SUPPLEMENTAL MATERIAL

Methods

Electroanatomical mapping and ablation strategy

All antiarrhythmic drugs (except for amiodarone) were withdrawn at least 5 half-lives before the electrophysiological study, and ablation was conducted with local anesthesia and conscious sedation with dexmedetomidine hydrochloride at 0.2-0.7 μg/kg per hour. Standard multielectrode catheters were placed in the His bundle region and the right ventricular apex. High-density substrate mapping during basal rhythm (sinus rhythm or atrial paced rhythm) and/or ventricular paced rhythm was performed using the CARTO system (Biosense Webster, Diamond Bar, CA) with a multielectrode catheter; Pentaray (2-6-2-mm interelectrode spacing; Biosense Webster), or DecaNav (2-8-2-mm interelectrode spacing; Biosense Webster). Pace-mapping was simultaneously attempted at the location with late or fragmented potentials.

Electrograms (EGMs) were automatically collected during sinus rhythm and/or ventricular paced rhythm using a Wavefront Annotation (Biosense Webster), which fixes on the points with maximal unipolar dV/dt only within the window demarcated by the beginning and end of the bipolar EGM complex [6]. However, at sites with multicomponent or fractionated local electrograms, automated mapping systems typically annotated far-field potentials that often generate a more negative dV/dt value than that of near-field late potentials. We reviewed the annotated local potentials during the procedures and manually moved the annotation to the reproducible latest potentials to mark the maximal discontinuity as necessary. In a diseased porcine model of infarct demonstrated that the conduction velocity decreased ≤ 25.0 cm/s [5]. Hence, the activation maps were displayed as isochronal maps of 10 msec steps, and the IC was defined as ≥4 isochrones within 10 mm [7].

Following completion of substrate mapping, VT induction was attempted with programmed ventricular stimulation from the right ventricular apex at two base cycle lengths (CLs) (400 and 600 ms), with up to three extra stimuli decremented to ventricular refractoriness. When hemodynamically stable VT was induced, electroanatomical activation mapping was performed to depict the tachycardia circuit and superimposed on the anatomic reconstruction of the ventricle. When rapid VT was induced, activation mapping was attempted by bolus intravenous dopamine or noradrenalin (5-10 μg). However, in the case of hemodynamically unstable VT, ablation of the VT substrate guided by pace-mapping and targeting local abnormal potentials was performed.

A 3.5 mm open irrigated tip ablation catheter (ThermoCool Smart Touch; Biosense

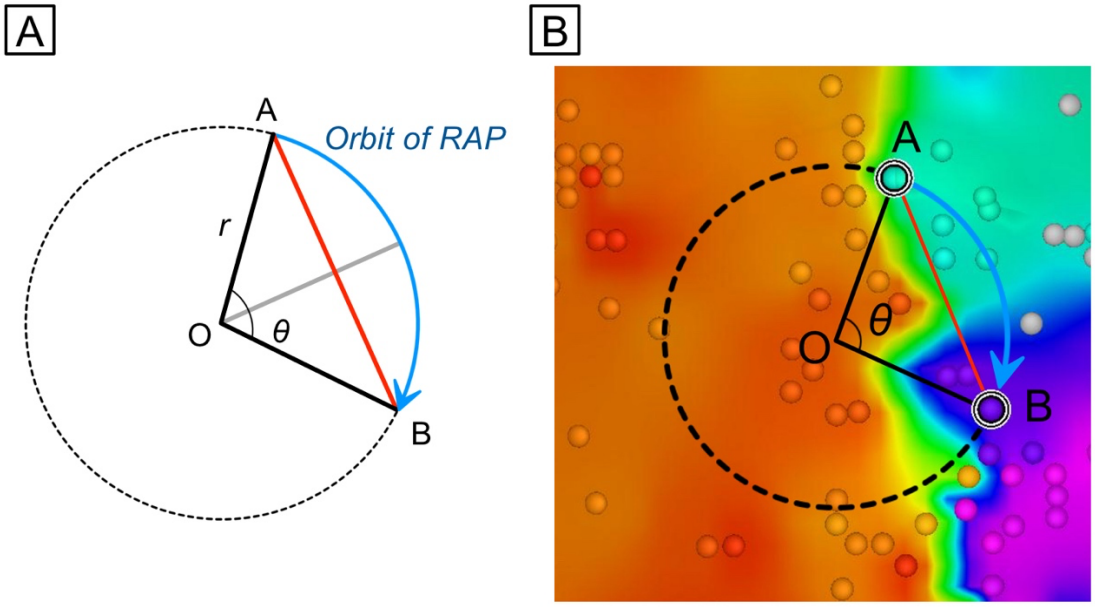
Webster) was advanced to the left ventricle (LV) or right ventricle (RV) using a femoral venous or arterial approach with either the retrograde aortic or transseptal approach. Radiofrequency (RF) energy was delivered at 30–50 W with saline irrigation, with the maximum temperature limit set at 45°C for 60–90 s per application. When the clinical VTs were not suppressed via the endocardial approach or the cases experienced acute recurrence after the previous procedure, epicardial mapping and ablation were performed. Complete success was defined as non-inducibility of any VT, while partial success was defined as the non-inducibility of clinical VT. This was evaluated with repeated programmed stimulation in patients with a stable hemodynamic status.

Characterization of the critical sites for the VT circuit

The VT termination site during RF energy application without an ectopic beat was determined as a critical site for the reentrant circuit. The geometry of the heart chambers can change during VT and sinus rhythm, and the points of termination sites were manually acquired immediately after the VTs were interrupted and recovered to sinus or paced rhythm. Depending on the time phase in relation to the diastolic interval, the termination site was assigned to one of the three main segments of VT: the entrance (inward curvature), common pathway (pathway between both lateral isthmus boundaries or a stimulus [S]-QRS/tachycardia CL [TCL] between 30% and 70%), and exit (outward curvature or S-QRS/TCL ≤30%). Entrainment pacing was not always performed because of the possibility of acceleration, morphological changes, and termination. However, pacing sites which demonstrated concealed fusions, post-pacing intervals (PPI)-TCL ≤30 ms, and S-QRS ≤70% was defined as a location on the critical circuit [8], in whom the entrainment pacing was performed.

In addition to direct termination with RF application, we also defined sites with good pacemap scores and either multiple exit sites or pace-mapping induction of VT as a part of critical VT circuits [9-10]. After RF energy applications by pace-mapping guidance, target VT became non-inducible. The pacemap score was calculated by software equipped with a 3D-electroanatomical mapping system (PASO Module, Biosense Webster), and a good pacemap score was defined as a correlation value of 89% or more [11]. When pacemap scores of 89% or higher were recorded at two or more points, the point with the longest stimulus to QRS was adopted as the critical site.

Supplemental Figure I. Measurement of the RAP angle.



To delineate the qualitative characteristics of the RAP, we adopted the Newton-Raphson method to approximate the angle of the RAP's orbit (θ)[12].

A, The earliest and the latest excitation sites (A and B) that form the RAP were identified. Assuming that the RAP has a regular circular trajectory, the trajectory and the shortest distance of the excited propagation are expressed as arcs ($\widehat{AB}$) and chords ($\overline{AB}$). The distance of the arc ($\widehat{AB}$, *blue trajectory*) and the chord ($\overline{AB}$, *red straight line*) were measured on the 3-dimensional electroanatomial mapping system. Although the radius of the RAP's orbit (r) is not measurable, it can be expressed from the arc and chord by the following equation:

$$r = \frac{\frac{\overline{AB}}{2}}{\sin\frac{\theta}{2}} = \frac{\frac{\widehat{AB}}{2}}{\frac{\theta}{2}}$$

then

$$\frac{\sin\frac{\theta}{2}}{\frac{\theta}{2}} = \frac{\frac{\overline{AB}}{2}}{\frac{\widehat{AB}}{2}} = \frac{\overline{AB}}{\widehat{AB}}$$

When we make the letter 'n' equal to the ratio of $\overline{AB}/\widehat{AB}$,

$$\frac{\sin\frac{\theta}{2}}{\frac{\theta}{2}} = n$$

We then get the following equation.

$$f(x) = \sin\frac{\theta}{2} - n \cdot \frac{\theta}{2}$$

The Newton-Raphson method is a root-finding algorithm, which is described by the following formula:

$$x_{n+1} = x_n + \frac{f(x_{n)}}{f'(x_{n)}}$$

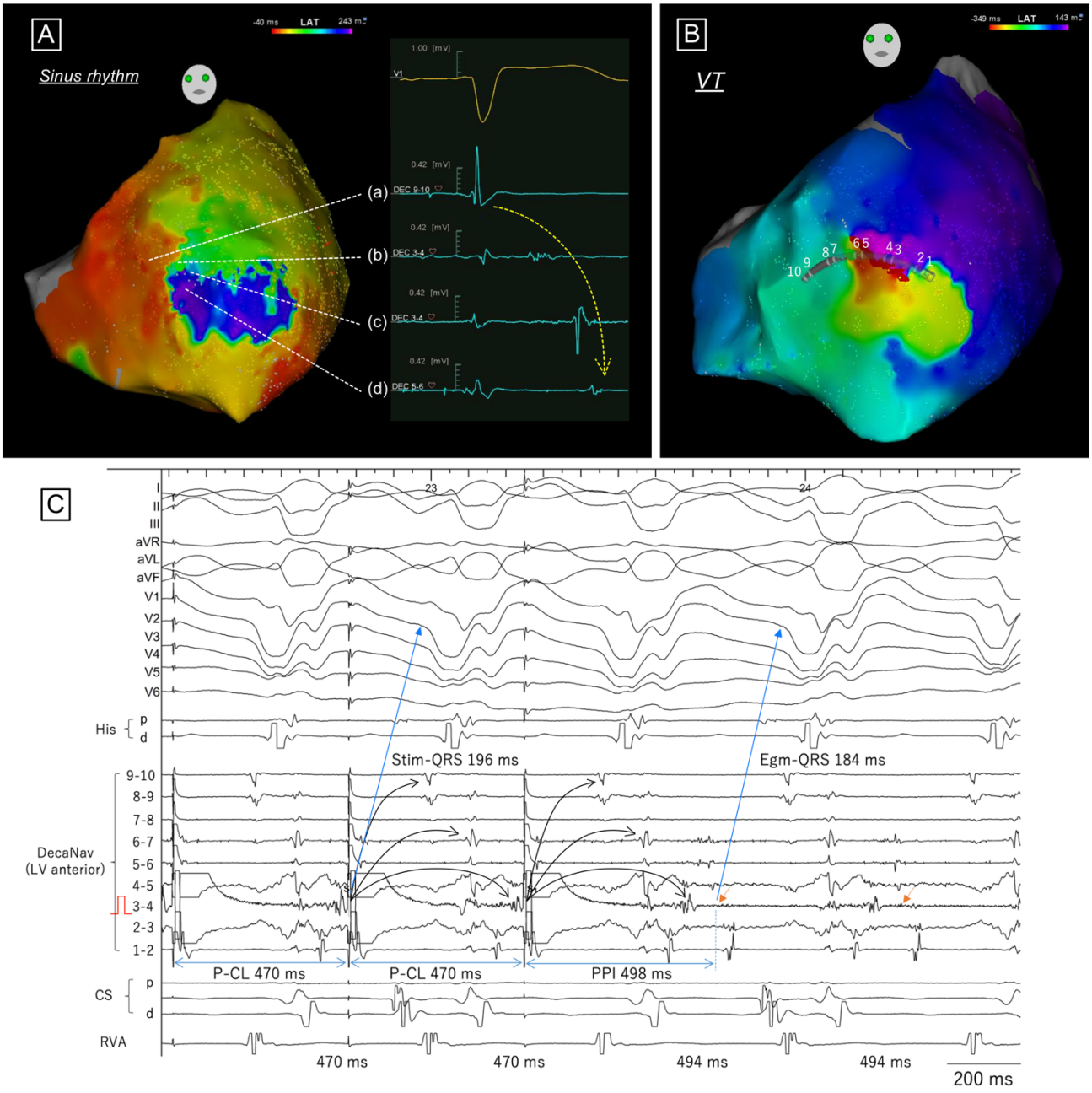
The derivative of *f(x)* is

$$f'(x) = \cos\frac{\theta}{2} - n = 0$$

Starting with the assumption that x_0 is 1 rad, the calculation was repeated until the solution for x_{n+1} was identical to x_n with three significant digits. The RAP angle was finally expressed in degrees by multiplying θ by 180/π.

B, Approximation of the RAP' angle in a clinical setting. Based on the measured arc length of 98 mm and string length of 87 mm, the central angle of the RAP was calculated to be 95 degrees.

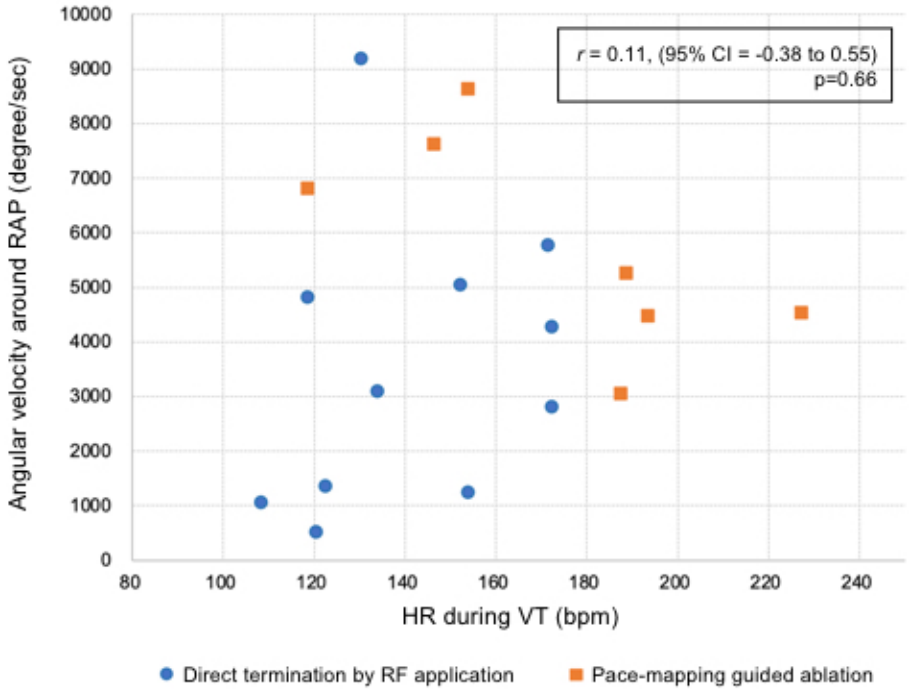
Supplemental Figure II. Entrainment at the isthmus site.



A, The local bipolar potentials recorded on the trajectory of the RAP during intrinsic QRS activation demonstrated a fragmented, long-duration and low-amplitude electrogram (b) followed by the wide split double potentials (c and d). **B,** The linear multielectrode catheter was vertically placed on the isthmus during VT. **C,** The bipolar electrogram recorded on DecaNav 3-4 showed an early diastolic potential in larger amplitude followed by a smaller fragmented mid-diastolic potential during VT (orange arrow). Entrainment pacing from this site captured the smaller fragmented potential (Stimulus to QRS of 196 ms; 39% of the TCL) and resulted in concealed fusion with PPI-TCL of 4 ms. The morphology of the larger

potentials recorded from DecaNav 3-4 to 9-10 remained unchanged during pacing, indicating an orthodromic capture of the potentials (black arrows). The CS indicates coronary sinus catheter; PPI, postpacing interval; TCL, tachycardia cycle length; and RVA, right ventricular apex.

Supplemental Figure III. Comparison of the RAP's angular velocity and TCL.



Scatter plot of the angular velocity of the RAP and the tachycardia cycle length (TCL). Red dots indicate VTs whose critical sites were identified by direct termination by radiofrequency application or by pace-mapping with a high correlation score (≥ 89% of the PASO score).

Supplemental Video Legends

Supplemental movie I. Videos illustrating the functional substrate mapping during sinus rhythm (left) and the activation mapping during VT (right) in a patient with remote anterior infarction who underwent epicardial ablation. The activation mapping during VT demonstrates figure-eight reentry, where the activation wavefront propagates down from the mid-anterior toward the apex where the impulse bifurcates, and two distinct wavefronts travel in the opposite direction. The VT was terminated in one second by RF energy application at the RAP site demonstrated by the mapping during sinus rhythm (green tag).

Supplemental movie II. Videos illustrating the functional substrate mappings acquired during sinus rhythm (left) and ventricular-paced rhythm from anterior interventricular vein (right). VT termination site (green tag) was located at the wavefront collision site and was not accompanied by the RAP during sinus rhythm. The mapping during ventricular-paced rhythm unveiled the RAP at the VT termination site.