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Fusion During Entrainment Of Orthodromic Reciprocating Tachycardia Is Enhanced For Basal Pacing Sites But Diminished When Pacing Near Purkinje System Endpoints

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Abstract

Background—In the EP lab, orthodromic atrioventricular reciprocating tachycardia (ORT) can be distinguished from atrial tachycardia and atrioventricular node (AVN) reentry tachycardia by identifying orthodromic and antidromic wavefront fusion during ventricular overdrive pacing (VOP). Previous work has shown that basal VOP near the accessory pathway (AP) increases the likelihood of observing fusion; however, in a third of cases, fusion is not appreciable regardless of VOP location.

Objective—We sought to explore the hypothesis that pacing near His-Purkinje system (PS) endpoints reduces fusion quality, which may explain non-responsive ORT patients.

Methods—In a novel computer model of ORT, simulations were performed with a variety of AP locations and pacing sites; results were analyzed to assess factors influencing fusion quality in pseudo-ECG signals.

Results—Entrainment by basal VOP near the AP was more likely to produce fusion visible on simulated ECGs compared to entrainment by apical VOP, but this advantage was dramatically diminished when the pacing site was also near PS endpoints. Prediction of fusion quality based on AP proximity alone was dramatically improved when corrected to penalize for PS proximity.

Conclusion—These results suggest that basal VOP near the AP and far from the PS is optimal; this could be tested in patients. A denser basal ramification of PS fibers is known to exist in a minority of human hearts; our findings indicate that this unusual PS configuration is a plausible explanation for ORT cases where fusion is never observed in spite of entrainment by basal VOP near the AP.

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Conflicts of interest:

PMB and GDV have nothing to declare.
EJV is President of Cardiosolv LLC

Keywords

Orthodromic atrioventricular reciprocating tachycardia; Purkinje system; ventricular overdrive pacing

Introduction

The first step during a transvenous catheter ablation procedure for supraventricular tachycardia (SVT) is establishing whether the SVT mechanism is orthodromic atrioventricular reciprocating tachycardia (ORT), atrioventricular nodal reentrant tachycardia (AVNRT), or atrial tachycardia (AT). In the most comprehensive study of SVT diagnosis,[1] Knight et al. concluded that multiple pacing maneuvers are required for an accurate diagnosis. Indeed, only 65% of SVTs could be accurately diagnosed with the best combination of SVT features and pacing maneuvers. Ventricular Overdrive Pacing (VOP) has since emerged as an attractive single diagnostic pacing maneuver, yielding a firm SVT mechanism in $\approx 90\%$ of cases.[2] Nonetheless, diagnostic challenges remain, particularly when atrial activation is central and the ventricular-atrial interval is long.

By examining the sequence of activations in the atria, His bundle, and ventricles, following VOP, the disorder can be divined. ORT can be identified if QRS complex fusion (explained below) is observed on the ECG during entrainment; unfortunately, fusion is fairly insensitive for ORT,[2] so its absence cannot be used to confirm AVNRT. During entrainment of ORT by VOP, the orthodromic wavefront emanates from the pacing site, excites the atria via the accessory pathway (AP), then re-enters the ventricles through the atrioventricular node (AVN) and His-Purkinje System (PS); the antidromic wavefront travels oppositely, through the ventricles and retrogradely into the PS. Thus, each antidromic wavefront collides with the orthodromic wavefront from the previous stimulus. When this collision occurs in ventricular tissue, a “fused” QRS complex, with morphology that differs visibly from that of a fully-paced beat, reliably identifies ORT; QRS complex fusion is impossible in AVNRT since the entire circuit resides within the AVN. Sometimes, the antidromic wavefront collides with the orthodromic wavefront so close to the pacing site that the entrained QRS complex cannot be distinguished from that of the conducted SVT, resulting in “entrainment with concealed fusion”, also reliably identifying ORT. When this collision occurs in the conduction system, the QRS complex will be that of a fully paced beat (“concealed entrainment”), and other diagnostic features are required to distinguish AVNRT from ORT. Fig. 1 highlights the difference between manifest fusion and concealed entrainment.

Basal VOP sites near the AP are more likely to result in fusion than apical ones.[2] Earlier AP activation within the entrained VOP circuit results in an orthodromic wavefront arriving sooner at the ventricular myocardium relative to the antidromic wavefront from the next pacing stimulus, increasing the likelihood of wavefront collision in ventricular myocardium, resulting in fusion. Other potential factors influencing fusion quality, including proximity to terminals of the PS, have not been investigated in detail and may help explain ORT cases where fusion is never observed.

We constructed a 3D electrophysiological computer model of ORT to better understand how VOP location influences the presence of fusion during entrainment. In particular, we considered apical versus basal sites, and the effects of pacing near the PS. Pacing there should produce earlier retrograde activation by the antidromic wavefront, moving the collision point into the conduction system, and diminishing the likelihood of visible fusion.

Methods

2.1 Conduction system and ventricles

The ventricular mesh (Fig. 2A) was the same as used previously [3, 4, 5] with updated membrane kinetics [6]. A cable model of the rabbit AVN (Fig. 2B) was implemented as specified by Inada et al.[7]. The PS was modeled as a branching 1D network as in previous studies [8, 4, 3, 5] but with rabbit PS membrane kinetics.[9]

Electrical propagation was governed by the monodomain formulation; the longitudinal-to-transverse anisotropic conductivity ratio was 9.16, with histological fiber orientation. As in previous studies, PS-myocardial coupling was tuned to match known delay characteristics. [3, 4, 5] Details regarding the numerical approach used in the simulation software (CARP) have been presented elsewhere.[10]

2.2 Accessory pathways

ORT requires a conductive pathway between the atria and ventricles. For each AP, a node representing the entry point was specified in the ventricles. Upon excitation of this node, the proximal AVN was stimulated after a fixed delay representing AP and atrial conduction times. The distal AVN node was connected to the proximal His bundle using a similar scheme without delays, allowing for both anterograde and retrograde AVN excitation. Fig. 2C shows a schematic for these interconnections.

2.3 Pseudo-ECG signals

Pseudo-ECG signals were generated from electric potentials recovered at discrete points near the LV, RV, and apex. The associated equation is:

$$\phi(P) = \frac{1}{4\pi\sigma_b} \int_V \frac{i_m}{r} dV \quad (1)$$

where ϕ is potential, σ_b is bath conductivity, P is the measurement point, i_m is the membrane current, and r is the distance from the measurement point for each node in the volume V . Standard limb lead approximations were obtained by differencing these signals.

In Fig. 2A, potential recovery sites and pseudo-ECG leads are superimposed on the image of the ventricles. Importantly, the signals recovered using this technique differ dramatically from standard ECG leads; in addition to closer proximity and the absence of torso filtering effects, the heart's orientation in the body means that recordings from the left and right arms do not correspond to signals recovered near the basal LV and RV. Nonetheless, pseudo-ECGs capture distinct ventricular depolarization (QRS complex) and repolarization (T wave) phases, so analysis of VOP site quality based on resulting QRS complex fusion is reasonable.

2.4 Quality of fusion

ORT identification by QRS complex fusion requires that the entrained ECG is distinct from its purely paced counterpart. For a high-quality VOP site in the ORT model, pacing from the same location with and without the AP should result in distinct ventricular activation. Entrained and purely paced responses were quantitatively compared using the cross-correlation coefficient[11]:

$$\gamma(x, y) = \frac{\sum_{n=0}^{N-1} [x(n) - \bar{x}] [y(n) - \bar{y}]}{\sqrt{\sum_{n=0}^{N-1} [x(n) - \bar{x}]^2 \sum_{n=0}^{N-1} [y(n) - \bar{y}]^2}} \quad (2)$$

γ varies between -1 and $+1$ (identical: $\gamma = 1$, statistically uncorrelated: $\gamma = 0$). γ was computed between entrained and paced QRS complexes for each lead and the three values were averaged. Finally, this value was subtracted from unity to produce the fusion quality index (Γ):

$$\Gamma = 1 - \frac{1}{3} \sum_{i=I}^{III} \gamma(ECG_{en,i}, ECG_{pp,i}) \quad (3)$$

where $ECG_{en,i}$ and $ECG_{pp,i}$ are entrained and purely paced segments from lead i , aligned by pacing time; Γ is higher for pairs of signals that are more distinct.

2.5 Simulation protocol

Four AP entry points were defined in basal ventricles: RV freewall and septal, LV freewall and septal. For each AP, four endocardial VOP sites (base to apex) were chosen (see Fig. 2C). Each AP was simulated with four conduction delays: 100, 150, 200, and 250 ms. For each configuration, ORT was simulated for 2 s to determine Tachycardia Cycle Length (TCL). Then, entrainment was performed from each VOP site by applying seven 3 ms-long pulses with Pacing Cycle Length (PCL) of $TCL - 30$ ms, after which native ORT returned. Finally, each VOP scenario was repeated without the AP to obtain the purely-paced response.

The same protocol was applied using APs chosen for their proximity to PS endpoints; these APs were in the RV freewall, LV posteroseptal, and LV anteroseptal regions. We carefully chose VOP sites with equivalent pace-to-AP and pace-to-PS delays.

Finally, for one configuration (RV septal, 100 ms delay, basal VOP), we performed entrainment with VOP rates progressively approaching TCL to ensure agreement with clinical results.

Each simulated second took approximately 2.5 h on a cluster of 12 CPUs at 2.67 GHz, resulting in cumulative wall time of approximately 10 days.

Results

3.1 Analysis of ORT simulations

Simulated ORT (Fig. 3) qualitatively resembled clinically-observed ORT. Native ORT (A), entrainment (B), and pure pacing (C) phases were distinct and could be identified in both pseudo-ECG signals and voltage maps. A distinct QRS complex is observed during entrainment since orthodromic excitation of the PS and ventricles occurs early enough that collision with the antidromic wavefront occurs in myocardium. In general, a given VOP site always stabilized to the same entrainment pattern, regardless of initial stimulus timing within the ORT cycle. With PCL of $TCL - 30$ ms, entrainment always stabilized within 1 or 2 pacing cycles; with longer PCL s approaching TCL , stabilization required more cycles.

For some configurations, pseudo-ECGs from entrainment were nearly identical to those generated without an AP. Fig. 4 shows several examples. Basal pacing sites near the AP (A

and B) resulted in fused ECGs that were distinct from purely paced counterparts; in contrast, apical pacing sites (C and D) produced entrained QRS nearly identical to purely paced segments, suggesting collision of orthodromic and antidromic wavefronts in the conduction system.

In basal sites, the distinction between entrainment and pure pacing was clearer for the RV septal case (B) than the RV freewall. Leads II and III were distinct but lead I was quite similar. This is notable because there were few PS junctions in the basal septum, suggesting that their absence near VOP sites is beneficial. Movie 1 in the Online Supplement highlights differences between basal and apical VOP sites in simulated ORT.

For septal pathways (B and D), ECGs during ORT and entrainment were comparable. This effect is particularly striking for RV septal pacing (B), where leads II and III hardly change during VOP, despite significant activation rate acceleration. This demonstrates entrainment with concealed fusion where relatively little ventricular tissue is depolarized by the paced antidromic wavefront.

In all simulations, qualitative comparisons of entrained and purely-paced segments agreed with quantitative analysis using Eqn. 3. Notably, for basal sites in Fig. 4, the configuration with three distinct leads (B) had appreciably higher $\Gamma = 0.715$ compared to one where lead I complexes were not distinctly fused (A; $\Gamma = 0.477$). This demonstrates our metric's fusion quality stratification capability.

3.1.2 Special entrainment scenarios reveal the importance of pacing far from PS terminals—ORT simulations confirmed the general clinical observation that pacing farther from the AP increases similarity between entrained and purely paced QRS complexes,[2] but this was not universal. In particular, VOP sites near PS terminals tended to produce low quality indices, regardless of AP proximity. To investigate further, simulated entrainment was performed in VOP site pairs that were roughly equidistant from the AP but varied considerably in proximity to PS terminals.

Fig. 5 shows a posteroseptal AP with VOP sites I and II, which both had pace-to-AP activation delay of $\Delta t_{AP} \approx 24$ ms; however, the pace-to-PS delay (using earliest retrograde PS activation) was considerably shorter for site I ($\Delta t_{PS} = 7.129$ ms) compared to site II ($\Delta t_{PS} = 22.78$ ms), since the latter was in a region devoid of PS endpoints. In both quantitative and qualitative (red-circled QRS complexes) terms, fusion quality was much higher for site II than site I.

Detailed investigation of activation sequences for the two VOP scenarios in Fig. 6 revealed the underlying mechanism of fusion quality differences. For site I, retrograde PS activation occurred very quickly following stimulation (A2); shortly afterward, the left bundle branch was activated antidromically just as the His bundle was being excited by the orthodromic wavefront from the previous pace (A3). Consequently, wavefronts collision was in the left bundle branch and ventricular differences between the entrained and purely paced scenarios were limited to the RV, where PS excitation was slightly accelerated.

For site II, the orthodromic AP excitation occurred concomitantly with site I (compare B2 and A3) but the earliest antidromic PS activation occurred much later. In fact, the first retrograde excitation of the PS occurred in the anterior fascicle of the left PS (located “behind” the cutaway portion of the ventricles in Fig. 6), far from the reentrant circuit being entrained. Consequently, orthodromic PS activations in the ORT circuit (excitations emanating from posterior LV terminals) fused with antidromic activity (B4), giving rise to a completely different depolarization pattern from the purely-paced scenario, hence the

increased fusion quality compared to site I. Movie 2 in the Online Supplement uses simultaneous maps of V_m in the PS and ventricles to reinforce the observations made in Figs. 5 and 6.

Results show that pacing near PS terminals can be extremely detrimental to fusion quality during entrainment. These “special” scenarios did not include any VOP sites very close to the AP due to the lack of abnormal basal PS terminals in our model; however, the implication is that fusion quality may be dramatically lower in patients with denser ramifications of PS endpoints near the most desirable VOP sites.

3.2 Improving prediction of pacing site quality

The existing metric for predicting VOP quality is AP proximity ($\Delta t_{AP} = t_{AP} - t_{pace}$), which is one component of the orthodromic pathway; a smaller value predicts higher fusion quality. To develop a more robust measurement reflecting the reduced effectiveness of VOP near the PS, we performed a thorough decomposition of the orthodromic and antidromic components, as presented in Fig. 7.

First, schematic representations of typical entrainment configurations were constructed (Fig. 7A); as expected, lengthening phase O1 (Δt_{AP}) pushes the sequence of orthodromic events forward, increasing the likelihood of fusion in the conduction system by forcing the end of phase O4 (orthodromic PS terminal activations) towards phase A1 (Δt_{PS} , the time spent in the ventricles by the antidromic wavefront). Clearly, shortening phase A1 (i.e. pacing closer to the PS while holding other VOP factors equal) should have a similarly detrimental effect on fusion quality. Thus, the pacing site quality metric must be derived from some combination of O1 and A1 durations!

To confirm this, orthodromic and antidromic delay times for several stable entrainment configurations were plotted. Horizontal bars in Fig. 7B were constructed as follows: starting from pace n , the orthodromic wavefront consisted of Δt_{AP} (O1: dark blue) followed by time spent in the AP (O2: orange), the AVN (O3: light red), and the PS (O4: dark green). The final orthodromic component was the delay between the first anterograde PS activation and AP excitation *in the absence of entrainment* (O5: olive green). Note that the sum of the latter four components (O2+O3+O4+O5) is equal to the TCL. The sequence of events in the antidromic wavefront was constructed similarly: the interval between pace n and pace $n+1$ (PCL, pink) was followed by the pace-to-PS retrograde activation delay (A1: light blue, $\Delta t_{PS} = t_{PS} - t_{pace}$), and the delay before His bundle excitation (A2: navy blue).

Orthodromic and antidromic wavefronts must collide in the myocardium for fusion to be manifest. Schematically, this is *guaranteed* if phase O4 (orthodromic PS activation) ends before phase A1 ends, as in VOP configuration B1. Examining configuration B2, increasing Δt_{AP} by moving the pacing site away from the AP clearly diminished fusion quality by shifting the end of phase O4 far to the right. Unsurprisingly, wavefront diagrams for B3 and B4, which correspond, respectively, to sites II and I in Figs. 5 and 6, show that decreasing Δt_{PS} had a comparable effect, moving the overlap point (*) to the left and decreasing fusion quality.

Thus, since VOP sites are penalized by proximity to PS junctions, simply using Δt_{AP} as a fusion quality predictor is inadequate. We found empirically that the corrected metric $(\Delta t_{AP})^2 - (\Delta t_{PS})^2$, which heavily weights the largest distances from the AP or PS terminals, was highly effective. Scatter plots comparing predicted and actual quality of fusion before (A) and after (B) correction for PS proximity are shown in Fig. 8. Observations associated with $\Gamma < 0.05$ were omitted, since they always corresponded to increasingly large Δt_{AP} values. Predictor values were horizontally reflected, then normalized between 0 and 1; with

these transformations, higher predictor values should correspond to improved fusion quality with an increasing linear relationship. Best-fit lines (solid black) were generated using linear regression; for each VOP scenario, we calculated the deviation of the prediction from its ideal value based on this line (Δx). Dotted lines represent $\Delta x \pm 0.2$ thresholds on both plots.

Qualitatively, it is obvious that the uncorrected predictor (A) performs poorly for numerous VOP scenarios, most of which were associated with stimuli near the AP but also near PS terminals. When a penalty factor was included for proximity to conduction system endpoints (B), points clustered near the best-fit line, with few observations exceeding $\Delta x \pm 0.2$. A paired Students' t-test was performed on 60 observations of corrected versus uncorrected predictions, revealing an improvement in mean deviation of $\Delta x_A - \Delta x_B = 0.17$, ($p < 0.0001$).

Finally, since Δt_{PS} may be difficult to measure clinically, the procedure was repeated with $\Delta t_{His} = t_{His} - t_{pace}$ used as a surrogate quantity; the resulting improvement in Δx was comparable to that illustrated in Fig. 8B (not shown).

Discussion

This study uses a computer model of ORT in rabbit geometry to investigate the relationship between pacing site location and the extent of QRS complex fusion during entrainment for diagnostic purposes. Its principle findings are as follows: (1) The model recreates clinical phenomena accurately enough to allow for detailed analysis of the mechanisms that underlie clinically confounding problems; (2) As seen clinically, the model demonstrates that VOP sites near the AP are more likely to result in entrainment with high-quality fusion; (3) VOP sites should also be distant from endpoints of the PS, since early retrograde propagation influences the point at which wavefronts collide, which can diminish the degree of fusion; and, (4) The presence of basal PS branches may explain why fusion does not occur in some ORT patients despite VOP from basal locations.

To the best of the authors' knowledge, this study is the first detailed 3D model of ORT and the first time computer simulation has been used to examine VOP outcomes. One model-based investigation used 1D tissue rings to examine ORT termination; [12] a low-resolution 3D model has been implemented before, but VOP was not performed because the primary objective of the associated study was to identify factors affecting ORT inducibility. [13] In this work, reentrant circuit components are implemented with realistic spatial and detailed biophysical properties, including the ventricles with fiber orientations, a PS, and a detailed AVN model.

Our results replicate the clinical finding that performing VOP at basal sites is conducive to clearly visible QRS fusion. [2] It was much easier to differentiate between entrained and purely-paced QRS complexes when pacing at sites closer to the base. Nonetheless, as shown in Fig. 8A, there were numerous cases where the predictive power of AP proximity was reduced, resulting in inaccurately-estimated fusion quality. These discrepancies could be mitigated by introducing a compensatory factor to penalize sites far from the PS, as illustrated in Fig. 8B. Even with this correction, some VOP configurations resulted in predictor values that under- or over-estimated the actual quality of the site; many points in Fig. 8B remain associated with large Δx values. Simply put, some VOP sites are simply better (or worse) than others. Δt_{AP} and Δt_{PS} provide information about important details of stabilized entrainment, but other factors contribute to the true quality of a VOP configuration. In particular, spatial factors, such as the location of initial antidromic PS excitation with respect to the ORT circuit, will have an important effect.

4.1 Clinical implications

Part of the explanation for the relatively low sensitivity of fusion as a diagnostic marker of ORT, even with basal VOP, is that some patients may have basal PS branches in regions desirable for VOP. Distribution of PS endpoints is known to differ significantly between individuals; in some cases, VA index times suggest unusually dense basal ramifications.[14] This hypothesis could be tested in the clinical setting by determining whether the delay between His excitation and basal activation is abbreviated in patients where entrainment is always concealed compared to the rest of the ORT cohort, where basal VOP results in manifest or concealed fusion.

4.2 Model limitations

Notably, the PS model used here is simplistic compared to the true physiological system; however, as discussed in previous studies, it retains certain key characteristics: namely, rapid propagation within the network, isolation from ventricular cells except at distributed terminals, and two-way propagation at PS-myocardial junctions, with asymmetric safety factor.[4] Moreover, the network's relative geometric simplicity simplifies construction of ORT simulations. Pacing electrodes can be placed with pinpoint accuracy, allowing for the exact proximity and timing of VOP events with respect to stimulus, AP, and PS to be observed with fine spatial and temporal resolution.

Instead of explicitly modeling the AP and atria, it was assumed that each activation at the ventricular entry point resulted in a distal AVN excitation after some fixed interval. This simplification is plausible because: (1) AP excitation contributes negligibly to far-field potentials; and, (2) beat-to-beat AP delay variance is minimal in stabilized ORT.

Although the components of our ORT model are based on rabbit physiology and our pseudo-ECG signals are constructed from potentials recovered near the heart surface, the importance of our findings extends to humans. While the scale of delay times and electrical measurements in a human model would differ, the important determinants of QRS complex fusion quality identified here—namely, proximity to the AP and distance from PS terminals—remain the same.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Glossary

AP	Accessory Pathway
AT	Atrial Tachycardia
AVN	Atrioventricular Node
AVNRT	Atrioventricular Nodal Reentrant Tachycardia
ORT	Orthodromic Atrioventricular Reciprocating Tachycardia
PCL	Pacing Cycle Length
PS	His-Purkinje System

SVT	Supraventricular Tachycardia
TCL	Tachycardia Cycle Length
VOP	Ventricular Overdrive Pacing

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Figure 1.

ECG recordings from clinical ORT cases illustrate the difference between manifest fusion and concealed entrainment. **A:** For a basal VOP site, the QRS complex during entrainment is readily distinguished from its purely-paced counterpart; **B:** pacing apically in a different patient, the QRS complexes are nearly identical.

Figure 2.

The ORT model (C) combines the ventricles (A), PS, and AVN (B) with a bulk AP model. Connections between model components are described in the text. Near-field measurement sites and lead orientations for pseudo-ECGs are shown in A. Regions of AVN ionic heterogeneity are shown in B. Stars in C represent several possible AP entry points; VOP sites for an RVFW AP are shown.

Figure 3.

Cutaway slices (V_m in ventricles and PS) with pseudo-ECG lead I illustrate the QRS complex fusion principle. Snapshots are from QRS onset (*). During ORT (A), PS breakthroughs close the reentrant circuit by activating the AP (star). In entrainment (B), antidromic and orthodromic wavefronts interact in the ventricles. In pure pacing (C), the AP is not included so there is no orthodromic wavefront. Each scenario results in a distinct QRS morphology.

Figure 4.

Basal and apical VOP sites are compared for two simulated RV APs (delay 200 ms). Pseudo-ECG recordings are shown before, during, and after entrainment and for pure pacing. Higher Γ indicates increased quality of QRS complex fusion, as described in the text. **A, B:** Basal VOP (near AP) resulted in distinct QRS complexes in all phases. **C, D:** Purely paced and entrained phases were difficult to distinguish for apical pacing sites (far from AP).

Figure 5.

Comparison of VOP near and far from the PS. Sites I and II are equidistant from the AP (pace-to-AP delays of Δt_{AP} = 24.01 and 23.93 ms) but site I is much closer to the PS. Whereas entrained and purely paced QRS complexes for site I are nearly identical due to wavefront fusion in the His bundle, depolarization patterns are distinct for site II; these differences (circled in red) are quantitatively confirmed by Γ values.

A: Entrainment by VOP at Site I

B: Entrainment by VOP at Site II

Figure 6.

For the AP configuration from Fig. 5, stabilized activation sequences are shown for entrainment by VOP from sites I (A) and II (B). In both cases, AP activation by orthodromic activity from the current pace (pace n) is imminent 20 ms after stimulation (A3 and B2). In contrast, retrograde PS activation by antidromic activity from pace n occurs much sooner for site I than for site II. Inset views of the PS in A3 show how this leads to fusion near the His bundle, accounting for low quality of fusion during entrainment from Site I. As shown in B3 and B4, Site II VOP results in wavefront collision in the ventricles. 3D maps show V_m on the same scale as in Fig. 3.

A: Schematic representations of typical stabilized entrainment configurations

B: Orthodromic and antidromic delay times for different VOP scenarios

Figure 7.

A: Stabilized entrainment configurations for different fusion locations are shown in terms of orthodromic and antidromic components. Arrow lengths are not to scale. For a particular AP, the duration of phases O1 and A1 depend on VOP site, i.e., stimulus proximity to the AP and PS endpoints. Note that not all phases are present in all scenarios. **B:** Orthodromic and antidromic timing diagrams for four pacing sites. For each site, (B1–B4), the orthodromic wavefront course is illustrated on top, while the antidromic is immediately below. The VOP stimulus for pace n+1 is delivered at the start of phase A1 (pale blue). As explained in the text, fusion quality can be approximated by examining the end of A1 with respect to orthodromic events, which is marked by an asterisk. For an AP on the RV freewall, the apical site (B2) is inferior to the basal site (B1), mostly due to the dramatically lengthened O1 delay; in contrast, for the posteroseptal AP from Figs. 5 and 6, the sites have nearly identical O1 delays but the overlap point is still shifted to the left since one site has a much shorter A1 delay than the other (B4 compared to B3).

Figure 8.

Actual versus predicted quality of fusion for VOP scenarios without (A) and with (B) correction for proximity to the PS. Best-fit lines (solid) for both sets are flanked by dashed lines that indicate $\Delta x \pm 0.2$; Δx is the deviation of a predicted quality value from the corresponding point on the best-fit line for the actual quality of that pacing site. Mean deviation was 0.17 lower ($p < 0.0001$) when VOP sites were penalized for proximity to the PS (B).