# Atypical lower loop reentrant tachycardia associated with multiple hepatic veins

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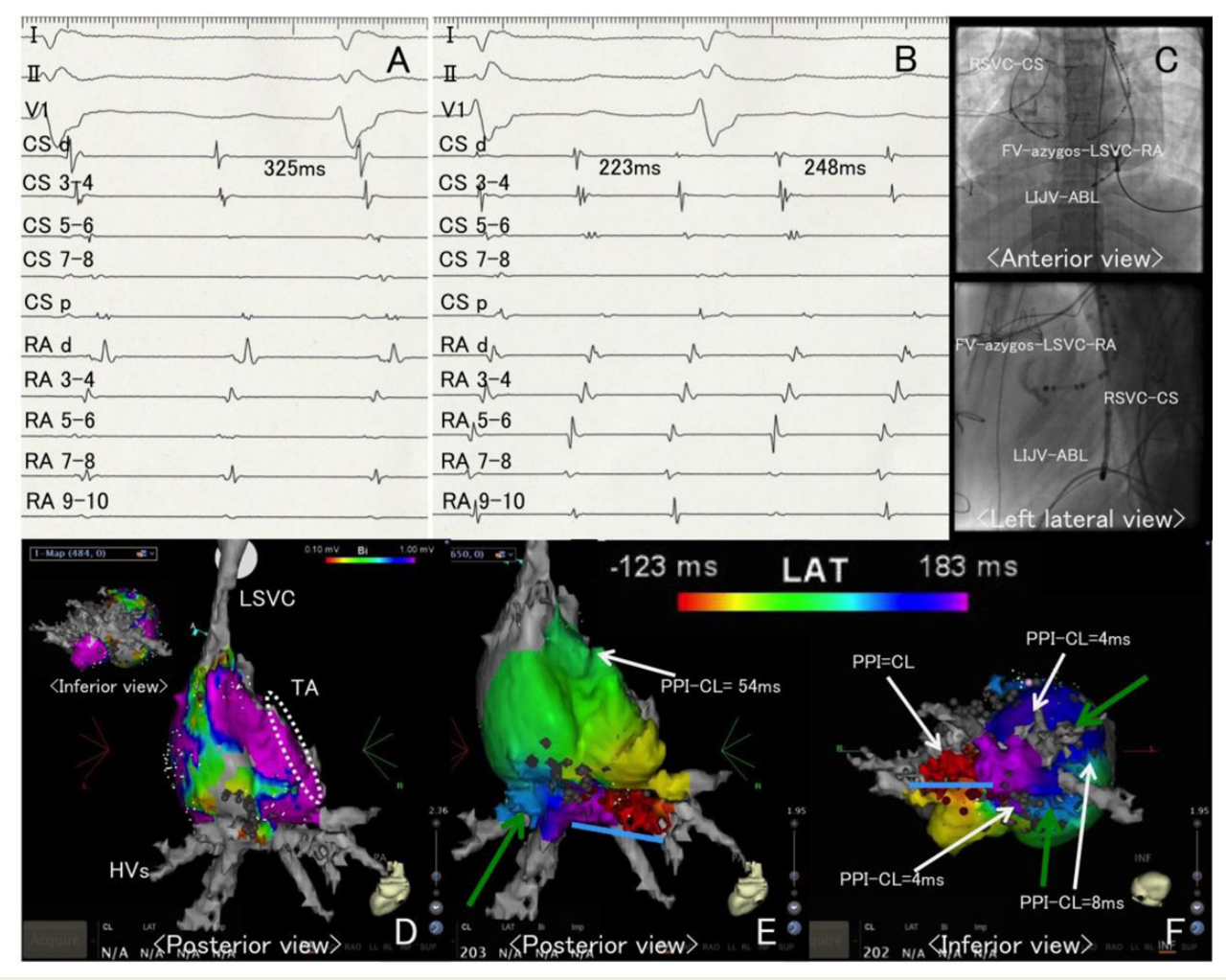


**Figure 1** (*A*) Intracardiac electrocardiogram during the stable atrial tachycardia with a cycle length of 325 ms. (*B*) Intracardiac electrocardiogram during the unstable atrial tachycardia with a cycle length ranging from 223 to 248 ms with a similar activation sequence recorded by multiple electrodes placed at the coronary sinus and the RA. CS, the electrode potentials recorded from the catheter placed into the coronary sinus; RA, the electrode potentials recorded from the catheter placed at the right atrium. Numbers are given from distal to proximal pairs in order. (*C*) Fluoroscopic images in the anterior and left lateral view. FV-azygos-LSVC-RA, the catheter placed at the right atrium via in order of femoral vein, azygos vein, and left superior vena cava; RSVC-CS, the catheter placed at the coronary sinus via right superior vena cava; LIJV-ABL, the ablation catheter placed at bottom of the right atrium via left internal jugular vein. (*D*) The voltage map of the right atrium from the posterior view and inferior view (inset). LSVC, left superior vena cava; TA, tricuspid annulus; HV, hepatic vein. (*E* and *F*) The activation map of the right atrium during the stable atrial tachycardia with a cycle length of 325 ms obtained from the posterior view (E) and inferior view (F). The entrainment study suggested a macroreentrant mechanism around the HVs directly connected to the bottom of RA: PPI − CL = 0 ms at the posterior of the TA, 4 ms at the anterior and posterior bottom of the RA, and 8 ms at the lateral bottom of the RA. On pacing from the anterior (upper) portion of TA, the PPI was prolonged (PPI−CL = 54 ms). White arrows show the sites where the entrainment study was performed. A blue line shows a linear ablation site from the tricuspid annulus to the bundle of hepatic veins. Green arrows show additional ablation sites for the fragmentation potentials. HV, hepatic vein; RA, right atrium; TA, tricuspid annulus; LAT, local activation time. PPI, post-pacing interval; CL, cycle length.

A 32-year-old female presented with paroxysmal palpitation for 5 years. She underwent Rastelli operation three times for mirror-imaged Tetralogy of Fallot, pulmonary atresia, interruption of IVC with azygos continuation, and persistent right superior vena cava with dextrocardia. A substrate map showed a long narrow strip of low voltage zone (<0.5 mV) at the postero-lateral wall of the RA (suggesting the incision lines produced by previous surgeries) and an area with poor electrical potential at the bottom of the RA (*Figure 1D*). Programmed stimulation at the RA induced atrial tachycardia (AT). The tachycardia showed predominantly a stable cycle length of 325 ms (*Figure 1A*), although sometimes beat-by-beat variations with a similar activation sequence were observed (*Figure 1B*). When the tachycardia cycle length was stable, entrainment mapping at different sites in the area assumed as the area of the lower loop reentry, the postpacing interval almost equaled the tachycardia cycle length (*Figure 1F*). On the other hand, the return cycle was longer (+54 ms) when the anterior right atrium was entrained (*Figure 1E*). Moreover, the activation time obtained from the CARTO map was 306 ms (123 + 183), which corresponded to 94% of the tachycardia cycle length (*Figure 1E* and *F*). During the ablation for the isthmus between the tricuspid annulus (TA) and a bundle of HVs, AT terminated with prolongation of the CL. We could not confirm the bidirectional block line at the isthmus because of the poor electrical potential at the bottom of the RA. Therefore, the additional ablation for the fragmented potentials was performed just in case. Finally, both stable AT and unstable AT were no longer inducible.

To the best of our knowledge, this is the first case of the LLR in the absence of IVC. The AT in this patient would be designated as atypical LLR. This reentrant circuit depends on conduction through the TA-HVs isthmus.

In general, the HVs directly drain to the RA either separately or through a common ostium in the absence of IVC.[1] Our case was the former based on the 3D computed tomography shown in *Figure 1D*. If there is some distance between the HVs, muscular tissue could spread over these gaps. In fact, Ihara *et al*. reported a case of AT originating from the hepatic segment of the venous tissue in the absence of IVC.[2] In agreement with their case report, we detected the electrical activation at the limited area between the HVs (*Figure 1E* and *F*). Because the voltage was very low over this area (*Figure 1D*), it would serve as the critical slow conduction responsible for reentry. Therefore, this macroreentrant tachycardia appears to have rotated around a bundle of HVs and/or several of them in some instances, thereby altering its cycle length as shown in *Figure 1B*.

**Conflict of interest:** none declared.

## References

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