A novel pacing manoeuvre to diagnose atrial tachycardia

Andrea Sarkozy1,2*, Sergio Richter1, Gian-Battista Chierchia1, Carlo De Asmundis1, Christos Seferlis1, Pedro Brugada1, Leonard Kaufman3, Ronald Buyl1, Paul Dorian2, and Iqwal Mangat2

1Heart Rhythm Management Center, Cardiovascular Center, UZ Brussel—Vrije Universiteit Brussel, Laarbeeklaan 101, Brussels 1090, Belgium; 2Cardiology Department, St Michael's Hospital, Toronto, Canada; and 3Department of Biostatistics and Medical Informatics, UZ Brussel—Vrije Universiteit Brussel, Laarbeeklaan 101, Brussels 1090, Belgium

Received 1 December 2007; accepted after revision 19 January 2008; online publish-ahead-of-print 25 February 2008

Aims Currently used diagnostic manoeuvres at the electrophysiology study do not always allow for consistent identification of atrial tachycardia (AT), either because of inapplicability of the technique or because of low predictive value and specificity. The aim of this study was to determine whether overdive atrial pacing during paroxysmal supraventricular tachycardia (SVT) with the same cycle length from both the high right atrium and the coronary sinus can accurately identify or exclude AT by examining the difference between the V–A intervals of the first returning beat of tachycardia between the two pacing sites.

Methods and results Fifty-two patients were included; 24 patients with atrioventricular nodal re-entry tachycardia (AVNRT), 13 patients with atrioventricular re-entry tachycardia (AVRT), and 15 patients with AT. Comparing the 37 non-AT patients with the 15 AT patients, there was a highly significant difference between the mean V–A interval difference, (delta V–A) 2.1 ± 1.8 ms (range 0–9 ms) vs. 79.1 ± 42 ms (range 22–267 ms) (P, 0.001), respectively. None of the patients in the non-AT group had a delta V–A. In contrast, all 15 patients with AT had a delta V–A interval .10 ms. Thus, the diagnostic accuracy of the delta V–A interval cut-off of .10 ms was 100%, with a 95% confidence interval of 93.1–100% for AT. In 11 (73%) of the 15 AT patients, the standard ventricular overdrive pacing manoeuvre was not possible. In 14 of the 15 patients (93%) in the AT group, standard atrial overdrive pacing showed variable V–A intervals, correctly diagnosing AT. In all 52 patients, this measurement was repeated during pacing from the other location. In five patients from the AT group, the result of the second attempt was different from the result of the first attempt.

Conclusion We found that atrial differential pacing during paroxysmal SVT without termination of tachycardia and the finding of variable returning V–A interval was highly sensitive and specific for the diagnosis of AT. The manoeuvre can be easily performed in all patients with SVT and is highly reproducible. It is a useful adjunct to the currently available ventricular and atrial pacing manoeuvres.

KEYWORDS
Atrial tachycardia; Atrial overdrive pacing; Paroxysmal supraventricular tachycardia

Introduction
For the successful ablation of supraventricular arrhythmias, it is essential to differentiate between focal atrial tachycardia (AT) and the other types of re-entrant supraventricular tachycardias (SVTs), namely atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reciprocating tachycardia (AVRT). Typically, baseline observations, tachycardia characteristics, and ventricular and atrial pacing manoeuvres during tachycardia are used in an attempt to diagnose the arrhythmia.1–3 However, the diagnosis of AT, especially septal AT, is sometimes challenging and time consuming in spite of the available pacing manoeuvres.

Currently, the standard manoeuvre is the analysis of the response following ventricular overdrive pacing with entrainment but without termination of the tachycardia.1 It has been described that the A–A–V response to ventricular overdrive pacing is highly specific and sensitive for the diagnosis of AT.1 However, this manoeuvre may result in pseudo A–A–V response in some forms of AVNRT and AVRT.4–6 In addition, the manoeuvre is only feasible in 80% of SVTs.1 A second useful pacing manoeuvre described for the differential diagnosis of AT is atrial overdrive pacing during tachycardia. Following the overdrive pacing, the V–A interval between the last paced conducted ventricular beat and the first spontaneous atrial beat is compared with the V–A interval during tachycardia.1,3 The same V–A interval is expected in AVRT and AVNRT due to the V–A linking, whereas a variable V–A interval is expected in AT.
However, this manoeuvre was found in a prospective study to be moderately sensitive and specific for the diagnosis of AT.\(^2\) This was due to accidental V–A linking in AT and V–A interval changes of the first beat compared with the subsequent beats in AVNRT.\(^2\)

We propose a new method of analysing the response to atrial overdrive pacing during SVT to better differentiate AT from the other types of re-entrant arrhythmias, namely AVRT and AVNRT. The objective of this study was to determine whether overdrive atrial pacing during SVT with the same cycle lengths from both the high right atrium (HRA) and the coronary sinus (CS), with atrial capture and without termination of tachycardia, can accurately identify and the coronary sinus (CS), with atrial capture and

synchronous atrial extrasystoles. The absolute differences between the V–A times of the return beat when pacing from the HRA (V–AHRA) and CS (V–ACS) locations were measured on each attempt. This difference was called the delta V–A (DVA) interval and was calculated as \(|V–AHRA – V–ACS|\) in milliseconds. The same reference points on the same channels were used for the V–A interval measurements. Subsequently, the V–A interval during the tachycardia was also measured following the pacing attempt from both the HRA and the CS. As described previously,\(^2\) if the difference between the returning V–A interval and the tachycardia V–A interval was \(< 10\) ms, V–A linking was diagnosed. If the V–A interval difference was \(> 10\) ms, variable V–A was diagnosed.

**Methods**

After informed consent, electrophysiological studies were performed using standard techniques. All antiarrhythmic drug therapies were discontinued at least five half-lives prior to the study. Standard multipolar electrophysiology catheters were introduced via the right femoral and right jugular veins and advanced to the HRA, CS, His bundle region (HIS), and right ventricular apex. Intracardiac electrograms were filtered at 30-500 Hz. After baseline measurements were made, pacing manoeuvres were performed to evaluate atrioventricular and ventriculoatrial conduction. Overdrive atrial and ventricular pacing or premature extrastimuli were used to induce sustained SVT. Isoproterenol was infused at a rate of 2–4 mcg/min if the tachycardia was non-sustained or non-inducible at baseline. Once tachycardia was induced, standard criteria were used to establish the diagnosis of AT, AVNRT, or AVRT. In each patient, the following observations were made: at baseline—(i) presence of anterograde delta wave, (ii) response to parahisian pacing (nodal vs. extranodal); during tachycardia—(i) septal V–A activation time, (ii) tachycardia termination with spontaneous or paced ventricular premature beats (with or without atrial activation), (iii) presence of eccentric atrial activation during tachycardia, (iv) premature ventricular extrasystole during His bundle refractoriness causing atrial pre-excitation, and (v) premature atrial extrasystole during His bundle refractoriness causing ventricular pre-excitation. Only patients with a definite diagnosis were included in the study. In all patients, ventricular overdrive pacing at a cycle length 10–40 ms less than the tachycardia cycle length was attempted at least three times. The presence of atrial entrainment and the A-A-V or A-V response to ventricular overdrive pacing were determined. Subsequently, the novel pacing manoeuvre was performed. Specifically, during SVT, atrial pacing from the HRA at a cycle length of 10–40 ms less than the tachycardia cycle length was attempted. Atrial capture was confirmed, but ventricular entrainment was not necessary. If the same tachycardia continued, the atrial overdrive pacing was repeated with the same cycle length from the proximal or mid-CS. If the tachycardia was reproducibly inducible, these pacing manoeuvres were repeated to assess reproducibility. If the tachycardia terminated during any of the pacing manoeuvres, it was re-induced, and the manoeuvre starting with the HRA pacing was repeated. All patients with at least one successful HRA and CS pacing attempt during the same tachycardia episode were included in the analysis. On all attempts, the presence of atrial capture was confirmed, and the V–A interval from the QRs following the last conducted paced atrial beat and the first returning spontaneous atrial beat was measured. It was confirmed that the first atrial beat had the same activation sequence as during tachycardia to exclude atrial extrasystoles. The absolute differences between the V–A times of the return beat when pacing from the HRA (V–AHRA) and CS (V–ACS) locations were measured on each attempt. This

**Statistical analysis**

Continuous variables are expressed as mean ± SD. The two-sided unpaired Student’s \(t\)-test was used to compare continuous variables. The measurement of agreement between the two attempts of the standard atrial overdrive pacing manoeuvre (for the presence or absence of V–A linking) was calculated using the kappa coefficient. A \(P\)-value <0.05 was considered statistically significant.

**Results**

**Patient population**

Fifty-two patients were included in the study. The baseline clinical characteristics of the patients are given in Table 1. The diagnosis was AVNRT in 24 patients; 22 patients had typical slow and fast AVNRT, one patient had atypical AVNRT, and one patient had both typical and atypical AVNRT. Thirteen patients had AVRT. Eleven patients had exclusively orthodromic tachycardia involving in eight patients a left lateral and in three patients a left posterolateral accessory pathway. Two patients had exclusively antidromic tachycardia: both these patients had anterograde decrementally conducting right-sided atriofascicular (Mahaim) accessory pathways. Altogether 37 patients had non-AT arrhythmia mechanism diagnosed (Group A). Fifteen patients had focal AT (Group B). The focal AT originated from the right atrium in 13 patients and from the left atrium in 2 patients. In one patient, a bystander accessory pathway was present.

**Novel atrial differential pacing manoeuvre**

The novel atrial differential pacing manoeuvre was performed in all 52 patients at least once. In order to assess the reproducibility of the manoeuvre, it was performed twice in 35 patients. For statistical analysis, the mean of the two measurements for each patient was calculated. In the AT group, the 15 patients had a mean DVA interval of 79.1 ± 42.0 ms, with a range between 22 and 267 ms (Figure 1). In the AVNRT patients, the mean DVA interval

| Table 1 Patients’ baseline clinical characteristics (n = 52) |
|-----------------|---------------------|
| Age (years)     | 47 ± 17             |
| Gender, n (%)   |                     |
| Male            | 13 (25)             |
| Female          | 39 (75)             |
| Heart disease, n (%) | 8 (15)          |
| CAD             | 3                   |
| Hypertension    | 2                   |
| Brugada syndrome| 1                   |
| Cor pulmonale   | 1                   |
| Mitral valve prolapse | 1               |
was 2.1 ± 2.0 ms, with a range between 0 and 9 ms (Figure 2). In the AVRT patients, the measured mean DVA interval was 2.2 ± 1.2 ms, with a range between 0 and 8 ms. Comparing the 37 non-AT patients with the 15 AT patients, there was a highly significant difference between the mean DVA interval; 2.1 ± 1.8 vs. 79.1 ± 42.0 (P < 0.001), respectively. More importantly, none of the patients on any of the 59 measurements in Group A had a DVA interval >10 ms. In contrast, all of the 15 patients from the AT group during 28 measurements had a DVA interval >10 ms. The cut-off value of >10 ms difference between the V-A intervals from the two pacing sites was 100% diagnostic for AT, with a 95% confidence interval between 93.1 and 100%. A V-A interval difference of ≤10 ms correctly excluded AT in 100% of the cases. The results of the second attempt in each of the 35 patients
with repeated measurements confirmed the results of the first attempt.

Standard ventricular overdrive pacing manoeuvre

The ventricular overdrive pacing manoeuvre was attempted at least three times in all study patients. In 41 (79%) of the 52 patients, atrial entrainment without tachycardia termination was achieved and the manoeuvre was feasible (Table 2). Ten of the remaining 11 patients had baseline no or poor retrograde V–A conduction, rendering atrial entrainment with ventricular pacing impossible and thus the manoeuvre inapplicable. Importantly, all 10 of these patients belonged to the AT group. In one additional patient from this group, atrial entrainment was on some attempts achieved, but the tachycardia terminated on all of these attempts. In summary, in 11 (73%) of the 15 AT Group B patients, the ventricular overdrive pacing manoeuvre was not feasible. In each of the remaining four AT patients, the ventricular response was A–A–V, correctly diagnosing AT. In the non-AT Group A, the manoeuvre was feasible in all 37 patients (100%). All of these patients had an A–V response, correctly excluding the diagnosis of AT on at least one attempt. However, one patient (3%) in Group A, who had both typical and atypical AVNRT, showed a pseudo A–A–V response on one attempt. This was due to retrograde double firing on both the fast and slow pathway with the last ventricular paced beat and re-initiation of an episode of atypical AVNRT.

Standard atrial overdrive pacing manoeuvre

During the first atrial overdrive pacing manoeuvre from the hRA, the V–A interval of the first returning beat was compared with the V–A interval of the tachycardia beats to assess V–A linking. In the presence of <10 ms difference, V–A linking was diagnosed. This measurement was repeated with atrial overdrive pacing from the proximal mid-CS, and the result was interpreted similarly as the presence or absence of V–A linking. The results of the second attempt were correlated to the first attempt to assess reproducibility of the manoeuvre. In Group B, 14 of 15 patients (93%) had variable V–A intervals on the first attempt and the manoeuvre correctly diagnosed AT (Table 2). However, in one patient in this group, the apparent V–A linking falsely ruled out the diagnosis of AT. In Group A, 36 of the 37 patients (97%) had V–A linking, correctly ruling out AT. However, in one patient (3%) with typical AVNRT, the V–A interval varied >10 ms, falsely suggesting the diagnosis of AT. In all 52 patients, the measurements were repeated following the second atrial overdrive pacing manoeuvre. Interestingly, in five patients, the result of the second measurement did not correspond with the first one (measurements of agreement: kappa coefficient 0.751, P ≤ 0.001). All of these patients belonged to the AT Group B. Four of these patients, who on the first attempt had variable V–A interval correctly diagnosing AT, showed apparent V–A linking on the second attempt when pacing from the other right atrial site. In contrast, one patient who showed apparent V–A linking on the first attempt had variable V–A intervals on the second attempt.

Discussion

Novel atrial differential pacing manoeuvre

In the current study, we tested a novel atrial differential pacing manoeuvre. We postulated that the absolute value of the difference in the V–A intervals between HRA and CS pacing will be minimal (<10 ms) in AVNRT or AVRT, since in these arrhythmias the first spontaneous atrial activation and V–A interval are dependent on the retrograde V–A conduction of the previous ventricular beat (Figure 3). In contrast, in AT, the V–A interval between the last conducted QRS complex after atrial pacing and first spontaneous AT beat is not linked. The V–A interval in this case is determined by the difference between the post-pacing interval (PPI) of the AT and the AV nodal conduction time of the last paced atrial beat. It has recently been shown that the PPI in focal AT is proportional to the distance of the pacing site from the focus irrespective of the mechanism of the tachycardia (automatic vs. non-automatic).7 We expected that when pacing is performed close to the AT focus, the PPI would be short, whereas pacing further from the tachycardia focus would result in a longer PPI. This difference in the PPI was expected to be accompanied with minor changes in the A–V conduction of the last atrial paced beat, resulting in a larger difference (>10 ms) between the two V–A intervals (Figure 4). After confirming these expectations first in a small patient population,8 now we report our results in larger patient population. In our study population of 52 patients with paroxysmal SVT, a V–A interval variability >10 ms with atrial differential pacing was 100% sensitive and specific for the diagnosis of AT. The advantage of this manoeuvre compared with the ventricular pacing manoeuvre is that it can be performed also in patients without retrograde V–A conduction. Furthermore, it can be used in all patients with focal AT irrespective of the tachycardia mechanism (automatic, re-entry, or triggered automacy). The difficulty while performing the manoeuvre might be the termination of tachycardia or conversion of one tachycardia to another, for example to atrial fibrillation.

Table 2 Standard atrial and ventricular overdrive pacing manoeuvres for the diagnosis of atrial tachycardia in our study population

<table>
<thead>
<tr>
<th>Pacing manoeuvre</th>
<th>Non-AT group, n (%)</th>
<th>AT group, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular overdrive pacing performed</td>
<td>37 (100)</td>
<td>15 (100)</td>
</tr>
<tr>
<td>Atrial entrainment without tachycardia termination</td>
<td>37 (100)</td>
<td>4 (27)</td>
</tr>
<tr>
<td>A–A–V response</td>
<td>1 (3)</td>
<td>4 (100)</td>
</tr>
<tr>
<td>A–V response</td>
<td>37 (100)</td>
<td>None (0)</td>
</tr>
<tr>
<td>Atrial overdrive pacing performed</td>
<td>37 (100)</td>
<td>15 (100)</td>
</tr>
<tr>
<td>V–A linking on first attempt</td>
<td>36 (97)</td>
<td>1 (7)</td>
</tr>
<tr>
<td>V–A linking on second attempt</td>
<td>36 (97)</td>
<td>4 (27)</td>
</tr>
</tbody>
</table>

Non-AT group, non-atrial tachycardia group; AT group, atrial tachycardia group.
Atrial overdrive pacing manoeuvre

Kadish and Morady\(^1\) first investigated atrial overdrive pacing as a method for diagnosing AT. In 53 patients with SVT, atrial overdrive pacing with progressively shorter cycle lengths was attempted. The curve relating the pacing cycle length to the V–A interval on the first beat following cessation of atrial pacing was analysed. They found that the curve was variable in patients with AT and was upsloping or flat in patients with AVNRT or AVRT.\(^3\) Subsequently, in a more recent prospective study by the same group, the diagnostic value of a similar atrial overdrive pacing manoeuvre has been investigated.\(^2\) Following high right atrial overdrive pacing, if the tachycardia continued upon cessation of pacing, the V–A interval of the first returning tachycardia beat following atrial overdrive pacing is only dependent on the retrograde V–A conduction and is independent from the site from which the overdrive pacing was achieved. On the inset, an example of the measurements in a patient with orthodromic atrioventricular re-entry tachycardia involving a left lateral accessory pathway is shown. The V–A interval is measured between the beginning of the QRS complex on the surface electrocardiogram and the beginning of the atrial activation on the Csd on both pacing attempts. The V–A hRA is measured at 86 ms. The V–A CS is measured at 86 ms. The delta V–A interval (DVA) is calculated as the difference between the V–A hRA and V–A CS, which is in this case 0 ms, excluding atrial tachycardia.

**Standard pacing manoeuvres**

**Atrial overdrive pacing manoeuvre**

Kadish and Morady\(^1\) first investigated atrial overdrive pacing as a method for diagnosing AT. In 53 patients with SVT, atrial overdrive pacing with progressively shorter cycle lengths was attempted. The curve relating the pacing cycle length to the V–A interval on the first beat following cessation of atrial pacing was analysed. They found that the curve was variable in patients with AT and was upsloping or flat in patients with AVNRT or AVRT.\(^3\) Subsequently, in a more recent prospective study by the same group, the diagnostic value of a similar atrial overdrive pacing manoeuvre has been investigated.\(^2\) Following high right atrial overdrive pacing, if the tachycardia continued upon cessation of pacing, the V–A interval of the first returning tachycardia beat was analysed. If this interval was within 10 ms of the V–A interval during tachycardia, it was categorized as fixed, otherwise it was defined as variable. A fixed V–A interval was expected in AVNRT and AVRT, when the first atrial activation following cessation of atrial pacing is linked to the previous ventricular activation. A variable interval would suggest the absence of V–A linking and thus the diagnosis of AT. In this study, the sensitivity of the fixed V–A interval was 97 and 98%, for AVNRT and AVRT, respectively. In the current study, we observed very similar results in the AVNRT/AVRT group: 1 of the 37 patients (3%) had a variable V–A time, falsely suggesting the diagnosis of AT. The variable V–A time in these patients is explained by the fact that although the retrograde atrial activation pathway in AVNRT and AVRT is stable and the same, there is sometimes variation in the conduction times. It has been described that changes in V–A interval during AVNRT mostly occur on the first beat after induction of tachycardia.\(^9\) In theory, the change in the V–A interval on the first beat compared with tachycardia might be due to the longer anterograde atrioventricular conduction on the last fast atrial-paced beat, allowing more time for the retrograde limb to recover, as during tachycardia. This problem

---

**Figure 3** Atrial differential pacing in atrioventricular nodal re-entry tachycardia (AVNRT) and atrioventricular re-entry tachycardia (AVRT). HRA, high right atrial; CS, coronary sinus; V–A interval, ventriculo-atrial conduction interval. On the insets, depicted are surface leads V1, V6, hRAd and hRAP (distal and proximal bipolar electrogram from a quadripolar catheter positioned in the high right atrium), and Csd and CSp (distal and proximal bipolar electrogram recorded from a quadripolar catheter positioned in the coronary sinus). In both conditions, the V–A interval on the first returning tachycardia beat following atrial overdrive pacing is only dependent on the retrograde V–A conduction and is independent from the site from which the overdrive pacing was achieved. On the inset, an example of the measurements in a patient with orthodromic atrioventricular re-entry tachycardia involving a left lateral accessory pathway is shown. The V–A interval is measured between the beginning of the QRS complex on the surface electrocardiogram and the beginning of the atrial activation on the Csd on both pacing attempts. The V–A hRA is measured at 86 ms. The V–A CS is measured at 86 ms. The delta V–A interval (DVA) is calculated as the difference between the V–A hRA and V–A CS, which is in this case 0 ms, excluding atrial tachycardia.
is avoided by the use of our pacing manoeuvre when we compare the V–A interval of the first returning beats with each other following pacing with the same cycle length. In the aforementioned study, the specificity of the fixed V–A interval for AVNRT and AVRT was 27 and 18%, respectively.2 The negative predictive value for AT in the presence of a fixed V–A interval was 71%. This was felt to be due to coincidental atrial and ventricular activation during AT, resulting in apparent V–A linking on the first returning beat.2 In the current study, we observed the same phenomenon, although less frequently. One of the 15 patients (7%) with AT had apparent atrial linking. In addition, in this study, we describe for the first time that the reproducibility of the atrial pacing manoeuvre especially in patients with AT is imperfect. It seems that the diagnostic accuracy of this method in AT is dependent on the location of the atrial overdrive pacing site. This finding is not surprising, considering the fact that the V–A interval is strongly dependent on the PPI. Thus, pacing close or far from the focus will result in different PPIs. Pacing on the second attempt closer to the focus and the A–V node, the post-pacing interval will be significantly shorter (PPI = 446 ms), with less shortening of the A–V conduction (Stim-V = 215 ms). Therefore, the V–A interval will be variable on the two attempts. The post-pacing interval calculation in the onset is for illustration, since it is measured between the stimulus and the local electrogram far from the stimulation channel.
Ventricular pacing manoeuvres
In two studies of patients with spontaneous and stimulated SVTs, the atrial response following the cessation of ventricular overdrive pacing during SVT resulting in 1:1 ventriculoatrial conduction has been investigated.1,2 The A-A-V response was highly specific and sensitive for the identification of AT. However, in these studies, 22% of patients were excluded from analysis because ventricular pacing resulted either in ventriculoatrial dissociation or termination of tachycardia. It was suggested that inability to achieve 1:1 ventriculoatrial conduction during SVT is highly suggestive, but not proof of AT.1,2 In our current study, we found similar results. Atrial entrainment was achieved and thus ventricular overdrive pacing was feasible in 41 (79%) of the 52 patients. However, we found in the current study that when considering only the patient population with AT, the manoeuvre was feasible only in 4 of the 15 patients (27%). These data suggest that ventricular overdrive pacing during SVT is very useful in non-AT patients to exclude AT, but it is frequently not applicable to confirm the diagnosis in patients with AT.

In addition, recent evidence suggests that in several situations, the A-A-V response can falsely suggest the diagnosis of AT. Pseudo A-A-V response has been described in patients with typical AVNRT and long H-V intervals and in patients with typical AVNRT and short H-A intervals.4,5 Furthermore, in atypical AVNRT when the V-A conduction during atrial entrainment is longer than the V-V cycle length, it is important to identify which is the last entrained atrial beat to avoid erroneous A-A-V labelling.4 In addition, pseudo A-A-V response can be evoked in patients with retrograde double firing, in the presence of a slow and fast conducting retrograde limb.6 This was the case in 1 of our 37 AVNRT/AVRT patients. Finally, the method might not be useful in antidromic tachycardia or bystander accessory pathway conduction.

Thus, overall, present diagnostic manoeuvres do not allow for consistent identification of ATs, either because of inapplicability of the technique or because of low predictive value and specificity.

Limitations
Theoretically, there are two situations in which our new manoeuvre might erroneously exclude AT. First, it is possible that pacing from the CS and the hRA is equally far from both the AT focus and the AV node, resulting in exactly the same PPIs and last atrial-paced beat to QRS conduction times, and thus no difference in the V-A intervals. To avoid this potential problem, pacing should be performed once close and once far from the presumed focus. Since in our experience the most frequent diagnostic problem is the differentiation of atypical AVNRT vs. septal AT, the suggested hRA and proximal CS pacing locations might avoid this problem. However, in the current study, we have included only one patient with atypical AVNRT. Therefore, an additional study with larger number of patients with atypical AVNRT is necessary to confirm the high diagnostic value of the new pacing manoeuvre in this patient population. Secondly, since the V-A interval is dependent on the AV conduction times and the PPIs, it is possible that pacing once close to the focus and once far away from the focus will result in exactly the same and reciprocal change in the AV conduction times and PPI. This would result in the same V-A intervals on the two pacing attempts. Although this is very unlikely to happen, changing the position of one of the catheters closer or farther from the focus and repetition of the manoeuvre should solve this problem. In addition, although the manoeuvre is expected to predict the diagnosis accurately in antidromic tachycardias and ATs with bystander accessory pathways, the manoeuvre needs further validation in these groups of arrhythmias since we only had two and one patient, respectively, in each of these groups. Furthermore, in the current study, isoproterenol was not administered to improve V-A conduction. The routine administration of isoproterenol for this purpose would have likely improved the usefulness of the ventricular overdrive pacing manoeuvre in the AT group. In addition, the ventricular pacing manoeuvre provides useful information also to exclude AVRT when the tachycardia continues despite dissociation of the ventricular activation. Finally, in our non-AT group, in one patient on one attempt the V-A interval difference was 9 ms. Although this value was under our cut-off value of 10 ms, the difference is within the error of measurement, thus it is likely that in a larger patient population, the specificity of our novel manoeuvre would be <100%.

Conclusions
We found that atrial differential pacing during paroxysmal SVT without termination of tachycardia and the finding of variable return V-A interval (>10 ms difference) was highly sensitive and specific for the diagnosis of AT. The manoeuvre can be easily performed in all patients with SVT and is highly reproducible. We also report that in our study population of unselected patients with spontaneous AT, the currently used gold standard manoeuvre of ventricular overdrive pacing is only feasible in the minority of patients. In addition, we found that the reproducibility of the other standard manoeuvre of atrial overdrive pacing and analysis of V-A linking is questionable in patients with AT. This is due to the fact that the result is strongly dependent on the pacing location. We conclude that the novel atrial differential pacing manoeuvre described is a useful adjunct to the currently available ventricular and atrial pacing manoeuvres.

Conflict of interest: none declared.

References
