

Similarities and differences between atrial flutter and atrial fibrillation

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Atrial flutter and atrial fibrillation have a complex relationship that has mechanistic, diagnostic, therapeutic and prognostic components. While thromboembolic risk management and pharmacological strategies share many similarities, there are important differences. Radiofrequency ablation should be considered to be an early, first-line alternative to pharmacological strategies for many patients with atrial flutter.

Key Words: Atrial fibrillation; Atrial flutter; Guidelines; Pharmacological strategy; Radiofrequency ablation

The electrocardiographic pattern of atrial flutter (AFL) (which consists of regular atrial activity at a rate of 240 beats/min to 340 beats/min usually with no intervening isoelectric periods) reflects the dominant macroreentrant electrophysiological mechanism of AFL, and distinguishes AFL from atrial fibrillation (AF). Nevertheless, there is a strong clinical relationship between AFL and AF. A single patient may have, at different times, paroxysms of both AFL and AF (1,2). Antiarrhythmic drugs are known to facilitate the transformation of disorganized AF into more organized AF (3-5), and patients who have undergone successful ablation procedures to cure AFL may go on to develop AF (6-10).

The precise nature of the relationship between these two rhythm disturbances is not fully understood. At the time of spontaneous conversion from AF to AFL, the AF cycle length changes just before AFL develops (11). AF may induce electrophysiological remodelling of atrial tissue, facilitating the development and persistence of the reentrant circuit of typical AFL (12-14). AFL, in turn, may give rise to AF if the cycle length is sufficiently short enough to provoke fibrillatory conduction (15). This may be particularly true of atypical AFL (16-19). Ectopic beats from the pulmonary veins have been postulated to be the obligatory triggers for activation of the AFL reentrant circuit (20) and have also been implicated in the transition from AFL to AF (21). In some patients, AFL and AF may even occur simultaneously (22).

Classical AFL (also known as typical or counterclockwise AFL) is due to a macroreentrant right atrial circuit in which the wavefront proceeds up the interatrial septum, down the right atrial free wall and through the cavotricuspid isthmus. This results in the classic electrocardiographic pattern of negative sawtooth flutter waves in leads II, III and aVF, along with positive flutter waves in lead V1. Reverse typical AFL (also known as clockwise AFL) uses the same circuit but in the reverse direction. This produces positive flutter waves in leads II, III and aVF, along with negative flutter waves in lead V1. Atypical AFLs are

Les ressemblances et les différences entre le flutter auriculaire et la fibrillation auriculaire

Le flutter auriculaire et la fibrillation auriculaire ont un rapport complexe, qui comporte à la fois des éléments mécanistes, diagnostiques, thérapeutiques et pronostiques. Même si le traitement du risque de thrombo-embolie et la pharmacothérapie ont plusieurs ressemblances, il existe également des différences importantes. L'ablation par courant de radiofréquence devrait être considérée comme une première bonne solution de rechange au traitement médicamenteux chez de nombreux patients atteints de flutter auriculaire.

those that have less characteristic macroreentrant wavefronts and other regular electrocardiographic atrial activation patterns. In the present report, the term 'AFL' is used in a generic way that includes all of these electrophysiological mechanisms.

RECOMMENDATIONS

Pharmacological management

Class I

- 1) When pharmacological management for patients with AFL is selected, either the rate-control strategy or the rhythm-control strategy is appropriate (level of evidence C).
- 2) The pharmacological agents used for rate control and rhythm control for patients with AFL are the same as those used for patients with AF (level of evidence C).
- 3) When a class IC or IA agent is chosen to treat a patient with AFL, an atrioventricular (AV) node blocking agent should generally be used concurrently (level of evidence C).

As with AF, the pharmacological treatment of AFL can be directed to achieve ventricular rate control (the rate-control strategy) or to attempt to restore and maintain sinus rhythm (the rhythm-control strategy). Because rate control can be more difficult to achieve in patients with AFL versus patients with AF, the rhythm-control strategy is often the primary approach to therapy. Nevertheless, the objective benefits of this strategy over effective rate control are unproven.

For macroreentrant AFL, including typical and reverse typical AFL, antiarrhythmic drug therapy that prolongs the refractory period within the reentrant circuit would be expected to inhibit the advancing wavefront (23-26) and, thereby, prevent the initiation and maintenance of AFL. The class III drugs (amiodarone, dofetilide, ibutilide and sotalol) are widely used both for the conversion to and maintenance of normal sinus

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rhythm. For the acute termination of AFL, ibutilide appears to be the most effective (27,28). Antiarrhythmic drugs in class IA (disopyramide, procainamide and quinidine) and those in class IC (flecainide and propafenone) may also be useful for either the conversion to or maintenance of sinus rhythm.

The shortcoming of each of these antiarrhythmic drug therapies is antiarrhythmic drug-related proarrhythmia. Class III drugs can produce torsade de pointes in 1% to 4% of patients (29). Class I drugs (particularly class IC) can slow the AFL rate dramatically to the point where the unencumbered AV node can conduct the slower AFL to the ventricles in a 1:1 ratio, leading to a substantial increase in the ventricular response rate (30). Accordingly, patients should be treated with an AV node-blocking agent concurrently whenever class I drugs are selected, except when AV node conduction is known to be poor.

When a rate-control strategy is chosen for the treatment of patients with AFL, drugs that prolong the refractory period of the AV node are effective (beta-blockers, nondihydropyridine calcium channel blockers and digitalis), just as they are in patients with AF. Nevertheless, rate control in patients with AFL may be more difficult to achieve than in patients with AF.

RECOMMENDATIONS: THROMBOEMBOLIC RISK MANAGEMENT

Class I

- 1) As with AF, AFL patients at high risk for systemic emboli should receive chronic oral anticoagulation therapy (level of evidence B).
- 2) Patients should have therapeutic international normalized ratio (INR) measurements on warfarin for at least three weeks before and at least three weeks following the restoration of sinus rhythm (whether by pharmacological therapy, electrical cardioversion or catheter ablation). Alternatively, cardioversion may be accomplished without prior long-term anticoagulation therapy if the atria have been cleared by low-risk findings on a transesophageal echocardiogram. Following a transesophageal echocardiogram-guided strategy, patients should be subsequently anticoagulated for at least four weeks (level of evidence C).

There are no randomized controlled trials that have examined the efficacy of any antithrombotic strategy in patients with AFL. However, several lines of evidence suggest that patients with AFL face an increased risk of thromboembolic events, which can be stratified on the basis of traditional risk factors (age 65 years or older, clinical evidence of left ventricular systolic dysfunction, history of hypertension, history of diabetes mellitus or history of a previous thromboembolic event).

In several series, the risk of thromboembolism has been found to be elevated in patients with AFL (30-34), particularly after conversion to normal sinus rhythm. One study (35) has suggested that at least some of this excess risk may be attributed to those patients with AFL who also have paroxysms of AF. This elevated thromboembolic risk may also be expressed at the time of catheter ablation for cure of AFL (36,37). Patients with AFL and impaired left atrial appendage function have also been reported to be at higher risk for thromboembolic events (38,39). Independent of left atrial appendage function, left atrial thrombus and spontaneous contrast ('smoke') appear to occur with higher frequency in AFL patients (40,41). Finally, one patient series (42) reported that all AFL patients who had

suffered a postcardioversion thromboembolic event were either not taking warfarin or were suboptimally anticoagulated. In contrast, none of the patients in this series who were well anticoagulated suffered a thromboembolic complication. These reports and clinical experiences demonstrating that many (perhaps most) patients with AFL also have periods of AF lead to a recommendation that the antithrombotic considerations for patients with AFL should be similar to those of patients with AF. When warfarin therapy is chosen, the target INR is 2.0 to 3.0.

RECOMMENDATIONS: CATHETER ABLATION

Class I

- 1) Curative catheter ablation for symptomatic AFL may be offered as a first-line therapy and presented as a reasonable alternative to pharmacological therapy (level of evidence B).
- 2) AV node ablation with permanent pacing should be reserved for patients with symptomatic AFL despite optimal medical therapy when curative ablation is not feasible (level of evidence C).

Catheter ablation of the cavotricuspid isthmus has emerged as a safe and effective strategy for the management of patients with isthmus-dependent AFL (43-48). Technologies that allow for larger atrial myocardial lesions appear to generate the best results (49). A large, prospective, randomized trial (50) has established the superiority of isthmus ablation to antiarrhythmic drug therapy in terms of success rate, quality of life and lower recurrence of AFL in follow-up. A single-centre study (51) examining catheter ablation in older patients found it to be safe and efficacious even in the elderly. Given the potential for electrical remodelling in AFL, similar to that seen in patients with AF (52), a compelling argument can be made for early intervention with catheter ablation for the treatment of patients with problematic isthmus-dependent AFL.

Notwithstanding the advantages of catheter ablation for the treatment of selected patients with AFL, early and late occurrences of AF following successful isthmus ablation are common (46,53,54). A previous history of AF, the presence of significant mitral regurgitation, and left ventricular systolic dysfunction all predict the occurrence of AF following successful ablation of AFL. Therefore, AFL ablation may not be appropriate for these patients with a high risk of subsequent AF in the absence of demonstrated failure of antiarrhythmic drug therapy. Emerging 'hybrid' strategies, however, are currently exploring combinations of pacing, antiarrhythmic drugs, isthmus ablation and pulmonary vein isolation; these strategies appear promising for patients who have both AFL and AF (55).

On occasion, usually after the failure of both pharmacological therapy and isthmus ablation, patients with problematic AFL may be treated with catheter ablation of the AV node to create an iatrogenic complete AV block, with the ventricular rate then governed by a permanent pacemaker.

RECOMMENDATIONS: ELECTRICAL CARDIOVERSION OF AFL

Class I

- 1) Electrical cardioversion of AFL should be carried out for the same indications as AF. The technique is the same as that for cardioversion of AF (level of evidence B).

Electrical cardioversion of persistent AFL is a safe, effective and economical procedure (56-58). Patients presenting with acute onset AFL who are unstable should be promptly cardioverted with synchronized direct current energy, as with a patient with AF. More stable patients may undergo elective or semi-elective cardioversion in the same way as a patient with AF, with similar attention to precardioversion anticoagulation (at least three weeks with therapeutic INRs). Postcardioversion anticoagulation also appears to be important (as it is for the patient with AF) as discussed in the above thromboembolic risk management section of the present article.

The only difference between the cardioversion of AFL and AF is the recommended starting energy. Many authorities (59-61) have recommended a starting energy of 50 joules (J). However, these recommendations were based on anecdotal experience and consensus. Since these recommendations were made, one trial (53) has reported that an initial energy of 100 J is superior to an initial energy of 50 J, with 100 J reducing the number of shocks required per case and having a first shock success rate of 85% (compared with 70% for a first shock of 50 J). Another study (62) in patients with long-standing AFL that

had been present for more than 30 days reported that an initial 100 J shock was successful 68% of the time.

Atypical AFL

Patients with substantially disordered atrial electrophysiology, including those with congenital heart disease and previous cardiac surgery, may develop atypical AFL. Interatrial reentrant circuits can form around atrial septal defects, atriotomy scars, the crista terminalis, AV valves, venae cavae and pulmonary veins. New mapping technologies permit the delineation of these circuits, making catheter ablation feasible for these complex arrhythmias (18,62-68).

CONCLUSIONS

AFL and AF have a complex relationship that has mechanistic, diagnostic, therapeutic and prognostic components. Pharmacological management and thromboembolic risk management considerations are similar for the two rhythm abnormalities. Radiofrequency ablation should be considered as an early alternative for many patients with problematic AFL.

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