Targeting Common Sites of Atrial Tachycardia Increases Ablative Success

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Successful ablation of focal atrial tachycardia (AT) requires knowledge of common sites, the ability to identify and localize P waves, use of multipolar mapping, and the knowledge to map and visualize neighboring structures, recognize intracardiac signals of interest, and consider alternative approaches. Anita Wokhlu, MD, HealthEast Heart Care, Minneapolis-St. Paul, Minnesota, USA, discussed how to maximize ablative success.

Dr. Wokhlu reported that the right atrium is the most common anatomic site for AT. Others include the crista terminalis and the tricuspid annulus. Although the majority of left atrial tachycardia (LAT) originate around the ostia of the pulmonary veins, the mitral annulus is an unusual but important site of origin for focal AT, with a propensity to be localized to the superior aspect [Kistler PM et al. *J Am Coll Cardiol* 2003]. Atrial appendage sites are associated with a high incidence of incessant tachycardia (84%) and left ventricular (LV) dysfunction (42%) [Medi C et al. *J Am Coll Cardiol* 2009].

In a 2006 report, Kistler et al. [*J Am Coll Cardiol* 2006] performed a detailed analysis of the P-wave morphology in AT and developed a detailed algorithm characterizing the likely location of a tachycardia associated with a P wave of unknown origin. Highly specific and sensitive, the P-wave algorithm correctly identified the tachycardia origin in 93% of cases.

While multipolar mapping hones the region of interest, risks can include choice of the inappropriate sinus and phrenic injury. Options, according to Dr. Wokhlu, include high-output phrenic pacing (10 to 20 mA; pulse 2 ms); cryomapping and ablation; and insertion of an epicardial balloon to displace the phrenic nerve [Lee JC et al. *Heart Rhythm* 2009].

Dr. Wokhlus noted that atrial appendage tachycardia ablations can be challenging due to complex relational anatomy, variant anatomy (especially left), pectinates interspersed within tissue, low flow, and a perforation risk that may limit endocardial approach. Her presentation also covered mapping and visualizing neighboring structures (eg, Bachman's bundle, fossa ovalis, coronary sinus ostia), how to recognize intracardiac signals of interest, and alternative ablative approaches (eg, epicardial ablation after endocardial fails, lasso-guided ablation).

MANAGING AND PREVENTING POST AF ABLATION TACHYCARDIA

Dipen Shah, MD, Hopital Cantonal de Geneve, Geneva, Switzerland, presented several studies on AT after ablation. The first report identified discrete isthmuses critical to LATs that may simplify their elimination by catheter ablation [Shah D et al. *J Cardiovasc Electrophysiol* 2006].

Fifteen patients (all male, 56±8 years) with 15 reentrant LATs following atrial fibrillation (AF) ablation underwent activation and entrainment mapping. Eleven patients (11 LATs) had a single localized site with low amplitude (0.16±0.05 mV), fractionated long duration (131±23 msec) electrograms coinciding with an isoelectric interval of 106±24 msec between flutter waves on all 12 electrocardiogram (ECG) leads [Shah D et al. *J Cardiovasc Electrophysiol* 2006]. Three-dimensional mapping and entrainment revealed this site to be a narrow, markedly slow-conducting isthmus adjacent to ablated left (n=8) or right (n=3) pulmonary vein ostia, and critical to nine small diameter (15±3 mm) and two large diameter (49±2 mm) circuits.

One radiofrequency (RF) application on this isthmus eliminated LAT in all 11 patients. Four patients (4 LATs) with large circuits around the mitral annulus and/or PV ostia lacked isoelectric ECG intervals and slow-conducting isthmuses and required multiple RF applications across anatomically wide, rapidly conducting isthmuses [Shah D et al. *J Cardiovasc Electrophysiol* 2006].

Focally ablatable narrow isthmuses of slow conduction are critical for the majority of reentrant LATs occurring after ablation for AF [Shah D et al. *J Cardiovasc Electrophysiol* 2006]. The role and presence of these isthmuses can be anticipated by observing significant isoelectric intervals between the flutter waves on all 12-surface ECG leads. Distinctive electrophysiological characteristics allow their identification and elimination by simple RF ablation.

The other study dealt with AT after linear lesions, with Prof. Shah covering atrial reentry tachycardia after LA linear lesions [Shah D et al. HRS 2010], pseudo-atypical flutter, perimitral LA flutter with a discrete gap, and small reentry in the mitral isthmus.

Prof. Shah reported that nearly a quarter of all patients develop AT/atrial flutter (AFL) after pulmonary vein isolation plus linear lesions, with LA dilation perhaps disposing to the development of AT/AFL. Development of AT/AFL is not associated with conduction block across linear lesions at index ablation and occurs despite complete conduction block in many patients. However, sites of successful ablation are most commonly on or beside linear lesions.

POSTSURGICAL ATRIAL TACHYCARDIA: ANATOMIC DEFECTS AND SURGICAL APPROACHES

Li-Wei Lo, MD, and Shih-Ann Chen, MD, National Yang-Min University, Taiwan, China, discussed the high prevalence of AT in patients with congenital heart

disease and the diversity of ATs that can develop after open heart surgery (ie, cavotricuspid isthmus [CTI]-dependent AFL, non-CTI-dependent AFL [intra-atrial reentrant tachycardia], and focal AT. They said that AT may develop in as many as 25% of patients who are difficult to manage with antiarrhythmic drug therapy.

Mechanisms of postsurgical AT include anatomical characteristics that promote macro-reentry, subtle changes in the electrophysiological substrate of the atrial myocardium (eg, cellular hypertrophy, fibrosis, and co-occurrence of sinus node dysfunction).

The types of AT and congenital heart disease include isthmus-dependent AFL and intra-atrial reentrant flutter. AF in congenital heart disease is relatively low, seen in end-stage heart disease and/or left-sided cardiac lesions. Types of surgical incisions are shown in Table 1. The ablation site should be selected according to the location of the incisional scar and the stability of the catheter [Nakao M et al. *Circ J* 2005].

Other subjects covered during the presentation were postsurgical mapping techniques of AT, including entrainment, voltage and activation mappings, entrainment versus 3D activation mapping, and case presentations of postsurgical AT.

Professors Lo and Chen concluded that electrophysiologists need to recognize late arrhythmia after cardiac surgery. In patients with surgically corrected congenital heart disease, AT is most often caused by macroreentrant mechanisms. They recommend a strategy of careful, anatomically-based mapping of the reentrant circuit and validation of acute conduction block.

Table 1. Surgical Incisions in Heart Disease

Atrial Incision	**Operaton**
RA appendage cannulation	CABG Aortic valve CABG and aortic valve Pulmonary valve
RA free wall atriotomy	Retrograde cardioplegia (CABG and/or aortic valve) ASD VSD Tricuspid valve
Transseptal LA atriotomy	Mitral valve Mitral valve and CABG Mitral valve and aortic valve Mitral valve and tricuspid valve LA myxoma
Direct LA atriotomy	Mitral valve Mitral valve and aortic valve

ASD=atrial septal defect; CABG=coronary artery bypass graft; LA=left atrial; RA=right atrial; VSD=ventricular septal defect.

Together, these presentations summarize the common sites of focal AT; how to prevent post-AF ablation tachycardias as well as atrial tachycardia and flutter after linear LA lesions; and the importance of anatomic defect and surgical approach in the management of postsurgical AT.