REVIEW

QRS Subtraction and the ECG Analysis of Atrial Ectopics

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The recognition of the ubiquitous role of short coupled ectopics in the initiation of paroxysmal atrial fibrillation has renewed interest in their localization with the ultimate aim of facilitating their neutralization by catheter ablation. The P-waves resulting from such ectopics are partly or completely concealed by the QRST of the preceding beat and therefore their morphology has been underutilized for localization purposes. Subtraction has been the most commonly used technique for QRST suppression and though an averaged template offers a higher signal-to-noise ratio, the immediately preceding QRST provides the best match between template and target and allows simple, nearly real-time, and accurate subtraction without distorting the underlying P-wave. Algorithms derived from clinical tachycardias as well as pacemapping have permitted a rational analysis and accurate prediction (81%) of the correct pulmonary vein of origin. Other nonpulmonary sources may also be similarly localized, particularly with the help of a catalogue of pacemaps from various sites. Incessant arrhythmia and frequent aberrancy limit the effectiveness of QRST suppression by subtraction. Further improvements in the localizing resolution of the P-wave may depend upon knowledge of the relationship between recording electrodes and the underlying atria. **A.N.E. 2004;9(4):389–398**

atrial ectopics; T wave; subtraction; pulmonary veins; atrial fibrillation.

Atrial ectopics, frequently considered the benign consequence of too much caffeine or smoking, have recently been recognized as playing a ubiquitous role in initiating paroxysms of atrial fibrillation (AF).¹ Since the great majority of these ectopics have been found to originate from within and around the pulmonary veins, these structures have become a logical target for ablative treatment.^{2,3} However, their frequently multiple sites of origins as well as their capricious manifestation have made it difficult to eliminate them by radiofrequency ablation targeting the earliest activation and limited procedural success as well as significant recurrence rate have frequently been the consequence.⁴ On the other hand, surgical procedures performed either using knife-based lesions (which guarantee transmural and continuity) or visually guided endocardially delivered RF, even if limited to the left atrium, have been cited as being more effective. This is despite the fact that an EP lab is clearly the

ideal environment to study the mechanism of atrial fibrillation, to verify the outcome of antiarrhythmic interventions, and to promptly correct any deficiencies in the curative strategy.

STRATEGIES FOR CURATIVE AF ABLATION

High recurrence rates after AF ablation call for renewed effort in optimizing the procedure in light of the current understanding of the mechanism of atrial fibrillation. The present strategy has been to broaden the original technique of focal targeting to an isolating ablation of the myocardial tissue within a culprit pulmonary vein and more recently to isolating all or as many of the pulmonary veins as possible.^{3,4} Despite such "anatomic" strategies, there may still be advantages to identifying and tracking specific sources of ectopic triggers. Because of the many variables affecting lesion size, it may be

easier to achieve effective stable isolation (particularly with currently available technology) for fewer culprit pulmonary veins by identifying them rather than by distributing the ablation effort over more numerous anatomically chosen (and perhaps less critical) targets.

THE SURFACE ECG : A SIMPLE TOOL

A simple strategy toward localization may be to use the information contained in the surface ECG—which has, however, been frequently ignored or underutilized. The surface ECG represents the total of the potentials generated by the heart as recorded on the body surface at specific sites and therefore contains information on all electrical events occurring in the heart. It has the advantage over intracardiac catheters of being independent of the limitations of positioning and stabilization but several limitations as far as assessing atrial activity is concerned. Because of their lower muscular mass, the atria generate lower voltages producing a low-amplitude low-frequency P wave. In the case of ectopics with short coupling intervals, the greater amplitude of ventricular depolarization obscures underlying atrial activity. The lack of clearcut differences in the frequency spectra of atrial and ventricular electrical activity also makes distinction between the two more difficult.⁵

Over the years, while the combination of the 12 standard ECG leads/derivations has remained unchanged, advances in recording systems, signal processing, and better noise reduction algorithms⁶ have allowed greater appreciation of subtle P wave variations. Notches in the P wave, low-amplitude initial or terminal negativity or positivity juxtaposed with a dominant deflection of opposite polarity are difficult to appreciate with a poor signal-to-noise ratio. In contrast, the greater electrical forces generated by the larger ventricular myocardial mass have allowed the recognition of various notches, and other features of intraventricular conduction defects almost since the advent of electrocardiology even with inferior signal-to-noise ratios.

P wave analysis is also affected by interindividual variations in the properties of the volume conductor (the patient's thorax and body), in the relationships between electrode body surface positions, and the underlying heart as well as differences in electrode placement on the body surface. The latter issue can make comparison of ECGs taken at different times even in the same patient troublesome.

For example, the surface ECG used in the interventional or electrophysiology lab is characterized by differences in precordial transition because of variations in chest electrode positions (in order to improve fluoroscopic visualization of the heart).

P WAVE ANALYSIS

In order to optimally analyze the surface ECG ectopic P wave, a good-quality tracing with a stable baseline, correct, standard electrode positioning, and a noise-free and sensitive recording within the bandwidth of interest are essential. Suppression or elimination of obscuring ventricular activity—both depolarization as well as repolarization—is necessary when short coupled ectopics are encountered and finally so is a reasoned analysis of the surface ECG P wave.

Various algorithms have been described in order to remove baseline wander and eliminate power line interference and this progress has been an important source of amelioration of signal-to-noise ratios. It is, however, uncertain whether the eliminated bandwidth contains useful information, for example, about repolarization.

The positioning of surface ECG electrodes as well as their relationship to the atria, the underlying source of the P wave, may result in significant but spurious variations. While the standard limb lead electrodes outside the EP or cath lab are usually placed far enough away from the thorax for small differences in positioning to have minor or inconsequential effects, the positioning of the unipolar chest lead electrodes is much more variable, possibly due to factors such as obesity, large breasts, torso size, simple miscalculation, or emphysema. Artifactual changes in the site of QRS transition can occur as a result of malpositioned unipolar chest leads.

QRST SUPPRESSION BY SUBTRACTION

Perhaps the most significant insight into the mechanism of AF has been the recognition of a common and reproducible mechanism of its initiation.¹⁻³ Mapping data from patients with paroxysmal atrial fibrillation studied in the EP lab have shown that the ectopics initiating atrial fibrillation have a short coupling interval—a mean of 210 ms.¹ This coupling interval (measured from local left atrial activity in sinus rhythm to the earliest

endocardial activity) results in the ectopic P wave coinciding in most cases with the T wave, which conceals its onset as well as its morphology. In many cases, careful comparison of adjacent beat T waves is necessary to even discern the presence of underlying ectopic P waves; and while it may be possible to detect maximum positivity or negativity by such comparison, detecting the P wave onset or analyzing its morphology are usually completely impossible. A proper assessment of the P waves produced by short coupled ectopics is only possible if ventricular activity can be suppressed without distorting the underlying atrial activity. In the digital equivalent of comparing the ectopic concealing T wave with a normal T wave (for evidence of an underlying ectopic P wave), the ectopic P wave can be unmasked by subtracting the signal of a matching normal QRS (Fig. 1). A digitized ECG tracing, which consists of a time series of voltage measurements representing the ECG trace, makes it possible to minus the arithmetic value of the voltage measurement of a suitable template QRS. This is graphically shown in Figure 1.

AVERAGED VERSUS PRECEDING QRS AS A TEMPLATE

In one approach, a series of beats were used to generate an averaged beat to be used as a template.⁷ Though the averaging process eliminates nonsynchronous noise in order to obtain the best possible signal-to-noise ratio, temporal variations occurring over the averaging period are modified or reduced and as a result reduce its match to the target QRS to be subtracted. Temporal variations include transient differences in repolarization (and therefore in

depolarization) as a result of changes in posture, autonomic tone, or activity. Clearly evident changes in repolarization (such as aberrant ventricular conduction or intermittent bundle branch block) produce major secondary changes in depolarization but the less obvious changes in repolarization (occurring even within a period of a few beats) are important for subtraction. They are reflected in variations in the QT interval as well as differences in the T wave contour. Since these changes can even occur on a beat-to-beat basis (e.g., with sinus arrhythmia), any averaged beat inevitably has significant differences from the target beat and the result of subtraction using such a template is inevitably distorted. In fact it is likely that this distortion may be exaggerated by the use of a longer time series for generating an averaged template. Generating an averaged template is also time consuming and makes it difficult to base rapid therapeutically relevant decisions, for example, during an EP ablation procedure.

On the other hand, the immediately preceding QRS (temporally the closest possible) to the target beat should possess the best match of conditions that significantly affect the QT morphology and most closely resemble the target QRS in terms of the surface ECG signature of ventricular de and repolarization.⁸ It is possible that respiratory variations which have longer periodicities (each cycle spanning about 4–6 beats of a normal sinus rhythm) can affect beat-to-beat repolarization (and depolarization?) and in that case the best QRS match may be a few beats before the target beat.

VALIDATION OF PRECEDING QRS AS SUBTRACTION TEMPLATE

We tested the above hypothesis on a group of patients undergoing ablation for various supraventricular arrhythmias including atrial fibrillation.⁸ Programmed stimulation was introduced through a roving map catheter bipole with one extrastimulus at a starting coupling interval of 500 ms after a series of drive beats at a cycle length just fast enough to overdrive sinus rhythm. The extrastimulus was decremented down to the atrial effective refractory period so that with shortening coupling intervals, the short coupled extrasystolic P wave was concealed by various phases of the preceding beat's T wave. The QRS of the preceding beat was selected as a template and within a manually selected window extending from the PR interval to

Figure 1. A schematic representation of subtraction of the preceding QRS (A) from a target QRS (B) which conceals a short coupled extrasystole within the T wave (shown in dotted outline above "B"). The digitized nature of the trace (where every sampled point has a given numerical voltage value encoded in bits) allows the operation $B - A$ so as to eliminate the QRS concealing the ectopic as long as the match between the two beats is good. The quality of the match is also indicated by the complete elimination of the QRS in the end result, $B - A$.

Figure 2. An example of the extrasystole simulation protocol used in order to evaluate the fidelity of subtraction using the preceding QRS as a template. On the left is shown the tracing before processing. Following a drive cycle, an extrastimulus is introduced (from the same site as the drive) at a coupling interval of 400 ms which conducts with RBBB type of aberration. On the right is shown the result of subtraction of the QRS (template taken from the beat preceding the last drive beat) from the last drive beat. The QRS is completely eliminated across the full 12-leads as highlighted by the two vertical lines indicating the subtracted segment. Note that the morphology of the underlying paced P-wave in this zone is exactly the same as during the drive beats.

the end of the apparent T wave (concealing the extrastimulus), the QRS was automatically detected based on amplitude and slope. The template QRS was aligned with the target QRS using a cross correlation function between the two QRSs and subtraction performed. The QRS was used as an index of matching because of the close dependence of ventricular repolarization upon depolarization. Since the drive cycle and the extrastimulus were introduced from the same atrial site, the fidelity of the derived subtracted beat using the preceding QRS as a template was tested by comparison with the morphology of the drive beat (Fig. 2).

When QRS subtraction was performed on consecutive beats in a tracing in normal sinus rhythm (without underlying ectopics), the excellent quality of matching was indicated by the straight (isoelectric) line derived in place of the subtracted QRS. This finding also underlined the close correspondence of the match between ventricular depolarization (the QRS) and repolarization (ST-T). As a consequence, the degree of match between repolarization segments could be inferred from the match between depolarization, that is, the resid-

ual QRS after subtraction or the correlation coefficient between the two QRSs before subtraction. When short coupled extrasystoles were simulated by programmed simulation, they were "uncovered" by subtracting the previous QRS from the complex (Fig. 2). Comparison with the drive beat revealed that a very good quality match (both amplitude and morphology as judged visually) was observed in 82% and a good quality match in 92% of beats. Among those beats that did not match, coupling intervals close to the refractory period at the site elicited lower amplitude and prolonged P waves but with similar morphology. This was observed in 2% of all analyzed beats.⁹

P WAVE PARAMETERS

The maximum P wave duration,¹⁰ the P wave vector magnitude calculated from the orthogonal Frank leads, signal-averaged P wave durations, P wave dispersions, root mean squared amplitude of the last part of a high-frequency signal-averaged P wave¹¹ have been compared in controls and patients with paroxysmal atrial fibrillation. However,

because of their limited discriminative aptitude, there is considerable interest in morphology-based parameters. Patients with paroxysmal atrial fibrillation frequently have distinctive P waves—notched or biphasic in standard leads.¹⁰ Paced P waves have also been used as markers of intraatrial block in the treatment of atrial flutter,¹² or to localize the site of retrograde atrial activation during orthodromic atrioventricular reentry.¹³ The understanding of the mechanism of initiation of paroxysmal atrial fibrillation and the recognition of the pivotal role of PV ectopy has renewed interest in the use of the P wave to localize their origin. Though some studies have concluded that the surface ECG P wave is not useful for determining the site of origin of impulse formation,¹⁴ it is clearly possible to discern polar sites of impulse origin (e.g., "coronary sinus rhythm") and it follows that other less polar, intermediate sites should be distinguishable though perhaps with limited resolution.

SINUS RHYTHM P WAVE

The P wave in sinus rhythm can be analyzed as a starting point. Mapping studies in sinus rhythm have shown that right atrial activation begins from near or at the junction of the superior vena cava with the right atrium and is then conducted downward so that right atrial activation terminates at or near the cavotricuspid isthmus. Left atrial activation begins near the insertion of Bachman's bundle situated in the high anterior and septal part of the left atrium though other sites—in the midseptal and fossa ovalis region may also serve as secondary inputs.¹⁵ Activation then proceeds around the mitral valve annulus to terminate close to the left inferior pulmonary vein at the level of the mitral annulus. During sinus rhythm, therefore, the standard limb lead P wave is composed of an initial inferiorly directed right atrial component (positive in leads II, III) followed by a significant overlap by a similarly inferiorly directed left atrial component as this chamber is activated by the superior interatrial input near or at the site of Bachmann's bundle. The right-sided origin of sinus node activation and the left-sided position of terminal left atrial activation result in a right-to-left net vector in the frontal plane, which translates into a greater amplitude in lead II over lead III. Among the chest leads, V1 in sinus rhythm typically consists of a small initial positivity reflecting the postero-anterior activation of the right atrial appendage followed by a negativ-

ity resulting from left atrial activation originating from the upper interatrial septum and moving posteriorly to complete activation of that chamber.

P WAVE MORPHOLOGY ANALYSIS IN THE LITERATURE

The early animal and human studies examined P-wave polarity and morphology on the 12-lead ECG as well as P wave vectorcardiograms during pacing from a variety of right and left atrial sites. However, because of the difficulty of accessing the left atrium, these studies focused essentially on distinguishing right from left atrial sites. Several distinct criteria were proposed, including the "dome and dart" P wave proposed by Mirowski to indicate a left atrial origin.¹⁶ MacLean et al.¹⁷ found that a negative P wave in leads I and avL was elicited only by pacing from the left atrium and that too when pacing from near the left pulmonary veins. They also observed that a bifid P wave in V1 indicated a left atrial origin from near the inferior pulmonary veins and the coronary sinus.

More recent endocardial pacing studies looked at various (percutaneous catheter) accessible sites with the intention of deriving an algorithm to distinguish left from right atrial foci. Tang et al.¹⁸ used tachycardia P waves during either spontaneous or pharmacologically induced atrioventricular block and evaluated their polarity and certain morphological features against the site of successful ablation radiofrequency delivery. Although a variety of sites were ablated a relatively limited number of P waves were available for evaluation of the pulmonary veins. The authors concluded that negativity in avL and positivity in V1 were indicative of a left atrial origin and that an inferiorly directed vector in the frontal leads was characteristic of a superior origin. These inferences are reasonable, though only a limited number of sites, based on the tachycardias studied, were actually evaluated.

The study by Man et al.¹⁹ used a multipolar catheter placed in the lateral right atrium and within the coronary sinus to perform unipolar pacing from each of the electrodes while recording a 12-lead ECG. The authors concluded that the spatial resolution of unipolar atrial pacemapping was about 17 mm. However, the unipolar pacing artifact obscures approximately the first half of the P wave; moreover, their evaluation was limited to the lateral right atrium and the coronary sinus as well as by the interelectrode spacings on the

catheters used. It is therefore possible that they underestimated the resolution of "pacemapping."

PACEMAPPING AND QRST SUBTRACTION

With this background, Yamane et al.²⁰ performed a pacemapping study in order to look at the ECG morphology of P waves elicited by pacing from the pulmonary veins. They performed bipolar pacing from within all the four pulmonary veins including two different sites within the two superior veins. In 30 patients with various supraventricular tachycardias including atrial fibrillation, they found distinct patterns of paced P waves that indicated the pacing site with considerable accuracy. Right from left pulmonary veins could be distinguished with greater accuracy than superior from inferior veins. A positive P wave in lead avL, a positive lead I P wave with an amplitude ≥ 50 microvolts indicated a right pulmonary vein origin with high specificities with, however, a positive P in lead I having a higher sensitivity than in avL (72% vs 38%). Similarly, a notched P wave in lead II indicated a

left pulmonary vein origin with high specificity but lower sensitivity. The authors also proposed two new criteria: the ratio of peak P wave amplitudes in lead III to those in lead II and the duration of positivity in V1. A lead III/II peak voltage ratio of ≥ 0.8 identified left pulmonary vein origin with high sensitivity, while a positivity in V1 of ≥ 80 ms also indicated left pulmonary vein origin but with a slightly lower sensitivity (Figs 3 and 4). The lead III/II amplitude ratio represents a simple refinement of the frontal plane axis of the P wave, which is useful in distinguishing a P wave originating from the left extremity of the atria from a P wave that has a central or slightly rightward origin. Similarly, the longer duration of positivity in V1 in case of a left pulmonary vein origin reflects their more posterior position. These results were used to develop an algorithm, which when evaluated in 20 patients in a blinded fashion succeeded in identifying correctly 93% of left versus right pulmonary vein pacing but only 79% of all pulmonary veins.

However, this study made no attempt to compare the P waves elicited by pacing from within the left atrium (or right atrium) to those during pulmonary

Figure 3. A clinical example from a patient undergoing catheter ablation for paroxysmal atrial fibrillation. Again on the left is the unprocessed ECG which shows normal sinus rhythm with induction of atrial fibrillation beginning with a short coupled extrasystole occurring coincidentally with pacing from the distal coronary sinus. On the right is the same tracing after subtraction of the preceding QRS. The pacing spike is retained but the QRS has been eliminated thus exposing the initiating beat which has a morphology indicating a left superior pulmonary vein origin.

Figure 4. Spontaneous induction of atrial fibrillation analyzed by subtraction. On the left is the baseline tracing and on the right is the same tracing after QRS elimination by subtraction. Note that the P wave has a morphology indicating an origin from the right superior pulmonary vein (lead III/II amplitude ratio < 0.8 , V1 positivity < 80 ms) which was verified by intracardiac mapping and ablation.

vein pacing. Furthermore, the P waves were obtained during a pacing rate only slightly faster than sinus rhythm in order to avoid overlap by preceding T waves and therefore, the effects of increasing rates were not studied. As was pointed out earlier, short coupling intervals are practically the rule in the clinical situation and so-called aberrant intra- or interatrial conduction may play a significant role in determining ectopic P wave morphology. The data also indicated that intraindividual differences in P waves from different pulmonary veins were more obvious than interindividual differences, suggesting that variations in individual activation and anatomy are probably responsible for reducing the accuracy of P wave predictions made in a group of different individuals.

The same group then studied patients with spontaneous ectopy, using the previously described software to eliminate the overlying QRS to analyze the ectopic P wave and compare it with a catalogue of pacemaps from the pulmonary veins and various atrial locations; the latter in order to correct for confounding variations in anatomy and/or associated activation. Choi et al.⁹ studied 44 patients with paroxysmal atrial fibrillation and 56

different ectopic morphologies. Based on endocardial activation mapping followed by successful ablation, 37 were determined to originate from the pulmonary veins while 19 ectopics observed after successful pulmonary vein disconnection were atrial in origin. QRS subtraction was simply and rapidly performed using the preceding beat as a template. Applying Yamane et al.'s algorithm allowed correct identification of pulmonary vein origin in 30 of 37 ectopics (81%). When comparison with the pacemap catalogue was allowed, 34 of 37 (92%) ectopics were correctly attributed to the appropriate pulmonary vein. Four ectopics observed following successful pulmonary vein isolation matched a PV pacemap and were found to originate from the atrial side of that isolated vein (ostial ectopy). The remaining 15 did not match any PV pacemap and were found to originate variously from the left atrium, right atrial septum, the superior vena cava or the inferior vena cava. Comparison with the pacemap catalogue indicated that the pulmonary veins were unlikely to be sites of origin and helped further localize the site of origin so that successful ablation was achieved in all but three instances. Successful localization of non-PV foci was

defined as being within a radius of 1 cm (of the best pacemap) without transgressing anatomical boundaries.

A practical definition of nonpulmonary venous foci was used in the above study: those that manifested after successful PV isolation. This definition circumvented the still-unresolved issue of where the left atrium ends and the pulmonary veins begin. The pacemap catalogue used in the study was obtained by bipolar pacing from the ostium of the coronary sinus, the superior vena cava, the inferior vena cava, the left atrial appendage, the left atrial roof between the two superior pulmonary veins, and the mid and distal coronary sinus. This catalogue included more left than right atrial sites but depending upon clinical suspicion, the catalogue could be readily modified or extended, for example, in order to refine the resolution within a region of interest. The authors attributed one instance of a nonmatch during pacing from the successful ablation site to aberrant intraatrial conduction at short coupling intervals.

It is clear though that subtraction as described above requires normal sinus beats (or junctional beats without retrograde conduction), which match the QRS concealing the ectopic; therefore incessant arrhythmia or frequent and varying aberrant conduction render subtraction difficult. While a noisy tracing makes the P wave morphology that much more difficult to discern, accurate subtraction can be achieved even in the immediate aftermath of an electrical cardioversion in spite of the resulting drift in baseline.

ECTOPIC P-WAVE ANALYSIS WITHOUT ADJUNCTIVE SUBTRACTION

Other groups also analyzed the P wave morphology of short coupled ectopics. Kolb et al.²¹ used a 12-lead 24-hour Holter monitoring system to document and analyze the spontaneous onset of atrial fibrillation episodes. Of a total of 297 episodes in 33 patients, 93% were initiated by atrial premature complexes. The authors classified 77.5% of ectopics to be of left atrial origin, 2% of right atrial origin, and the remaining 13.5% were indeterminate. However, since this analysis was performed on the raw ectopic-QRS combination, that is, without any form of QRS suppression, their conclusions can be considered approximate at best.

O'Donnell et al.²² examined ectopic P wave morphology during spontaneous ectopy and compared it with pulmonary vein pacing. Though the authors did not use QRS suppression in any form, they found a good match at comparable coupling intervals. However, in about 25% of the patients, at more rapid pacing rates, changes in amplitude, duration, and morphology were observed compared with the P wave at baseline. These changes reduced the predictive accuracy using Yamane et al.'s as well as two other algorithms. Moreover, all the algorithms assessed in this study were found to be less accurate in patients with enlarged atria, abnormal P waves in sinus rhythm, or persistent atrial fibrillation. Again, however, these inferences were limited by the confounding influence of overlying T waves that were not eliminated by subtraction.

Kistler et al.²³ recently described a group of patients with pulmonary vein tachycardias in whom they correlated P wave morphology with the site of successful ablation. Using Yamane et al.'s algorithm, they were able to identify 78% of the pulmonary veins correctly, and the P waves of incorrectly identified veins suggested that comparison with a pacemap catalogue might have been of help. When the authors evaluated a paced endocardial activation map in 10 of 27 patients as an aid to identify the PV source, eight veins were correctly identified but in the two remaining patients catheter movement and inability to capture prevented comparison.

ECTOPY ORIGINATING FROM OTHER SITES

Apart from left atrial and pulmonary vein ectopy, the superior vena cava, and rarely the inferior vena cava have also been described as giving rise to ectopy in patients with atrial fibrillation.²⁴ Ectopy and atrial fibrillation initiation from a persistent left superior vena cava draining into the coronary sinus have also been described. In each case, the resulting P wave morphology reflects the site of atrial insertion. Thus ectopy or tachycardias originating from the superior vena cava resemble sinus rhythm (lead II amplitude greater than lead III) although with greater amplitude in the inferior leads than during sinus rhythm. Lead V1 also reflects sinus rhythm-like activation with a biphasic or dominant negative deflection.²⁵ Interestingly, ectopy from the nearby right superior vena cava can be distinguished by its characteristic positive deflection in lead V1.²⁶ In

the case of the persistent left superior vena cava, the P wave reflected a site of atrial insertion between the two left-sided pulmonary veins—with a notched positive deflection in lead V1 and low amplitudes in inferior leads. Similarly, the P wave morphology of ectopy from the inferior vena cava reflects its connection near the cavotricuspid isthmus with relatively deep negativity, which is more negative in lead III than in lead II (in accordance with its rightward origin) and is not positive in lead V1.^{9,24}

CLINICAL IMPLICATIONS

The above studies provide strong support for the use of ectopic P wave morphology in order to localize or help localize the site of ectopy origin. In case of short coupled ectopy, most common in case of patients with atrial fibrillation, this is only feasible using subtraction to eliminate the overlying QRST. Patients with frequent ectopics or tachycardias originating from the pulmonary veins require rapid assessment of the target vein followed by isolation of either that vein alone or supplemented thereafter in stable sinus rhythm by isolation of the remaining pulmonary veins. The above technique can be of help even before intracardiac catheters are in place and once they are in place, the clear recognition of the P wave onset made possible by QRST subtraction allows the operator to judge whether the local activation at a particular intracardiac site actually precedes the surface ECG P wave onset (Fig. 5). However, in patients with low-amplitude P waves—frequently as a result of degenerative “silent” areas—it is difficult to discern specific patterns and one has to rely, instead, on intracardiac recordings.

Using the preceding QRST as a template for subtraction, published algorithms provide 81% accuracy in identifying the pulmonary vein of origin based on a standard but digitized 12-lead ECG format. Anatomic interindividual variations can be simply overcome in large part by the use of a pacemap catalogue resulting in an increase in predictive accuracy (in clinical situations) reaching 92%. These results argue against the need for complex and less widely applicable technology such as body surface mapping systems for improving localizing resolution. The correction of other confounding factors, including differences in electrode positioning and standardizing their relationship to the

Figure 5. An example of QRST elimination aiding intracardiac mapping. On the left is shown the baseline tracing during a short coupled extrasystole. Intracardiac mapping shows that activation in the circumferential mapping catheter (Lasso placed in the left superior pulmonary vein) is the earliest with later activation in the ablation (in the right superior pulmonary vein) and coronary sinus catheters. However, only after QRST elimination does it become evident that activation recorded in the ablation catheter follows the ectopic P wave onset and that Las activation is almost 80 ms before the ectopic P wave onset which is otherwise completely concealed by the overlying T wave.

atria, may allow further improvements in reliability and resolution.

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