4:2:1 – a thoracic vein tachycardia

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A 27-year old woman presented to the Foothills Medical Centre (Calgary, Alberta) with a two-year history of intermittent episodes of rapid heart action. There was no history of cardiovascular disease, and a transthoracic echocardiogram revealed no evidence of structural heart disease. She underwent a four-catheter transvenous electrophysiological study and catheter ablation procedure for recurrent atrial tachycardia.

On the day of the procedure, a 12-lead surface electrocardiographic recording (Figure 1) documented clinical tachycardia. Intracardiac recordings from the superior vena cava (SVC), right atrium and surface electrocardiographic leads II and V1 at 100 mm/s paper speed (Figure 2) were recorded during activation mapping. The recording from the SVC revealed that structure was the origin of the arrhythmia, whereby the

tachycardia exhibited a rate of 460 beats/min. Every other beat of the tachycardia conducted to the atria, and every other atrial depolarization (230 beats/min) conducted to the ventricles, resulting in an overall conduction ratio of 4:2:1 and a ventricular rate of 115 beats/min.

Because conduction to the atria occurred in a regular 2:1 fashion, the tachycardia manifested electrocardiographically as an atrial tachycardia. However, one can understand how more rapid or variable conduction can produce less organized atrial activation, or atrial fibrillation. The arrhythmogenic potential of the pulmonary veins is well recognized (1), but all thoracic veins can, in fact, be arrhythmogenic, including the SVC, as demonstrated in the present case (2-4). The SVC was electrically isolated, and the patient had no symptoms after one month of follow-up.

Figure 1) A surface 12-lead electrocardiogram during atrial tachycardia

Figure 2) Intracardiac recordings from the superior vena cava (SVC), right atrium (RA) (P wave), and surface leads II and V1 (QRS) during atrial tachycardia (paper speed was 100 mm/s)

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