CATHETER ABLATION OF TYPICAL ATRIAL FLUTTER

Allesandro De Bortoli and Peter M. Schuster,
Institutt for indremedisin, Haukeland universitetsykehus, Bergen.

Typical atrial flutter (AFL) (also known as type I, common, right-sided) is a supra-ventricular tachycardia with an atrial rate between 240 to 340 beats per minute and typically a 2:1 to 4:1 rate of heart block. An American study estimated its incidence to 88 cases per 100.000 inhabitants per year. Male sex, age over 70 years, co-existing presence of cardiac disease and obstructive lung conditions are well acknowledged risk-factors (1). Depending mostly to the degree of heart block, AFL can go unnoticed or lead to symptoms such as palpita-tions, dizziness, dyspnoea and reduced tolerance to physical exercise. Typical AFL classi-cally depends on a re-entrant circuit located in the right atrium with a critical pas-sage in the cavo-tricuspidal isthmus, a fibrous area com-prised between the entrance of the inferior caval vein and the posterior aspect of the tricuspid annulus. According to the direction of the re-entry, typical AFL can manifest in a counterclockwise (CCW) or in a far less common clockwise (CW) fashion. Typical AFL can be recognized in the 12-lead ECG for the "sawtooth pat-tern" of the flutter waves with negative polarity in the inferior leads (II, III and aVF) (figure 1) whereas CW AFL shows a positive polarity (figure 2). An additional criteria is to measure the ratio between the voltage in aVF and lead I: aVF/lead I ratio of the flutter waves >2.5 is diagnostic for CCW (2). The diagnosis of CCW AFL with 2:1 AV conduction can be challenging because the flutter waves are buried in the QRS and in the T waves. Increasing the degree of block with vagal manoeuvres or

Figure 1 – Surface ECG of typical counterclockwise (CCW) flutter. To be noted the negative polarity of flutter waves in the inferior leads and the difference in amplitude of flutter waves between lead I (almost isoelec-tric) and aVF.
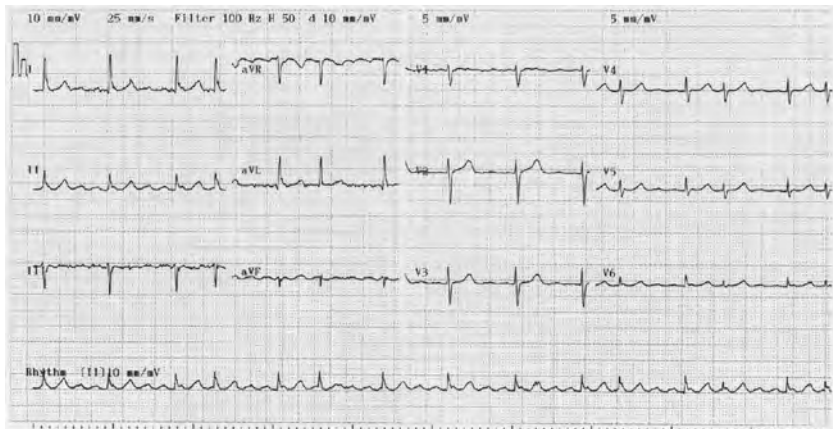
Figure 2 – Surface ECG of typical clockwiseflutter. To be noted the positive polarity of flutter waves in the inferior leads.

adenosine facilitates the diagnosis. History of previous heart surgery or catheter ablation in the atria increases the likelihood of atypical scar-related AFL, even despite typical AFL ECG findings. Intracardiac recording of typical AFL leaves no doubts on the diagnosis showing the classic pattern with CCW activation (figure 3).

Atrial fibrillation (AF) is closely linked to AFL. A large study evaluated retrospectively 750.000 patients over 65 years of age that were diagnosed with either AF or AFL. Among the AFL group, after 6.5 years of follow-up more than 50% of the patients developed also clinical AF. When compared to a control group, the risk of developing stroke during the follow-up was increased among those with isolated AFL compared to the control group (RR 1.406) and even more among those with AF or combined AF/AFL (RR 1.642) (3).

The interrelation between AF and AFL may also be pathogenetical. Based on previous animal and human studies Waldo hypothesizes that virtually all the episodes of AFL are initiated by a short burst of AF (4).

Typical AFL is commonly a paroxysmal arrhythmia, usually self-terminating within 48 hours. In rare cases it can become persistent over weeks or months requiring pharmacological or electric cardioversion. The rapid contraction of the atria determines poor flow in the left atrial appendage (LAA) that can lead to thrombus formation. Despite the fact that several studies showed lower incidence of thrombi in the LAA among pure AFL patients (5), long-
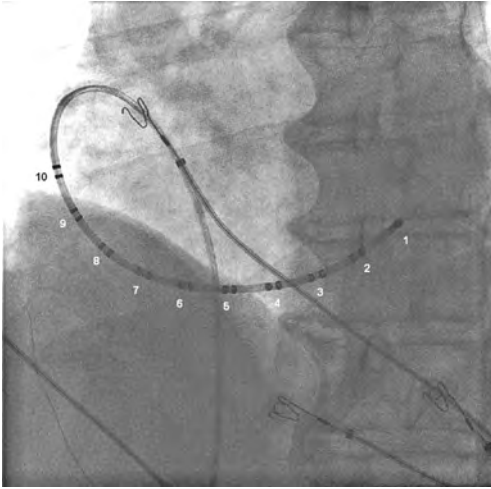

Figure 3 – Intracardiac recording of a typical CCW AFL. As noted by the arrows, the atrial activation follows a proximal to distal CCW direction from HALO10 to HALO1. The atrial cycle length is 214 msec. The three upper electrograms are lead I, lead II and V1 respectively.

Figure 4 – Coronary sinus catheter (LAO 30°). As described in the text the catheter is lopped in the right atrium, around the tricuspid valve with the distal end in the coronary sinus. The electrode 1 is the most distal and 10 is the most proximal. The septum is approximately located at electrode 4 level.

term anticoagulation management should be regarded as AF patients.

Pharmacological treatment of AFL can aim either maintenance of sinus rhythm (SR) (flecainide, amiodarone) or control of ventricular rate (calcium channel blockers, digitalis, beta-blockers). Due to the variable response to pharmacological therapy, catheter ablation is increasingly performed in these patients. At our facility we insert through femoral vein access, a 20-poles diagnostic catheter in the right atrium looped around the tricuspid valve and with the distal end in the coronary sinus (CS) (figure 4). An irrigated ablation catheter is then inserted with the same modality in the right atrium. Radiofrequency energy is applied on the cavo-tricuspidal isthmus to create a line of block in the re-entrant circuit.

The procedural endpoint is the creation of bidirectional conduction block. This can be assessed by pacing manoeuvres (to be performed in SR). Pacing from the distal CS normally shows a progressive activation of the proximal electrodes in a regular linear fashion (figure 5). The cavo-tricuspidal isthmus a critical passage for the conduction of the impulse. When a block is obtained the impulse will travel from the distal CS until the line of block, then it will activate the septum and finally the electrodes of the 20-poles catheter that is proximal to the block (figure 6).

With the introduction of open-irrigated ablation catheter that allows greater lesion depth, a typical AFL procedure takes between 60 and 90 minutes and offers high success and low complication rate. A previous study reported a 90% acute success of the procedure. Complications, such as complete AV block and right coronary artery damage are very rare, with an incidence below 2% (6).

Despite these positive results, a considerable amount of patients remains symptomatic following AFL ablation; the reason for this is to be attributed mainly to the co-morbidity with AF. As previously noted, experimental studies show that AF tends to organize in typical AFL. In presence of combined AF/AFL it seems that the elimination of the AFL substrate rather leads to more prolonged AF episodes (7).

An evaluation of 62 patients that received exclusively AFL ablation at our centre between 2009 and 2010 reveals that 41 (66%) had documented AF before the procedure and 29 (46%) after the procedure. Among those with AF recurrences
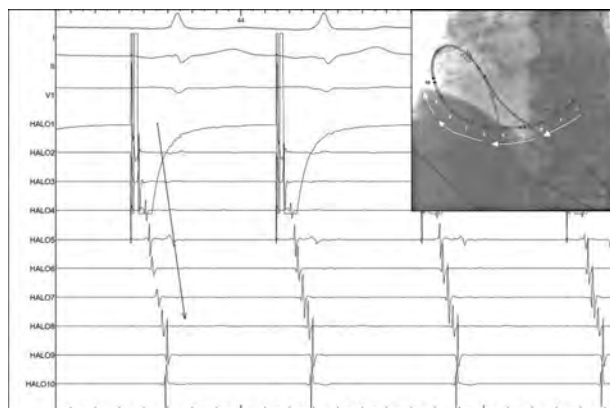

Figure 5 – Normal CS activation. In absence of conduction block over the cavo-tricuspidal isthmus, pacing from the distal CS leads to progressive activation, distally to proximally, of all the electrodes (see arrows). In the upper right corner is given a visual explanation of the activation sequence.
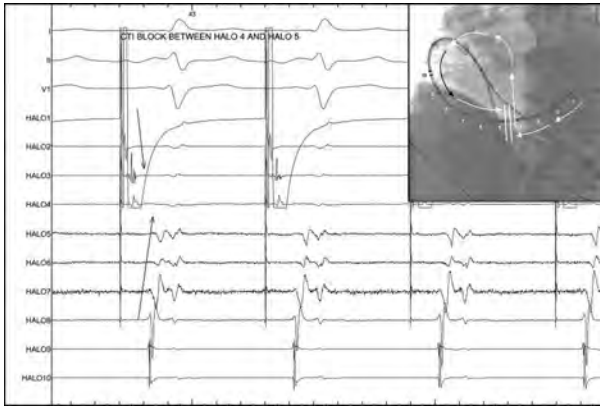
Figure 6 – Cavo-tricuspidal isthmus block. In presence of conduction block over the cavo-tricuspidal isthmus pacing from the distal CS leads to progressive activation that is interrupted in HALO4. The impulse then activates the septum and finally reaches the proximal part of the HALO catheter from the other direction (see arrows). In the upper right corner is given a visual explanation of the activation sequence.

after the AFL ablation, 18 (62%) were referred and underwent AF ablation in the following 2 years. Only 5 patients (8%) had recurrent typical AFL and had to undergo a repeat ablation of the cavo-tricuspidal isthmus.

The combined presence of AF and AFL in suitable patients may receive indication for a combined ablation that targets both the arrhythmias. We would recommend an isolated AFL procedure only in the presence of lone AFL (rare) or in those patients that, despite AF documentation, are exclusively symptomatic for AFL.

Typical AFL ablation is a fast, safe and effective procedure to eliminate the substrate for this arrhythmia. The presence of co-existing AF and its relationship to the patients' symptoms must be investigated prior to referral for ablation.

References

1. Granada J, Uribe W, Chyou PH, Maassen K, Vierkant R, Smith PN, Hayes J, Eaker E, Vidaillet H. Incidence and predictors of atrial flutter in the general population. J Am Coll Cardiol. 2000;36:2242-6.

2. Lai LP, Lin JL, Lin LJ, Chen WJ, Ho YL, Tseng YZ, Chen CH, Lee YT, Lien WP, Huang SK. New electrocardiographic criteria for the differentiation between counterclockwise and clockwise atrial flutter: correlation with electrophysiological study and radiofrequency catheter ablation. Heart. 1998;80:80-5.

3. Biblo LA, Yuan Z, Quan KJ, Mackall JA, Rimm AA. Risk of stroke in patients with atrial flutter. Am J Cardiol. 2001;87:346-9, A9.

4. Waldo AL, Feld GK. Inter-relationships of atrial fibrillation and atrial flutter mechanisms and clinical implications. J Am Coll Cardiol. 2008;51:779-86

5. Schmidt H, von der Recke G, Illien S, Lewalter T, Schimpf R, Wolpert C, Becher H, Lüderitz B, Omran H. Prevalence of left atrial chamber and appendage thrombi in patients with atrial flutter and its clinical significance. J Am Coll Cardiol. 2001;38:778-84.

6. Calkins H, Canby R, Weiss R, Taylor G, Wells P, Chinitz L, Milstein S, Compton S, Oleson K, Sherfesee L, Onufer J; 100W Atakr II Investigator Group.

7. Results of catheter ablation of typical atrial flutter. Am J Cardiol. 2004;94:437-42.

8. Gilligan DM, Zakaib JS, Fuller I, Shepard RK, Dan D, Wood MA, Clemo HF, Stambler BS, Ellenbogen KA. Long-term outcome of patients after successful radiofrequency ablation for typical atrial flutter. Pacing Clin Electrophysiol. 2003;26:53-8.