Circulation
Volume 95, Issue 10, 20 May 1997; Pages 2416-2422
https://doi.org/10.1161/01.CIR.95.10.2416

ARTICLE

Local Capture by Atrial Pacing in Spontaneous Chronic Atrial Fibrillation

Claudio Pandozi, Leopoldo Bianconi, Mauro Villani, Antonio Castro, Giuliano Altamura, Salvatore Toscano, Anna P. Jesi, Giuseppe Gentilucci, Fabrizio Ammirati, Francesco Lo Bianco, and Massimo Santini

ABSTRACT: *Background* Atrial fibrillation (AF) is considered to be maintained by multiple reentrant circuits without or with a very short excitable gap. However, the possibility of local atrial capture has been shown recently in experimental AF or induced AF in humans.*Methods and Results* This study was undertaken to evaluate the feasibility of atrial capture—suggestive of an excitable gap—in spontaneous chronic AF. Decremental pacing was performed in 47 right atrial sites in 14 patients with chronic AF, not taking antiarrhythmic drugs. A Franz catheter (for pacing and monophasic action potential recording) and a recording quadripolar catheter positioned about 10 mm apart were used. Local capture was achieved in 41 (87.2%) sites for a total of 100 captures. In 71 episodes the capture was lost within 15 seconds, while in the remaining 29, pacing was stopped after 15 seconds of stable capture. The AF types immediately before capture were type 1 in 83 and type 2 in 17 episodes. Type 3 AF was never captured. Pacing cycle at capture was 175.7±20.9 ms. The baseline atrial interval (FF) was 185.4±24.5, significantly longer than the FF recorded during pacing immediately before capture (176.0±19.8 ms) (P<.02).*Conclusions* During spontaneous chronic AF in humans, (1) local capture by atrial pacing is possible up to at least 15 mm from the pacing site, (2) regional entrainment is possible during type 1 and type 2 AF but not type 3 AF, and (3) pacing before capture accelerates AF, probably by transient or local capture. These findings suggest that an excitable gap is present in chronic AF, therefore supporting the hypothesis that leading circle reentry is not the unique electrophysiological mechanism maintaining the arrhythmia.

Key Words: fibrillation ■ atrium ■ arrhythmia ■ reentry

Atrial fibrillation is a complex arrhythmia maintained by multiple wandering wavelets continuously reentering themselves.[1 2 3] Atrial cells are therefore believed to be reexcited immediately after their recovery from refractoriness. Indeed, this concept has recently been challenged by some reports[4 5 6 7] showing the possibility of local atrial capture during atrial fibrillation. This phenomenon has been demonstrated during induced, short-lasting atrial fibrillation in the dog,[4 5 6] and even in humans,[7] direct evidence of local transient entrainment during

induced atrial fibrillation has been reported. However, at the moment, the possibility of atrial pacing capture during spontaneous chronic atrial fibrillation in humans still has not been investigated.

The present study was conducted in patients with spontaneous chronic atrial fibrillation and was designed to assess the following: (1) the possibility of local atrial pacing capture, which would be consistent with the presence of an excitable gap, (2) the correlation between the electrophysiological characteristics of atrial fibrillation and local pacing capture features, and (3) the modality of the loss of capture during entrainment by atrial pacing.

METHODS

Patient Selection

The study was carried out in 14 consecutive patients with chronic atrial fibrillation as a part of a protocol intended to study the efficacy and safety of low-energy internal cardioversion immediately before the cardioversion procedure. The study was approved by our Institutional Ethical Committee, and all the patients gave their written informed consent.

Diagnosis of atrial fibrillation was based on the surface ECG with the following criteria: presence of fluctuation of the baseline without regular P or F waves, with totally irregular RR intervals. These criteria had to be validated by endocardial recordings showing irregular atrial activation not separated by an isoelectric line or discrete atrial complexes separated by an isoelectric line but with irregular atrial intervals (FF) (standard deviation >10 ms). Moreover, no periodic pattern of the FF intervals could be present.[8 9]

Eleven patients were men and 3 were women; mean age was 60.8±9.4 years; atrial fibrillation duration was >30 days in all the patients (mean, 293.6±462.1 days; median, 90 days). The mean left atrium size was 41.5±4.9 mm. The underlying heart diseases were valvular heart disease in 5 patients, hypertension in 3, hypertrophic cardiomyopathy in 1, sick sinus syndrome in 1, and coronary heart disease in 1. Three patients had lone atrial fibrillation. Thyroid dysfunction had been ruled out in all patients. No patient was on amiodarone treatment for at least 3 months before the procedure; all other antiarrhythmic drugs including verapamil and digoxin were stopped at least 5 half-lives before the procedure.

Electrophysiological Study

At least three catheters were used for each patient. A quadripolar standard lead with 2-mm spacing (Bard-USCI Inc) was positioned in the right atrium, allowing contemporary recording of bipolar electrograms from the distal and proximal pairs as well as unipolar recording from the distal electrode. A second catheter for monophasic action potential (MAP) recording was also positioned in the right atrium. This lead (Franz catheter, EP Technologies) has two nonpolarizable electrodes, one at the tip of the catheter and the second 5 mm proximally.[10] Between these two electrodes, in the orthogonal position, a second pair of platinum electrodes is embedded for pacing purposes. This configuration makes the pacing and recording sites almost identical. A third decapolar catheter (Elecath Inc) was positioned in the coronary sinus for shock delivery. The quadripolar and the MAP catheters were always positioned close to each other at a distance of ≈10 mm and were used contemporarily for right atrial mapping.

In each patient, in the 30 degree left anterior oblique view, up to seven right atrial sites were mapped, depending on the time needed for the procedure. The sites were mapped in the following

order: mid lateral, low lateral, high lateral, atrial roof, high septal, mid septal, and low septal. In each mapped point, basal unipolar and bipolar electrograms from the quadripolar catheter as well as the MAP from the Franz catheter were recorded for a period of 60 seconds on a seven-channel Mingograph polygraph (Siemens-Elema AB) at a standard paper speed of 100 mm/s. The unipolar recordings were unfiltered, whereas the bipolar electrograms were filtered between 30 and 300 Hz. Only the sites in which it was possible to obtain stable electrograms and MAPs were selected for pacing.

Stimulation Protocol

The Franz catheter was used for pacing by delivering a square wave of 4 mA and 2-ms duration. At each site, atrial stimulation was carried out by starting at a cycle length of 250 ms and then decreasing the cycle by 10 ms every 10 seconds until a stimulation cycle of 100 ms or a stable local atrial capture (at least 5 consecutive beats) was achieved. If local atrial fibrillation entrainment was lost before 15 seconds, the pacing was continued by reducing the cycle length by 10 ms. In the case of constant local atrial capture, the pacing cycle was maintained at the same rate for 15 seconds (long-lasting atrial capture) and then stopped. After 30 seconds, the pacing was resumed following the above-mentioned protocol, starting at a cycle 10 ms shorter. After a maximum of 3 atrial capture sequences (stable or long-lasting), the catheter position was changed according to the mapping procedure and the stimulation protocol was repeated.

Local atrial capture was assumed to occur when all the following criteria were present: (1) MAP, unipolar, and bipolar electrograms phase-locked to the stimulus artifact, (2) local atrial cycle length equal to the pacing cycle length, (3) constant morphology of the unipolar and bipolar atrial electrograms and MAPs, (4) constant activation sequence of the electrograms recorded from the distal and proximal pairs of the atrial catheter, and, finally, (5) the appearance of the typical endocavitary recordings of atrial fibrillation (variable morphology, amplitude, cycle length, and activation sequence of local atrial electrograms and MAPs) at the loss of capture or at cessation of pacing.

Endocavitary Recording Analysis

The Wells classification,[11 12] applied to endocardial electrograms, was used to identify the type of atrial fibrillation according to the morphology of the endocardial bipolar recording: type 1, atrial electrograms showing discrete complexes of variable morphologies separated by an isoelectric baseline free of perturbation; type 2, discrete beat-to-beat atrial electrogram complexes of variable morphology but with an isoelectric baseline showing perturbations of varying degree; and type 3, highly fragmented atrial electrograms showing no discrete complexes or isoelectric intervals. Local unipolar and bipolar electrograms and MAPs were analyzed both in the basal conditions and during atrial pacing.

The beat-to-beat FF intervals (assumed as the distance between atrial electrograms and not between MAPs) were manually calculated with a caliper, assuming the local activation time in the following ways[13 14] : (1) for the unipolar recording: the steepest intrinsic deflection of the electrogram and (2) for the bipolar recording: the peak of the first rapid deflection in a predominantly monophasic electrogram or the time of the intrinsic deflection in a predominantly biphasic electrogram. In the case of discordance between bipolar and unipolar recordings, the latter was always chosen for calculations. The interval between two MAPs was assumed as the distance between their 0 phase.

Parameters Evaluated

In the basal condition, the following were evaluated: (1) type of atrial fibrillation during the 60 seconds of basal recording, (2) mean local FF interval with the standard deviation of 20 consecutive beats after 10 seconds from the recording start, and (3) the difference between the 5th and 95th percentile (P5-95) of FF intervals.[15]

During pacing, the type of local atrial fibrillation, the FF intervals with the standard deviation, and their variations (P5-95) were calculated for the 20 beats preceding capture and for the 20 beats after either the loss of capture or cessation of pacing. Therefore, close capture episodes not preceded or followed by at least 20 consecutive local FF intervals were not considered for analysis.

During atrial capture, we evaluated (1) the duration of local capture (up to 15 seconds, when the pacing was stopped anyway), (2) the pacing rate at capture, (3) the interval between the last noncaptured and the first captured electrogram, and (4) the interval between the last captured and the first noncaptured beat at the loss of local atrial fibrillation entrainment. Obviously, the parameters mentioned in basal conditions and during pacing could not be calculated if type 3 atrial fibrillation was present after 10 seconds from the basal recording starting or immediately before the capture, respectively.

Statistical Analysis

Data are presented as mean±SD. Differences in continuous variables were analyzed by paired or unpaired Student's *t* test or ANOVA as appropriate, and comparison between groups was performed by multiple Bonferroni test. Differences in categorical variables were analyzed by χ^2 test, with Yates' correction if needed. A value of $P<.05$ was considered statistically significant.

RESULTS

Sites of Capture

A total of 58 atrial sites were mapped in the 14 patients (mean, 4.3 per patient). Forty-seven of these sites were considered for pacing and 11 were excluded because of absence of stable recordings from the quadripolar and/or the MAP catheters. During the basal recording, a type 3 atrial fibrillation was never present after 10 seconds from the recording starting. The numbers of mapping, pacing, and captures in relation to the catheter position in the right atrium are reported in Table 1. At least one stable capture was observed in 41 sites (87.2%). Capture was achieved more frequently in the lateral wall (30/31=96.8%) than in the atrial roof (3/4=75%) or septum (8/12=66.7%) ($P<.05$).

The type of atrial fibrillation during all the 60 seconds of the baseline recording in the three explored atrial walls (lateral, septal, and roof) and the respective captures observed are reported in Table 2. Pure type 1 atrial fibrillation was more frequently observed in the lateral wall (21/31=68%) than in the other atrial sites (6/16=38%) ($P<.05$). However, local capture was not affected significantly by the pattern of the baseline arrhythmia (92.5% in sites with basal pure type 1 atrial fibrillation versus 80% in sites with phases of type 2 and 3 atrial fibrillation in the basal 60 seconds recording, *P*=NS).

Episodes of Capture

A total of 100 capture sequences suitable for complete analysis were obtained. The captured sites were high lateral (17), mid lateral (41), low lateral (21), atrial roof (5), high septum (10), mid

septum (3), and low septum (3). Another 15 capture episodes were excluded because of the absence of at least 20 FF intervals between an episode of loss of capture and the next capture phase (6 cases) or because of the presence of phases of type 3 atrial fibrillation during the 20 beats considered for measurement (7 before and 2 after capture).

In 29 of the 100 episodes, pacing was stopped after 15 seconds of entrainment; loss of capture was observed in the other 71 (mean capture duration, 6403.7±3231.3 ms).

Atrial Fibrillation Type at Capture

The form of atrial fibrillation at the moment of capture was type 1 in 83 episodes and type 2 in 17 (Figs 1 and 2). In 7 circumstances, type 3 atrial fibrillation was present until a few hundred milliseconds before the capture, when transformation in type 1 atrial fibrillation occurred (Fig 3). Type 3 atrial fibrillation was never captured.

FF Intervals Before and After Atrial Capture

The mean FF intervals in basal conditions and during pacing before capture were 185.4±24.5 ms (P5-95, 52.3±21.5 ms) and 176.0±19.8 ms (P5-95, 41.7±18.0 ms) (P<.02).

In the capture episodes considered, the mean pacing cycle at capture was 175.7±20.9 ms. The interval between the last noncaptured and the first captured electrogram was significantly longer: 187.7±30.6 ms (P<.0001).

Table 3 shows the pacing cycle at capture and the FF interval at baseline, before capture, and after capture by comparison of the cases with capture loss during pacing with those in which pacing was stopped after long-lasting entrainment. No significant differences were found regarding the basal FF interval and the FF interval before capture between the episodes of capture loss and those in which pacing was stopped. In the 29 occurrences in which pacing was stopped, the FF interval after cessation of pacing was 196.1±24.1 ms, significantly longer than that observed at the loss of capture (169.0±19.1 ms) (P<.0001). The FF cycle shortening between baseline and the period preceding the capture was statistically significant only in the cases in which capture was lost and not in those in which a long-lasting entrainment was attained.

In the 71 episodes of capture loss, the interval between the last captured and the first noncaptured beat was 164.8±18.4 ms, and it was invariably shorter than the pacing cycle length. An example of loss of capture is reported in Fig 4. In Fig 5, an example of pacing cessation during constant capture is given.

Reported data suggest the following observations: (1) the basal FF interval was longer than that recorded during pacing before capture; (2) the atrial fibrillation acceleration during pacing was statistically significant only in patients in whom capture was lost and not in those in whom the entrainment was maintained until pacing cessation; (3) the loss of capture was never due to capture failure but always to the presence of a spontaneous atrial activation preceding the stimulus artifact; (4) the FF interval after pacing cessation was longer than the mean FF interval during pacing before capture; (5) the mean FF interval was similar before capture and after capture loss; and (6) the mean FF interval after pacing cessation was similar to that recorded in basal conditions.

Atrial Fibrillation Characteristics Before and After Pacing

By considering the atrial fibrillation type during the 60 seconds of basal recording, three groups of episodes of atrial capture can be distinguished: pure type 1 atrial fibrillation (63 episodes of local pacing capture, group 1); presence of phases of type 2 atrial fibrillation (11 episodes, group 2); and phases of type 3 atrial fibrillation (26 episodes, group 3). In Table 4, FF intervals at baseline

and before capture in each group are summarized. The FF intervals were longer at baseline in group 1 than in groups 2 and 3, and a significant shortening of the baseline FF interval was observed only in group 1.

DISCUSSION

Excitable Gap in Atrial Fibrillation:Previous Studies

High-resolution mapping studies performed both in animals[3] and humans[15 16] have confirmed Moe's hypothesis,[1] according to which atrial fibrillation is maintained by multiple independent wavelets irregularly activating atrial cells at very high rates. Nevertheless, some of these studies have shown that random reentry[15 16] and leading circle reentry,[15 17] respectively, with a short excitable gap and without an excitable gap, are not always present under the mapped area. This implies that an excitable gap of variable duration exists even in the most complex forms of atrial fibrillation. This should allow local electrical stimuli to be able to capture at least a small portion of the atria. At the moment, evidence of local atrial capture has been obtained only in induced fibrillation in animals[4 5 6] and in humans.[7] However, the clinical relevance of the above observations is limited by the fact that pacing-induced atrial fibrillation could have electrophysiological properties different from those of the spontaneously occurring arrhythmia.

Local Capture in Chronic Atrial Fibrillation

In the present study, local atrial capture was obtained in 87.2% of the sites considered for pacing, for a total of 100 episodes of capture. This means that an excitable gap is present, at least in some phases, in the fibrillating right atrium, thus implying that leading circle reentry (without excitable gap) is not the only electrophysiological mechanism maintaining atrial fibrillation in humans. Although the presence of random reentry cannot be excluded, it is indeed unlikely that the very short excitable gap of random reentry can account for all the capture episodes we observed, at least in type 1 or 2 atrial fibrillation, since type 3 was never captured. However, since the local atrial fibrillation pattern varies frequently from one type to another, the likelihood of obtaining local capture during prolonged pacing was not affected by the baseline atrial fibrillation type. Nevertheless, the lateral wall, where we found the atrial activity to be more steadily organized, was the site where atrial entrainment was more frequently achieved.

It should be stressed that in order to increase the specificity of our results, the definition of local capture was based on very strict criteria, taking into account the contemporary presence of several precise conditions. On the other hand, this might have reduced the sensitivity of the method by possibly missing episodes of inconstant capture or capture limited to the MAP catheter.

Characteristics of Baseline Atrial Fibrillation

In agreement with a recent observation,[18] we found atrial fibrillation to be more organized in the lateral wall than in other areas of the right atrium. This could be explained by the recent experimental finding[19] that the atrial refractory period is shorter in the lateral and anterolateral wall than in other atrial sites. An additional explanation could involve the peculiar architecture of the right atrium, in which the crista terminalis could act as an anatomical-functional barrier (as in atrial flutter[20]), partially insulating the lateral wall, thus reducing the number of the wave fronts entering this area.

Characteristics of Atrial Fibrillation Preceding Atrial Capture

The mean 20 FF intervals preceding atrial capture (stable or long lasting) were shorter than the basal ones. This observation implies that pacing itself induces an acceleration of the local atrial fibrillation. The acceleration of the fibrillation waves was more marked when the basal atrial fibrillation was less complex and with a longer mean FF interval. In fact, this phenomenon was significant when in basal conditions type 1 atrial fibrillation was recorded but not when phases of type 2 or 3 atrial fibrillation were present.

Moreover, atrial fibrillation acceleration was marked and statistically significant only in the episodes in which capture was lost and not in those in which long-lasting entrainment was obtained. This indicates that the atrial fibrillation acceleration before capture, either spontaneous or induced by the pacing itself, might make more difficult the achievement of stable capture.

Kirchhof et al,[5 6] in induced atrial fibrillation in dogs, showed that pacing-induced acceleration of local atrial fibrillation before stable capture is due to the induction of small, temporary leading circle reentrant circuits by stimuli temporarily capturing only a very limited portion of atrial tissue. The acceleration we observed in our patients may be explained by the same mechanism occurring during pacing after a transient or local capture.

The acceleration of local atrial fibrillation by pacing is limited to the pacing period. In fact, after the end of the stimulation, the FF intervals, which are shortened during pacing before capture, return immediately to the prepacing values.

The mean pacing interval at capture was almost identical to the mean FF interval preceding capture. Moreover, the mean interval between the last noncaptured and the first captured electrogram was similar to the basal mean cycle length and longer than the pacing cycle length at capture. These findings show that atrial capture by a pacing cycle identical to the mean local fibrillation cycle length occurs when the stimulus falls during one of the spontaneously occurring long FF intervals (P5-95, 41.46±18.01 ms). This observation is in agreement with Kirchhof and colleagues' experience[5 6] in induced atrial fibrillation, in which regional capture was possible by pacing the center of the mapped area at intervals equal to or slightly shorter than the mean atrial fibrillation local interval.

Characteristics of Atrial Fibrillation at Loss of Capture

In most of the cases, capture was lost before pacing cessation (15 seconds after the beginning of capture). The loss of capture, despite the high stimulation rate, was never due to the failure of one of the stimuli to excite the atrium but always to the fact that the interval between the last captured and the first noncaptured beat was invariably shorter than the pacing cycle length.

No atrial fibrillation acceleration was noted in the 20 beats after the loss of capture with respect to the mean FF interval immediately before capture. The presence of a short interval between the last captured and the first noncaptured beat, in the absence of any acceleration of the atrial fibrillation, can be explained easily by the spontaneous variations in the fibrillation intervals. These findings are somehow different from those reported by Kirchof et al[5 6] during capture of induced atrial fibrillation in dogs. In their study, atrial fibrillation probably accelerated because after capture achievement, the pacing was performed continuously at increasing rates, reaching very high frequencies relative to the atrial fibrillation rate before capture, thus inducing a new, faster reentry circuit of the leading circle type. In our study, the mean pacing cycle length at capture was similar to the atrial fibrillation cycle length during pacing before capture; moreover, during capture, the pacing cycle was fixed, preventing further atrial fibrillation acceleration during capture.

Study Limitations

Our findings about the possibility of obtaining atrial capture during atrial fibrillation are limited to the right atrium, since the left atrium was not considered in our study.

Our stimulation protocol, with the use of a fixed pacing rate after capture, does not allow us to draw any insight about the influence of pacing at increasing rates on captured atrial fibrillation. Thus, the possibility of a further acceleration of atrial fibrillation by continuous local pacing capture at increasing rates could not be investigated. This further acceleration was demonstrated to be responsible for the subsequent loss of regional control in experimentally induced atrial fibrillation.[5][6]

Moreover, our protocol, though very specific for capture assessment, does not permit any evaluation of the spatial resolution of atrial capture beyond the proximal pair of the right atrial quadripolar electrodes (15 mm).

Finally, no attempt was made to evaluate the possibility of atrial fibrillation interruption by capturing, simultaneously and at increasing rates, several atrial sites.

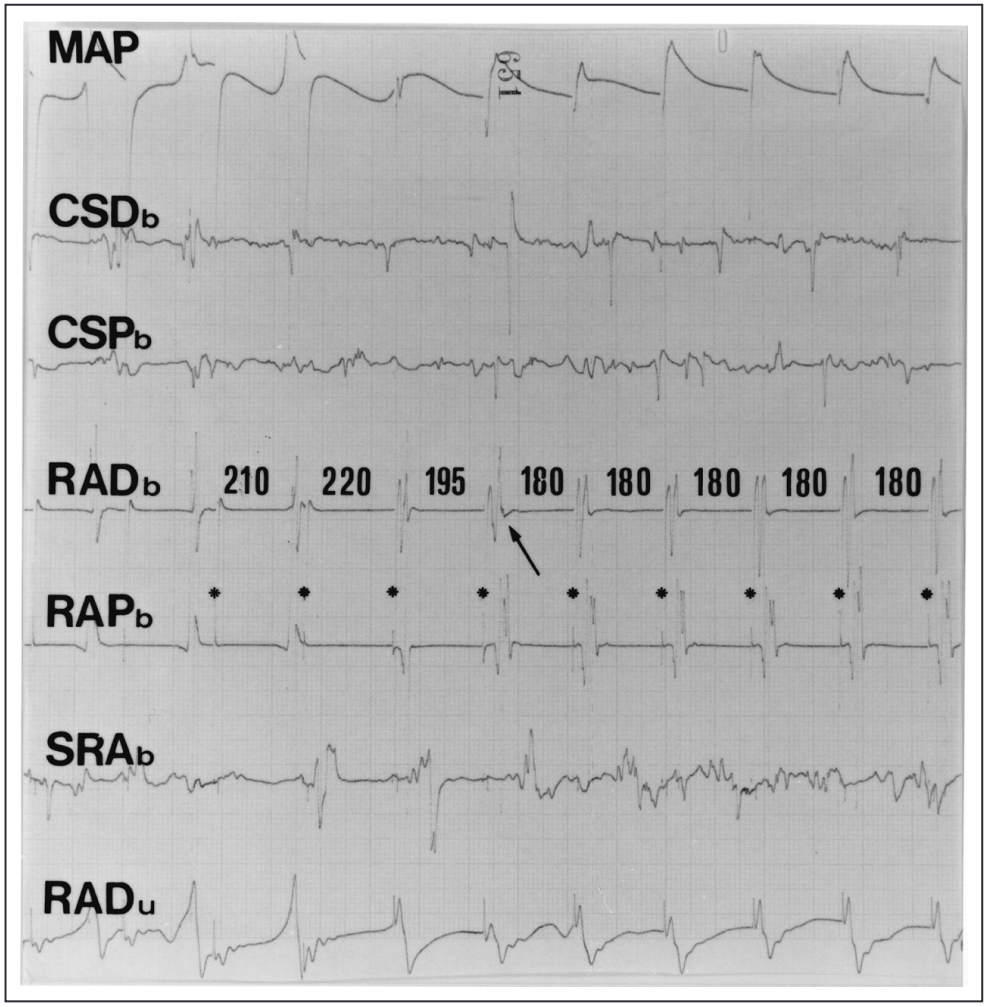

Figure 1. Endocavitary recording showing local capture during type 1 atrial fibrillation. During pacing at the mid lateral right atrial wall level (cycle, 180 ms), the spikes (asterisks) initially fall in the absolute atrial refractory period, as clearly shown by the monophasic action potential (MAP). The spikes are evidently out of phase in respect to the MAP and to the local unipolar and bipolar electrograms. At the moment indicated by the arrow, the MAP, the unipolar, and both the proximal and distal bipolar electrograms from the lateral right atrial wall become constantly phase-locked to the stimulus artifact. The configuration of the electrograms becomes uniform as well as the local activation sequence, indicating that capture has occurred. Capture does not extend to the septal right atrium and the coronary sinus, which show autonomous electrical activity. CSD indicates coronary sinus distal; CSP, coronary sinus proximal; RAD, lateral right atrium distal; (lateral) RAP, right atrium proximal; SRA, septal right atrium; b, bipolar recording; and u, unipolar recording.

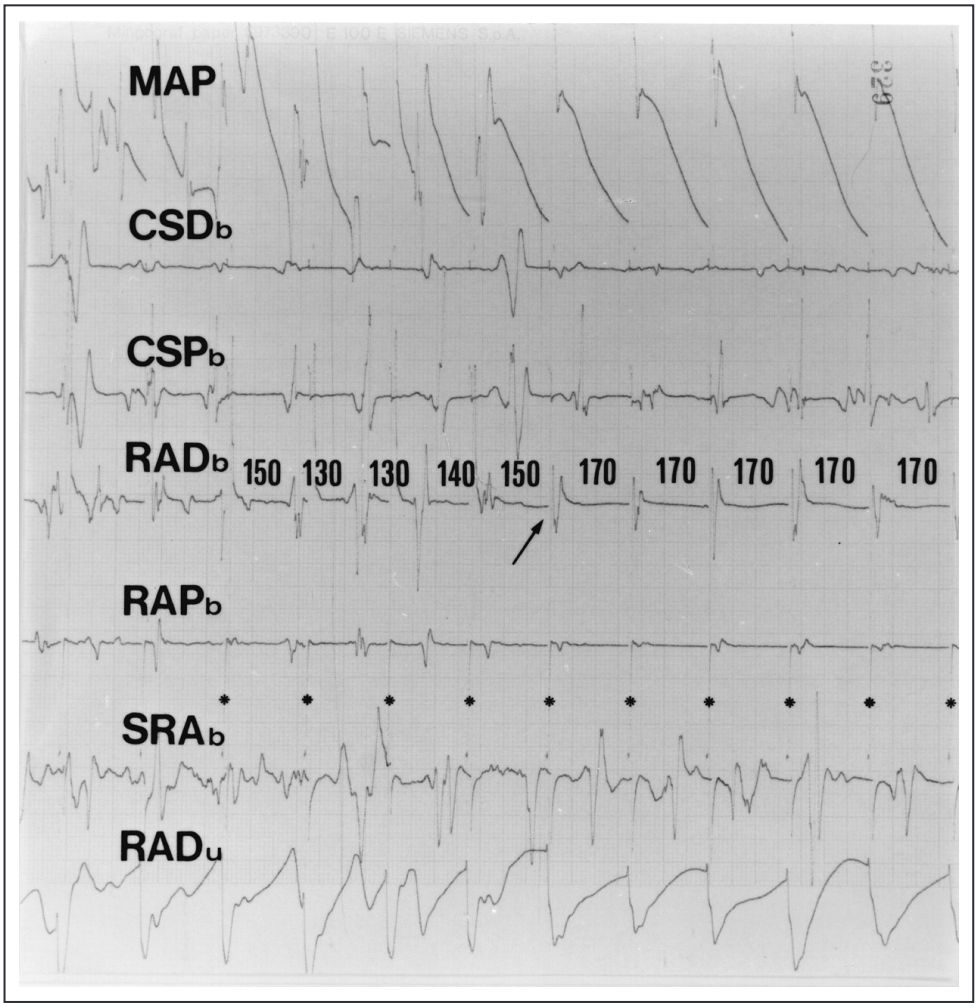

Figure 2. Example of local capture during type 2 atrial fibrillation. During pacing at the low lateral right atrial wall level (cycle, 170 ms), at the moment indicated by the arrow, the MAP, the unipolar, and the bipolar atrial electrograms of the lateral atrial wall become phase-locked to the stimulus artifacts (asterisks) and the configurations of the electrograms become constant, as well as the local activation sequence, suggesting that local atrial capture has occurred. Septal right atrium and coronary sinus show autonomous electrical activity. For abbreviations, see Fig 1.

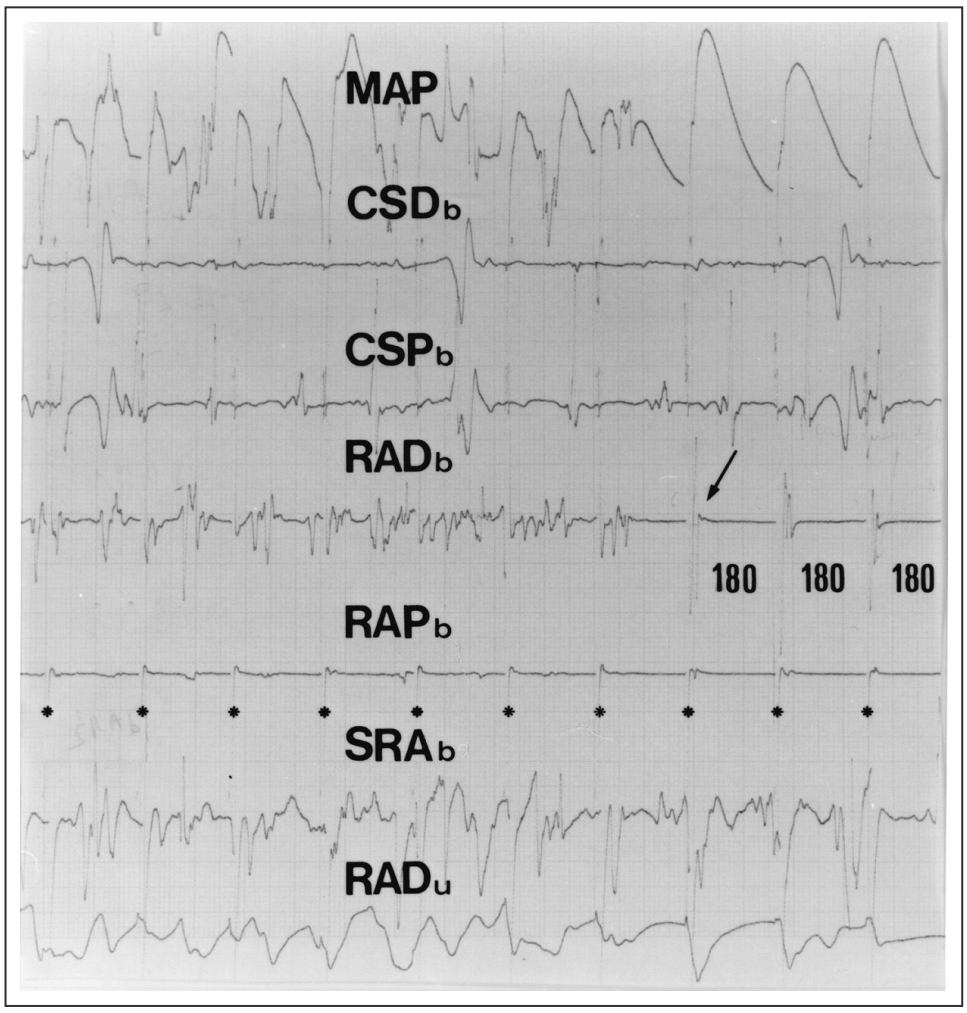

Figure 3. Example of atrial capture occurring during type 3 atrial fibrillation. During pacing at the atrial roof level (cycle length, 180 ms), type 3 atrial fibrillation is recorded by the distal bipolar atrial pair without evidence of capture (in fact, even MAPs are not discernible). At the moment indicated by the arrow, the MAP, the distal unipolar (RADu), and the distal and proximal bipolar (RADb and RAPb, respectively) atrial electrograms become phase-locked to the stimuli (asterisks) and the configurations of the electrograms become constant, as well as the local activation sequence. This seems to suggest that local atrial capture has occurred during type 3 atrial fibrillation. A careful examination indeed reveals that the type 3 atrial fibrillation was never captured and that capture actually occurred just 125 ms after the transition from type 3 to type 1 atrial fibrillation, as shown by the presence of a regular baseline free of perturbations immediately before capture. For abbreviations, see Fig 1.

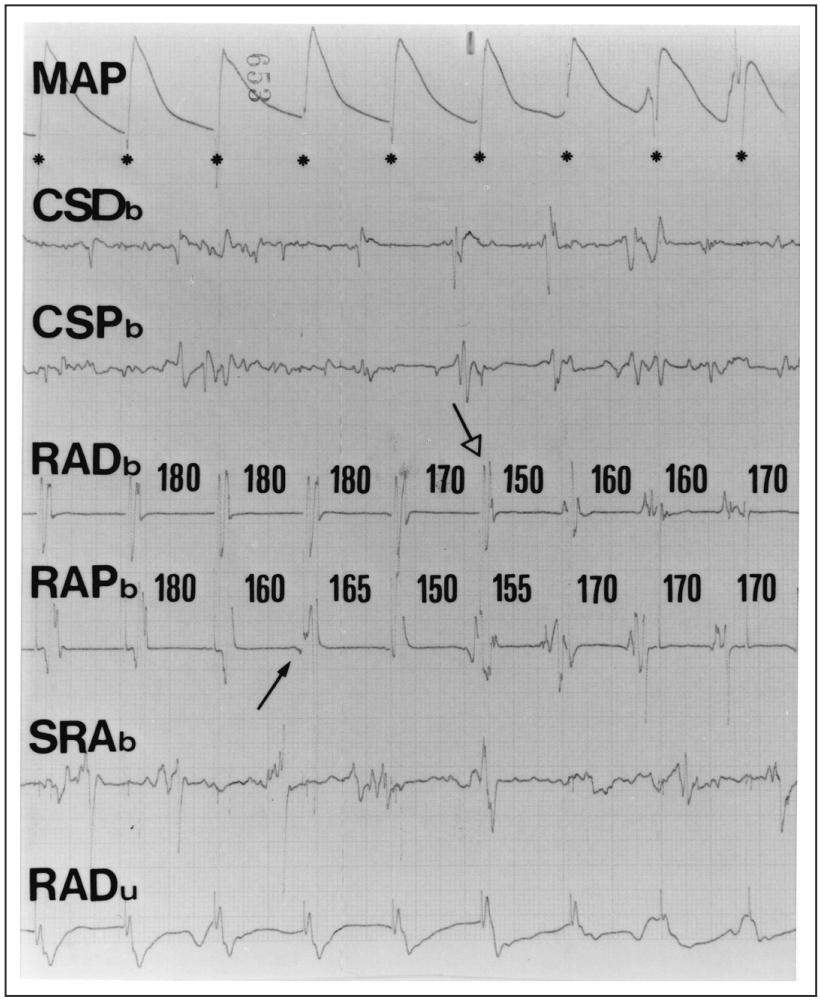

Figure 4. Example of capture lost. A constant capture by right atrial pacing at the septal level (cycle length, 180 ms) is initially present, as evident on MAP, distal unipolar (RADu), and distal and proximal bipolar (RADb and RAPb, respectively) atrial electrograms. The closed arrow indicates a spontaneous atrial electrogram in RAPb preceding the stimulus (asterisks) and leading to capture loss. The loss of capture is subsequently evident also in RADb (fusion beat is marked by an open arrow), RADu, and MAP. The interval between the last captured and the first noncaptured beat in RAPb is 160 ms, shorter than the pacing cycle length. Because of the fixed pacing rate used, the failure of a single stimulus to excite the atrium leads to complete loss of capture, since the following stimuli and the spontaneous atrial activation are thrown out of phase. For abbreviations, see Fig 1.

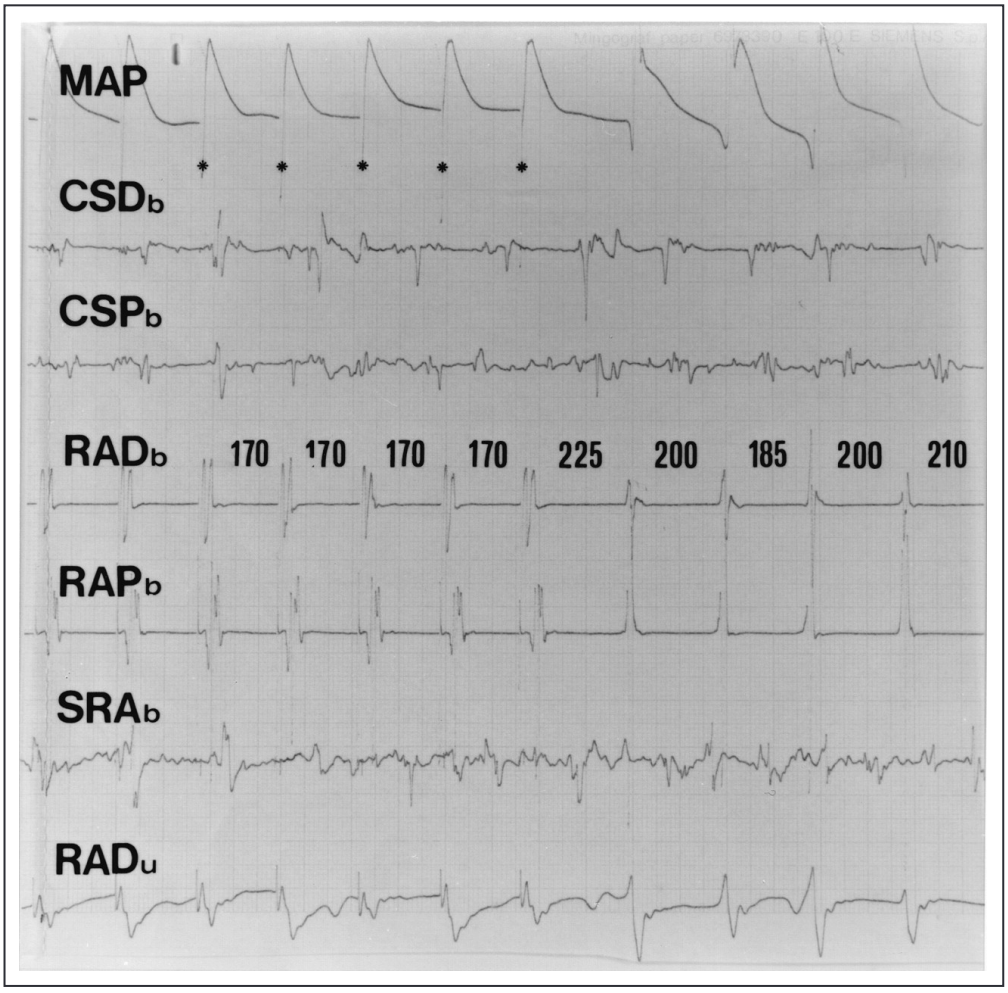

Figure 5. Example of pacing cessation after constant capture at mid lateral wall level. Local activation constantly following the stimuli (asterisks) is evident in MAP, RADb, RADu, and RAPb. At cessation of pacing, atrial electrograms and MAPs of variable morphology, amplitude, cycle length, and activation sequence resume. For abbreviations, see Fig 1.

Table 1. Table 1.

TABLE 1. Number of Different Sites That Were Mapped, Paced, and Captured

	Sites		
	Mapped, No.	Paced, No.	Captured, No.
Lateral wall			
Mid	14	14	14
Low	9	9	9
High	8	8	7
Total	31	31	30 (96.8%)*
Atrial roof	8	4	3 (75%)
Septal wall			
High	7	5	5
Mid	6	3	2
Low	6	4	1
Total	19	12	8 (66.7%)
All sites	58	47	41 (87.2%)

*$P<.05$ vs atrial roof and septal wall.

Table 2. Table 2.

TABLE 2. Type of Atrial Fibrillation During All 60 Seconds of the Baseline Recording and the Respective Captures in the Different Right Atrial Walls

	Atrial Fibrillation Type			
	Type 1	Type 2	Type 3	All
Lateral wall	21	4	6	31
Capture	20	4	6	30 (96.8%)*
Atrial roof	2	0	2	4
Capture	2	0	1	3 (75%)
Septum	4	5	3	12
Capture	3	2	3	8 (66.7%)
All sites	27	9	11	
Capture	25 (92.5%)†	6 (66.7%)	10 (90.9%)	

Type 1 indicates type 1 only; types 2 and 3 indicate phases of types 2 and 3.

*$P<.05$ vs atrial roof and septum.

†P=NS vs atrial roof and septum.

Table 3. Table 3.

TABLE 3. Pacing Cycle at Capture and FF Intervals at Baseline, Before Capture, and After Capture in the Cases of Capture Loss Compared With Those in Which Pacing Was Stopped After 15-Second Capture (Long-Lasting Capture)

	Capture Loss (n=71)	Long-Lasting Capture (n=29)	*P*
Pacing cycle at capture, ms	174.7±19.0	176.1±21.6	NS
FF cycle, ms			
Baseline	184.7±22.9	187.7±33.6	NS
Before capture	174.3±19.2*	180.6±21.4†	NS
After capture	169.0±19.1‡	196.1±24.1§	.0001

*$P<.02$ vs baseline.
†P=NS vs baseline.
‡P=NS vs before capture.
§$P<.01$ vs before capture.

Table 4. Table 4.

TABLE 4. FF Interval at Baseline and Before Capture in the Different Types of Atrial Fibrillation at Baseline: Pure Type 1 (Group 1), Phases of Type 2 Atrial Fibrillation (Group 2), and Phases of Type 3 Atrial Fibrillation (Group 3)

		FF Cycle, ms		
	No.	Baseline	Before Capture	*P*
Group 1	63	192.6±22.8	179.2±19.2	<.01
Group 2	11	176.3±18.7*	172.7±21.5	NS
Group 3	26	176.7±28.8*	169.6±19.8†	NS
Total	100	185.4±24.5	176.0±19.8	<.02

*$P<.05$ vs group 1; †$P<.005$ vs group 1.

ARTICLE INFORMATION

Received August 29, 1996; revision received November 18, 1996; accepted December 14, 1996; published online May 20, 1997.

Correspondence

Correspondence to Dr Claudio Pandozi, Via Madonna Di Fatima, 22, 00147 Roma, Italy.

Affiliations

the Department of Cardiac Diseases, San Filippo Neri Hospital, and Arrhythmia Control Unit (M.V.), I Clinica Medica La Sapienza Univerisity, Rome, Italy.

REFERENCES

1. Moe GK. On the multiple wavelet hypothesis of atrial fibrillation. *Arch Int Pharmacodyn Ther.* 1962;*140*:183-188.
2. Allessie MA, Lammers WJEP, Bonke FIM, Hollen J. Experimental evaluation of Moe's multiple wavelet hypothesis of atrial fibrillation. In: Zipes DP, Jalife J, eds. *Cardiac Electrophysiology and Arrhythmias*. Orlando, Fla: Grune & Stratton; 1985:265-275.
3. Allessie MA, Lammers WJEP, Rensma PL, Bonke FIM. Flutter and fibrillation in experimental modes: what has been learned that can be applied to humans. In: Brugada P, Wellens HJJ, eds. *Cardiac Arrhythmias: Where to Go From Here*. Armonk, NY: Futura Publishing Co Inc; 1987:67-82.
4. Allessie M, Kirchhof C, Scheffer GJ, Chorro F, Brugada J. Regional control of atrial fibrillation by rapid pacing in conscious dogs. *Circulation.* 1991;*84*:1689-1697. Crossref. PubMed.
5. Kirchhof C, Wijffels M, Allessie M. Pace termination of atrial fibrillation. In: Olsson SB, Allessie MA, Campbell RW, eds. *Atrial Fibrillation: Mechanisms and Therapeutic Strategies*. Armonk, NY: Futura Publishing Co, Inc; 1994:251-271.
6. Kirchhof C, Chorro F, Scheffer GJ, Brugada J, Konings K, Zetelaki Z, Allessie M. Regional entrainment of atrial fibrillation studied by high-resolution mapping in open-chest dogs. *Circulation.* 1993;*88*:736-749. Crossref. PubMed.
7. Daoud EG, Pariseau B, Niebauer M, Bogun F, Goyal R, Harvey M, Ching Man K, Strickberger A, Morady F. Response to type 1 atrial fibrillation to atrial pacing in humans. *Circulation.* 1996;*94*:1036-1040. Crossref. PubMed.
8. Lammers WJEP, Ravelli F, Disertori M, Antolini R, Furlanello F, Allessie MA. Variations in human flutter cycle length induced by ventricular beats: evidence of a re-entrant circuit with a partially excitable gap. *J Cardiovasc Electrophysiol.* 1991;*2*:375-387. Crossref.
9. Ravelli F, Disertori M, Cozzi F, Antolini R, Allessie MA. Ventricular beats induce variations in cycle length of rapid (type 2) atrial flutter in man: evidence of leading circle reentry. *Circulation.* 1994;*89*:2107-2116. Crossref. PubMed.
10. Franz MR. The role of monophasic action potential recording in atrial fibrillation. In: Olsson SB, Allessie MA, Campbell RW, eds. *Atrial Fibrillation: Mechanisms and Therapeutic Strategies*. Armonk, NY: Futura Publishing Co Inc; 1994:109-125.
11. Wells JL Jr, Karp RB, Kouchoukos NT, MacLean TN, Waldo A. Characterisation of atrial fibrillation in man: studies following open heart surgery. *Pacing Clin Electrophysiol.* 1978;*1*:426-438. Crossref. PubMed.
12. Waldo AL. Atrial fibrillation following open heart surgery: mechanism and treatment. In: Olsson SB, Allessie MA, Campbell RW, eds. *Atrial Fibrillation: Mechanisms and Therapeutic Strategies*. Armonk, NY: Futura Publishing Co Inc; 1994:211-223.
13. Niwano S, Ortiz J, Abe H, Gonzalez X, Rudy Y, Waldo A. Characterization of the excitable gap in a functionally determined reentrant circuit: studies in the sterile pericarditis model of atrial flutter.

Circulation. 1994;*90*:1997-2014. Crossref. PubMed.

14. Pieper CF, Blue R, Pacifico A. Simultaneously collected and discrete bipolar electrograms: comparison of activation time detection algorithms. *Pacing Clin Electrophysiol.* 1993;*16*:426-433. Crossref. PubMed.
15. Konings KTS, Kirchhof CJHJ, Smeets JRLM, Wellens HJJ, Penn OC, Allessie MA. High-density mapping of electrically induced atrial fibrillation in humans. *Circulation.* 1994;*89*:1665-1680. Crossref. PubMed.
16. Allessie MA, Konings, Kirchhof C. Mapping of atrial fibrillation. In: Olsson SB, Allessie MA, Campbell RW, eds. *Atrial Fibrillation: Mechanisms and Therapeutic Strategies*. Armonk, NY: Futura Publishing Co Inc; 1994:37-49.
17. Allessie MA, Bonke FIM, Schopman JG. Circus movement in rabbit atrial muscle as a mechanism of tachycardia, III: the 'leading circle' concept: a new model of circus movement in cardiac tissue without the involvement of an anatomical obstacle. *Circ Res.* 1977;*41*:9-18. Crossref. PubMed.
18. Shah DC, Haissaguerre M, Jais P, Lavergne T, Hocini M, Gencel L, Garrigue S, Clementy J. Chronic atrial fibrillation: the relevance of organised atrial activity. *Eur J Cardiac Pacing Electrophysiol*. 1996;6(suppl 5):65. Abstract.
19. Li H, Hare J, Mughal K, Krum D, Biehl M, Deshpande S, Dhala A, Blanck Z, Sra J, Jazayeri M, Akhtar M. Distribution of atrial electrogram types during atrial fibrillation: effect of rapid pacing and intercaval junction ablation. *J Am Coll Cardiol.* 1996;*27*:1713-1721. Crossref. PubMed.
20. Olgin JE, Kalman JM, Fitzpatrick AP, Lesh M. Role of atrial endocardial structures as barriers to conduction during human type I atrial flutter: activation and entrainment mapping guided by intracardiac echocardiography. *Circulation.* 1995;*92*:1839-1848. Crossref. PubMed.