Atrial Flutter: Mechanisms, Clinical Features and Management

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In the last decades, the management of cardiac arrhythmias has known remarkable advances. The advent of new therapeutical options made it possible to target arrhythmogenic substrates. A large amount of knowledge about tachyarrhythmia mechanisms had previously accumulated, that opened the way to the curative approaches. Moreover the availability of potentially effective tools also contributed to stimulate research and promote a better understanding of rhythm disorders. Nowadays, catheter ablation is routinely performed in electrophysiology laboratories. In drug-refractory tachyarrhythmic patients, the recourse to ablative methods has become a well-accepted alternative option. Thus, the management of many arrhythmias is conducted in a quite different way from that used in the past. For patients with the Wolff- Parkinson-White and those with intranodal reentrant tachycardias, the field of antiarrhythmic agents has markedly decreased. Due to high success rates and an optimal benefit/ratio, radiofrequency current ablation tends to be considered at increasingly earlier stages and even as first intent approach. The treatment of atrial flutter has undergone similar changes with some delay. The first attempts at ablation were mitigate due to uncertainties regarding the appropriate target and also occasional severe adverse effects like atrioventricular block caused by high energy shocks (1). Subsequently the critical zone for atrial flutter maintenance was better defined. In the mean time, the generalization of radiofrequency current techniques led to significantly increase ablation efficacy and to minimize the risks (2). Thus the same trend towards a preferential use of nonpharmacological treatment also involved atrial flutter. This was reinforced by the notion of poor efficacy of antiarrhythmic drugs in this setting. Interestingly, the therapeutic progress in the management of atrial flutter was accompanied by further elucidation of the underlying mechanisms.

MECHANISMS OF ATRIAL FLUTTER

The electrical phenomena underlying atrial flutter were first studied in experimental models. Several decades ago, Lewis had demonstrated the role of a single intraatrial circus movement in canine surgically induced atrial flutter. The creation of lesions impairing conduction at critical sites within the right atrium provided the basis for the initiation of reentry. More recently Allessie, using left atrium preparations in rabbits, stressed the possibility of stable reentry phenomena developed around functional obstacles, this being combined electrocardiographically to flutter-like rapid atrial rhythms. However the advent of endocardial mapping techniques in humans was decisive in elucidating the electrophysiologic process involved in the clinical arrhythmia. Puech's pioneer work in the late fifties set the foundations of our current knowledge of atrial flutter (3). Subsequently Cosio's studies opened the way to electrical ablation of this disorder (4).

The basic mechanism of atrial flutter is a macroreentrant process whose revolution time determines the atrial rate. There is a continuous electrical activity all along the atrial cycle length. The duration of the entire circus movement ranges from 200 from 350 ms. An excitable gap lies between the head and the tail of the depolarization wave. For atrial reentry to occur, the role of anatomical obstacles whatever the type (atrial structures or fibrosis) is essential. The atrioventricular conduction system usually blocks the rapid atrial impulses, thus decreasing the ventricular response. This may help improve the hemodynamic tolerance. Coexisting atrial fibrillation is of common occurrence in atrial flutter patients. The transformation into atrial fibrillation following high atrial rates can always take place triggered by elevated sympathetic tone. Conversely bursts of atrial fibrillation have been shown to promote atrial flutter induction.

Most often Atrial Flutter Is of The Common Type

The relevant electrophysiologic process is then a circular movement confined to the right atrium (5). The left atrium is passively depolarized and is not a necessary limb of the circuit. The pathological substrate comprises the septum and the right atrial free wall as the main components. Both are connected by the isthmus lying between the orifice of the inferior cava vein and the tricuspid annulus. The septal end of the isthmus includes the triangle of Koch bordered by the coronary sinus ostium, the tendon of Todaro and the tricuspid valve. The triangle of Koch is an area of slow conduction (6). Anisotropy due to propagation perpendicular to the long axis of the fibers is invoked in the genesis of slow conduction within the postero-inferior part of the septum. The maintenance of reentry during common atrial flutter is made possible by the presence of barriers protecting the circulating wave (7). In this view the crista terminalis plays an important role. Any transverse depolarization emerging from the main reentrant process is blocked against this structure and is then prevented from colliding with the head of the flutter wave. Again anisotropy is likely to be involved in this phenomenon. In the lower part of the right atrium, the Eustachian ridge intervenes as an additional barrier. Anyway the architecture of the right atrium further reinforces the possibility of macroreentry. In this view, the role played by the cava veins orifices, the tricuspid annulus, the coronary sinus ostium and the fossa ovalis is to be stressed. Usually common atrial flutter is counterclockwise. The wavefront proceeds along the septum towards the roof of the right atrium, then changes its direction to invade the pectinae muscle cephalocaudally and returns to the lower septum through the cavotricuspid isthmus. Clockwise atrial flutter exhibits an inverted pattern. The septal depolarization is cephalocaudal, and is followed by a reverse propagation within the right atrial free wall

Scar Flutter Is a Variant Form Associated with an History of Congenital Heart Disease

These subjects have undergone surgery for cardiac abnormalities (mainly atrial septal defect repair, but also Fontan, Mustard or Senning procedures for complex congenital diseases). The post-incisional right atrial scar provides a substrate for the subsequent development of a reentrant mechanism (8). Right atrial dilatation may favor atrial flutter maintenance. Endocardial mapping in humans has confirmed the possibility of circus movement around atrial scars. However common atrial flutter can also occur in this setting. Careful electrophysiologic studies are required to differentiate true scar flutter from the isthmus-dependent common forms.

Atrial Flutter Can Also Involve The Left Atrium (9)

Most patients have a structural heart disease with marked left atrial dilatation. Usually there is a single loop reentrant circuit, the impulse rotating around the mitral annulus in a counterclockwise or clockwise fashion. Figure 1.- 8 reentrant circuits have been described. In this situation, from a common channel, 2 loops are rotating in opposite directions. Rarely the reentry circuit is small, with the rest of the left atrium passively activated. Use of 3D computerized mapping has brought a significant contribution to the understanding of left atrial flutter and paved the way to ablation therapy.

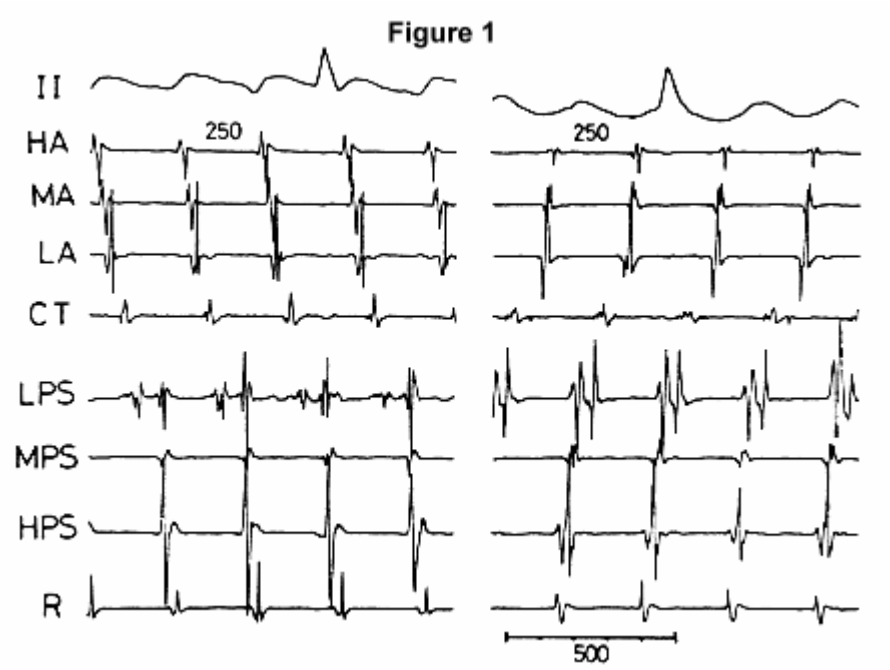

Figure 1 : Endocardial mapping data during counterclockwise (left panel) and clockwise atrial flutter (right panel). Lead II is simultaneously represented. HA, MA and LA correspond to electrograms recorded from the high, middle and lower part of the right atrium anterior wall. CT is the electrical activity derived from the cavotricuspid isthmus. LPS, MPS, HPS and R are endocardial leads related to the local activity of the lower, middle, high posteroseptal wall and of the atrial roof. Clearly the impulse proceed cephalocaudally along the right atrial free wall and then in a caudocephalic fashion during common counterclockwise flutter. The direction of rotation is reversed in clockwise flutter (from Cosio, ref 12)

Type 2 Atrial Flutter Is a Particular Entity which Is To Be Separated from the Other Forms

The atrial rate is high and can exceed 350 beats per minute. Pacing techniques are unable to entrain the arrhythmia, a response which does not fit a macroreentrant atrial process (see further). Use of atrial mapping supports the role of localized circus movement coexisting with atrial fibrillation areas. Type 2 atrial flutter is likely to represent a transitional disorder preceding transformation into sustained atrial fibrillation.

CLINICAL FEATURES

Clinically Atrial Flutter May Give Rise To a Wide Range Of Symptoms

Surprisingly the perception of palpitations is infrequent. The most encountered signs are fatigue, exertional dyspnea and general discomfort. However this intolerance is often mild. Certain forms are even asymptomatic. Conversely the occurrence of atrial flutter in severe cardiac patients may be accompanied by congestive heart failure and pulmonary edema. Syncopal attacks are exceptional in relation to very rapid rates. Aetiologies include valvular, myocardial and coronary heart diseases. Acute causes like infection or pulmonary disease can trigger atrial flutter attacks. In a significant number of patients, no patent abnormality is detected. However some of them exhibit overweight and/or hypertension. Furthermore echocardiography may unveil left atrial dilatation, whose significance remains unsettled. Atrial flutter is occasionally observed in the setting of sinus bradycardia. In the bradycardia -tachycardia syndrome, atrial flutter or fibrillation alternates with slow sinus rhythm or sinus pauses. In these patients, symptoms combine dizziness, syncope and palpitations.

Short-lasting episodes of atrial flutter are rare. Usually the arrhythmia is sustained and has a propensity to self-perpetuation. After a first attack, atrial flutter tends to recur. Time to recurrence markedly varies according to individuals and the risk factors for relapses are ill-known. During the clinical course, atrial flutter patients may also develop atrial fibrillation. Both arrhythmias are likely to share a common substrate. The transformation of atrial flutter into fibrillation and vice-versa is a well accepted notion (10).

The Electrocardiographic Features Characterize Atrial Flutter

The atrial rates range from 200 to 350 beats per minute. The atrial activity results in a continuous undulation of the baseline seen in the peripheral leads, while discrete P waves can be recorded in the precordial leads. This sawtooth aspect is variable according to atrial flutter mechanisms and the underlying pathologic substrates. Common counterclockwise atrial flutter is associated with biphasic P waves in leads II, III and aVF. The initial negative component corresponds to the upward progression of the depolarizing wavefront within the atrial septum. The subsequent activation of the atrial free wall causes a positive overshoot of the atrial waves in the inferior leads. Then is apparent a downsloping plateau simultaneous with the invasion of the cavo-tricuspid isthmus. At the end of this process a new atrial activity appears indicating the reinitiation of the circus movement. In clockwise atrial flutter, the bipolar leads show a similar undulating aspect of the atrial rhythm. However the P waves look predominantly positive. Anyway common atrial flutter can also be combined to less characteristic patterns and even to atrial tachycardia-like morphologies. Similarly scar flutter has variable electrocardiographic features, the diagnosis being supported by the context. For left atrial flutter, the coexistence in sinus rhythm of marked P waves abnormalities evoking interatrial block may be of help to suspect this rare form. In so-called type 2 atrial flutter, the very fast atrial activity occasionally shows changes in form and amplitude suggesting alternation with atrial fibrillation.

In response to rapid atrial rhythm, the atrio-ventricular conduction system results in blockade of atrial impulses. The usual atrial rate is 300 beats per minute. Two to one atrial flutter is of common occurrence associated with a ventricular rate of 150 beats per minute. Spontaneously or following drug therapy, there may be higher degrees of atrioventricular block causing 3:1, 4:1 atrial flutter or forms with variable ventricular response. Atrial flutter can also coexist with complete heart block and escape ventricular rhythm. Carotid sinus massage may transiently aggravate the atrioventricular block The resulting decrease in ventricular rate is followed after a few seconds by the resumption of the initial frequency. Furthermore this maneuver sometimes helps unmask the flutter waves in electrocardiographically difficult cases. Rarely atrial flutter shows a 1:1 ventricular response combined to heart rates ranging from 200 to 300 beats per minute. This may involve children due to the peculiarities of their atrioventricular conduction system. Adults on antiarrhythmic drug therapy occasionally develop 1:1 atrial flutter. Such paradoxic response is well-known after administration of class 1 antiarrhythmics in patients with atrial fibrillation. Although being able to prevent the fibrillatory process, the effect of these agents on the atrial electrophysiology may still let emerge slow atrial flutter. Decrease in atrial rate then permits the resumption of 1:1 atrioventricular conduction.

As a supraventricular tachycardia atrial flutter usually exhibits narrow QRS complexes. However fast rate and irregularity predispose to ventricular aberration. Myocardial impairment also contributes to rate-dependent widening of the ventricular beats. In this situation atrial flutter attacks may mimic ventricular tachycardia. Under electrocardiographic control, vagal maneuvers or adenosine administration can be used to identify the nature of the arrhythmia. Coexistence of ventricular tachycardia and atrial flutter is also possible. Evidence for atrioventricular dissociation is to be found to support the diagnosis of dual tachycardia.

The electrocardiogram in sinus rhythm often exhibits notched P waves, mainly seen in lead II, whose duration may be of 0.12 sec or more. This feature indicates some degree of intraatrial conduction disorder, an abnormality supposedly underlying the genesis of atrial flutter. In rare cases, the P waves are comprised of two distinct components showing in some leads opposite polarities. The total duration is markedly prolonged. The latter aspect is viewed as reflecting a conduction block between both atria. Interatrial block appears to be associated with forms of atrial flutter involving the left atrium.

MANAGEMENT

Asymptomatic forms of atrial flutter do not necessarily impose any treatment, especially if the spontaneous ventricular response is within normal range. In case of excessive heart rate, pharmacologic control is recommended in order to preserve myocardial function. In this view atrioventricular nodal

depressant drugs like digitalis, beta-blockers or verapamil can be used. Prescribing antithrombotic agents is debated since the embolic risk in this setting remains unproven. However the coexistence of atrial fibrillation attacks may lead to give aspirin or oral anticoagulants.

Recent Onset Atrial Flutter with Clinical Intolerance Requires Medical Management

Rarely immediate DC countershock is mandatory because of cardiac decompensation. In the usual forms, significant improvement can be achieved through rate control. Digitalis is preferably used for this purpose. Chemical cardioversion using class 3 antiarrhythmic agents (amiodarone, ibutilide or dofetilide) has been proposed for very recent episodes of less than 24 hours. Conversion rates of 50 to 70 per cent are reported following drug therapy (11). For most cases, attempts at restoring sinus rhythm are performed after rate control.

Transesophageal pacing is an option. The procedure consists of stimulating the atria with a catheter introduced via the esophageal route. The pacing rate is slightly higher than the spontaneous atrial rate. To capture the atria, high intensities and increased impulse duration may be required. However discomfort and pain often accompany transesophageal pacing. Right atrial pacing through an endocardial electrode-catheter has a better tolerance but requires a catheterization. Conversion rates are relatively high. Resumption of sinus rhythm can be preceded by transient transformation into atrial fibrillation. Finally electrical countershock remains the privileged alternative. Efficacy is remarkable with success rates approximating 100 per cent. Transthoracic delivery of 50 joules is usually sufficient to cardiovert the patients. Prolonged oral anticoagulation therapy is not needed before cardioversion. However the recourse to anticoagulants is licit in patients with advanced heart disease or an history of atrial fibrillation. In case of uncertainty, transesophageal echocardiography can be performed before cardioversion.

Drug Prophylaxis of Atrial Flutter Lacks Firm Bases

There is no evidence for the preventive efficacy of any antiarrhythmic agent. The class 1 drugs are not recommended because they may occasionally favor the emergence of 1:1 atrial flutter. Concomitant prescription of beta-blockers or verapamil aims at minimizing such a risk. Class 3 antiarrhythmic effect seems to be more appropriate in this setting. Amiodarone is commonly used for atrial flutter prevention. Actually amiodarone possesses all classes of antiarrhythmic action. Besides increasing atrial refractory periods, the drug also depresses atrioventricular nodal conduction, which is a desirable response to avert 1:1 atrial flutter. Efficacy of amiodarone is likely although unproven. Similarly sotalol, a beta-blocking agent with class 3 effects, is utilized in atrial flutter patients. Thus far there are no convincing data to support this choice. Doubts are reinforced due to the poor efficacy of sotalol for converting patients with ongoing atrial flutter. Drug management of atrial flutter is purely empirical. However antiarrhythmics are still used as first intent option in patients with recurrent and/or poorly tolerated attacks of atrial flutter. A first well tolerated episode does not necessarily require prophylaxis, especially if the role of acute factors is suspected.

Nonpharmacologic Management Remains an Alternative Option in Drug-Refractory Patients

Cardiac pacing can be proposed in case of sinus node dysfunction. Antibradycardia pacing has been shown to reduce the incidence of atrial arrhythmia episodes in those subjects. Outside sinus node dysfunction, use of pacemakers may address the control of atrial flutter attacks. In this view devices with atrial lead are able, following arrhythmia detection, to deliver bursts of electrical impulses for cardioversion purposes. However the recourse to this technique in atrial flutter patients has remained marginal. In subjects with repeated, disabling and ill-controlled attacks, radiofrequency catheter ablation of the atrioventricular node-His pathway aims at eliminating excessive frequencies and equalizing the ventricular response. Concomitant insertion of a DDD pacemaker with automatic mode switching significantly contributes to the functional improvement. Currently atrioventricular nodal ablation tends to be replaced by curative interventional means.

Ablation of the atrial tissue has been a major step in the management of atrial flutter patients (12). In the common forms, for reentry to occur, the impulse has to proceed along the atrial isthmus lying between

the inferior cava vein orifice and the tricuspid annulus. This relatively narrow path represents an ideal target for ablation. Evidence for the role of the cavo-tricuspid isthmus can be provided by entrainment techniques (13). Following isthmus stimulation, the local post-pacing interval is to equal atrial flutter cycle length, which indicates that the pacing site belongs to the circuit. Endocardial mapping is helpful to delineate the reentry limbs and the sense of rotation. Radiofrequency current can be applied during atrial flutter or in sinus rhythm. One minute applications are delivered sequentially from the tricuspid ring up to the inferior cava vein, the catheter being withdrawn step by step. Local temperatures of 50 to 60 degrees are to be achieved. Perfused catheters may avoid excess temperature and high impedance due to the formation of coagulum at the catheter tip. The objective of ablation is to create a line of block within the isthmus. The result can be assessed by stimulating the coronary sinus area and the lower part of the right atrial free wall successively. The sequence of activation is then compared to that seen before ablation. Evidence for the diversion of the wavefront due to the isthmus block is sought. In this view, appraisal of local electrogram polarities before and after ablation is helpful. The detection of split potentials along the line of ablation is also an accurate marker of complete isthmus block. Acute success rate nears 90 per cent (14). Recurrences of atrial flutter are reported in 20 per cent of the cases and can be controlled by repeat ablation. Subsequent emergence of atrial fibrillation may impair the clinical course in 10 to 20 per cent of patients leading to prescribe antiarrhythmic drug therapy (15). Conversely the role of isthmus ablation in eliminating concomitant atrial fibrillation in some patients is uncertain.

In scar flutter, efficacy of ablation is dependent on careful atrial mapping which aims to define the contours of reentry. Usually the circus movement is evolving around the scar. The ablation line is then drawn from the scar to the tricuspid valve (16). Ablation targeting the isthmus may be required in occasional isthmus dependent forms. Left atrial flutter is even more complex with multiple potential mechanisms. Use of 3D mapping may be needed to accurately delineate the circuits within the left atrium and locate the ablation areas. In these subsets, radiofrequency ablation is still efficacious (9). However the reported series are limited.

CONCLUSION

Atrial flutter is a familiar entity for the cardiologist. The benign nature of this arrhythmia is acknowledged. However, like any tachycardia, atrial flutter may aggravate the status of cardiac patients. Thromboembolic risk is uncertain, but deserves attention in patients with coexisting episodes of atrial fibrillation. Currently the mechanisms of atrial flutter appear to be elucidated. A macroreentrant process within the atria underlies the tachyarrhythmia. For the most common forms, the circus movement is restricted to the right atrium, the rotation being counterclockwise or less often clockwise. If acute conversion, mainly using DC shocks, is highly effective, effect of drug prophylaxis is not established. In case of recurrence, rate control may be envisaged. However, in symptomatic patients, the recourse to ablation is increasingly considered. Common atrial flutter can be cured by radiofrequency current applications resulting in a line of block across the cavotricuspid isthmus. Long term efficacy is remarkable. However, the clinical course may be impaired by the occurrence of atrial fibrillation. Isthmus ablation might well evolve in the future towards a first intent use in patients with recurrent atrial flutter.

REFERENCES

1. Saoudi N, Atallah G, Kirkorian G et al. Catheter ablation of the atrial myocardium in human type I atrial flutter. Circulation 1991;81:762-71.

2. Feld GK, Fleck RP, Chen P-S et al. Radiofrequency catheter ablation for the treatment of human type 1 atrial flutter. Identification of a critical zone in the reentrant circuit by endocardial mapping techniques. Circulation 1992;86:1233-40.

3. Puech P. L'activité électrique auriculaire normale et pathologique. Masson & Cie, Paris, France, 1956.

4. Cosio FG, Arribas F, Palacios J et al. Fragmented electrograms and continuous electrical activity in atrial flutter. Am J Cardiol 1986; 57:1309-14.

5. Puech P, Latour H, Grolleau R. Le flutter et ses limites. Arch Mal Cœur 1970; 63:116-44.

6. Olshansky B, Okumura K, Hess PG et al. Demonstration of an area of slow conduction in human atrial flutter.

J Am Coll Cardiol 1990; 16:1639-48.

7. Cosio FG, Arribas F, Barbero JM et al. Validation of double spike electrograms as markers of conduction delay or block in atrial flutter. Am J Cardiol 1988; 61:775-80.

8. Triedman JK, Saul JP, Weindling SN et al. Radiofrequency ablation of intra-atrial reentrant tachycardia after surgical palliation of congenital heart disease. Circulation 1995; 91:707-14.

9. Jais P, Shah DC, Haissaguerre M et al. Mapping and ablation of left atrial flutters. Circulation 2000; 101:2928-34.

10. Waldo A, Cooper TB. Spontaneous onset of type I atrial flutter in patients. J Am Coll Cardiol 1996; 28:707-12.

11. Ellenbogen KA, Stambler BS, Wood MA et al. Efficacy of intravenous ibutilide for rapid termination of atrial fibrillation and atrial flutter : a dose-response study. J Am Coll Cardiol 1996; 28:130-6.

12. Cosio FG, Arribas F, Lopez-Gil M et al. Atrial flutter mapping and ablation II. Radiofrequency ablation of atrial flutter circuits. PACE 1996; 19:965-75.

13. Cosio FG, Lopez-Gil M, Arribas F et al. The mechanisms of entrainment of human common flutter studied with multiple endocardial recordings. Circulation 1994; 89:2117-26.

14. Fischer B, Jais P, Shah D et al. Radiofrequency catheter ablation of common atrial flutter in 200 patients. J Cardiovasc Electrophysiol 1996; 7:1225-33.

15. Fischer B, Haissaguerre M, Cauchemez B et al. Frequency of recurrent atrial fibrillation after successful radiofrequency catheter ablation of common atrial flutter : results in 100 consecutive patients (abstracts). PACE 1995; 18:856.

16. Kalman JM, VanHare GF, Olgin JE et al. Ablation of "incisional" reentrant atrial tachycardia complicating surgery for congenital heart disease. Circulation 1996; 96:502-12.

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