al have demonstrated that the application of radiofrequency energy to the site of double potentials results in abolition of the slow pathway and cure of AVJRT.

The finding that the area covered by double potentials is much larger than the area covered by the 3-millisecond isochron during retrograde slow pathway conduction suggests that these potentials are not specific markers for the atrial insertion of the slow pathway. In our opinion, the high frequency component of the double electrogram probably represents the local atrial electrogram, and the accompanying low frequency potential is probably a “far-field” signal from nearby regions (see Fig 12) or from fibers deep in the endocardium. The likely explanation for the genesis of double potentials is the complex three-dimensional anatomy of the posterior septal space. Thus, double potentials may simply be markers for the posterior septal space in which the slow pathway lies rather than markers for the slow pathway itself. Nevertheless, our findings confirm the observation of previous investigators that complex atrial electrograms are present at the point of earliest atrial excitation during retrograde slow pathway conduction.

**Limitations of This Study**

An interelectrode distance of 3 mm was used in this study. It is possible that more closely spaced electrodes would have yielded more information, but we estimate that the differences in activation times of adjacent electrodes in a more closely spaced array would approach 1 to 1.5 milliseconds, which is close to the spontaneous variability in activation time. Thus, it is unlikely that a more closely spaced electrode array would have been useful. Similarly, the use of a lower high-pass filter may have allowed the recording of AV nodal potentials, but when we attempted to use a wider bandpass, we found that variations in the baseline voltage often made the recordings uninterpretable. Our inability to record AV nodal potentials prevented us from defining the route by which atrial impulses entered the AV node. Thus, although the sequence of atrial activation of Koch’s triangle did not alter appreciably whether anterograde conduction occurred via the fast or slow pathway, it is probable that the site of entry to the AV node did, in fact, change. The use of bipolar recording electrodes would have reduced the effects of “far-field” signals, but the magnitude of deflections recorded by bipolar electrodes are dependent on the direction of the wave front of activation, and this effect may have obscured electrical activity in electrode pairs perpendicular to the direction of activation.

Finally, the use of subendocardial or left atrial endocardial electrodes may have allowed us to better define the excitation of the atrial septum, a three-dimensional structure. However, given the time constraints of normothermic cardiopulmonary bypass and the increased risk of systemic embolism engendered by opening the left atrium, we did not attempt to record from these sites.

**Other Observations**

In 1976, Josephson and Kaster demonstrated that in some cases of AVJRT it was possible to advance low atrial activation using atrial premature stimuli without affecting the tachycardia cycle length. They suggested that this indicated that atrial tissue was not a part of the reentrant circuit. A possible alternative explanation of this observation is that a thin rim of atrium that formed a part of the reentrant circuit was not detected by their electrodes. This alternative explanation seems less likely in view of our finding that atrial activity could be advanced in all electrodes overlying the whole of Koch’s triangle without affecting the subsequent ventricular cycle length. If atrial tissue does indeed participate in the reentrant circuit, it is likely that the atrial advancement did not advance His or ventricular activity because of an exact compensatory delay in anterograde AV nodal conduction. It is probable that in these cases the AV nodal anterograde conduction curve has a slope of close to 45°, and thus advancement of atrial activity produces an equal increase in AV conduction time. Furthermore, Brugada and Wellens have demonstrated that a similar phenomenon occurs in accessory pathway-mediated tachycardia in which atrial tissue undoubtedly forms part of the reentrant circuit.

**Conclusions**

At least two distinct groups of atrionodal connections exist. The site of retrograde conduction during anterior AVJRT is similar to that of fast pathway conduction during ventricular pacing. The corresponding atrionodal connection is close to the His bundle–AV node junction. The path of retrograde conduction during posterior AVJRT is similar to that of slow pathway conduction during ventricular pacing. This site is near the coronary sinus orifice, approximately 15 mm from the His bundle. The anterograde slow pathway appears to be different from the retrograde slow pathway in some patients. Complex atrial electrograms are recorded from the right atrial endocardium overlying the posterior septal space and are present at the site of earliest atrial excitation during retrograde slow pathway conduction. These potentials, however, are an imprecise guide to the site of the slow pathway.
Acknowledgments

This work was supported in part by a grant-in-aid from the National Heart Foundation of Australia, ACT, Australia. M.A.McG. is a recipient of a Postgraduate Medical Research Scholarship from the National Health and Medical Research Council of Australia, ACT, Australia. J.P.B. was the 1989 Anglo-Australian fellow of the British Heart Foundation, London. The authors wish to acknowledge the technical assistance of Barbara Dewsnop, Elisabeth Wallace, and Alan Steirn.

References

High resolution mapping of Koch's triangle using sixty electrodes in humans with atrioventricular junctional (AV nodal) reentrant tachycardia.
M A McGuire, J P Bourke, M C Robotin, D C Johnson, W Meldrum-Hanna, G R Nunn, J B Uther and D L Ross

Circulation. 1993;88:2315-2328
doi: 10.1161/01.CIR.88.5.2315
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1993 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/88/5/2315