JACC Vol. 15, No. 4
March 15, 1990:833–41

ELECTROPHYSIOLOGIC STUDIES

Characterization of Double Potentials in Human Atrial Flutter: Studies During Transient Entrainment

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Double potentials, defined as atrial electrograms with two discrete deflections per beat separated by an isoelectric interval or a low amplitude baseline, have been observed during right atrial endocardial mapping of human atrial flutter. In this study, bipolar atrial electrograms were recorded during atrial flutter (mean cycle length 235 ± 27 ms [± SEM]) from the high right atrium, the His bundle region, the coronary sinus and at least 30 right atrial endocardial mapping sites in 10 patients. Double potentials were recorded from the right atrium in all patients during atrial flutter.

Double potentials were evaluated during transient entrainment of atrial flutter by rapid high right atrial pacing in 5 of the 10 patients. In four of these five patients during such transient entrainment 1) one deflection of the double potential was captured with a relatively short activation time (mean interval 89 ± 45 ms) and the other deflection was captured with a relatively long activation time (mean interval 233 ± 24 ms), producing a paradoxical decrease in the short interdeflection interval from a mean of 75 ± 20 ms to a mean of 59 ± 24 ms; and 2) the configuration of the double potential remained similar to that observed during spontaneous atrial flutter. On pacing termination 1) the two double potential deflections were found to be associated with two different atrial flutter complexes in the electrocardiogram (ECG); 2) the previous double potential deflection relation resumed; and 3) when sinus rhythm was present, the double potentials were replaced by a broad, low amplitude electrogram recording at the same site. These functional double potentials probably represent collision of activation wave fronts in a functional center of the atrial flutter reentrant circuit and therefore may serve as a marker for an area of functional block. In one of the five patients, double potentials were recorded from the same site during transient entrainment of atrial flutter, during spontaneous atrial flutter and during sinus rhythm. These were called persistent double potentials and were associated with the same atrial flutter complex in the ECG, indicating that not all double potentials recorded during atrial flutter represent the same phenomenon.

(J Am Coll Cardiol 1990;15:833–41)

Double potentials have been observed during mapping of various tachyarrhythmias in human and in animal models (1–7). Recently, double potentials have been recorded in the right atrium during atrial flutter in patients (6,7). Various mechanisms have been postulated for generation of these double potentials (1–3,7), but their etiology is not known. Data from our canine model of atrial flutter (5) and the rabbit and canine atrial models of leading circle reentry of Allessie et al. (1,3) have shown that double potentials seem to occur in the functional center of a reentrant circuit.

We postulated that transient entrainment, defined as pacing capture of a tachycardia recognized by any of four criteria previously outlined (8–11), would allow a better understanding of the nature of double potentials that occur in human atrial flutter. We therefore used the principles of transient entrainment (8–24) to 1) gain insight into the nature of these double potentials, 2) characterize the relation of double potentials to the atrial flutter complex in the electrocardiogram (ECG), and 3) test the hypothesis that these double potentials recorded in the right atrium during human atrial flutter are secondary to two different wave fronts of activation colliding in the center of a reentrant circuit (3,4).

From the University Hospitals of Cleveland/Case Western Reserve University, Cleveland, Ohio. This study was supported in part by Grant R01HL38408-01 from the Department of Health and Human Services, Public Health Service, National Heart, Lung, and Blood Institute, Bethesda, Maryland and by a Research Initiative Award from the American Heart Association, Northeast Ohio Affiliate, Cleveland, Ohio.

Manuscript received March 20, 1989; revised manuscript received September 8, 1989, accepted October 20, 1989.

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0735-1097/90/$3.50

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Table 1. Data in 10 Patients

Patient	Age (yr)/ Gender	Diagnosis	AFl CL Before PA (ms)	AFl CL After PA (ms)	PA Dose (mg)	Double Potential Sites	
						Septal	Free Wall
1	55/M	COPD, R-CHF	205	—	None	—	E4, B5, C4, C5
2†	59/M	CMP	210	250	500	—	C5, A4
3*	65/M	CA s/p rupture	215	—	None	E3, E4	B1, B2, C2, C3, D3
4	50/M	CAD s/p CABG	235	280	1,000	—	A5
5†	70/M	SSS, neck mass	240	360	1,000	E2	B5, C4, D4, D5
6†	76/M	MVP	240	285	1,000	E3	C4, D5
7	33/M	ETOH-CMP, VSD repair	265	320	500	E1	A3, C3, C4
8†	48/M	CAD s/p CABG	210	270	1,000	—	C4
9	71/F	CAD	235	370	500	—	B4
10	63/M	CAD/CMP	290	370	1,000	E3	B4, B5, C4, C5, D5
Mean SD	59 ± 13		235 ± 27	313 ± 48			

*Patient with persistent double potentials; †patient with functional double potentials in whom transient entrainment was performed. AFl = atrial flutter; CA = esophageal cancer; CABG = coronary artery bypass grafting; CAD = coronary artery disease; CL = cycle length; CMP = cardiomyopathy; COPD = chronic obstructive pulmonary disease; ETOH = alcohol; F = female; M = male; MVP = mitral valve prolapse; PA = procainamide; R-CHF = right-side heart failure; s/p = status post; SSS = sick sinus syndrome; VSD = ventricular septal defect. (Data from Patients 2 and 3 used for Fig. 3 to 6 and Fig. 7 to 9, respectively.)

Methods

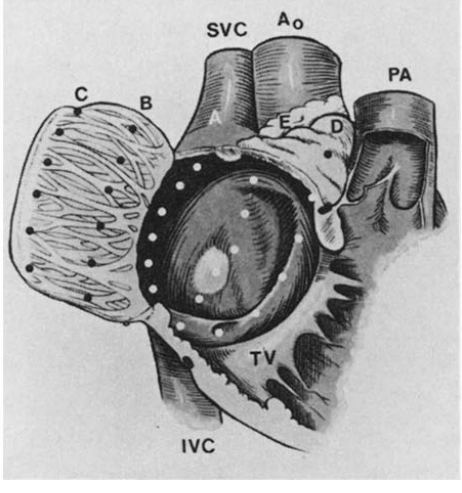
Study patients (Table 1). Ten consecutive patients with spontaneous atrial flutter present for >24 h were studied. No patient was in the immediate period following open heart surgery. Atrial flutter was first diagnosed from the surface ECG and confirmed from subsequently recorded intra-atrial electrograms (25). Stable hemodynamics prevailed and a controlled, clinically satisfactory ventricular response rate was present in all patients.

Electrophysiologic Study

Protocol. Informed consent was obtained using a protocol approved by the Institutional Review Board. Patients were studied in the cardiac catheterization laboratory with standard electrophysiologic techniques in the absence of all antiarrhythmic medications for more than five half-lives. However, all patients were receiving digoxin. An exploring quadripolar catheter electrode with a 2 mm space between the electrodes of each pair and a 5 mm space between the electrode pairs was used to map the right atrium during atrial flutter. Custom-designed Brockenbrough steerable catheters were used in four patients, but we found no advantage in the use of these catheters over the use of standard quadripolar catheters. The exploring catheter was used to search for double potentials by recording bipolar electrograms sequentially from 30 right atrial endocardial sites (Fig. 1). Double potentials were defined as bipolar atrial electrograms with two discrete potentials (deflections labeled x and y for purpose of identification) per beat either separated by an isoelectric baseline or a low amplitude interval (Fig. 2). Single plane fluoroscopy was used to guide the placement of the exploring catheter.

Atrial mapping. Mapping was performed systematically in all patients. The catheter was initially placed in the high right atrium to coincide with the region of site A_1, and the atrium was explored from its superior to its inferior aspect. Then the catheter was torqued more medially, again placed superiorly and the process repeated with each row mapped in turn. Each recording site was separated by a 1 to 2 cm

Figure 1. Mapping grid used for determination of the atrial activation sequence and investigation of double potentials during atrial flutter. **Row A** represents the most lateral right atrial free wall. **Rows B and C** represent the right atrial free wall. **Row D** involves the most medial aspect of the right atrium as well as the tricuspid ring, the His bundle region and the right atrial appendage. **Row E** is the interatrial septum. The sites in each row were numbered 1 through 6 from the cranial to the caudal aspect. A_o = aortic valve; IVC = inferior vena cava; PA = pulmonary artery; SVC = superior vena cava; TV = tricuspid valve.



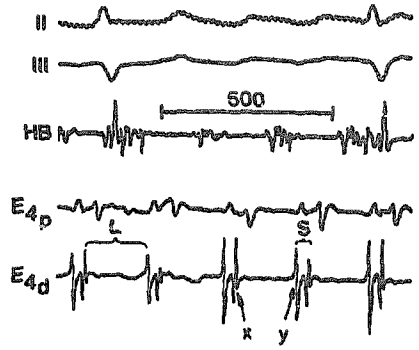


Figure 2. Electrocardiographic leads II and III recorded simultaneously with bipolar atrial electrograms during atrial flutter (cycle length 220 ms) from near the His bundle (HB) position and from the proximal pair (p) and distal pair (d) of a quadripolar electrode catheter located at site E_4 (a low right atrial septal site). Double potentials (x and y) are recorded from the E_{4d} site. L, the long isoelectric interdeflection (x–y) interval between double potentials, is 180 ms. S, the short interdeflection (y–x) interval (note: not isoelectric) between the double potentials, is 35 ms. Fractionated, low amplitude atrial electrograms recorded at the His bundle position occur temporally during the x–y isoelectric interval. Site E_4 is in close proximity to the His bundle catheter (located at site D_4) in the right atrium. The bar indicates 500 ms. See text for discussion.

distance. Within the constraints of the technique, the goal was to map the entire right atrium from as many different sites as possible. Mapping was performed to search systematically for abnormal atrial electrograms, not to evaluate the atrial activation sequence for purposes of this study. The size of the reentrant circuit, the circuit center and the zone of slow conduction was not assessed for this study. Multipolar catheter electrodes were also placed in the high right atrium (10 patients), low lateral right atrium (10 patients), coronary sinus (9 patients) and the His bundle region (8 patients) to be used for either bipolar recording or bipolar pacing. The coronary sinus catheter was always placed with the recording electrode located at the proximal coronary sinus. The catheter near the His bundle region was purposely placed to record the right atrial septal electrogram (but across the tricuspid ring); the gain was set to obtain an atrial electrogram, not to obtain a His bundle electrogram.

During each study, all bipolar electrograms were filtered between a band pass of 10 and 500 Hz and recorded simultaneously with ECG leads I, II, III and V_1 on photographic paper at 100 mm/s on an Electronics-for-Medicine VR16 switched-beam oscilloscopic recorder. All data were simultaneously recorded on a Honeywell 5600C FM tape recorder for subsequent playback and data analysis. A Medtronic 1349A programmable stimulator was used for pacing.

Atrial pacing: transient entrainment. First, sequential site atrial mapping during atrial flutter was performed. Then rapid atrial pacing was performed during atrial flutter at twice the stimulus threshold from the high right atrium at selected rates progressively faster than the spontaneous rate of the atrial flutter in order to demonstrate criteria for transient entrainment (8–11). Pacing was always initiated at a cycle length 10 ms shorter than the spontaneous atrial flutter cycle length. After establishing atrial capture (acceleration of all recorded atrial electrograms to the pacing rate), atrial pacing was continued in all patients for at least 15 s. On cessation of pacing, if the atrial flutter continued, the pacing cycle length was shortened by 10 ms or the pacing rate was increased by 10 beats/min and pacing was again performed in the same manner. Pacing was performed at a mean of 6 (range 4 to 8) incrementally faster pacing rates.

For the purposes of this study, transient entrainment of atrial flutter was considered present 1) if during pacing at a constant rate faster than the atrial flutter rate, constant atrial fusion beats were demonstrated in the ECG except for the last captured atrial beat that was not fused, or 2) if progressive fusion of atrial beats was demonstrated in the ECG (that is, if constant atrial fusion beats occurred in the ECG during rapid atrial pacing at each of two or more constant rates faster than the atrial flutter rate, but with different degrees of fusion at each pacing rate) (8,13,15). During rapid atrial pacing of atrial flutter in five patients, criteria for transient entrainment were demonstrated when double potentials could be recorded from the distal two electrodes of the exploring catheter electrode stabilized at a recording site in the low right atrium.

Atrial mapping in sinus rhythm. Following the demonstration of transient entrainment, atrial flutter was interrupted in all patients by rapid atrial pacing (after administration of 500 or 1,000 mg of procainamide given intravenously, as a rule, at 50 mg/min, in eight patients) (Table 1). Procainamide was administered even more slowly when necessary, to maintain adequate blood pressure. Then the patients had atrial mapping performed during sinus rhythm to search for the presence of double potentials. In addition, rapid atrial pacing was performed in seven patients during sinus rhythm from the high right atrium at a rate similar to the previous atrial flutter rate to compare the activation time and electrogram configuration at selected atrial recording sites (especially where double potentials were previously observed) with those during atrial flutter.

Results

Presence of double potentials (Table 1). During sequential site endocardial atrial mapping, double potentials (Fig. 2 and 3) were recorded during atrial flutter (mean cycle length 235 ± 27 ms; range 205 to 290 ms) in all 10 patients. Double potentials were recorded in each patient from at least one site in the low right atrium and remained stable during the recording period. Such sites were always in relatively close proximity to sites from which fractionated electrograms were recorded (Fig. 2). The distance between fractionated

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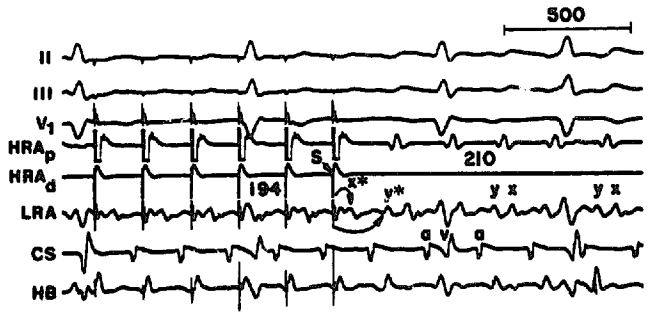


Figure 3. Patient 2. Representative example demonstrates the use of transient entrainment (via high right atrial pacing) during atrial flutter to evaluate the deflections of recorded double potentials. Electrocardiographic leads II, III and V_1 were recorded simultaneously with bipolar atrial electrograms from the proximal pair (p) and distal pair (d) of a quadripolar electrode catheter located at a high right atrial (HRA) site, at a low right atrial (LRA) free wall site, at a coronary sinus (CS) site and at a site near the His bundle (HB) where atrial activity was demonstrated, at the termination of high right atrial pacing (pacing cycle length 194 ms). The last pacing stimulus (S) activates the deflections of the double potentials, x* and y*, recorded at the low right atrial site. The activation time from the stimulus to the x* deflection is relatively short (55 ms), whereas activation time from the stimulus to the y* deflection is relatively long (205 ms). Y* is the last paced (captured) deflection of the double potential because it occurs at the pacing cycle length and not at the atrial flutter cycle length. The CS and HB atrial electrograms are activated by long activation times (140 and 200 ms, respectively), but the CS atrial electrogram precedes y*. During atrial flutter, the long interval between deflections (x–y) is 140 ms and the short interval (y–x) is 70 ms. The apparent shortening in the y–x interval is not an actual shortening of the interdeflection interval because the two potentials are associated by the long interval (x–y), which does not shorten. All numbers are in milliseconds. See text for discussion.

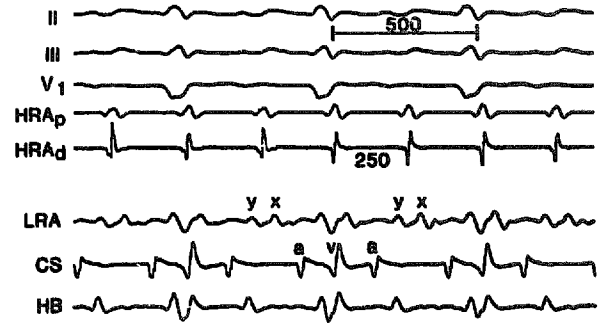


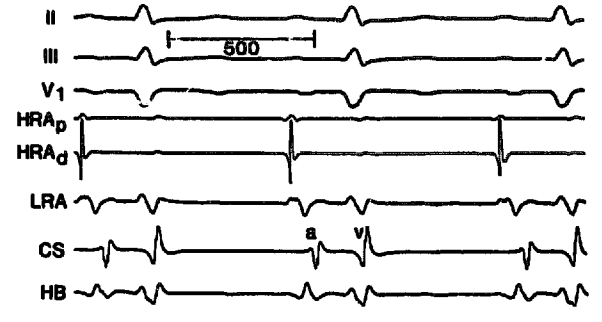
Figure 4. Same patient as in Figure 3. Although the atrial flutter rate has slowed after administration of intravenous procainamide, double potentials are still present at the same site. The long coupling interval, x–y, lengthened to 170 ms and the short coupling interval, y–x, lengthened to 80 ms, but the configuration of the two deflections did not change. All numbers are in milliseconds.

and double potentials was no more than two sites on the mapping grid (Fig. 1). Although single plane fluoroscopy may have limited our ability to map precisely the exact double potential location, double potentials were reproducibly recorded at least twice at a similar site for each patient studied.

In seven patients, double potentials could be recorded from multiple low right atrial free wall sites (mean 3.6 ± 1.3, range two to five sites/patient). In five patients, double potentials were also recorded from the interatrial septum (mean 1.2 ± 0.4, range one to two sites/patient). These septal double potentials were discrete and spatially separated from the free wall double potentials. In those patients in whom procainamide was administered before interruption of the atrial flutter, the double potentials remained present at the same site until pacing interrupted the atrial flutter (Fig. 4 to 6).

Transient entrainment of atrial flutter. In all patients, transient entrainment criteria (first criterion in all patients, second criterion in seven patients, see Methods section) were demonstrated, thereby establishing that reentry was the most likely cause for atrial flutter (13–19). During transient entrainment of atrial flutter by high right atrial pacing, there was no change either in the relative sequence of atrial activation at the electrode recording sites (coronary sinus, His bundle position and low right atrium) or in the atrial electrogram configuration from those observed during spontaneous atrial flutter (Fig. 3). Also a long activation time was demonstrated from the pacing site to the atrial electrograms recorded at the coronary sinus (228 ± 54 ms, n = 9). This finding is consistent with the presence in the atrial flutter reentrant circuit of an area of slow conduction somewhere between the high right atrium and the coronary sinus recording site. An alternative explanation is the presence of a very long pathway of activation between these sites. However, the latter explanation is unlikely in view of the anatomy of the atria, the limited possible activation pathways between these two sites and the fact that analysis of activation of the various grid sites during atrial flutter was not consistent with such a long pathway. Atrial pacing was not performed at

Figure 5. Recorded from the same patient as in Figures 3 and 4. Note that in the recordings from the same low right atrial recording site, no double potentials were observed during sinus rhythm after administration of procainamide from the same low right atrial (LRA) site as in Figures 3 and 4. Instead, the atrial electrogram is broad. Both atrial (a) and ventricular (v) electrograms are shown. The bar indicates 500 ms.



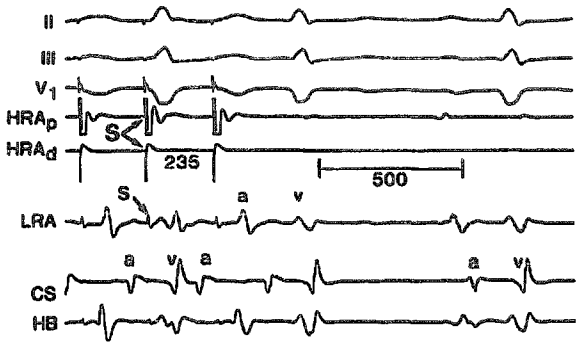


Figure 6. Same patient as in Figures 3, 4 and 5. Rapid atrial pacing performed during sinus rhythm after procainamide administration demonstrates no double potentials. From the same low right atrial recording site as seen in Figures 3, 4 and 5, rapid high right atrial pacing (S) at a rate similar to atrial flutter produces only broad, low amplitude atrial (a) and ventricular (v) electrograms at this same low right atrial site, but no double potentials. All numbers are in milliseconds.

multiple sites during atrial flutter, a potential limitation of this study.

Observation of double potentials during transient entrainment. In four patients, double potentials, which were recorded in the right atrial free wall during transient entrainment of atrial flutter, demonstrated a consistent finding: An unexpected relation was noted between the deflections of the double potential. For these patients, the mean cycle length of their atrial flutter was 225 ± 17 ms (range 210 to 240 ms) (Table 1). During atrial flutter, the double potentials had a mean short interdeflection time (y–x interval) of 75 ± 20 ms (range 55 to 100 ms), and a mean long interdeflection time (x–y interval) of 155 ± 12 (range 145 to 170 ms) (Fig. 2). When high right atrial pacing achieved transient entrainment, the configuration of the y deflection of the double potential did not change (Fig. 3), suggesting that it was activated orthodromically. It was difficult to ascertain whether the x deflection of the double potential always had a configuration identical to that during atrial flutter because it was often temporally within the stimulus artifact (an inherent limitation of the recording technique) (Fig. 3), but when it could be dissociated from the stimulus artifact during transient entrainment, the configuration was the same, suggesting orthodromic activation of that deflection. All double potential interdeflection intervals (x–y and y–x) resumed their previous relation after pacing termination if atrial flutter was still present (Fig. 3).

In one patient, the double potentials recorded during atrial flutter and high right atrial pacing of atrial flutter responded differently from those of the four patients described. The results of atrial pacing in this patient are described in a subsequent section.

Effect of rapid pacing in the double potentials. During transient entrainment with high right atrial pacing (mean pacing cycle length 192 ± 17 ms) of atrial flutter, one deflection of the double potential (x) was captured with a relatively short activation time (mean stimulus to x interval 89 ± 45 ms) and the other deflection (y) was captured with a much longer activation time (mean stimulus to y interval 233 ± 24 ms). This reference resulted in an apparent decrease of the short (y–x) interdeflection interval from a mean of 75 ± 20 ms to a mean of 59 ± 24 ms during high right atrial pacing. This confirms the recent observations of Cosio et al. (7). Actually, the apparent decrease resulted only because the atrial rate increased from the spontaneous atrial flutter rate to the atrial pacing rate during transient entrainment. The long interdeflection interval (x–y) represents the interval by which the deflections were associated during high right atrial pacing. This interval did not lengthen with more rapid pacing. Only the y deflection (which occurred with a long activation time from the pacing impulse, that is, the stimulus to y interval) was associated with the next atrial flutter complex on the ECG (Fig. 3). This is consistent with activation of the y deflection by a wave front that had traveled through an area of slow conduction, and with activation of the x deflection by a wave front, either in or outside the reentrant circuit, that had not. The long activation time could also be explained by conduction over a very long pathway but, as indicated, this is unlikely. The fact that the x–y interval did not lengthen with increasingly more rapid pacing suggests that the properties of the area of apparent slow conduction were not identical electrophysiologically to those of the atrioventricular (AV) node.

Are the x and y deflections immediately proximal and distal to an area of slow conduction? The notion of "bracketing" an area of slow conduction was recently advanced to explain double potentials recorded in a patient during ventricular tachycardia (26). An observation addresses this question: With high right atrial pacing during atrial flutter, the coronary sinus atrial electrogram was always activated with a long activation time, but before the y deflection of the double potential (Fig. 3). The long activation time indicates that the wave front of activation to the coronary sinus site also traveled through an area of slow conduction. But activation of the coronary sinus site before the y deflection makes it rather difficult to consider that the y deflection is immediately distal to the area of slow conduction. The proximal coronary sinus atrial electrogram always appeared to be the earliest atrial activation measured compared with the negative deflection of the flutter wave seen on the surface ECG.

Double potentials during sinus rhythm and rapid pacing of sinus rhythm. In 9 of the 10 patients, interruption of atrial flutter by rapid atrial pacing was associated with disappearance of all double potentials (Fig. 5 and 6). During sinus rhythm in these nine patients, sequential site atrial mapping was performed expressly to search for the presence of double potentials, but none were found. Also, the double

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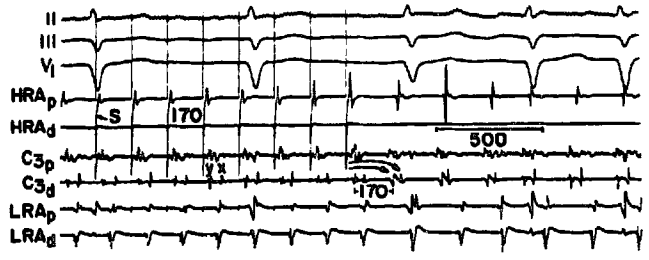

Figure 7. Patient 3. Persistent double potentials. High right atrial pacing performed during atrial flutter demonstrates the influence of pacing on double potentials recorded at a low right atrial free wall site (C_3) during atrial flutter in the one patient whose double potentials responded differently to high right atrial pacing. ECG leads II, III and V_1, as well as electrograms recorded from the proximal (p) and distal (d) electrode pairs from electrode catheters located at a high right atrial (HRA) site, at C_3 (a free wall site) and a low right atrial (LRA) site. The interval between the deflections was greater during rapid atrial pacing than during spontaneous atrial flutter. At the C_{3_d} site, during pacing, the double potential deflections (y and x) are associated by the short interval (y–x) and both deflections are captured with a long stimulus (S) to electrogram time. The first deflection of the double potential was captured with a stimulus to electrogram time of 215 ms, and the second deflection at 255 ms, consistent with activation of both deflections after the wave front of activation traversed an area of slow conduction. All numbers are in milliseconds.

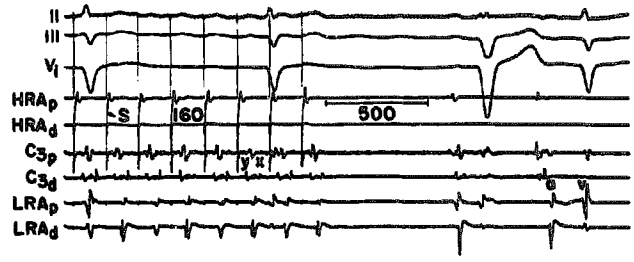

Figure 8. Same patient as in Figure 7. Electrocardiographic leads and electrogram recordings are the same as in Figure 7. High right atrial pacing at a short cycle length (160 ms) interrupted atrial flutter. The stimulus to electrogram time to each of the double potentials (y and x) as measured at C_{3_d} is now shorter than in Figure 7. Double potentials are present in sinus rhythm (right) at site C_{3_d}, but with a shorter interdeflection (y–x) interval. During sinus rhythm, the deflections are not separated by an isoelectric baseline. a = atrial electrogram, v = ventricular electrogram. All numbers are in milliseconds.

potentials were not seen during sinus rhythm or during overdrive atrial pacing of sinus rhythm at rates similar to the rate of the atrial flutter in these patients. The electrograms recorded in the low right atrium where double potentials had occurred during atrial flutter were broad and of low amplitude during sinus rhythm (Fig. 5). High right atrial pacing during sinus rhythm at a cycle length similar to the atrial flutter cycle length resulted in broad, low amplitude electrograms at the site where double potentials occurred during atrial flutter (Fig. 6). Because these double potentials were present only during atrial flutter, we have chosen to call them functional double potentials.

Effects of procainamide. Although procainamide may have influenced double potentials during atrial flutter, it is unlikely that the drug caused obliteration of double potentials on the termination of flutter. Double potentials remained even after procainamide infusion as long as atrial flutter persisted. Procainamide never obliterated double potentials during atrial flutter, suggesting that absence of double potentials during sinus rhythm was due instead to the conversion of sinus rhythm.

Not all double potentials are the same: persistent double potentials. In one patient (Patient 3), double potentials recorded from one site during atrial flutter behaved differently (Fig. 7 to 9). In this patient, during transient entrainment of atrial flutter, the two deflections of the double potential (y and x) were associated with the same atrial flutter complex (Fig. 7), remained present after termination of the atrial flutter with rapid atrial pacing (Fig. 8) and were present during rapid pacing of sinus rhythm and during sinus rhythm (Fig. 9). Because these double potentials were always present, we have chosen to call them persistent double potentials.

In this patient, high right atrial pacing of atrial flutter (transient entrainment) produced results different from those in the other patients. In contradistinction to the previously described double potentials, both deflections of the double potentials remained associated by the short interdeflection (y–x) interval during rapid high right atrial pacing of atrial flutter (Fig. 7), and there was a long activation time to each deflection (215 and 255 ms, respectively). The narrowly spaced interval between the two deflections of the double potentials became more widely spaced during high right pacing of atrial flutter and both deflections were captured with a long stimulus to electrogram time, suggesting conduction from the pacing site through an area of slow conduction to activate both deflections (Fig. 7). When pacing was performed during sinus rhythm and during interruption of atrial flutter (Fig. 8 and 9), the double potentials remained associated by the narrow interdeflection interval. This interval widened further with pacing, yet both deflections were captured with a shorter stimulus to electrogram interval than that noted during high right atrial pacing of atrial flutter. The marked difference in response of the double potentials in this patient suggests that not all double potentials recorded during atrial flutter represent the same phenomenon.

Discussion

In this study we have gained insight into the nature of double potentials recorded in the atrium during atrial flutter. We have characterized the relation of double potentials to the atrial complex in the ECG and have presented data that

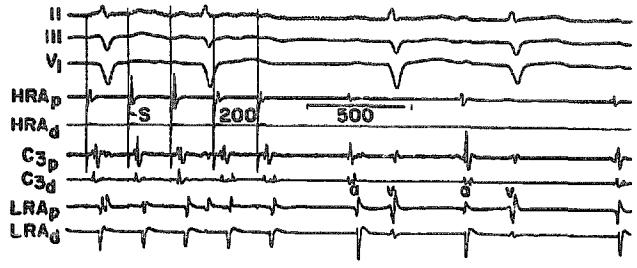

Figure 9. Same patient as in Figures 7 and 8. Electrograms and ECG recordings made at the termination of rapid high right atrial (HRA) pacing initiated during sinus rhythm. There is a short activation time from the stimulus (S) to each double potential deflection during rapid pacing. At site C_{3_d} during sinus rhythm, a low amplitude abnormal electrogram is present that appears to be a double potential (but there is no clear isoelectric baseline between deflections). The degree of separation of the deflections is accentuated by high right atrial pacing in this tracing and the degree of separation appears similar to that during atrial flutter and rapid high right atrial pacing of atrial flutter. Abbreviations as in previous figures.

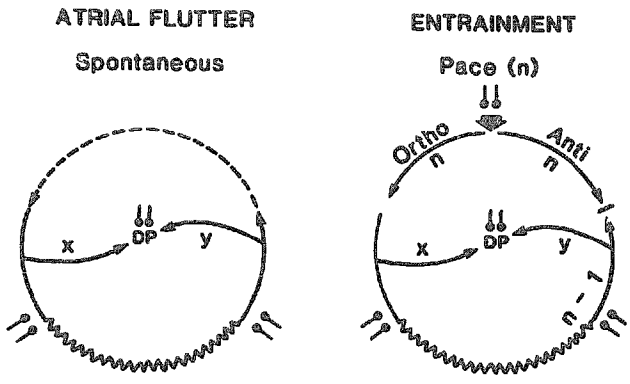

Figure 10. Diagram illustrates suggested explanation of the observations at the functional double potential recording site during spontaneous atrial flutter and its entrainment. The panel at left shows the components of a reentrant tachycardia, including an area of slow conduction (**serpentine line**), areas of normal conduction (**smooth line**) and an excitable gap (**dashed line**). Bipolar electrogram recording sites are shown. A double potential (DP) is represented as collision of two wave fronts of activation in a zone of functional block. The panel at right shows the mechanism of transient entrainment whereby each pacing wave front enters into the reentrant circuit by way of the excitable gap. The wave front from the pacing impulse enters the excitable gap of the reentrant circuit orthodromically (Ortho) and antidromically (Anti). The antidromic wave front of each pacing impulse (n) collides with the orthodromic wave front of each previous beat (n-1). During transient entrainment, the interval related to the x deflection of the DP (as seen in Fig. 3) has a short activation time from the stimulus artifact, because it is proximal to the slow conducting area (**serpentine line**), but the interval related to the y deflection (as seen in Fig. 3) has a long activation time from the stimulus artifact (and the x deflection), because it must first traverse the area of slow conduction.

although not conclusive are consistent with the hypothesis that functional double potentials recorded in the inferior right atrium during atrial flutter are secondary to two different wave fronts of activation colliding in the center of a reentrant circuit.

Characterization of double potentials during atrial flutter. These data confirm that double potentials are frequently present in the right atrium during atrial flutter in humans but are usually not present during sinus rhythm. The data further demonstrate that two distinct types of double potentials may occur: functional and persistent. Functional double potentials are not present during sinus rhythm or rapid atrial pacing of sinus rhythm at rates similar to the atrial flutter rate, whereas persistent double potentials are present under these conditions. Furthermore, the data recorded during high right atrial pacing of atrial flutter demonstrate that each deflection of functional double potentials, in contrast to persistent double potentials, is associated with different (sequential) atrial flutter waves.

One functional double potential deflection (y) was associated with a long activation time during transient entrainment, but the other deflection (x) was associated with a short activation time. This response is consistent with activation of the y deflection by way of a wave front that first traveled through an area of slow conduction in the reentrant circuit, whereas the x deflection was not activated through such an area (Fig. 10). The possibility that activation of the y deflection occurred via a very long pathway is an alternative but unlikely explanation for this long activation time.

During transient entrainment of atrial flutter by high atrial pacing, the configuration of the y deflection (and the x deflection when it was sufficiently discernible) of the functional double potential did not change, indicating orthodromic capture of this deflection (activation occurred from the same direction during entrainment as during the atrial flutter). Functional double potentials occurred during atrial flutter (in the presence or absence of procainamide) but did not occur at the same atrial site during rapid atrial pacing of sinus rhythm at rates similar to those of atrial flutter or during sinus rhythm. For this reason they appear to represent a functional property of the atria found only during atrial flutter.

Persistent double potential deflections responded differently to pacing than did functional double potentials in the one patient in whom the former were found. In this one patient the two deflections (x and y) were associated by the short interdeflection interval. Most important, the double potential was also present during sinus rhythm in this patient alone, the only patient in this series with this finding. This indicates that not all double potentials represent the same phenomenon.

Other investigators (7) have reported that double potentials occur in patients with atrial flutter, but the mechanism has not been clarified. Double potentials have been reported to be present at various sites in the right atrium, generally in

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the low right atrium. Cosio et al. (7) reported double potential electrograms in the atria during human atrial flutter, but unlike our findings, none in the anterior right atrial free wall. Perhaps that was because they recorded at fewer sites. Cosio et al. speculated that double potentials could be explained by either anisotropic conduction or collision of wave fronts.

Use of transient entrainment to understand double potentials: implications. The apparent change in both the timing and the relation of each of the two deflections of the functional double potentials during transient entrainment of atrial flutter invites careful examination of the implications. The ability to entrain a tachycardia implies that the mechanism of the tachycardia is reentry with an excitable gap (8–24). Pacing at a rate faster than the spontaneous rate of the atrial flutter results in entry of the wave front from the pacing impulse into the excitable gap, both orthodromically and antidromically (Fig. 10), resulting in transient entrainment of the tachycardia (8–18). When pacing orthodromically proximal to an area of slow conduction in a reentrant circuit, if the relative sequence of activation and electrogram configuration at a given recording site is preserved, then activation is occurring during pacing as it does during the spontaneous tachycardia (8–24). An electrogram representing activation proximal to an area of slow conduction in a reentrant circuit during pacing from a site that is even more orthodromically proximal to the area of slow conduction will demonstrate a short activation time with preserved electrogram configuration at that recording site. When pacing proximal to an area of slow conduction, orthodromic activation of an area involved in a reentrant circuit that is just distal to the area of slow conduction will demonstrate no change in electrogram configuration but will demonstrate a long activation time from the pacing site to that recording site (9–24) (Fig. 10).

Using these principles of transient entrainment to analyze the response of each of the functional double potentials during rapid pacing, we suggest that during atrial flutter the functional double potentials represent collision of activation wave fronts. These collisions create an area of functional block around which the reentrant wave front circulates. One deflection represents a wave front proximal to a discrete area of slow conduction and the other a wave front distal to an area of slow conduction in a reentrant circuit but not immediately bracketing an area of slow conduction (Fig. 10).

Alternative explanations are possible but less plausible. Perhaps, for instance, the double potentials are not recorded from a portion of the reentrant circuit but represent a bystander phenomenon or a dead-end pathway. If so, the double potentials appear to immediately bracket an area of slow conduction (possibly due to anisotropic conduction), but they are not part of the actual reentrant circuit responsible for atrial flutter. Other investigators have postulated that double potentials recorded during tachycardias represent an area of slow conduction (perhaps secondary to anisotropy) or bracket an area of slow conduction (6,7,26–28). This may be possible, but for the reasons discussed does not explain the double potentials recorded in our study.

Although these alternatives are possible, our data suggest that they are not likely explanations in human atrial flutter. Functional double potentials seem to be critically related to the reentrant circuit. They are not present during sinus rhythm or during rapid atrial pacing from the high right atrium at rates approximately those of atrial flutter, but only during atrial flutter (before and after procainamide infusion). A dead-end pathway or an innocent bystander phenomenon is unlikely but possible.

The functional double potentials do not immediately bracket an area of slow conduction in the reentrant circuit. This is demonstrated by our data: During transient entrainment of atrial flutter from the high right atrium, the coronary sinus site was activated via an area of slow conduction but before activation of the y deflection of the double potential. It is possible that the y deflection alone represents a dead-end pathway, but if this were the case, double potentials would also remain during sinus rhythm or the pacing of sinus rhythm. Procainamide did not appear to be the critical factor that eliminated double potentials because they were present after procainamide infusion until atrial flutter was interrupted.

Whether collision of activation wave fronts outside or inside a reentrant circuit or very slow conduction in the area of the recording electrode explains double potentials, they almost certainly do not represent active depolarization of the same tissue. The short interdeflection interval (mean 75 ms) during atrial flutter, which is even shorter during transient entrainment (mean 59 ms), makes active depolarization of the same tissue impossible because of atrial refractoriness.

Relation to double potentials in canine atrial flutter. In studying their acetylcholine canine model of atrial flutter (the mechanism was thought to be leading circle reentry), Allessie et al. (3) noted that characteristically during atrial flutter the double potentials (they called them double complexes) were recorded from the center of the reentrant circuit. These double potentials were recorded with a unipolar electrode that, they said, permitted interpretation of the recordings as "indicative of functional conduction block." They further suggested that the area of functional block serves as "a central arc around which the impulse circulates."

Double potentials were previously observed in our sterile pericarditis canine model of atrial flutter (5), which has as its clinical counterpart postoperative human atrial flutter. In these dogs, using the concepts of both transient entrainment and epicardial atrial mapping, we showed induced stable atrial flutter to be secondary to reentry. In this animal model, double potentials were frequently recorded in the free wall of the right atrium and were always recorded in the functional center of the reentrant circuit. However, they have not yet

been evaluated during transient entrainment. The relation between the data from our canine model of atrial flutter and the data reported here requires more corroboration, but is certainly intriguing.

Conclusions. Double potentials were shown to occur during atrial flutter in all 10 patients studied. Double potential deflections (functional type) were present only during atrial flutter, demonstrated a changing interrelation during transient entrainment of atrial flutter and were not present during sinus rhythm. In one patient, double potentials (persistent type) were present during sinus rhythm and during atrial pacing of sinus rhythm. The data suggest that most double potentials during atrial flutter are a phenomenon related to reentry that can be explained by collision of activation wave fronts in the functional center of a reentrant circuit around which the reentrant wave front circulates.

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