

## Case Report

# Use of the Local Activation Time Histogram “Trough” to Identify the Slow Conduction Channel in Complex Congenital Heart Disease Macro-re-entrant Arrhythmias

Nathan C. Denham, BM, PhD,<sup>a</sup> Robert D. Anderson, MBBS, PhD,<sup>a</sup>

Raja Selvaraj, MBBS, MD, DM,<sup>b</sup> Andrew C.T. Ha, MD,<sup>a</sup> and Krishnakumar Nair, MD<sup>a</sup>



<sup>a</sup> University Health Network Toronto, Peter Munk Cardiac Centre, and University of Toronto, Toronto, Ontario, Canada



<sup>b</sup> Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry, India

**Performing and interpreting activation maps of atrial flutter in patients with complex congenital heart disease is challenging due to a combination of anatomic barriers and electrophysiological remodelling. We report a case where we used the spread of local activation time across the left atrium to identify the critical isthmus and design a successful ablation line, which was not highlighted by conventional activation mapping techniques. We outline the potential benefits to local activation time “trough” analysis in patients with congenital heart disease and its limitations.**

A 49-year-old gentleman, status after repair of a total anomalous pulmonary venous connection and closure of an atrial septal defect (ASD), underwent an electrophysiology study for recurrent symptomatic atrial flutter. The original anatomy consisted of an intracardiac total anomalous pulmonary venous connection with drainage into the coronary sinus (CS) and a small secundum ASD. At 6 months of age, he underwent unroofing of the CS with pericardial patch closure of the ASD and CS os. The patient was in ongoing atrial arrhythmia at the time of ablation. Two sheaths were inserted in the left femoral vein as the right femoral vein was occluded. A right atrial activation map was created using the CARTO-3 system (Biosense Webster, Irvine, CA) and a 3-3-3 mm OctaRay catheter (Biosense Webster). The reference catheter was a 5-5-5 mm decapolar catheter (Inquiry; Abbott, Chicago, IL) placed in the oesophagus as the right atrial anatomy (ie, absence of a patent CS os) precluded stable positioning of an intracardiac reference catheter. A window of interest (WOI)

was used fulfilling the conventional De Ponti method,<sup>1</sup> setting the WOI to mid-diastole after identification of the surface P-wave spanning 100% of the tachycardia cycle length (TCL: 240 ms). Only 70% of the TCL was observed in the right atrium with septal breakout (Fig. 1A), indicating a left atrial (LA) source.

A trans-septal puncture was performed under intracardiac echocardiography guidance (SOUNDSTAR; Biosense Webster) with an SL-0 sheath (Abbott) and an NRG radio-frequency trans-septal needle (Baylis Medical, Mississauga, ON), subsequently exchanged for a large curl Agilis (Abbott). An LA activation map contained the entire TCL; however, centering the flutter wave in the activation window demonstrated a broad early-meets-late (EML) region from the right pulmonary venous (RPV) ostium to the mitral annulus obliquely across the posterior wall (Fig. 1B; Video 1 , view video online). Analysis over this region showed fractionated, long-duration electrograms (EGMs) with a duration of up to 50 ms (21% of the TCL) and the shortest distance across this anatomic isthmus measured 6.7 cm from the RPV ostium to the mitral annulus. An LA voltage map performed during atrial flutter demonstrated widespread septal, posterior, and inferior patchy low-voltage zones suggestive of scar (Fig. 1C and D; Video 2 , view video online). Application of coherent mapping with slow or no conduction zones and conduction velocity vector direction and speed now revealed 2 potential circuits:

- (1) Roof-dependent macro-re-entry encircling the RPV ostium (Fig. 1E; Video 3 , view video online)
- (2) Macro-re-entry around focal scar in the inferoposterior region of the interatrial septum (Fig. 1F, Video 4 , view video online).

Analysis of the local activation time (LAT) histogram distribution yielded useful information for identifying the critical isthmus and an improved ablation target over that suggested by the anatomic isthmus in the EML region. A total of 5435 LAT points were annotated, and the point density was visually

Received for publication November 18, 2023. Accepted December 30, 2023.

Corresponding author: Dr Krishnakumar Nair, Peter Munk Cardiac Centre, Toronto General Hospital, University of Toronto, 200 Elizabeth St, 3rd Floor, Toronto, Ontario M5G 2C4, Canada. Tel.: +1-647-993-6247; fax: +1-416-595-1811.

E-mail: [Krishnakumar.Nair@uhn.ca](mailto:Krishnakumar.Nair@uhn.ca)

### Novel Teaching Points

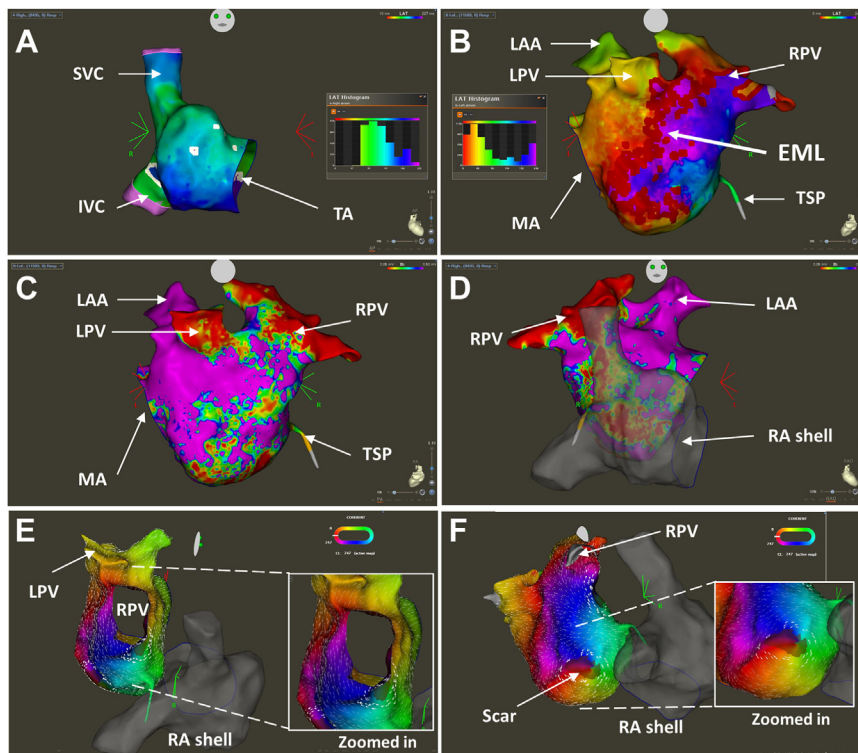
- In complex atrial substrates that characterize congenital heart disease, identifying a small number of activation points over a small area of the atrium can be used to find the slowly conducting isthmus, particularly when this percentage of the tachycardia cycle length is not represented elsewhere.
- Locating this local activation time “trough” can be successfully used in real time as an alternative to design optimal ablation lines, particularly when this narrow channel is shorter than the early-meets-late region using the De Ponti method.<sup>1</sup>
- For this method of mapping to be valid, a complete and uniform density of activation points should be taken from the relevant atrial chamber(s).

evenly distributed across the LA (Supplemental Fig. S1). Segmentation of the total activation time into 10 isochrones (each representing 10% of the mapped TCL) highlighted a “trough” in the LAT histogram, characterized by the smallest isochrone

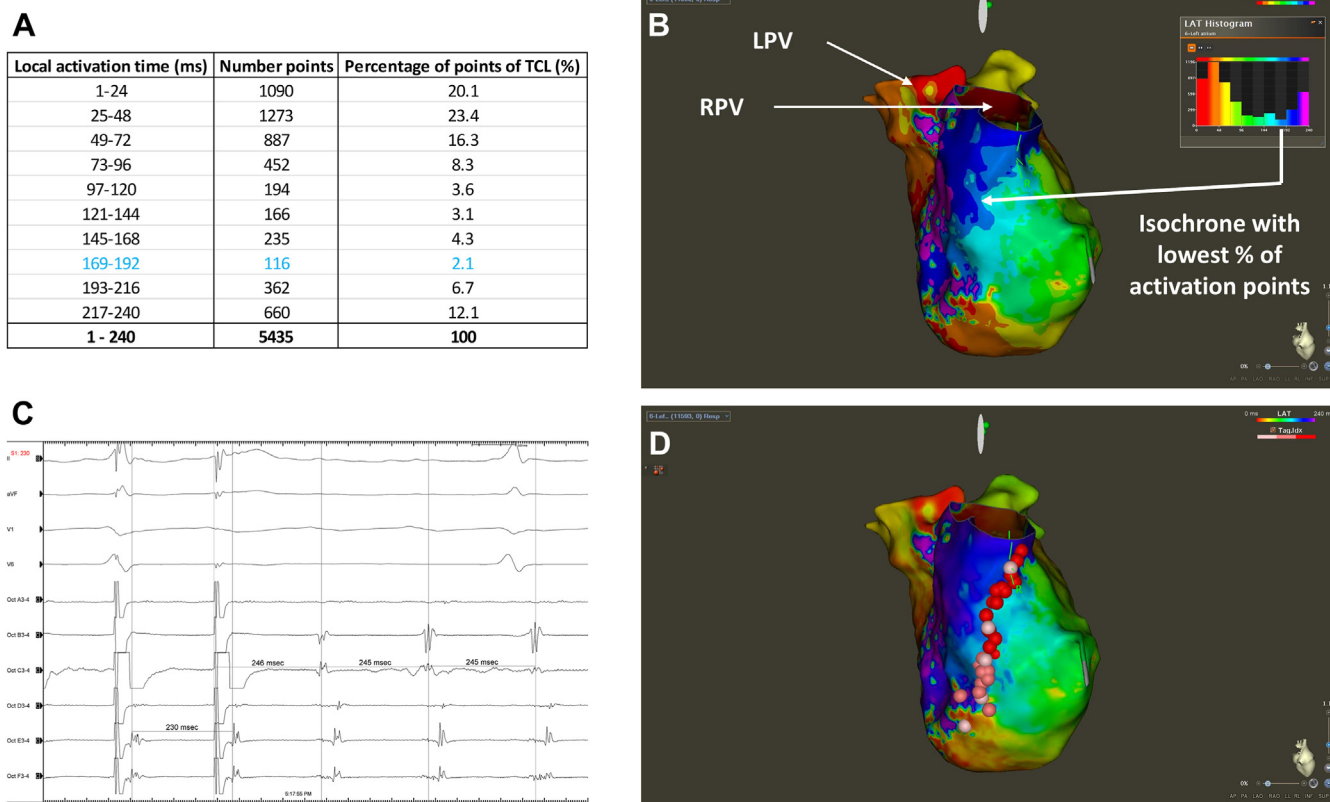
(116 LAT points [2.1% of total LAT points], Fig. 2A). In the activation map, this corresponded to a narrow common channel through which the re-entrant wavefront must propagate for perpetuation of both putative arrhythmia circuits (Fig. 2B). This critical common isthmus was proven with concealed entrainment and a postpacing interval approximating the TCL (Fig. 2C). The anatomic basis of this isthmus was felt to be a region of slowed conduction arising from patchy scar on the posterior interatrial septum between the RPV ostium and the unroofed CS. A 5-cm linear ablation lesion across this isthmus linking 2 patches of electrically inert scar (target ablation index: 550) resulted in successful termination of the flutter and subsequent noninducibility (Fig. 2D).

### Discussion

The archetypal surgically repaired congenital patient with atrial arrhythmias epitomizes the most challenging for electrophysiologists: anatomic barriers, diffuse, and heterogeneous substrate secondary to incisional lines and structural remodeling. Conventional mapping tools are often restrictive such as CS access to allow stable atrial reference and low-amplitude surface P-waves to configure an accurate WOI. Furthermore,



**Figure 1.** Activation maps of the atria in flutter and the potential circuits. **(A)** An activation map of the right atrium (RA) in the anteroposterior projection. The window of interest was set at 100% of the tachycardia cycle length (240 ms). The local activation time histogram is missing the initial 30% of the tachycardia cycle length. **(B)** An activation map of the left atrium in the posteroanterior projection. The histogram shows that the entire cycle length was mapped with a broad area of early-meets-late (EML) (deep red) on the posterior wall. **(C)** A corresponding voltage map of the left atrium in the posteroanterior projection showing the pulmonary vein anatomy and patchy posterior scar (bipolar voltage cutoffs 0.05-0.5 mV). **(D)** A voltage map in the right anterior oblique projection demonstrating scar on the interatrial septum. An anatomic shell of the RA (grey) is shown for orientation. **(E)** A coherent map of the left atrium showing a potential roof-dependent flutter encircling the RPV ostium (cutout). The region of interest has been enlarged on the right. **(F)** A coherent map of the left atrium showing an alternative flutter mechanism around scar on the inferoposterior aspect of the interatrial septum. The region of interest has been enlarged on the right. IVC, inferior vena cava; LAA, left atrial appendage; LPV, left pulmonary veins; MA, mitral annulus; RPV, right pulmonary veins; SVC, superior vena cava; TA, tricuspid annulus; TSP, trans-septal puncture.



**Figure 2.** Using the spread of local activation time points to identify the critical isthmus. **(A)** A table showing the distribution of local activation points across the tachycardia cycle length (TCL) by dividing into 10 equally spaced isochrones. The minimum represented isochrone is highlighted in blue. **(B)** An activation map of the left atrium shown in the right lateral projection. The isochrone with the lowest percentage of activation points (mid-blue) forms a narrow channel of activation over the septum. **(C)** Entrainment from the narrow channel using OctaRay bipoles C3-4 demonstrates concealed entrainment and a postpacing interval approximating the TCL. **(D)** The linear ablation lesion set shown in a right lateral projection. LPV, left pulmonary veins; RPV, right pulmonary veins.

activation mapping of complex circuits in this patient group is challenging due to limitations of LAT detection and annotation of long duration, usually low-amplitude EGMs, multiple regions of bystander slow conduction, wavefront collision, lines of functional and anatomic block, and the propensity for multiple circuits often sharing common isthmuses. Finally, discerning the mechanism and site participation with standard manoeuvres such as entrainment are open to error due to substrate complexity.

Analogous to highlighting conduction slowing in sinus rhythm with isochronal late activation mapping (ILAM), identifying the zone of slowest conduction in a re-entrant circuit is clinically valuable and has previously been shown to be correlated with critical circuitry components in atypical flutters without complex congenital heart disease.<sup>2-4</sup> It has also recently been shown to be a useful tool to guide ablation targets in patients with biatrial tachycardias in congenital heart disease by localizing the shortest anatomic interatrial connections through overlapping of the activation histogram graphs,<sup>5</sup> as well as helping to identify the common shared isthmus in dual loop tachycardias.<sup>6</sup> The LAT histogram “trough” (also referred to as a “valley”) is a novel visualizable technique to easily emphasize the region of slowest conduction during re-entrant tachycardia, based on the presumption that a lower point density denotes a smaller region of substrate

activated for each equally spaced isochrone. A deeper “trough” has been associated with decreased isthmus dimensions (particularly a narrower width) and a slower conduction velocity, making it an attractive ablation target.<sup>6</sup> Unlike the EML region, which can be modified depending on the WOI:P-wave ratio, the LAT “trough” remains fixed irrespective of the WOI.<sup>2</sup> Static EGM analysis is often limited by finding regions of long, fractionated EGMs that constitute a significant portion of the diastolic interval and appear critical but are noncritical bystanders due to wavefront collision. A major advantage of LAT “trough” analysis is the ability to differentiate these sites and avoid unnecessary ablation.<sup>2</sup> In addition, a review of the LAT histogram may help identify alternative isthmuses amenable to ablation when the primary target cannot be selected, for example, proximity to the phrenic nerve, concerns over the ability to achieve transmural lesions in hypertrophied/scarred chambers, or poor ablation catheter stability. In our case, although the LAT “trough” helped us target the critical isthmus, a lower ablation index target (400-450) was required at the inferior end of the linear lesion set to avoid injuring the left-sided conduction system, highlighting that the “trough” does not always equate to the ideal place to ablate.

Accurate interpretation requires a number of prerequisites including, and most importantly, the assumption that the

arrhythmogenic mechanism is macro-re-entrant and contains a slowly conducting isthmus. Non-re-entrant sources will typically not harbour slow conduction at their sites of origin. It also assumes that the propagating wavefront is planar to the mapped surface; intramural or epicardial circuit components with short bridging activation gaps may falsely appear erroneously slow or blocked. It is therefore unlikely that this method of LAT analysis can be applied as successfully in the ventricle. Furthermore, complete chamber mapping encompassing the total activation time and homogeneous point density is essential, although no current study has defined the minimum point density per unit surface area to make LAT histogram analysis valid. We chose 10 isochrones in this case as it appears to be suitable to visually differentiate the “trough,” although this could be modified depending on chamber size and point density.

The educational aspect of this case is that we used the LAT histogram “trough” in real time to identify and ablate the critical isthmus in a patient with complex congenital heart disease. The majority of currently published case series using this type of analysis are retrospective;<sup>2–4</sup> however, Moore et al.<sup>6</sup> have previously demonstrated prospectively in a small series the benefit of targeted ablation to the “trough” highlighted by the global activation histogram using the Rhythmia HDx mapping system. It has yet to be shown however in a randomized, prospective trial whether histogram-guided ablation has comparable outcomes to other methods of LAT mapping (eg, Ripple mapping). We found that the histogram simplified the LAT map, allowed for focused entrainment of the potential circuit, and helped design a shorter ablation line through a critical isthmus compared with the anatomic line suggested by the EML zone.

One area of future interest is whether the LAT “trough” identified in atrial flutter corresponds to the same regions of conduction slowing or deceleration highlighted by ILAM mapping in sinus rhythm or with atrial pacing. In our case, we did not perform an ILAM map for comparison, so we were unable to explore this further. However, if this relationship could subsequently be demonstrated, ILAM mapping could provide an additional tool to help design ablation in congenital patients in whom the clinical atrial flutter cannot be induced or is poorly haemodynamically tolerated.

### Acknowledgement

We would like to thank Alannah Lax-Vanek for her assistance in creating images for this case.

### Ethics Statement

The authors confirm the research reported has adhered to the relevant ethical guidelines.

### Patient Consent

The authors confirm that patient consent forms have been obtained for this article.

### Funding Sources

No funding was received for this study.

### Disclosures

The authors have no conflicts of interest to disclose.

### References

1. De Ponti R, Verlato R, Bertaglia E, et al. Treatment of macro-re-entrant atrial tachycardia based on electroanatomic mapping: identification and ablation of the mid-diastolic isthmus. *EP Europace*. 2007;9:449–457.
2. Yang S, Waks JW, Galvao MET, et al. The use of local activation timing histogram in ablation of focal and re-entrant atrial tachycardias. *JACC Clin Electrophysiol*. 2023;9:2603–2614.
3. Takigawa M, Martin CA, Derval N, et al. Insights from atrial surface activation throughout atrial tachycardia cycle length: a new mapping tool. *Heart Rhythm*. 2019;16:1652–1660.
4. Adragão P, Matos D, Costa FM, et al. A new electrophysiologic triad for identification and localization of the critical isthmus in atrial flutter. *Rev Port Cardiol (Engl Ed)*. 2020;39:309–314.
5. Moore JP, Bowman H, Gallotti RG, Shannon KM. Mechanisms and outcomes of catheter ablation for biatrial tachycardia in adults with congenital heart disease. *Heart Rhythm*. 2021;18:1833–1841.
6. Moore JP, Buch E, Gallotti RG, Shannon KM. Ultrahigh-density mapping supplemented with global chamber activation identifies noncavotricuspid-dependent intra-atrial re-entry conduction isthmuses in adult congenital heart disease. *J Cardiovasc Electrophysiol*. 2019;30:2797–2805.

### Supplementary Material

To access the supplementary material accompanying this article, visit *CJC Pediatric and Congenital Heart Disease* at <https://www.cjpc.ca/> and at <https://doi.org/10.1016/j.cjpc.2023.12.004>