Variations in duration and composition of the excitable gap around the tricuspid annulus during typical atrial flutter

Variations dans la durée et la composition de la fenêtre d’excitabilité du flutter atrial typique autour de l’anneau tricuspidien

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Summary

Background. — Differences in the duration of the excitable gap along the reentry circuit during typical atrial flutter are poorly known.

Aim. — To prospectively evaluate and compare the duration and composition of the excitable gap during typical counterclockwise atrial flutter in different parts of the circuit all around the tricuspid annulus.

Methods. — The excitable gap was determined by introducing a premature stimulus at various sites around the tricuspid annulus during typical counterclockwise atrial flutter in 34 patients. Excitable gap was calculated as the difference between the longest resetting coupling interval and the effective atrial refractory period.

Abbreviations: AFL, atrial flutter; AFLCL, atrial flutter cycle length; AR, atrial roof; CSO, coronary sinus ostium; CTI, cavotricuspid isthmus; EAR, effective atrial refractory period; EG, excitable gap; EFG, fully excitable gap; HRA, high right atrium; LRA, low right atrium; PEG, partially excitable gap; PPI, post-pacing interval; RCI, resetting coupling interval; RF, radiofrequency; Sept, right atrial septum.

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Background

The mechanism of typical atrial flutter AFL in humans is based on a large macro-reentry located in the right atrium, with a counterclockwise reentrant wavefront rotating around the tricuspid annulus and surrounding a central obstacle formed by the inferior vena cava and adjacent areas of functional block [1,2].

Reentry in AFL displays an excitable gap (EG) between the tail of the refractory period of the previous wave and the front of the next activation [3–5]. The EG can be defined as the part of the cycle during which the tachycardia can be advanced by a premature stimulus [6], and can be clinically evaluated by analysing the resetting response after a premature stimulation [4,5,7].

In humans, the EG during typical AFL has a duration ranging from 30–100 ms, representing 15–40% of the atrial flutter cycle length (AFLCL) [3,4,7–14], and can be divided into fully and partially excitable parts [3–5,9,11,13]. The presence of a large EG is particularly relevant for the stability of the arrhythmia, for the ability to stop reentry acutely using antiarrhythmic drugs [15,16], and for the possibility of capture of the circuit by pacing for entrainment and termination of the tachycardia [4,6,17].

Determination of the duration and composition of the EG in humans has been performed mainly at the cavitricuspid isthmus (CTI) [4,8] or at the lateral right atrial wall [3,7]. To date, only one study has investigated the EG at various parts of the right atrium [9]. The aim of this study was to prospectively evaluate and compare the duration and composition of the EG during typical counterclockwise AFL in different parts of the circuit all around the tricuspid annulus.

Methods

Thirty-four consecutive patients referred for radiofrequency (RF) ablation of typical counterclockwise AFL were prospectively included. Only patients with typical AFL, with negative sawtooth waves in the inferior leads, counterclockwise right atrial activation and successful RF ablation at the CTI were included. Patients with a previous history of atrial RF ablation were not included.

AFL was present at the beginning of the procedure in each patient. One standard quadripolar electrode-catheter (interelectrode distance of 10 mm) and one roving RF catheter (Bard Stinger, with 8-mm tip and 10-mm inter-electrode distance at the distal dipole) were inserted percutaneously via the right femoral vein. The quadripolar catheter was positioned under fluoroscopic guidance along the tricuspid annulus at the right atrial lateral wall, to enable stable pacing/detection from the high right atrium.
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Figure 1. Schematic representation of the tricuspid annulus in left anterior oblique view: the quadripolar catheter allows pacing from the high right atrium (HRA; 10 o’clock) and the low right atrium (LRA; 8 o’clock), while the radiofrequency catheter is successively placed at different locations along the tricuspid annulus: at the cavotricuspid isthmus (CTI; 6 o’clock), at the coronary sinus ostium (CSO; 4 o’clock), at the right atrial septum (Sept; 2 o’clock) and at the atrial roof (AR; 12 o’clock). IVC: inferior vena cava; SVC: superior vena cava.

(HRA; 10 o’clock position in left anterior oblique view) and the low right atrium (LRA; 8 o’clock), using the proximal and distal dipoles, respectively (Fig. 1). The tip of the RF catheter was then successively placed at different locations along the tricuspid annulus: at the CTI (6 o’clock), at the coronary sinus ostium (CSO; 4 o’clock), at the right atrial septum (Sept; 2 o’clock) and at the atrial roof (AR; 12 o’clock), for resetting manoeuvres (Fig. 1). Filtered and amplified bipolar intracardiac electrograms were recorded on a Cardiolog® system (Prucka Eng., Houston, TX, USA). Measurements were made at a speed of 100 mm/s. Regularity of AFL was checked at baseline, and patients with any alternans in AFLCL or differences greater than 10 ms between successive or non-successive cycles were excluded.

The EG during AFL was determined once at each atrial pacing site before any RF application, by introducing a premature stimulus and analysing the effect on AFL as previously described [6,7,10]. Bipolar pacing and detection were always performed from the same pairs of electrodes from the quadripolar or RF catheter. Briefly, single extrastimuli (bipolar, 20 mA output, 2 ms duration) were delivered every eight sensed atrial complexes with progressively shorter coupling intervals, in 10 ms decrements, beginning 10 ms below the AFLCL and up to atrial refractoriness. Coupling intervals were measured between the intrinsicoid deflection of the last atrial activation and the spike artefact. Analysis of the following atrial intervals allows determination of when the reentry has been resetted (i.e. the tachycardia has been advanced by the premature stimulus [6]), indicating that the premature paced beat has entered the reentry circuit during the EG. Lack of atrial capture was easily detected by the lack of resetting of AFL and the lack of anticipated atrial beat, which was always clearly visible, at least in the remaining intracardiac leads.

RCI was defined as the longest coupling interval allowing resetting, and EAR as the longest coupling interval failing to result in an atrial depolarization. The duration of the whole EG was then calculated as the difference between RCI and EAR [7,10]. Furthermore, for each coupling interval leading to resetting, the duration of the PPI was measured between the spike artefact and the intrinsicoid deflection of the following atrial activation. The response pattern characterized the way the tachycardia is transiently entrained by the coupling intervals, leading to resetting, and is drawn from the evolution of the PPI according to the coupling intervals of the premature paced beat. Response pattern was considered as flat (< 10 ms difference between PPI for any coupling interval allowing resetting), increasing (prolongation of PPI by ≥ 10 ms when decreasing coupling intervals) or mixed (flat pattern for at least two successive coupling intervals and then an increasing pattern while still decreasing coupling intervals) [6,7]. In case of mixed pattern, the duration of the PEG was calculated as the difference between the longest coupling interval leading to an increased PPI and the EAR, whereas duration of the PEG was calculated as the difference between the duration of the PEG and the whole EG [6,7] (see examples in Figs. 2 and 3). A resetting response curve was constructed by plotting the duration of the PPI against the coupling interval, and the mean slope of the ascending part of the curve was evaluated by dividing the increase in PPI by the duration of the PEG [4,6,7].

Finally, to analyse the catheter location with regard to the reentry circuit, we evaluated the differences between PPI and AFLCL, and between AFLCL and RCI.

Statistical analysis

Statistical analysis was performed using the StatView® programme (Abacus Concepts Inc., version 4.57). Results are expressed as means ± standard deviations (ranges). Statistical comparison between categorical data was performed using Fisher’s exact test, while numerical variables were compared using Student’s unpaired t test. Analysis of variance repeated measures were used to analyse the differences between various pacing locations, and comparisons between sites were then performed using a post-hoc Bonferroni-Dunn test when a significant difference was found in the analysis of variance. A P value < 0.05 was considered statistically significant, except for post-hoc analysis (P < 0.0033 significant).

Results

Clinical characteristics of the population are listed in Table 1. Mean AFLCL was 248 ± 27 ms (210–300). RF ablation was successful in every case, with termination of AFL during RF application at the CTI and achievement of complete bi-directional CTI block in each patient. AFL recurred in one patient over a mean follow-up of 15 ± 7 months, who underwent a second and successful procedure.

Induction of atrial fibrillation or atypical AFL, change in AFLCL or termination of AFL did not occur because of the pacing protocol. Post-processing analysis revealed that resetting sometimes did not happen at some pacing sites, probably due to lack of atrial capture (eight patients, never
Figure 2. V1 lead and intracardiac recording (high right atrium [HRA]) depicting the technique used for determining the duration and composition of the excitable gap (EG). Atrial flutter cycle length (AFLCL) is 310 ms. A: late extrastimulus (coupling interval 290 ms) fails to advance the following atrial event (i.e. the interval encompassing the stimulus is exactly twice the AFLCL = 620 ms), implying that no resetting has occurred. B: resetting first occurs with a coupling interval of 240 ms (resetting coupling interval [RCI]), advancing the next atrial depolarization, with a post-pacing interval (PPI) = 360 ms. C: the same happens with a shorter coupling interval of 200 ms and PPI is 360 ms again. D: when the coupling interval is shortened to 190 ms, atrial flutter (AFL) is still resetted but PPI lengthens to 380 ms. E: for an early coupling interval of 160 ms, AFL is still resetted with a much longer PPI of 410 ms. F: resetting no longer happens with an earlier coupling interval of 150 ms because the effective atrial refractory period (EARP) is reached (spontaneous atrial event after the spike) and the encompassing interval is again twice the AFLCL. Duration of the whole EG is therefore RCI (240 ms) – EARP (150 ms) = 90 ms. Duration of the partially EG (PEG) can be calculated as the difference between the longer coupling interval leading to increased PPI and EARP = 190 – 150 ms = 40 ms. Duration of the fully EG can be calculated as the difference between the durations of the PEG and the whole EG = 90 – 40 ms = 50 ms. The slope of the resetting response curve is evaluated as 50 ms/40 ms = 1.25 ms/ms. See text for explanation.
Figure 3. Example of excitable gap (EG) with an increasing-type response at the low right atrium (LRA). A: the latest coupling interval has already reset atrial flutter (as the interval encompassing the stimulus is slightly less than twice the atrial flutter cycle length). B: following the next coupling interval, the post-pacing interval (PPI) already increases, indicating an increasing-type response. C: earliest coupling interval leading to atrial capture followed by very long PPI. D: a still earlier coupling interval reaches atrial refractoriness as there is no capture and there is no more resetting. The whole EG duration is therefore 100 ms in this example, without the FEG. The slope of the resetting response curve is evaluated as 85 ms/100 ms = 0.85 ms/ms. See text for explanation. Of note, the remaining intracardiac lead allows clear determination of atrial capture (arrows in C and D). Correct location of the catheter along the tricuspid annulus is attested by the recording of ventricular far-fields potentials (V). HRA: high right atrium.

Table 1 Clinical characteristics of the population (n = 34). Data are mean ± standard deviation or number (%), unless otherwise indicated.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Men/women (n/n)</th>
<th>Age (years)</th>
<th>Underlying heart disease</th>
<th>Preserved ejection fraction</th>
<th>Ejection fraction (if altered)</th>
<th>Previous atrial fibrillation</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>31/3</td>
<td>69 ± 8 (53—86)</td>
<td>28 (82)</td>
<td>20 (58)</td>
<td>35 ± 11 (range, 20—50)</td>
<td>14 (41)</td>
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<td>Chronic pericarditis</td>
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<tr>
<td>Miscellaneousa</td>
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<tr>
<td>Antiarrhythmic drugs (amiodarone)</td>
<td></td>
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</tbody>
</table>

a Association of more than one of the above listed heart diseases.

more than one site per patient, twice at the HRA, AR and Sept, once at the CTI and CSO, P = not significant; this was responsible for a total of 88 missing values (7% of the total data). However, resetting and therefore regular capture occurred consistently for each of the remaining pacing attempts.

Electrophysiological variables determined during AFL are depicted in Table 2. Durations of the whole EG (P = 0.042), EARP (P = 0.027) and RCI (P = 0.0006) differed significantly all along the tricuspid annulus (Table 2 and Fig. 4). Conversely, the differences in FEG, PEG and the slope of the resetting response curve between the different pacing sites did not reach statistical significance. When expressed as a percentage of the AFLCL, differences in EARP (P = 0.037), the whole EG (P = 0.044) and RCI (P = 0.0017) were still significant, while differences in PEG and FEG were not (Table 2).

There was no change in these results according to sex, preserved or altered ejection fraction, presence and type of underlying heart disease, previous history of atrial fibrillation, age older or younger than 65 years, or AFLCL longer or shorter than 250 ms.

Post-hoc analysis revealed a significantly longer duration of the whole EG at the LRA than at the CTI, whether in absolute values (P = 0.002) or relative to the AFLCL (P = 0.002). Longest EARPs were found at the CTI and shortest EARPs at the HRA, but with only borderline significant difference in
Table 2. Electrophysiological variables determined during atrial flutter at various pacing sites along the reentrant circuit.

<table>
<thead>
<tr>
<th></th>
<th>LRA</th>
<th>HRA</th>
<th>Sept</th>
<th>CSO</th>
<th>CTI</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCI (ms)</td>
<td>229 ± 34</td>
<td>234 ± 32</td>
<td>241 ± 32</td>
<td>219 ± 31</td>
<td>222 ± 31</td>
</tr>
<tr>
<td>RCI/AFCL (%)</td>
<td>96 ± 27</td>
<td>94 ± 26</td>
<td>94 ± 24</td>
<td>90 ± 25</td>
<td>93 ± 25</td>
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<tr>
<td>EARP (ms)</td>
<td>65 ± 21</td>
<td>66 ± 21</td>
<td>67 ± 22</td>
<td>64 ± 21</td>
<td>62 ± 20</td>
</tr>
<tr>
<td>EARP/AFCL (%)</td>
<td>86 ± 19</td>
<td>85 ± 20</td>
<td>84 ± 20</td>
<td>88 ± 19</td>
<td>87 ± 19</td>
</tr>
<tr>
<td>EG (ms)</td>
<td>74 ± 22</td>
<td>66 ± 23</td>
<td>74 ± 24</td>
<td>65 ± 21</td>
<td>63 ± 20</td>
</tr>
<tr>
<td>EG/AFCL (%)</td>
<td>73 ± 19</td>
<td>71 ± 20</td>
<td>72 ± 21</td>
<td>70 ± 20</td>
<td>68 ± 19</td>
</tr>
<tr>
<td>Slope (ms/ms)</td>
<td>1.03 ± 0.45</td>
<td>1.03 ± 0.49</td>
<td>1.05 ± 0.48</td>
<td>1.03 ± 0.47</td>
<td>1.02 ± 0.46</td>
</tr>
</tbody>
</table>

AFLCL: atrial flutter cycle length; AR: atrial roof; CSO: coronary sinus ostium; CTI: cavotricuspid isthmus; EARP: effective atrial refractory period; EG: excitable gap; FEG: fully excitable gap; HRA: high right atrium; LRA: low right atrium; PEG: partially excitable gap; RCI: resetting coupling interval; SD: standard deviation; Sept: right atrial septum.


difference would be present between pacing sites (\( p < 0.019 \)) by the difference between RCI and EARP, this was due to the low number of patients, similar trends were observed when the 10 patients without amiodarone were analysed separately. In patients without amiodarone, the EG was the shortest at the CTI (65 ± 21 ms) and the longest at the LRA (89 ± 22 ms; \( P = 0.007 \) with paired \( t \) test) while EAR was the shortest at the HRA (134 ± 33 ms) and one of the longest at the CTI (143 ± 20 ms; \( P = 0.1 \)).

Differences between PPI and AFLCL and between RCI and AFLCL at various pacing sites along the reentrant circuit are depicted in Table 3. Minor but significant differences were found for both variables.

The response curve was mostly of the mixed-type, present in each patient in at least one pacing site, observed at 146 pacing sites (77% of the analysable sites), while the increasing-type was observed at 38 pacing sites (20%) in 17 patients and the flat-type at six pacing sites (3%) in four patients. Examples of resetting responses curves are shown in Fig. 5. In 15 patients, the mixed-type response curve was consistently observed at each pacing site. Increasing-type responses occurred at only one site in six patients and at several pacing sites (two to five) in 11 patients. Flat-type responses were observed twice in two patients and once in two other patients. The increasing-type response pattern was observed at least once at each location and the flat-type response pattern was observed at each pacing site except at the CTI. There was no significant difference between the response patterns according to the pacing site.

Discussion

In the present study, duration of the whole EG during typical counterclockwise AFL was significantly different all along the tricuspid annulus, with shortest values at the CTI, the CSO and AR, and longest durations at the right atrial lateral wall, with significant differences between CTI and LRA. As EG was defined in this study (as in many others\cite{3,7,9,13}) by the difference between RCI and EARP, this was due to longer refractory periods at the CTI than at the lateral right atrium, because there was no significant difference in RCI between pacing sites in post-hoc analysis.

In the present study, we measured the "local" EG at the pacing/sensing site, which may be more representative of the "clinical" EG that can be used in clinical practice. An underestimation of the EG inside the circuit, which could be defined as the difference between cycle length and refractory period\cite{3,4,10}, is therefore not excluded. Changing the definition of EG would not have fundamentally modified our results: if EG had been defined by the difference between AFLCL and EARP, an even more significant difference would be present between pacing sites (\( p = 0.019 \)), with lowest values at the CTI (89 ± 17 ms) and higher values at the HRA (101 ± 20 ms), whereas CSO (100 ± 22 ms), LRA (97 ± 20 ms), AR (96 ± 21 ms) and Sept (90 ± 24 ms) would display intermediate values. Differences in our initial results were caused by the shorter RCI at the HRA, CSO and AR, probably due to the fact that these sites are slightly remote...
from the reentry path in at least some patients. Nevertheless, in this study, catheters were located close to the reentry circuit in all patients: firstly, RCI was close to the AFLCL (mean difference $24 \pm 15$ ms, $0–70$ ms), implying that there was no significant interposed tissue between the pacing site and the reentry circuit [5,9]; second, PPI duration in case of flat pattern was also close to the AFLCL [5] (mean difference $12 \pm 13$ ms, $0–60$ ms). Although significant, the existing differences in these variables between pacing sites were minor (Table 3), and can be attributed to variations in the upper part of the reentry circuit in a subset of patients or to the fact that pacing could have been performed inside the proximal coronary sinus in some patients.

| Table 3 Differences between resetting coupling interval and atrial flutter cycle length, and between post-pacing interval$^a$ and atrial flutter cycle length at various pacing sites along the reentrant circuit. |
|-----------------|---------|-----|-----|-----|-----|-----|--------|
|                 | HRA     | LRA | CTI | CSO | Sept | AR   | Mean ± SD (range)     | $p^b$ |
| RCI–AFLCL (ms)  | $28 \pm 15$ | $18 \pm 13$ | $21 \pm 13$ | $31 \pm 16$ | $20 \pm 11$ | $27 \pm 16$ | $24 \pm 15$ (0–70) | $0.0006$ |
| PPI–AFLCL (ms)  | $16 \pm 14$ | $10 \pm 15$ | $7 \pm 10$ | $17 \pm 15$ | $10 \pm 12$ | $12 \pm 11$ | $12 \pm 13$ (0–60) | $0.004$ |

AFLCL: atrial flutter cycle length; AR: atrial roof; CSO: coronary sinus ostium; CTI: cavotricuspid isthmus; HRA: high right atrium; LRA: low right atrium; PPI: post-pacing interval; RCI: resetting coupling interval; SD: standard deviation; Sept: right atrial septum.

Post-hoc analysis: for the differences between AFLCL and RCI, shortest values (LRA and Sept) both differed significantly from the largest one (CSO) ($p=0.0001$ and $0.001$, respectively); for the differences between PPI and AFLCL, the shortest value (CTI) differed significantly from both the largest ones (CSO and HRA, $p=0.001$ and $0.002$ respectively). Even if significant differences exist between pacing sites for both variables, caused by the more or less proximity of the pacing site to the reentry circuit, the values of these variables and their differences are, however, rather reduced, allowing the conclusion that pacing sites were generally included in or very close to the reentry circuit.

$^a$ PPI values are determined for flat response patterns only.

$^b$ $P$ value in analysis of variance repeated measures.
Figure 5. Examples of the three types of resetting response curve constructed by plotting the duration of the post-pacing interval (ms) against the coupling interval (ms): A: mixed. B: flat. C: increasing (see text for explanation).

Referring to previous studies using the same methodology, the duration of the EG during typical counterclockwise AFL in humans is in the range of 30–100 ms, representing 15–40% of the AFLCL [3,4,7–14] according to the various pacing sites and the presence or lack of antiarrhythmic drugs. Durations of the EG in our study were found to be relatively similar to those in many of these other studies when identical pacing sites were compared [3,7,13].

Refractory periods may vary from site to site in a reentrant circuit, so that duration and composition of the EG depend on where pacing is performed inside the circuit [6]. To date, only one study had compared the durations of the EG in different parts of the right atrium [9]. In this study, EGs were significantly longer at the posterior right atrium and septum than at the coronary sinus and lateral right atrium [9]. Discrepancies with our study can be explained, firstly, by the fact that some pacing sites were different (not located around the tricuspid annulus and therefore probably not located in the circuit, although PPI was generally close to the AFLCL) and, secondly, by the fact that pacing was not performed at the CTI. Moreover, EG was somewhat differently defined in this work, and AFCLs were shorter, while refractory periods were similar to our findings (possibly because none of the patients was receiving amiodarone) making the EG shorter and therefore difficult to compare [9].

The EG during experimental AFL in dogs is characterized by an incomplete recovery of excitability [18]. Conversely, in previous works in humans, a fully excitable part of the EG during AFL was present in the majority of patients [3,4,7,9,13] and, when determined, durations of FEG ranged between 10 and 40 ms [3,4,7], relatively similar to our findings. Duration of the PEG (30 ms) and the slope of the resetting response curve (1.1 ms/ms) in our previous study [7] were also similar to our current results. Durations of the PEG and the FEG as well as the slope of the resetting response curve did not statistically differ according to the pacing site, even if CTI displayed one of the shortest FEGs. For each pacing site, an FEG was present in the majority of patients, and there was no difference between sites according to the flat/mixed or increasing-type of response pattern. Therefore, composition of the EG and characteristics of the PEG do not seem to change significantly according to the pacing site in our study.

It is interesting to note that the site with one of the shortest EGs and the longest EARP is also the site that has displayed the slowest conduction in many previous reference works [19–21]. Long refractory periods at the CTI during AFL have already been mentioned [16]. Increased refractoriness and decreased conduction velocity may therefore both locate at the CTI, which is the protected isthmus of the reentry circuit during typical AFL.

It has been previously concluded that EG increases at the exit of the isthmus because of slow isthmus conduction allowing more time for the distal areas to recover [9]. We found that EG duration is indeed longer at sites distal to the CTI (CSO or septum) but also at sites proximal to the isthmus (LRA). Our interpretation is that areas showing altered conduction velocities also present with prolonged refractoriness, and that slow conduction into a particular area should increase AFLCL and therefore EG duration in any other part of the circuit, not only in the area distal to the isthmus. In other words, EG duration is only dictated by local refractoriness because AFCL is constant and equal, whatever the pacing site.

Two-thirds of our patients were receiving amiodarone, which may have led to changes in electrophysiological findings [7,22]. Therefore, our results apply mainly to patients receiving amiodarone. However, similar trends were found in the subgroup without amiodarone, even if they were sometimes non-significant due to the low number of cases: for example, CTI also displayed the shortest EG and one of the longest EARPs in this subgroup. Thus our results can be extended to patients who are not receiving antiarrhythmic drugs. Besides, it has been reported that modifications of the EG by drugs were not different when measurements were made at different pacing sites [10].
Clinical implications

Drug-induced AFL termination usually happens at the CTI, whether using class 1 agents to suppress conduction in the zone of slow conduction without abolition of the EG [16,23] or by eliminating the EG using class 3 drugs [16,24]. The association of increased refractoriness together with slow conduction at the CTI furnishes additional clinical arguments for explaining these observations.

Atrial overdrive pacing is a widely accepted technique for conversion of AFL. The duration of the EG has been shown to modify the success rate of overdrive pacing [1,2]. From our results, it appears that pacing at the LRA would enhance the success rate of overdrive pacing due to the longer local EG, as already mentioned [9]. Further studies are mandatory in order to evaluate this hypothesis, as this issue is still being debated. Discussion about this particular point can become even more complex, as it was also demonstrated that the success rate of overdrive pacing can be increased using class 1 drugs that increase the EG during AFL [4,10], but also by using class 3 drugs that, conversely, reduce the duration of the EG [14,25].

Study limitations

Increasing response pattern is provoked by an altered conduction velocity of the paced beat because of relative refractoriness from the previous activation, but this does not strictly mean that this slowed conduction locates at the pacing site. As we did not use multipolar catheters, we were unable to locate the area of slowed conduction in such cases. In fact, Callans et al. demonstrated that the increase in return cycle after pacing from the LRA or low septal right atrium was caused by conduction delay at the CTI [3]. This could explain the lack of difference in durations of the PEG and the FEG according to the pacing site in our patients.

High output pacing was consistently used to allow constant atrial capture in areas where contact was sometimes not optimal. This should have led to occasional decreased refractoriness and therefore to major EG duration — or to not optimal. This should have led to occasional decreased atrial capture in areas where contact was sometimes not optimal. This should have led to occasional decreased atrial capture in areas where contact was sometimes

Conflict of interest statement

None.

References


