Mechanisms of Entrainment of Human Common Flutter Studied With Multiple Endocardial Recordings

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Background The mechanisms of common atrial flutter entrainment have not been directly studied in humans. Methods and Results Endocardial mapping in six cases of common flutter showed large right atrial (RA) reentry circuits. Activation was craniocaudal in the anterolateral right atrium and caudocranial in the septum. The inferior vena cava–tricuspid isthmus (IVC-TV) closed the circuit. The high right atrium was paced at progressively shorter cycle lengths (CLs) in all, and the IVC-TV was paced in three cases. We recorded six to eight simultaneous RA electrograms from septum and anterior wall. Transient entrainment was recognized from all sites by capture of all electrograms at two or more paced CLs, with total or partial preservation of baseline flutter sequence and return to baseline after pacing. Antidromic circuit penetration was documented in five cases during high RA pacing and in one with IVC-TV pacing. Short CLs induced orthodromic conduction delays that resulted in a postpacing pause longer than basal flutter CL. ECG fusion with high RA pacing correlated poorly with antidromic septal penetration. This was related to overlap of orthodromic septal activation with anterior wall activation of the following cycle. Pace disorganized flutter into a brief irregular rapid rhythm in two cases and atrial fibrillation in one case. In two cases, complete antidromic septal penetration led to sudden flutter interruption, and in another case it led to circuit inversion.

Conclusions Direct recordings confirm orthodromic and antidromic penetration of flutter circuits by high and low RA pacing. Short CLs modify the circuit. Disorganization is the most common mode of flutter interruption. (Circulation. 1994;90:2117-2125.)

Key Words • atrial flutter • reentry • entrainment • mapping

Waldo et al described that high right atrial (RA) pacing at increasing rates in patients with common atrial flutter could capture the atrium while preserving all or part of the negative component of the flutter waves in lead II of the ECG. If, after pacing, flutter returned to baseline cycle length (CL) and morphology, it was called transient entrainment. The study of entrainment in accessory pathway atrioventricular tachycardia led to its explanation as continual reset of the circuit. The paced front enters the circuit in the orthodromic direction, advancing circuit activation, and in the antidromic direction, colliding with the previous orthodromic front before it completes the circuit. If pacing stops, the last paced orthodromic front restarts reentry.

Entrainment of other reentrant arrhythmias can be recognized from indirect criteria in the surface ECG or with the help of isolated endocardial recordings (Table 1), but the actual events in the circuit are difficult to study. Recent studies in atrial flutter have delineated large RA reentrant circuits that are easily accessible to endocardial recording. This report describes the changes in RA activation sequence during flutter entrainment in humans studied with multiple simultaneous recordings within the circuit, previously defined by mapping.

Received December 4, 1993; revision accepted January 26, 1994.
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Methods

Patients

We studied six patients (four men) aged 51 to 69 years with stable common flutter and clinical indication for conversion to sinus rhythm (Table 2). One had no heart disease, two had mild hypertension, one had sick sinus syndrome, one had chronic obstructive lung disease, and one had coronary artery disease with left ventricular dysfunction after bypass surgery.

Electrophysiological Study

The patients were fasting and were under light sedation with midazolam or midazolam and fentanyl. All antiarrhythmic drugs were interrupted five or more half-lives before the study. In case 5, digoxin was discontinued 48 hours previously. Two hexapolar catheters or one dodecapolar catheter with 1-cm electrode separation were advanced from the femoral vein to the anterolateral and posteroseptal right atrium (Fig 1). Recordings were bipolar (50 to 600 Hz) in cases 1 through 4 and unipolar (30 to 600 Hz) in cases 5 and 6. Amplification and recordings were carried out with an ink-jet polygraph (Minograf, Siemens-Elema). All data were stored on magnetic FM tape for subsequent analysis.

With the stable catheters, we obtained RA electrograms from three or four levels in the craniocaudal direction (Fig 2). A deflecting tip catheter electrode (5-mm separation), advanced from the femoral vein, was used to record sequentially local electrograms from three or four levels of the posterior, posterolateral, lateral, and septal walls. The inferior vena cava–tricuspid valve isthmus (IVC-TV) at the caudal right atrium was reached by rotating the catheter anteriorly (ventrally) and medially. A multipolar catheter was used to map the coronary sinus. Local activation time was taken at the first sharp deflection of bipolar electrograms or at the intrinsic deflection of unipolar electrograms. Simultaneous unipolar
and bipolar electrograms were recorded in cases 2 and 3 with the exploring electrode; the difference in local activation times between both was ≤10 milliseconds.

After mapping, the right atrium was paced at progressively shorter CLs, starting 10 milliseconds below flutter CL, and decreasing by 10-millisecond steps. The same deflector catheter used for sequential mapping was used for pacing. Stimulus intensity was the minimum ensuring continuous capture (6 to 12 mA). The high right atrium was paced in all cases and the IVC-TV in three. Six to eight recordings were obtained during pacing. The protocol ended with flutter interruption or the induction of fibrillation. In four cases (patients 1, 3, 4, and 5), flutter was reinduced and interrupted by radiofrequency ablation of the IVC-TV.12

Ethical Aspects

All patients gave written informed consent for the procedure. The study was approved by the Hospital Human Research Committee.

Results

Basal Activation Sequence

RA activation during common flutter was as described previously9-11: craniocaudal in the anterior wall and caudocranial in the septum (Figs 3 through 7). There was a line of functional block inscribing double electrograms in the posterolateral wall extending cranially from the inferior vena cava. The gap between low anterior and low septal activation was closed through the IVC-TV. Fragmented and/or double electrograms were recorded from the low or mid posteroseptal wall in all cases, and conduction appeared slower in this area (longer interelectrogram intervals).

The downstroke of the flutter wave in lead II coincided with caudocranial septal activation and the upstroke with craniocaudal anterior wall activation (Figs 3 through 7). The relatively flat component coincided with the interval between low anterior and mid septal activation, including the fragmented electrograms of the low or mid posteroseptal area.

Entrainment Mapping

The high right atrium was paced in all cases (Table 2), and in cases 4 through 6, the IVC-TV was also paced. Transient entrainment could be recorded from all pacing sites at two or more CLs by stable capture of all electrograms, with preserved activation sequence in at least part of the circuit and return to baseline after pacing.

Fig 3A illustrates changes induced by pacing the high anterior right atrium at a CL of 175 milliseconds in patient 5. Electrogram sequence and CL after pacing are identical to baseline flutter. Activation is craniocaudal from the RA “roof” toward the low anterior right atrium and caudocranial from the low to the high posteroseptal right atrium. During pacing, there is no change in electrogram sequence or morphology from the mid to the low anterior RA or through the posteroseptal right atrium. The high anterior and “roof” electrograms are changed in morphology and do not precede the mid anterior electrogram, as during flutter. Relative delay of the “roof” electrogram reflects anterograde penetration of the paced activation front but is not deep enough to reach the high septal right atrium. The last paced flutter wave in lead II is superimposed to a QRS complex, but its initial shape is clearly different from the entrained “fusion” waves. As soon as pacing stops, there is no collision with anterograde penetration, and circular activation is reestablished. This illustrates the first criterion of entrainment (Table 1).4

Fig 3B illustrates the end of pacing from the IVC-TV at 200-millisecond CL in the same patient. Electrogram sequences are identical during and after pacing. There is shortening of the interval between the low anterior and low posteroseptal electrograms, indicating advance

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<th>Sex</th>
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<th>Basic AF CL, ms</th>
<th>Pacing Site</th>
<th>Minimum CL Paced, ms</th>
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A indicates anterior; AF, atrial flutter; AL, anterolateral; CABG, coronary artery bypass graft surgery; CL, cycle length; COLD, chronic obstructive lung disease; IVC-TV, inferior vena cava-tricuspid valve isthmus; PL, posterolateral; and SSS, sick sinus syndrome.
of septal activation by orthodromic penetration of the paced activation. This would be the only evidence of fusion, since there is no discernible change in flutter waves in lead II. There is no change in morphology of the low anterior electrogram (other than that produced by the pacing artifact) or in its sequence with the mid anterior electrogram, indicating that antidromic penetration is not reaching the low anterior right atrium. When pacing stops, reentry is reestablished, with a return to a longer interval between the low anterior and low posteroseptal electrograms. This illustrates concealed entrainment.23

**Antidromic Penetration**

Antidromic penetration was variable from case to case and not strictly dependent on paced CL (Table 2). Fig 4A illustrates progressive antidromic penetration of the posteroseptal right atrium in case 4, pacing the high anterior right atrium with shortening CL. Pacing changes morphology and sequence of the high and mid anterior electrograms at all CLs, but sequence remains cranio-caudal from mid to low anterior right atrium. Posteroseptal electrogram sequence and morphology are unchanged pacing at 210-millisecond CL. At 190-millisecond CL, there is change in morphology of the high and mid posteroseptal electrograms that become simultaneous. The mid posteroseptal electrogram still follows the low posteroseptal electrogram. With a 180-millisecond CL, there is further change in morphology of the mid posteroseptal electrogram that now also precedes the low posteroseptal electrogram. Baseline flutter returned after each pacing run. This would be an illustration of the second criterion of entrainment on the ECG (see below) and the fourth criterion in the intracardiac recordings (Table 1).4

Fig 4B illustrates antidromic penetration of the anterior wall during IVC-TV pacing in the same patient. Pacing at CLs of 190- and 180-millisecond electrogram sequence and morphology in the anterior and posteroseptal right atrium remain unchanged from baseline flutter. At 170-millisecond CL, there is a change in morphology of the low and mid anterior electrograms and an advance of the low with respect to the mid anterior electrogram. This is compatible with antidromic penetration to the mid anterior right atrium and collision at this level. Baseline flutter returned after
Fig 3. Case 5, top to bottom: Lead II and unipolar right atrial recordings from roof (R), high anterior (HA), mid anterior (MA), low anterior (LA), coronary sinus ostium (CSO), low posterior septum (LPS), mid posterior septum (MPS), and high posterior septum (HPS). Values are in milliseconds. The drawings represent the right atrium (see Fig 1), with a line of functional block (cross-hatched) in the posterolateral wall. Black arrowheads indicate last paced activation. White arrowheads mark preceding entrained cycle. A, End of pacing at 175-millisecond cycle length. During pacing, the MA electrogram is advanced in relation to R. HA is activated later than LA and R, perhaps because of local anisotropy. Electrogram sequence and morphology through the circuit from MA to HPS remains identical to that during flutter (arrows). Schemes suggest events during entrainment (left) and flutter (right). Approximate location of each recording site is indicated on the drawings. B, End of pacing from the IVC-TV with 200-millisecond cycle length. There is no change in electrogram sequence or morphology during pacing throughout the circuit. Open arrows indicate sites of collision with the preceding and following activation fronts. Scheme on the left suggests activation during entrainment. The second component of the fragmented electrogram in MPS is delayed beyond the HPS electrogram, suggesting that it may be peripheral to the main activation pathway (see Fig 6). S indicates stimulation site.

Fig 4. Case 4, top to bottom: Lead II and bipolar electrograms from high anterior (HA) right atrium (RA), mid anterior RA (MA), low anterior RA (LA), low posterior septum (LPS), mid posterior septum (MPS), and high posterior septum (HPS). Values are in milliseconds. Schemes as in Fig 3. A, Left to right: Baseline flutter and pacing the right atrial appendage (RAA) at cycle lengths (CL) 210, 190, and 180 milliseconds. Note the long interval (150 milliseconds) between LA and MPS electrograms at baseline, covered by fragmented activity (with two main components) in LPS. With CL 190, this interval increases to 185 milliseconds, and there is antidromic capture of HPS and MPS. With CL 180, there is total inversion of septal sequence. Open arrows indicate antidromic penetration by the paced front following that marked by black arrows. Flutter waves start losing depth at CL 210, and there is marked change at CL 190, with little septal penetration. Boxes at the bottom of the figure mark the time frame of recorded anterior RA (upper) and septal (lower) electrograms, not including the fragmented electrograms at LPS. Note overlap during pacing. B, Left to right: Basal flutter and IVC-TV pacing at CLs 190, 180, and 170 milliseconds. Black arrows mark activation sequence. Open arrows mark activation from the previous paced cycle. Note increase in stimulus-MPS interval from 120 milliseconds at CL 190 to 200 milliseconds at CL 170. At CL 170, low anterior right atrial activation is reversed and electrogram morphology changed, indicating antidromic penetration. This would be an illustration of the fourth criterion, because flutter returned after pacing. Flutter waves in lead II, distorted by the stimulus artifact, remain negative.

each of these pacing runs. There are only minimal changes in ECG flutter morphology, which are even more difficult to interpret because of the large pacing artifact. From the surface ECG, these data do not fulfill the first or second criterion for entrainment (concealed entrainment) but do nicely illustrate the fourth criterion (Table 1).

Pacing-Induced Conduction Delay
In five cases (patients 1 through 4 and 6), we recorded lengthening of orthodromic conduction intervals by 20 to 80 milliseconds during pacing. This was localized between the low anterior and mid posteroseptal areas. Figs 4A and 4B illustrate this delay in patient 4. In Fig 4A (pacing high anterior right atrium), the interval from
low anterior to mid posteroseptal electrograms is noted. Baseline value was 150 milliseconds, increasing to 175 milliseconds at 210-millisecond paced CL and 185 milliseconds at 190-millisecond CL. The interval is not increased at 180-millisecond CL because it is now determined by antidromic capture, not orthodromic conduction. In Fig 4B, the interval between the pacing artifact and the mid posteroseptal electrogram is noted in the same patient during pacing at the IVC-TV. The interval increases from 190 milliseconds at a paced CL of 190 milliseconds to 200 milliseconds at a CL of 170 milliseconds, and this allows an increase in antidromic penetration. These recordings localize the delay to the low posteroseptal area beyond the pacing site in the IVC-TV.

Pacing-induced conduction delay could cause post-pacing (return) pauses longer than baseline flutter CL. Fig 5 illustrates this phenomenon in case 4. During pacing conduction, delay is accompanied by slight oscillation of CL at the high and mid septal electrograms but not at the anterior wall sites. The “return” pause (actually the last entrained cycle) is 20 milliseconds longer than baseline. Duration of fragmented electrogram in the low posteroseptal right atrium is clearly longer than in the following pause, when baseline CL is recovered. These recordings again localize conduction delays to the low posteroseptal right atrium.

In cases 2, 3, and 5, some components of posteroseptal fragmented electrograms appeared to represent activation of areas peripheral to the circuit. Fig 6 illustrates such an observation in patient 3. During flutter, after pacing, irregular fragmentation is recorded from the low posteroseptal right atrium, but a sharp deflection in this electrogram precedes the mid posteroseptal electrogram, and overall posteroseptal activation is caudocranial. During pacing from the RA roof at 180-millisecond CL, the sharp component of the low posteroseptal electrogram is still clearly recorded, but it is now later than the mid posteroseptal electrogram. This is not due to reversal of septal activation by antidromic penetration, as shown by the undisturbed sequence of high and mid electrograms. A similar observation can be made in Fig 3 (case 5) during baseline flutter and during entrainment. In case 3 (Fig 6), it also can be observed that some components of the low posterior septum electrogram are intermittently recorded. The myocardium generating the delayed and intermittent components would not belong to the circuit.

Mechanisms of Fusion

The extent of recorded septal antidromic penetration was not closely related to fusion in the ECG. Fig 6 shows marked loss of depth of flutter waves pacing the high right atrium in case 3, whereas posteroseptal wall recordings still show caudocranial activation, that is, lack of antidromic penetration. ECG fusion appeared related to overlap of cranio-caudal anterior wall activation advanced by pacing, with caudocranial septal activation from the previous cycle (Figs 4A, 7A, and 8). This effect was due to the long conduction times in the caudal right atrium accentuated by pacing.

During IVC-TV pacing in case 4 (Fig 4B), there was antidromic penetration of the anterior wall with partial reversal of cranio-caudal activation, but there was minimal or no change in flutter morphology in lead II. During IVC-TV pacing, there was no overlap of septal and anterior wall activation (Figs 3B, 4B, and 8).

This overlap of septal and anterior RA activation of consecutive cycles during pacing also seemed to have a role in double-spikewave electrogram fusion. Fig 7A illustrates double electrograms recorded from the high posterior right atrium in case 1. During high RA pacing at 190-millisecond CL, there is no change in electro-
Fig 7. Case 1: Bipolar electrograms (see Figs 4 through 6 for abbreviations). Pacing high anterolateral (HAL) RA. Values are in milliseconds. Schemes as in Fig 3. Boxes below lead II mark time frame of recorded anterior wall (upper) and septal (lower) electrograms, not including fragmented electrograms at LPS.

A. Left to right: Baseline flutter and pacing at 190-, 180-, and 170-millisecond cycle length (CL). Arrows mark direction of activation. Note baseline LA-MPS interval of 120 milliseconds increasing to 185 milliseconds at CL 180. At 185 milliseconds, there is loss of flutter wave depth without antidromic septal penetration. Note overlap of anterior wall and septal activation. At 180 milliseconds, there is further flattening of flutter waves, and the two components of the HP electrogram fuse. There is still no reversal of septal activation sequence, but note further overlap of anterior wall and septal activation (boxes). At 170 milliseconds, there is reversal of septal electrogram sequence, and HP shows only one component. Flutter returned after pacing, suggesting block of the antidromic front (cross-hatched zone in Koch's triangle).

B. Flutter interruption by a second pacing run at 170-millisecond CL. Left tracing illustrates progressive septal antidromic penetration (open arrows indicate activation from the previous paced cycle). Right tracing shows that antidromic septal penetration is maintained to the end of pacing. Activation of the septum and anterior wall in the first post pacing cycle (large arrowhead on top) shows a craniocaudal sequence as the following sinus beat but occurs early.

There is no change in electrogram sequence in this area. However, the 180-millisecond CL induced a conduction delay between the low anterior and mid posteroseptal electrograms that resulted in complete overlap of anterior wall and (preceding) posteroseptal activation. The sequence of the first component of the high posterior electrogram in relation with the mid posteroseptal electrogram and of the second component of the high posterior electrogram with the mid anterior electrogram are unchanged. This again supports that double potentials represent activation of the craniocaudal and caudocranial arms of the circuit.10,13

Flutter Interruption

Pacing led to flutter interruption in five patients (Table 2). In cases 1 and 6, interruption was sudden, at the end of pacing. In cases 2 and 4, pacing induced a fast, self-limited, irregular tachycardia leading to sinus rhythm in seconds. In case 3, pacing induced atrial fibrillation that led to sinus rhythm after 15 minutes. In case 5, pacing inverted circuit rotation, and the inverted flutter was interrupted suddenly by pacing.

Fig 7B shows sudden interruption in case 1. High RA pacing progressively reversed the septal activation sequence, and sinus rhythm resumed after pacing. The first postpacing cycle was close to flutter CL, with an activation pattern suggesting a sinus node origin. This could be explained by local or sinus reentry or by an early sinus escape in the presence of atrio-sinus block during pacing. A similar early postpacing cycle also was observed in case 6 at the time of sudden flutter interruption.

This mode of flutter interruption differs from the third criterion of entrainment (Table 1)4 only in that progressive penetration, not local activation block, was apparently responsible for septal activation inversion. A previous pacing run at the same CL did not interrupt...
flutter despite apparent complete septal antidromic penetration, thus illustrating the fourth criterion of entrainment (Table 1)4 (Fig 7A, far right tracing). A possible explanation, suggested in the figure, would be that the antidromic front could be blocked before it collided with the orthodromic front, leaving the later free to reenter when pacing stopped.

Fig 9 illustrates reversal of circuit rotation in case 5 (for reference, see baseline flutter in Fig 3A). The left tracing shows progressive antidromic septal penetration until septal activation was inverted completely. However, at the end of pacing, flutter is present but with a completely inverted electrogram sequence. Sequential mapping of the new flutter again demonstrated a line of conduction block in the posterolateral RA and closure of reentry through the IVC-TV. Pacing entrained and suddenly interrupted the inverted flutter. Flutter waves in lead II had a different morphology but still appeared negative, confirming previous observations that the direction of circuit rotation does not correlate well with ECG morphology.11 Circuit inversion by pacing is incompatible with collision of orthodromic and antidromic paced activation and suggests block of orthodromic activation before the slow conduction zone, allowing reentry of the last paced antidromic activation to reverse circuit direction.

Fig 10 illustrates the changes leading to flutter interruption in case 4 during IVC-TV pacing (for reference, see Fig 5B). During the last five paced cycles, the posteroseptal electrograms slightly precede the pacing artifact, and the interval from this artifact to the low and mid anterior electrograms decreases progressively. When pacing stops, a rapid, slightly irregular rhythm persists, with caudocranial activation of the septum and caudocranial activation of the low-to-mid anterior right atrium that progressively becomes caudocranial all the way to the high anterior right atrium. The two cycles after pacing termination show electrogram sequence and morphology almost identical to the last paced cycle, suggesting that this rhythm already was present at that time. This relatively organized tachycardia would be compatible with a new reentrant circuit located in the left atrium, with septal participation. The end of this tachycardia could be due to a lack of stability of reentry and perhaps is due to fatigue in some part of the new circuit. Note that the two last complexes are slower and show a definite change in electrogram sequence.

Discussion

Accepted entrainment criteria (Table 1) mainly are based on observations in atrioventricular or ventricular tachycardia.2-4 The effect of premature stimuli14,15 or pacing7,10,13,15-17 on human flutter circuits has been studied with atrial recordings, but the relation of the recording sites with the circuit was unknown. By defining the circuit previously and placing the recording electrodes selectively throughout the circuit, our study affords more complete data on the mechanisms of flutter entrainment.

Circuit Penetration

We can be reasonably certain that our recordings were obtained very close to or within the circuit because our RA maps showed circular activation covering 100% of the flutter cycle. Furthermore, the validity of the map was confirmed in four cases by the effect of local ablation of a critical zone of the circuit.12 In this setting, we can define entrainment directly, ie, by observation of actual capture of the circuit and return to baseline after pacing, as in animal experiments reported by others.18-21 This allows testing the limits of the currently used indirect criteria of entrainment.

Caudocranial septal activation during high RA pacing was a clear demonstration of orthodromic circuit penetration. When flutter was interrupted, high RA pacing
and/or sinus rhythm resulted in craniocaudal septal activation, ruling out fixed block as the cause of caudocranial septal activation during entrainment. In fact, we could also record antidromic septal capture in four cases during entrainment at shorter CLs. Recordings showed flutter reinitiation by the last paced orthodromic front.

**Collision Versus Block**

Block of entrained activation not caused by collision has been documented in animal flutter and can be the mode of interruption of the circuit. In our cases, we did not directly record local block leading to circuit interruption, as described by Henthorn et al. In most pacing runs, collision of antidromic and orthodromic activation could explain the sequence of constant reset followed by return to reentrant activation after pacing. However, some of our observations can be explained best by separate block of activation fronts. Reversal of circuit rotation in case 5 is compatible with block of orthodromic activation before the slow conduction zone followed by reentry of the antidromic front. In case 1, block of the antidromic front may also explain flutter reinitiation despite deep septal penetration (Fig 7A). In both cases, block apparently occurred at or close to the area of slow conduction, as emphasized by Schimizu et al.

**Pacing-Induced Conduction Delay**

Pacing modified the circuit by inducing rate-dependent conduction delays in orthodromic activation, as described by Waldo et al. The slow conduction zone in the low septal RA appeared to be the source of the delays. However, the meaning of fragmented electrograms in the low and mid posteroseptal areas must be interpreted with caution, as indicated by the dissociation of some of the electrogram components from circuit activation sequence (Fig 6). This finding is consistent with the data obtained by Saumarez et al. with fine mapping of Koch's triangle in a case of flutter during surgery.

Rate-dependent conduction delays could explain a postpacing pause longer than basal CL even when recording within the circuit, an effect also described with single extrastimuli. In one case, we recorded conduction interval oscillation (Fig 5). Rate-dependent conduction delays described during animal flutter entrainment can modify the length and location of areas of functional block forming the center of the circuit.

**Mechanisms of Fusion**

We recorded constant fusion as partial orthodromic and partial antidromic activation of the circuit; however, this did not correlate closely with changes in ECG morphology. Antidromic penetration of the anterior wall during IVC-TV pacing did not produce fusion (Fig 5B); there was ECG fusion during high RA pacing without inversion of septal activation, and, finally, fusion of double electrograms recorded in the posterior wall was not correlated with antidromic penetration. Fusion during high RA pacing was largely related to the overlap of anterior wall activation with septal activation from the preceding cycle (Figs 5A, 7A, and 8), an event to be expected but not emphasized previously. We obtained recordings only from three or four sites from each wall, but these were widely separated in the craniocaudal direction and appeared to encompass rapid caudocranial and craniocaudal activation coinciding with the downward and upward deflections of the flutter wave in lead II. Overlap was due to the location of the slow conduction area between the anterior and septal walls in the circuit and was accentuated by pacing-induced orthodromic conduction delay. During IVC-TV pacing, the slow conduction zone did not separate activation of the anterior and septal walls, and there was no overlap (and no ECG fusion) (Figs 3B, 4B, and 8). These observations emphasize the importance of the location of the slow conduction zone in relation to the pacing site for the appearance of fusion.

**Circuit Interruption**

The appearance of atrial fibrillation or a short-lived, faster, irregular tachycardia was the mode of flutter interruption in three of our cases. In two cases, the mode of interruption suggested complete antidromic and orthodromic circuit penetration with collision, as suggested by Waldo et al. These patients displayed good examples of the third criterion of entrainment. Our recordings showed progressive antidromic penetration rather than local block as the mechanism of the "third criterion"; however, if we had had only a mid or low posteroseptal electrogram, the difference between the two mechanisms would not be apparent. The mechanism of the postpacing short-cycle activation observed in these two patients is intriguing. An interesting possibility is the orthodromic activation front turning back on its path ("reflection and echo wave termination"), as recently described by Boersma et al. in experimental reentry.

In other patients, despite evidence of antidromic penetration as shown by changes in electrogram sequence and morphology, interruption occurred through the appearance of rapid transitional rhythms. It is possible that the pacing rate was increased too fast in our protocol, not allowing a full manifestation of the spectrum of entrainment. The production of irregular rhythms by pacing flutter is a common observation both in clinical practice and in the experimental laboratory. This could be due to the induction of functional reentry by rapid pacing, as shown by Allessie et al. We did not have enough recordings to obtain instantaneous maps, and the rhythms produced in our patients were too short for sequential mapping. Nevertheless, a certain regularity in activation sequence suggest that rapid, unstable, localized reentry could be the origin of these transitional rhythms. In experimental preparations based on large anatomic obstacles, disorganization is also a common form of interruption by pacing.

The stability of the flutter circuit is impressive. Through the changes produced by rapid pacing, the complex mix of anatomic and functional factors that make flutter possible may remain stable even to the point of supporting circuit inversion without total disruption. This would suggest a very solid structural base, probably the combination of anatomic "holes" and muscle bundle alignment.
Study Limitations

We obtained recordings from only a few points of the right atrium, and in most cases these were bipolar with a 10-mm interelectrode separation. Nevertheless, flutter circuits appear large enough to yield some information even with this recording method.5-7-10 The single dodecapolar catheter technique, using unipolar electrograms, appears promising in this regard, although there is more interference by ventricular activation.

Another limitation is the unknown “inner limit” of the circuit. Even accepting rotation around the inferior vena cava and a line of functional block in the postero-lateral right atrium, the width of anterior and septal RA walls still leaves room for multiple reentrant paths. In our study, both pacing and recording may have taken place outside the shortest activation path, and this may be the explanation of electrogram sequence distortion close to the pacing site in the high right atrium. Electrogram sequence in the absence of antidromic penetration was perfectly preserved by pacing the IVC-TV, the narrowest area of the circuit.

Finally, no left atrial recordings were obtained during pacing, and this may limit the value of our observations on the mechanisms of fusion patterns on the ECG, since left atrial activation is an important determinant of flutter wave morphology.15 With this lack of left atrial activation data, it is difficult to make a correlation of apparent flutter wave polarity with direction of circuit rotation.11,12 Mapping studies from our laboratory and others5-10,15 have shown that left atrial activation tends to coincide in time with septal activation, and septal penetration may thus reflect in part the events in the left atrium. In any case, this deficiency does not invalidate the observation that fusion in the ECG may occur in the absence of antidromic septal penetration during high RA pacing.

Acknowledgments

This study was supported by Grant PM89-0035 from Comisión Asesora de Investigación Científica y Técnica. Ministerio de Educación y Ciencia, Spain. We are indebted to nurses Pilar Adoué, Isabel de las Fuentes, and Pilar Gómez for their invaluable cooperation and to Ana Fernández for secretarial work. We thank Dr Jesús Almendral for his comments and review of the manuscript.

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_Circulation_. 1994;89:2117-2125
doi: 10.1161/01.CIR.89.5.2117
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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