Diagnosis and Management of Typical Atrial Flutter

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KEYWORDS

• Atrial flutter • Cavo-tricuspid isthmus • Ablation

Type 1 atrial flutter (AFL) is a common atrial arrhythmia that may cause significant symptoms and serious adverse effects including embolic stroke, myocardial ischemia and infarction, and rarely a tachycardia-induced cardiomyopathy as a result of rapid atrioventricular conduction. The electrophysiologic substrate underlying type 1 AFL has been shown to be a combination of slow conduction velocity in the cavo-tricuspid isthmus (CTI), plus anatomic and/or functional conduction block along the crista terminalis and Eustachian ridge (**Fig. 1**). This electrophysiologic milleu allows for a long enough reentrant path length relative to the average tissue wavelength around the tricuspid valve annulus to allow for sustained reentry.

Type 1 AFL is relatively resistant to pharmacologic suppression. As a result of the well-defined anatomic substrate and the pharmacologic resistance of type 1 AFL, radiofrequency catheter ablation has emerged in the past decade as a safe and effective first-line treatment. Although several techniques have been described for ablating type 1 AFL, the most widely accepted and successful technique is an anatomically guided approach targeting the CTI. Recent technological developments, including three-dimensional electro-anatomic contact and noncontact mapping, and the use of irrigated tip and large-tip ablation electrode catheters with high-power generators, have produced nearly uniform efficacy without increased risk. This article reviews the electrophysiology of human type 1 AFL, techniques currently used for its diagnosis and management, and emerging technologies.

ATRIAL FLUTTER TERMINOLOGY

Because of the variety of terms used to describe atrial flutter in humans, including type 1 AFL and type 2 AFL, typical and atypical atrial flutter, counterclockwise and clockwise atrial flutter, and isthmus and non-isthmus dependent flutter, the Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology convened and published a consensus document in 2001 in an attempt to develop a generally accepted standardized terminology for atrial flutter.[1] The consensus terminology derived from this working group to describe CTI-dependent, right atrial macroreentry tachycardia, in the counterclockwise or clockwise direction around the tricuspid valve annulus was ''typical'' or ''reverse typical'' AFL respectively.[1] For the purposes of this article, these two arrhythmias will be referred to specifically as typical and reverse typical AFL when being individually described, but as type 1 AFL when being referred to jointly.

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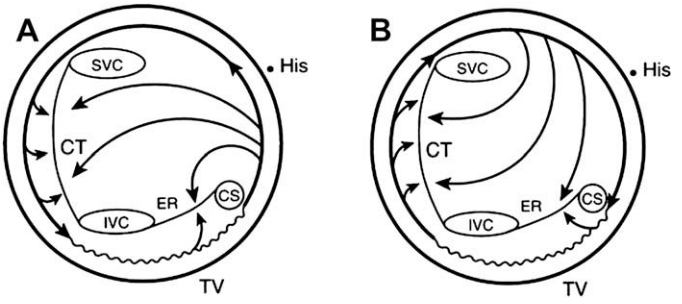

Fig.1. Schematic diagrams demonstrating the activation patterns in the typical (*A*) and reverse typical (*B*) forms of human type 1 AFL, as viewed from below the tricuspid valve annulus (TV) looking up into the right atrium. In the typical form of AFL, the reentrant wavefront rotates counterclockwise in the right atrium, whereas in the reverse typical form reentry is clockwise. Note that the Eustachian ridge (ER) and crista terminalis (CT) form lines of block, and that an area of slow conduction (*wavy line*) is present in the isthmus between the inferior vena cava (IVC) and Eustachian ridge and the tricuspid valve annulus. CS, coronary sinus ostium; His, His bundle; SVC, superior vena cava.

PATHOPHYSIOLOGIC MECHANISMS OF TYPE 1 ATRIAL FLUTTER

The development of successful radiofrequency catheter ablation techniques for human type 1 AFL was largely dependent on the delineation of its electrophysiologic mechanism. Through the use of advanced electrophysiologic techniques, including intraoperative and transcatheter activation mapping,[2–7] type 1 AFL was determined to be attributable to a macro-reentrant circuit rotating in either a counter-clockwise (typical) or clockwise (reverse typical) direction in the right atrium around the tricuspid valve annulus, with an area of relatively slow conduction velocity in the low posterior right atrium (see **Fig. 1**A, B). The predominate area of slow conduction in the AFL reentry circuit has been shown to be in the CTI, through which conduction times may reach 80 to 100 msec, accounting for one third to one half of the AFL cycle length.[8–10] The CTI is anatomically bounded by the inferior vena cava and Eustachian ridge posteriorly and the tricuspid valve annulus anteriorly (see **Fig. 1**A, B), both of which form lines of conduction block or barriers delineating a protected zone of slow conduction in the reentry circuit.[5,11–13] The presence of conduction block along the Eustachian ridge has been confirmed by demonstrating double potentials along its length during AFL. Double potentials have also been recorded along the crista terminalis suggesting that it also forms a line of block separating the smooth septal right atrium from the trabeculated right atrial free wall. Such lines of block, which may be either functional or anatomic, are necessary to create an adequate path-length for reentry to be sustained and to prevent short circuiting of the reentrant wavefront.[12–14] The medial CTI is contiguous with the interatrial septum near the coronary sinus ostium, and the lateral CTI is contiguous with the low lateral right atrium near the inferior vena cava (**Fig. 1**A, B). These areas correspond electrophysiologically to the exit and entrance to the zone of slow conduction, depending on whether the direction of reentry is counterclockwise (CCW) or clockwise (CW) in the right atrium. The path of the reentrant circuit outside the confines of the CTI consists of a broad activation wavefront in the interatrial septum and right atrial free wall around the crista terminalis and the tricuspid valve annulus.[11–14]

The slower conduction velocity in the CTI, relative to the interatrial septum and right atrial free wall, may be caused by anisotropic fiber orientation in the CTI.[2,8–10,15,16] This may also predispose to development of unidirectional block during rapid atrial pacing, and account for the observation that typical (CCW) AFL is more likely to be induced when pacing is performed from the coronary sinus ostium. Conversely, reverse typical (CW) AFL is more likely to be induced when pacing from the low lateral right atrium.[17,18] This hypothesis is further supported by direct mapping in animal studies demonstrating that the direction of rotation of the reentrant wavefront during AFL is dependent on the direction of the paced wavefront producing unidirectional block at the time of its induction.[19] In humans, the predominate clinical presentation of type 1 AFL is the typical variety, likely because the trigger(s) for AFL commonly arise from the left atrium in the form of premature atrial contractions or nonsustained atrial fibrillation.[20] Triggers arising from the left atrium or pulmonary veins usually conduct to the right atrium via the coronary sinus or interatrial septum, thus entering the CTI from medial to lateral, which results in clockwise unidirectional block in the CTI with resultant initiation of counterclockwise typical AFL.

The development of abnormal dispersion or shortening of atrial refractoriness as a result of atrial electrical remodeling may increase the likelihood of developing regional conduction block and