



Canadian
Cardiovascular
Society

**CANADIAN CARDIOVASCULAR SOCIETY
CONSENSUS CONFERENCE:
ATRIAL FIBRILLATION
2004**

EXECUTIVE SUMMARY

CO- CHAIRS:

Charles R. Kerr

ST. PAUL'S HOSPITAL, UNIVERSITY OF
BRITISH COLUMBIA, VANCOUVER, B.C.

Denis Roy

MONTREAL HEART INSTITUTE, UNIVERSITY OF
MONTREAL, MONTREAL, QUEBEC.

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PRIMARY PANELISTS:

CHAIRS:

CHARLES R. KERR,

PROFESSOR, DEPARTMENT OF MEDICINE,
UNIVERSITY OF BRITISH COLUMBIA,
St. Paul's Hospital, Room B344
1081 Burrard Street, Vancouver,
B.C., V6Z 1Y6

DENIS ROY

PROFESSOR, DEPARTMENT OF MEDICINE,
UNIVERSITY OF MONTREAL,
MONTREAL HEART INSTITUTE
5000 rue Belanger
Montreal, QC, H1T 1C8

PANEL:

STUART J. CONNOLLY

McMASTER UNIVERSITY
HAMILTON GENERAL HOSPITAL
Hamilton, Ontario

PETER G. GUERRA

UNIVERSITY OF MONTREAL
MONTREAL HEART INSTITUTE
Montreal, QC

SEAN P. CONNORS

MEMORIAL UNIVERSITY
HEALTH SCIENCES CENTRE
St. John's, NL

LOUISE HARRIS

UNIVERSITY OF TORONTO
TORONTO GENERAL HOSPITAL
Toronto, ON

EUGENE CRYSTAL

UNIVERSITY OF TORONTO
SUNNYBROOK AND WOMEN'S COLLEGE HEALTH SCIENCE
CENTRE
Toronto, ON

BRETT G. HEILBRON

UNIVERSITY OF BRITISH COLUMBIA
St. PAUL'S HOSPITAL
Vancouver, BC

PAUL DORIAN

UNIVERSITY OF TORONTO
St. MICHAEL'S HOSPITAL
Toronto, ON

GEORGE J. KLEIN

UNIVERSITY OF WESTERN ONTARIO
LONDON HEALTH SCIENCES CENTRE
London, ON

ANNE M. GILLIS

LIBIN CARDIOVASCULAR INSTITUTE OF ALBERTA
UNIVERSITY OF CALGARY
Calgary, AB

L. BRENT MITCHELL

LIBIN CARDIOVASCULAR INSTITUTE OF ALBERTA
UNIVERSITY OF CALGARY
Calgary, AB

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PIERRE PAGE

UNIVERSITY OF MONTREAL
SACRÉ-COEUR HOSPITAL
Montreal, QC

JOHN H. PARKER

CANADIAN CARDIOVASCULAR SOCIETY
Ottawa, ON

CHRISTOPHER S. SIMPSON

QUEEN'S UNIVERSITY
KINGSTON GENERAL HOSPITAL
Kingston, ON

ALLAN C. SKANES

UNIVERSITY OF WESTERN ONTARIO
LONDON HEALTH SCIENCES CENTRE
London ON

MARIO TALAJIC

UNIVERSITY OF MONTREAL
MONTREAL HEART INSTITUTE
Montreal, QC

D. GEORGE WYSE

LIBIN CARDIOVASCULAR INSTITUTE OF ALBERTA
UNIVERSITY OF CALGARY
Calgary, AB

NON-PANELIST AUTHORS:

ROBERT M. GOW,

UNIVERSITY OF OTTAWA,
CHILDREN'S HOSPITAL OF EASTERN ONTARIO
Ottawa, ON

SAMUEL C. SIU,

UNIVERSITY OF TORONTO,
TORONTO GENERAL HOSPITAL,
Toronto, ON

JOEL A. KIRSH,

UNIVERSITY OF TORONTO,
HOSPITAL FOR SICK CHILDREN
Toronto, ON

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SECONDARY PANEL MEMBERS:

DENIS BOUCHARD

MONTREAL HEART INSTITUTE
Montreal, QC

DAVY C. H. CHENG

UNIVERSITY OF WESTERN ONTARIO
London, ON

KENNETH M. FLEGEL

MCGILL UNIVERSITY
Montreal, QC

MARTIN S. GREEN

UNIVERSITY OF OTTAWA
OTTAWA HEART INSTITUTE
Ottawa, ON

PAUL J. HENDRY

UNIVERSITY OF OTTAWA
Ottawa, ON

MALCOLM HING

UNIVERSITY OF OTTAWA
Ottawa, ON

DANIEL W. HOWES

KINGSTON GENERAL HOSPITAL
Kingston, ON

JANE IRVINE

UNIVERSITY OF TORONTO
Toronto, ON

RICHARD A. LEATHER

ROYAL JUBILEE HOSPITAL
Victoria, BC

HEATHER KERTLAND

ST. MICHAEL'S HOSPITAL
Toronto, ON

PAUL KHAIRY

MONTREAL HEART INSTITUTE
University of Montreal

SHANE KIMBER

UNIVERSITY OF ALBERTA
Edmonton, AB

ANDREW KRAHN

UNIVERSITY OF WESTERN ONTARIO
London, ON

FRANCIS MARCHLINSKI

UNIVERSITY OF PENNSYLVANIA
Philadelphia, PA

JOHN PAWLOVICH

FAMILY PRACTICE
Fraser Lake, BC

STEVE SHALANSKY

ST. PAUL'S HOSPITAL
Vancouver, BC

KEVIN WOLFE

UNIVERSITY OF MANITOBA,
Winnipeg, MB

RAYMOND YEE

UNIVERSITY OF WESTERN ONTARIO
London, ON

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Background and Consensus Process

DENIS ROY, CHARLES R. KERR

Atrial fibrillation (AF) affects approximately 200,000 to 250,000 Canadians and is associated with many common clinical conditions such as aging, thromboembolism, hypertension, valvular heart disease and heart failure and is responsible for substantial morbidity and increased mortality. Consequently, AF places a tremendous burden on our health care resources. Thus, the management of AF is complex and has far-ranging implications making this an important challenge for treating physicians.

Atrial Fibrillation was the topic of the 1994 Consensus Conference. In the intervening decade, much of our knowledge about the management of AF has been solidified or modified by the enormous amount of research being performed on this disease. Unfortunately, there remain many issues for which there is little or no scientific evidence to guide clinical practice. The present Consensus Conference was developed to incorporate these new data and to update AF practice recommendations in the context of Canadian standards of practice and the Canadian healthcare system.

ORGANIZATION OF THE CONSENSUS

Major areas of interest in AF were assigned to primary panellists who prepared chapters.

Recommendations were then debated, revised, and voted on during a face-to-face meeting in February 2004. A secondary panel of physicians, cardiologists and arrhythmia experts reviewed the manuscripts during the Spring, 2004 and the documents were circulated to the CCS membership.

The final text and recommendations were presented at the Annual Meeting of the Canadian Cardiovascular Congress in Calgary in October 2004.

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RECOMMENDATIONS AND RULES OF EVIDENCE

Recommendations are expressed in the standard ACC/AHA format:

- Class I:** Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective.
- Class II:** Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.
- Class IIa:** The weight of evidence or opinion is in favour of the procedure or treatment.
- Class IIb:** Usefulness/efficacy is less well established by evidence or opinion.
- Class III:** Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful.

Evidence supporting the recommendations is ranked as:

- A (highest):** When the data were derived from multiple randomized clinical trials involving a large number of individuals.
- B (intermediate):** When the data were derived from a limited number of randomized trials, nonrandomized studies or observational registries.
- C (lowest):** When the primary basis for the recommendation was expert consensus.

We present here summaries of each chapter. The format is a statement of the recommendations for each section, followed by a brief description of issues in the chapters

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Chapter 1. Etiology and Initial Investigation

ALLAN C. SKANES, PAUL DORIAN

RECOMMENDATIONS

Class I:

1. **Baseline history, appropriate laboratory tests, 12-lead ECG and echocardiography should be obtained in all patients to identify potential etiology and other co-morbidities and to stratify for risk of stroke. Details are highlighted in tables 1 and 2. (Level of evidence: C)**
2. **Underlying causes or precipitating factors including underlying hypertension should be identified, eliminated or treated. (Level of evidence: C)**

Class IIa:

1. **Other ancillary tests should be considered under specific circumstances. Details are highlighted in table 1. (Level of evidence: C)**

The initial evaluation of a patient with atrial AF should include a comprehensive review of historical factors, a physical examination and initial investigations. This evaluation has many important purposes including developing a therapeutic strategy for symptom relief, assessing and managing thromboembolic risks, and identifying underlying etiology. This evaluation should also review management of risk factors for overall cardiovascular morbidity and its treatment.

The predominant pattern of AF should be determined as defined by the ACC/AHA/ESC guidelines for management of AF:

- ▶ **First detected AF**
- ▶ **Paroxysmal:** AF is self-terminating within 7 days of recognized onset
- ▶ **Persistent:** AF is not self-terminating within 7 days or is terminated electrically or pharmacologically
- ▶ **Permanent:** AF in which cardioversion has failed or in which clinical judgment has led to a decision not to pursue cardioversion.

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One may not be able to identify the pattern of AF at the time of initial presentation and the pattern may change over time. An assessment of the severity of symptoms and impact on quality of life should be performed. Symptoms associated with AF are highly variable with some patients truly asymptomatic while others having highly disruptive symptoms.

Table 1 outlines recommended initial routine investigation as well as additional investigation that may be appropriate in specific circumstances. Table 2 summarizes potential etiologies of AF that should be considered in the initial evaluation and that may guide further investigation.

Table 1. Initial Investigation: ROUTINE INVESTIGATION

- ▶ History and Physical Examination
 - Establish pattern (first detected, paroxysmal, persistent, permanent)
 - Establish severity including impact on quality of life
 - Identify potential etiology
 - Consider hypertension, alcohol abuse, thyroid disease, sleep apnea
 - Determine underlying thromboembolic risk
 - Develop a treatment strategy based on clinical risk factors
 - Evaluate likelihood of other arrhythmia – PSVT / atrial flutter
 - Document prior pharmacologic therapy aimed at rhythm and rate control including effectiveness and adverse effects

- ▶ 12-lead ECG
 - Evidence of hypertrophy, conduction disease, myocardial infarction, QT interval.

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- ▶ Transthoracic echocardiography
 - Chamber size, ventricular function, valve function
- ▶ CBC, electrolytes, renal function
- ▶ Thyroid function

ADDITIONAL INVESTIGATIONS OF POTENTIAL VALUE

- ▶ Chest Radiograph
- ▶ Ambulatory ECG monitoring

This includes 24 hour Holter monitor, event recorder, or loop monitor

 - Document arrhythmia and establish symptom-rhythm correlation - AF or alternative contributing arrhythmia (PSVT / flutter)
 - Assess rate control during AF
- ▶ Treadmill exercise test
 - Only in those with intermediate or high risk of coronary disease
 - To evaluate rate control
- ▶ Trans-esophageal echocardiogram
 - Assess LA size and rule out LA thrombus
- ▶ Electrophysiologic study
 - Documented or suspected underlying PSVT
 - Consider atrial flutter ablation in those where this forms a substantial part of the symptom burden

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Table 2. Etiology of Atrial Fibrillation

CARDIOVASCULAR CAUSES

- ▶ Hypertension
- ▶ Valvular Disease
- ▶ Coronary artery disease with prior myocardial infarction
- ▶ Cardiomyopathy
- ▶ Pericardial disease
- ▶ Electrical / Senescence
 - bradycardia-tachycardia (“sick-sinus”) syndrome
- ▶ Genetic / familial
- ▶ Post-operative
- ▶ Congenital heart disease

NON-CARDIOVASCULAR CAUSES

- ▶ Autonomically-mediated (vagal)
- ▶ Toxin – e.g., alcohol
- ▶ Endocrine – e.g., thyroid disease
- ▶ Pulmonary disease—COPD, pneumonia, sleep apnea
- ▶ Neurologic – usually associated with myopathic muscle diseases
- ▶ Idiopathic
 - Occult hypertension
 - Occult genetic causes

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Chapter 2. Rate Control versus Rhythm Control – Decision Making

D. GEORGE WYSE, CHRISTOPHER S. SIMPSON

RECOMMENDATIONS:

The following recommendations apply to recurrent AF outside the setting of reversible causes. Anticoagulation should be used according to subsequent sections of these guidelines, regardless of whether a rate control or rhythm control approach is used. The recommendations are based on a primarily pharmacologic approach.

Class I:

1. There is no evidence that rhythm control or rate control is superior to the other and both are recommended as acceptable initial approaches, except for permanent AF where rate control is recommended. (Level of Evidence: A).

Class IIa:

1. The choice of rate control or rhythm control for initial therapy should be individualized and is determined by a number of factors (Table) such as classification of AF, degree of symptoms. (Level of Evidence: C).

FAVOURS RATE CONTROL	FAVOURS RHYTHM CONTROL
Persistent AF	Paroxysmal AF
Recurrent AF	First Episode of AF
Less Symptomatic	More Symptomatic
>65 years of age	< 65 years of age
Hypertension	No Hypertension
No History of Congestive Heart Failure	History of Congestive Heart Failure
Previous Antiarrhythmic Drug Failure	No Previous Antiarrhythmic Drug Failure
Patient Preference	Patient Preference

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Class IIb:

1. **Crossover to the alternative strategy, return to the initial strategy and nonpharmacologic therapies should be considered when therapy fails due to adverse effects or failure to improve symptoms.**

(Level of Evidence: C).

Origin of the Rate versus Rhythm Question

There are two accepted general strategies for arrhythmia management in AF. The first is to control the heart rate without any specific attempt to restore and maintain sinus rhythm (The Rate Control Strategy). The second is to restore and attempt to maintain sinus rhythm, including repeated cardioversion for recurrences (The Rhythm Control Strategy). Either strategy must be accompanied by strict adherence to recommendations regarding antithrombotic therapy.

Initial practice favoured the rhythm management strategy, with the thought that maintenance of sinus rhythm would result in less morbidity and mortality and better quality of life. However, recent randomized trials have shown that, in a select population of patients, there is no major difference in outcome between the two therapeutic strategies. These studies mainly enrolled an older, relatively asymptomatic group of patients.

Therefore, the choice of rhythm versus rate control should be individualized, taking into account a number of clinical and laboratory variables and strongly focusing on symptomatic outcome. The table above outlines some of the factors that may favour one approach over the other.

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Chapter 3: Drug Therapy for Termination of Atrial Fibrillation and Maintenance of Sinus Rhythm

MARIO TALAJIC, DENIS ROY

RECOMMENDATIONS:

Conversion of Atrial Fibrillation

Class I:

1. Electrical or pharmacologic conversion should be considered in patients with AF who are hemodynamically stable (Level of Evidence: C)
2. Immediate conversion to sinus rhythm is recommended in patients with AF who are hemodynamically unstable. Electrical cardioversion is more effective and is preferred over pharmacologic conversion in these patients. (Level of Evidence: C)

Class IIA:

1. Rate-control with anticoagulation therapy alone is acceptable while awaiting spontaneous conversion in patients with AF of less than 48 hours duration. (Level of Evidence: B)
2. Pharmacological agents may be used to accelerate conversion of AF in patients with AF of less than 48 hours duration. (Level of Evidence: B). See table 1 for specific drug recommendations.
3. Antiarrhythmic drugs may be used to pre-treat patients before electrical cardioversion (to decrease early recurrence of AF and to enhance cardioversion efficacy). (Level of Evidence: B)

Class IIB

1. Blockade of the angiotensin-renin system may be considered in combination with amiodarone before electrical cardioversion in order to decrease the recurrence rate of AF (Level of evidence: B)

Table 1. Recommended Drugs for Conversion of Atrial Fibrillation

Class I

- Ibutilide (Level of Evidence: A)
- Flecainide (Level of Evidence: A)
- Procainamide (Level of Evidence: B)
- Propafenone (Level of Evidence: A)

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Class IIA

- Chronic Oral Amiodarone (Level of Evidence: B)

Class III

- Sotalol (Level of Evidence: B)

MAINTENANCE OF SINUS RHYTHM IN PATIENTS WITH AF

Class I:

1. Oral antiarrhythmic drugs may be used in patients with recurrent AF in whom long-term maintenance of sinus rhythm is desired and in whom a reversible cause of AF is not identified. (Level of Evidence: B)
2. The choice of an antiarrhythmic drug should be based on the safety profile of the different agents, taking in account the clinical characteristics of the patient. (Level of Evidence: B) Recommendations regarding specific agents are listed in table 2.

Class IIA:

1. In patients without risk factors for proarrhythmia, antiarrhythmic drugs may be initiated as outpatients. (Level of Evidence: B)
2. In patients with structural heart disease (including those with LV dysfunction) amiodarone may be initiated as outpatients. (Level of Evidence: B)
3. An AV nodal blocking agent is recommended in patients treated with a class 1C antiarrhythmic drug. (Level of Evidence: B)

Class IIB:

1. Patients treated with sotalol or dofetilide should be reassessed if QTc exceeds 480 msec. (Level of Evidence: C)

Class III:

1. Sotalol should not be used for rate-control alone in patients with permanent AF. (Level of Evidence: C)

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Table 2. Selection of Antiarrhythmic Drugs for Maintenance of Sinus Rhythm

1. PATIENTS WITH STRUCTURALLY NORMAL HEARTS

First Choices:	Propafenone
	Flecainide
	Sotalol*
Second Choice:	Amiodarone
Alternative Choices:	Disopyramide
	Dofetilide**

2. PATIENTS WITH STRUCTURALLY ABNORMAL HEARTS

A. CAD with normal ventricular function

First Choice:	Sotalol*
Second Choice:	Amiodarone
Additional Choices:	Dofetilide**
	Propafenone

B. Left Ventricular Dysfunction (with or without CHF)

First Choice:	Amiodarone
Second Choice:	Dofetilide**

HYPERTENSION WITH LVH

First Choices:	Sotalol
	Amiodarone
	Propafenone
	Flecainide

* contraindicated in females > 65 years of age taking diuretics

** Dofetilide is available in Canada through Health Canada's special access program

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When contemplating therapy for AF, one must initially decide on a strategy of cardioversion and attempted rhythm control or rate control (Chapter 2). Antiarrhythmic drug therapy to maintain sinus rhythm has not been demonstrated in randomized, clinical trials to improve prognosis or prevent thromboembolic complications in patients with AF. Therefore drug therapy to restore and maintain sinus rhythm should be limited to those patients who have a greater *symptomatic* burden of AF.

Drug therapy may be used for conversion in patients with hemodynamically stable AF. Therapy to control the ventricular rate response to AF should be initiated before or simultaneously with therapy to convert the arrhythmia. Before attempting drug conversion, patients should be adequately anticoagulated to prevent post conversion thromboembolic complications. Intravenous procainamide or ibutilide and oral propafenone or flecainide are the most effective agents for pharmacological cardioversion. Sotalol is ineffective in facilitating conversion to sinus rhythm.

In the absence of a reversible cause, AF is usually recurrent. Placebo controlled trials have shown that the one year recurrence rate of AF in the absence of an antiarrhythmic drug is approximately 75%. Antiarrhythmic drug therapy is usually necessary to decrease the number of episodes in patients with paroxysmal AF and to prevent recurrence in patients with persistent AF.

Of the presently available oral antiarrhythmic drugs, amiodarone has been demonstrated in comparative studies to be more efficacious than other drugs. However it also has significant non-cardiac side effects limiting its widespread use as an agent of first choice. Other agents have the potential for significant pro-arrhythmia when given to patients with underlying heart disease. As a result the choice of a chronic antiarrhythmic drug in an individual patient is usually guided by the safety profile of the drug with respect to the clinical characteristics of the patient (Table 2).

Patients with no underlying heart disease have a low risk of pro-arrhythmia. As a result, antiarrhythmic drug initiation generally can be started as an outpatient if sinus node dysfunction or AV conduction disturbances are not present. Sotalol may be initiated as an outpatient in a patient without risk factors for torsades de pointes. Patients with underlying heart disease have a higher risk of pro-arrhythmia; drugs should be initiated in-hospital with ECG monitoring if a drug other than amiodarone is used.

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Chapter 4: Pharmacologic and Non-pharmacologic Methods of Rate Control

PAUL DORIAN, SEAN P. CONNORS

RECOMMENDATIONS:

Class I:

1. Rate control should be undertaken for improvement of symptoms as well as control of ventricular rate. (Level of Evidence: C)
2. Administer non-dihydropyridine calcium channel blocking agents (diltiazem, verapamil) or beta blocking agents as initial rate slowing therapy in active and younger patients. (Level of Evidence: B)
3. Administer beta blocking agents combined with digoxin to control ventricular rate in patients with heart failure. (Level of Evidence: C)
4. Consider pacemaker implantation and atrioventricular nodal ablation for patients with persisting symptoms due to rapid or irregular ventricular rate, in whom oral drug therapy is ineffective or not tolerated. (Level of Evidence: A)
5. In patients with a rapid ventricular rate associated with pre-excitation over an accessory bypass tract (Wolff-Parkinson-White Syndrome), administer intravenous procainamide or ibutilide or perform DC cardioversion if unstable. (Level of Evidence: B)

Class IIa:

1. Assess ventricular rate at rest and during exercise and modify target rates depending on patients' symptoms. (Level of Evidence: C)
2. Administer digoxin as initial therapy in elderly, inactive patients (Level of Evidence: C) or as adjunctive therapy to calcium channel or beta blocking agents in younger and active patients. (Level of Evidence: C)

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Symptoms during AF are usually caused by the rapid and irregular rate. A persistent rapid rate can result in a tachycardia-induced cardiomyopathy. Thus, rate control should be undertaken both to improve symptoms and to prevent sustained high rates. Most patients will have a rapid rate during AF, unless there is concomitant AV nodal conduction disease, frequently seen in the elderly. Rate control may be needed in persistent or permanent AF but also should be considered in addition to antiarrhythmic drugs in paroxysmal AF, in the event that AF should recur. Drugs such as propafenone and flecainide do not adequately slow the ventricular rate and may, in fact, accelerate the rate.

In AF, rate control should be assessed at rest and with exercise as the rate often accelerates rapidly with minimal exercise. Calcium channel blocking drugs (such as diltiazem or verapamil) or beta blocking agents are recommended as first line therapy for rate control in younger, active patients. Digoxin may be added as adjunct therapy but is not useful in control of exercise heart rate in this population. In the elderly and inactive, monotherapy with digoxin may adequately control the rate. In patients with heart failure, beta blocking drugs in conjunction with digoxin are recommended.

In patients with persistent symptoms while on rate slowing drugs or in those with drug intolerance, pacemaker implant and AV Node ablation can provide excellent symptomatic relief and improvement in functional status. In all circumstances appropriate antithrombotic therapy must be administered.

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Chapter 5: Catheter Ablation Therapy for Atrial Fibrillation

PETER G. GUERRA, ALLAN C. SKANES

RECOMMENDATIONS FOR CATHETER ABLATION FOR RHYTHM CONTROL:

Class I:

1. In patients with AF and preexcitation, catheter ablation of the accessory pathway is recommended, particularly if associated with syncope, rapid ventricular rates, or if the accessory pathway has a short refractory period (Level of Evidence: B).

Class IIa:

1. In young patients with lone, paroxysmal AF, an electrophysiologic study should be considered to exclude a reentrant tachycardia as a potential etiology for AF, and if present, curative ablation should be performed (Level of Evidence: B)
2. Patients with highly symptomatic, paroxysmal AF refractory to medical therapy should be considered for an ablation procedure aimed at maintaining sinus rhythm (Level of Evidence: B).

RECOMMENDATIONS FOR CATHETER ABLATION FOR RATE CONTROL:

Class I:

1. Patients with highly symptomatic, permanent AF with rapid ventricular rates in whom oral rate control drug therapy is insufficiently effective or not tolerated should be considered for AV nodal ablation and pacemaker implantation (Level of Evidence: B).
2. Patients with highly symptomatic, paroxysmal AF in whom attempts at rhythm control have been abandoned and in whom pharmacologic rate control is insufficiently effective or not tolerated should be considered for AV nodal ablation and pacemaker implantation (Level of Evidence: B).

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Although a rate control strategy may provide adequate symptomatic improvement in many patients, others may continue to be highly symptomatic, particularly younger patients with paroxysmal AF. In these patients ablation therapy aimed at preventing AF may provide a good alternative to antiarrhythmic drugs. Ablation techniques are rapidly evolving and may be targeted at the factors initiating AF (“triggers”, often in the pulmonary veins) or of the mechanisms that maintain AF (multiple re-entry circuits) or may use techniques that approach both mechanisms. The efficacy has been improving as mapping and ablation techniques evolve. The patients best suited for ablation at the present time would be younger patients with paroxysmal AF and minimal underlying structural disease. Ablation will likely become more widely applicable as techniques and understanding of the underlying mechanisms improve. There are recent reports of the efficacy of ablation in older patients and in those with underlying heart disease, but this needs to be validated with randomized trials. In patients with recurrent, paroxysmal AF, other mechanisms may trigger AF, such as SVT due to an accessory pathway or AV node re-entry. These mechanisms should be looked for as elimination of the primary arrhythmia often eradicates the episodes of AF.

In patients who are refractory to rhythm control with drugs or ablation, in those in whom adequate rate control cannot be achieved, or in those who are intolerant of drugs, pacemaker implant and AV node ablation is a highly effective means of improving symptoms.

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Chapter 6: Surgical Treatment of Atrial Fibrillation

PIERRE PAGÉ, ALLAN C. SKANES

RECOMMENDATIONS

Class I:

1. Patients undergoing intraoperative ablation of AF should be anticoagulated postoperatively unless they have a strong contraindication to oral anticoagulation (Level of evidence: C)

Class IIa:

1. In patients undergoing mitral valve replacement or repair with a history of symptomatic persistent or paroxysmal AF, concomitant intraoperative AF ablation should be considered to increase the likelihood of restoration of sinus rhythm. (Level of evidence B)

Class IIb:

1. Patients with symptomatic persistent or paroxysmal AF undergoing other cardiac surgery (e.g. coronary artery bypass grafting, aortic valve replacement or both) maybe considered for intraoperative AF ablation. (Level of Evidence: C)
2. Patients with refractory, symptomatic AF not associated with organic heart disease and without co-morbidities may be considered for surgical ablation when other non-pharmacological procedures have failed. (Level of Evidence: C)
3. Patients who have undergone intraoperative AF ablation should be re- evaluated for anticoagulation after three months of follow-up according to the general recommendations post valvular surgery. (Level of evidence: C)

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After demonstration of the mechanism of maintenance of AF, surgical procedures were designed that effectively cut and re-sutured the atrium to prevent the maintenance of multiple re-entrant pathways. The Maze procedure has proved successful in stopping AF and has been shown to restore atrial contractile function. However, the procedure has required open heart techniques and its use has been limited.

Patients undergoing heart surgery have a high incidence of pre-operative and post-operative AF, particularly those undergoing mitral valve replacement. The presence of pre-operative AF is a strong predictor of AF following mitral valve replacement. Modifications of the Maze procedure and use of different energy sources (e.g. radiofrequency) have led to the ability to perform an effective ablation procedure during mitral valve surgery with minimal added surgical time and morbidity. Although randomized trials of the efficacy of this technique have not been performed, the results look promising and will likely continue to improve.

Therefore, concomitant Maze ablation should be considered in patients with previous AF who are undergoing mitral valve surgery. Less data is available on patients undergoing other heart operations but the procedure may be considered in these settings.

Operative AF ablation as a primary procedure in patients without other heart disease is uncommonly performed but may be considered in highly symptomatic, refractory cases. In patients undergoing intraoperative AF ablation, anticoagulation should be continued after surgery and ongoing use evaluated at 3 months.

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Chapter 7: Pacing for Prevention of Atrial Fibrillation

ANNE M. GILLIS, CHARLES R. KERR, EUGENE CRYSTAL

RECOMMENDATIONS

Class IIa

1. Atrial pacing (with or without a ventricular lead) should be considered in patients with symptomatic bradycardia to decrease the probability of developing AF and progressing to permanent AF (Level of Evidence: A).
2. The proportion of the time the ventricles are paced should be minimized in patients with intrinsic AV conduction to reduce the incidence of AF (Level of evidence: B).
3. Temporary atrial pacing should be considered following heart surgery to reduce the incidence of peri-operative AF. (Level of Evidence: B).

Class III

1. Atrial pacing for prevention of AF in the absence of symptomatic bradycardia is not recommended. (Level of Evidence: B).

Atrial fibrillation is common in patients with indications for pacing for bradycardia, particularly in those with sinus node dysfunction. There are theoretical reasons why atrial based pacing may be superior to ventricular pacing in reducing the incidence of AF in the general pacing population. Several large randomized trials of pacing mode selection have demonstrated a significantly lower incidence of AF with atrial based pacing modes (AAI or DDD) compared to VVI. Therefore, atrial based pacing should be considered in most patients requiring pacing for bradycardia. In patients with intact AV nodal conduction, efforts should be made to minimize pacing in the ventricle by selecting AAI pacing mode or prolonging the AV delay.

In patients without a bradycardia indication for pacing, studies have not shown a significant reduction of AF by atrial pacing. Several studies have evaluated specific algorithms to prevent AF but the results have shown minimal or no benefit. Alternate site atrial pacing has similarly shown no significant benefit. Dual site pacing has shown slight benefit but this is not generally recommended because of the complexity of multiple atrial leads. Thus, pacing for prevention of AF in the absence of a bradycardia indication is not recommended.

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Chapter 8: Atrial Fibrillation Following Cardiac Surgery

L. BRENT MITCHELL, EUGENE CRYSTAL, BRETT HEILBRON, PIERRE PAGÉ

RECOMMENDATIONS:

Class I:

1. Patients who have been receiving a beta-blocker prior to cardiac surgery should have that therapy continued through the operative period in the absence of the development of a new contraindication. (Level of Evidence: A)
2. Temporary ventricular epicardial pacing electrode wires should be placed at the time of cardiac surgery to allow backup pacing as necessary. (Level of Evidence: C)
3. Post-operative AF with a rapid ventricular response rate should be treated with a beta-blocker, a non-dihydropyridine calcium antagonist, or amiodarone to establish ventricular rate control. In the absence of a specific contraindication, the order of choice is as listed. (Level of Evidence: B)

Class IIa

1. Patients who have not been receiving a beta-blocker prior to cardiac surgery should be considered for prophylactic therapy to prevent post-operative AF with a beta-blocker or amiodarone (Level of Evidence A) or with temporary epicardial atrial pacing or magnesium. (Level of Evidence B)
2. Post-operative AF may be appropriately treated with either a ventricular rate-control strategy or a rhythm-control strategy. (Level of Evidence: A)
3. Consideration should be given to anticoagulation if post-operative AF persists for more than 48 hours. (Level of Evidence: C)
4. When anticoagulation therapy, rate-control therapy, and/or rhythm control therapy has been prescribed for post-operative AF, formal reconsideration of the ongoing need for such therapy should be undertaken six to eight weeks later. (Level of Evidence: B)

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Atrial fibrillation is extremely common following heart surgery and is associated with increased morbidity and prolonged hospital stay. Approximately 30% of those undergoing coronary bypass will develop AF or atrial flutter post-operatively. The incidence is higher following valve surgery (40%) or combined coronary bypass and valve surgery (50%).

Several strategies have been studied to reduce the incidence of AF following heart surgery. The most investigated is the use of beta blocking agents. Meta-analysis has shown a significant reduction in AF (Table 1). Therefore pre-operative beta blockers should be restarted post-operatively and instituted in all possible patients not on beta blockers pre-operatively. Pre-operative or immediate post-operative amiodarone has been shown to reduce AF. There is less compelling, but strongly suggestive, evidence that magnesium and epicardial atrial pacing (left, right, or dual site) may prevent AF and should be considered.

Treatment of AF in the post-operative setting should be similar to AF in other settings. However, because the AF (particularly new onset) usually resolves over 6-8 weeks, choice of a rate control strategy is acceptable. If a decision is made to restore sinus rhythm, consideration should be given to pharmacologic cardioversion or DC cardioversion after loading with antiarrhythmic medication. In those with persistent AF, anticoagulant therapy should be started and continued for a minimum of 6 weeks.

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TABLE 1. Prophylactic therapies for the prevention of post-operative atrial tachyarrhythmias

THERAPY	DOSAGE*	ODDS RATIO†	CAUTIONS	ADVERSE EFFECTS
pre-op beta blocker	any in usual therapeutic dose (i.e. metoprolol 50 mg PO q12h or q8h for at least 2 pre-op days, day of surgery, and at least 6 post-op days)	0.39 (0.28 – 0.52)	reactive airways disease, decompensated CHF	sinus bradycardia AV block Hypotension bronchospasm
pre-op amiodarone	10 mg/kg/day (rounded to nearest 100 mg) divided into two daily PO dosages for 6 pre-op days, day of surgery, and 6 post-op days	0.61 (0.50 – 0.74)	30%-50% reduction in the dosages of other drugs with antiarrhythmic or sinus/AV nodal effects and warfarin will be required	sinus bradycardia AV block hypotension torsade de pointes VT (rare) pulmonary toxicity (rare)
post-op amiodarone	900 – 1200 mg IV over 24 hrs beginning within 6 hours of surgery, then 400 mg PO tid each of the next 4 days	0.53 (0.39 – 0.71)	30%-50% reduction in the dosages of other drugs with antiarrhythmic or sinus/AV nodal effects and warfarin will be required	sinus bradycardia AV block hypotension torsade de pointes VT (rare) pulmonary toxicity (rare)
magnesium sulfate	1.5 gm IV over 4 hrs first pre-op day, immediately post-op, and next 4 post-op days. Other trials have omitted the pre-op dosage	0.83 (0.65 – 1.06)	renal failure	hypotension (rare) sedation (very rare) respiratory depression (very rare)
atrial pacing	right, left, or biatrial pacing for 3 – 4 days post-op . Rate set to overdrive sinus rate either manually or using sensing algorithms	0.67 (0.54 – 0.84)	may increase atrial tachyarrhythmias if pacing continues in setting of sensing malfunction	diaphragmatic stimulation, increased myocardial oxygen requirements, ?increased infection rate

* Dosages used in the randomized studies vary widely and the optimal dosages for this indication have not been established. The dosages provided are those used in the largest positive trial of that therapy and are referenced to that study.

† The odds ratios provided are from meta-analyses of the studies of each prophylactic approach (not for the single study referenced for dosage). Comparisons of the efficacies of various prophylactic approaches require randomized trials, which, for the most part, have not been performed. Accordingly, comparisons among the odds ratios provided in the Table should be avoided.

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Chapter 9: Atrial Arrhythmias and Special Circumstances

LOUISE HARRIS, ROBERT M. GOW, JOEL A. KIRSH,
GEORGE J. KLEIN, SAMUEL C. SIU

Management of AF in Hypertrophic Cardiomyopathy

RECOMMENDATIONS:

Class I

1. Anticoagulate patients with paroxysmal, persistent, or permanent atrial fibrillation with warfarin (INR 2 to 3). *(Level of Evidence: B)*

Class IIa

1. Strategies to maintain sinus rhythm are generally preferred over rate control. *(Level of Evidence: C)*
2. Amiodarone is generally the preferred antiarrhythmic agent for maintenance of sinus rhythm. *(Level of Evidence: C)*

Atrial fibrillation occurs in 20-25% of patients with hypertrophic cardiomyopathy (HCM), and is associated with increased risk of sudden and non-sudden death, heart failure and stroke. The results of AFFIRM notwithstanding, restoration and maintenance of sinus rhythm has been considered an important priority. Although rigorous comparative studies are not available, amiodarone has been considered the most effective and safest drug for maintenance of sinus rhythm. Other antiarrhythmics have been used but disopyramide has been recommended in the absence of large comparative trials, possibly due to its reported favourable hemodynamic effects in patients with obstruction. Rate-control may be achieved with beta blockers or calcium channel blockers such as verapamil. Non-pharmacological therapies including operative and catheter ablation have not been specifically evaluated in HCM but their role in management of AF in this context is likely to increase. Anticoagulation with warfarin is recommended.

Management of AF in patients with Wolff Parkinson White Syndrome

RECOMMENDATIONS

Class I

1. Catheter ablation of the accessory pathway is recommended in symptomatic patients with AF. *(Level of Evidence: B)*

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2. Operative ablation of the accessory pathway is indicated in patients with problematic or life-threatening AF where catheter ablation is not feasible. (*Level of Evidence: B*)
3. Antiarrhythmic therapy with amiodarone, sotalol, disopyramide, flecainide, propafenone, quinidine or procainamide is recommended when corrective ablation is not feasible. (*Level of Evidence: C*)
4. Immediate electrical cardioversion is recommended where AF occurs with a rapid ventricular response and hypotension. (*Level of Evidence: B*)
5. Intravenous procainamide or ibutilide is recommended in AF with predominantly preexcited complexes when the patient is hemodynamically stable. (*Level of Evidence: C*)
6. Verapamil, diltiazem or beta blockers are indicated for rate control when AF occurs without preexcitation. (*Level of Evidence: C*)

Class III

1. Intravenous beta-blocking agents are not generally useful and digitalis, diltiazem, or verapamil are contra-indicated in patients with a rapid ventricular response related to preexcitation. (*Level of evidence: B*)

In the WPW Syndrome accessory pathways may have extremely short effective refractory periods, allowing very rapid ventricular rates in the event of AF. This may result in ventricular fibrillation and sudden death. Although AF in patients with WPW may result from causes unrelated to the preexcitation, it is most frequently related to supraventricular tachycardia which then degenerates into AF. Accessory pathways are composed of working muscle fibers and drugs that usually prolong AV node refractoriness are contraindicated. Intravenous verapamil in particular may precipitate hemodynamic collapse due to its negative inotropic effect and by accelerating the ventricular rate probably due to a reflex sympathetic effect. Intravenous sodium and potassium blocking drugs such as procainamide and ibutilide prolong the refractory period of the accessory pathway and acutely slow the ventricular response in preexcited AF. They may also result in conversion to sinus rhythm. Intravenous amiodarone has not been extensively evaluated for acute treatment of arrhythmias related to WPW.

Catheter ablation of the accessory pathway is currently the treatment of choice for symptomatic WPW syndrome. Where catheter ablation is not feasible, surgical ablation of the accessory pathway is advised for patients with life-threatening AF.

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Management of AF In Pregnancy

RECOMMENDATIONS

Class I

1. Control the rate of ventricular response with digoxin, a beta-blocker, or a calcium channel antagonist. (*Level of Evidence: C*)
2. Perform electrical cardioversion in patients who become unstable due to AF. (*Level of Evidence: C*)
3. Administer antithrombotic therapy (anticoagulant or aspirin) throughout pregnancy in all patients with persistent or paroxysmal AF. (*Level of Evidence: C*)
4. In patients at risk of thrombo-embolism, administer heparin during the first trimester and after 36 weeks gestation. Unfractionated heparin may be administered by intravenous infusion or by twice a day subcutaneous injection (dose adjusted to maintain an activated partial thromboplastin time 2-3 times control value). Alternately, low molecular weight heparin can be utilized (dose adjustment guided by anti Xa levels). (*Level of Evidence: C*)
5. Administer warfarin or heparin during the second trimester to patients with AF and at high thromboembolic risk. (*Level of Evidence: C*)

Class IIa

1. For symptomatic patients or those with poorly tolerated AF, pharmacologic or electrical cardioversion may be considered. (*Level of Evidence: C*)

Atrial fibrillation during pregnancy is usually associated with the presence of maternal structural heart disease or hyperthyroidism. A rapid ventricular response to AF can have deleterious effects on both mother and fetus. Diagnosis and treatment of the underlying condition causing AF is the first priority. The ventricular rate should be controlled with digoxin, a beta-blocker, or a calcium channel antagonist. Cardioversion should be performed if there is severe hemodynamic compromise. All currently available antiarrhythmic drugs have the potential to cross the placenta and to be excreted in breast milk and should be used cautiously.

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Anticoagulation should be administered in pregnant women with structural heart disease and AF. Warfarin should be avoided especially in the first trimester and last month, during which time heparin or low molecular weight heparin should be used.

AF in the absence of structural heart disease is uncommon during pregnancy. As serum levels of several coagulation factors are increased during pregnancy, decisions for the treatment of lone AF during pregnancy (no treatment versus aspirin) need to be tailored for the individual patient.

Management of Atrial Arrhythmias in Patients with Congenital Heart Disease

PREAMBLE

Atrial tachycardias are being recognized increasingly as an important cause of morbidity in patients with congenital heart disease. Most frequently the arrhythmia is a macroreentrant atrial tachycardia. Although often labeled as atrial flutter, it is now preferably called intra-atrial reentrant tachycardia (IART). However, multiple mechanisms for atrial arrhythmias exist in these patients and AF is also well described. Many of the medical issues that are important in adults with AF are relevant to the patient with congenital heart disease and atrial tachycardia. Therefore, the more generic term *atrial tachycardia* (AT), to encompass these different arrhythmias, will be used for the purpose of the following recommendations.

RECOMMENDATIONS:

Cardioversion of AT: Class I

1. Immediately perform electrical cardioversion in patients with AT who are hemodynamically unstable. (Level of Evidence: C)

Class IIa

1. Electrical cardioversion for the early restoration of sinus rhythm is advisable in patients with newly diagnosed AT after appropriate anticoagulation. For those patients with pacemakers, cardioversion may also be accomplished by overdrive pace termination of atrial tachycardia. (Level of Evidence: C)

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2. All patients with congenital heart disease and atrial tachycardia should be managed as patients with AF and structural heart disease with respect to anticoagulation. (Level of Evidence: C)
3. In addition, all patients with complex heart lesions require a transoesophageal echocardiogram before elective cardioversion if no prior anticoagulation or if anticoagulation is sub-therapeutic, independent of arrhythmia duration. (A complex heart lesion in this setting is defined as one with excessive atrial enlargement and scarring, sluggish blood flow and predisposition to atrial thrombus formation even in sinus rhythm. Often it is accompanied by systemic ventricular dysfunction and /or right to left shunting. As such, it most commonly applies to the patient post-Fontan operation but can also be encountered in other clinical situations such as Ebstein's anomaly). (Level of Evidence: C)
4. Strategies to maintain sinus rhythm are generally preferred over rate control. (Level of Evidence: C)

Class IIb

1. Pharmacological cardioversion of AT may be considered in patients who are hemodynamically stable and who have a controlled ventricular rate. (Level of Evidence: C)

Longterm Antithrombotic Management In Patients With Congenital Heart Disease And AT:

Class I

1. Anticoagulation with adjusted dose warfarin is advised in patients with complex high risk lesions who have had an episode of AT. (Level of Evidence: C)

Class IIa

1. Anticoagulation with adjusted dose warfarin should be considered in all other patients with congenital heart disease and recurrent episodes of AT. (Level of Evidence: C)

Class IIb

1. The usefulness of anticoagulation or aspirin in patients with congenital heart disease who have minimal residual lesions and who have experienced a single episode of AT is uncertain. The decision to initiate anticoagulation with adjusted dose warfarin should then be based on conventional risk factors (see Chapter 12). (Level of Evidence: C)

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Surgery in Patients with Congenital Heart Disease and Recurrent AT: Class I

1. All patients presenting with AT require full clinical assessment and investigation to evaluate for anatomically correctable abnormalities. (Level of Evidence: C)
2. Concomitant atrial arrhythmia surgery is recommended in patients with symptomatic, recurrent AT who will be undergoing an operative procedure to correct anatomic abnormalities. (Level of Evidence: C)

Class IIa

1. Arrhythmia mapping and surgery as a primary indication for surgery is reasonable and may be considered in patients with arrhythmias refractory to medical and ablation therapy without a co-existing anatomic/haemodynamic indication for surgery. (Level of Evidence: C)

Discussion and recommendations about maintenance of sinus rhythm, rate control and non-medical management of AT in patients with congenital heart disease are included in the complete document.

Atrial Fibrillation and Flutter in the Paediatric Patient without Congenital Heart Disease

RECOMMENDATIONS:

Acute management

Class I

1. Perform electrical cardioversion if there is severe haemodynamic compromise.
2. Unless otherwise contraindicated, anticoagulate with heparin for urgent cardioversion in patients in whom the duration of arrhythmia is > 48 hours or is uncertain.
3. Administer beta blockers, calcium blockers and, less frequently, digoxin to achieve acute rate control. Intravenous calcium channel blockers should be avoided in infants who are more susceptible to their negative inotropic effects.
4. Consider transoesophageal atrial pacing, which has been shown to be particularly effective in terminating neonatal atrial flutter.

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5. In patients not on anticoagulation or subtherapeutically anticoagulated, perform transoesophageal echocardiography before cardioversion if arrhythmia has been present for > 48 hours or is of uncertain duration.
6. In stable patients with duration of AF > 48 hours or of uncertain duration in whom a decision has been made to attempt cardioversion, optimize rate control and anticoagulate to an INR of 2.0 – 3.0 for 3 weeks prior to cardioversion.

Chronic management

Class IIa

1. Consider drugs with class 1C and class III actions as preferred agents for prevention of recurrence of atrial arrhythmias. AV Node blockade should be considered as adjunctive therapy when using 1C drugs.
2. Consider radiofrequency ablation of recurrent atrial flutter.
3. Antithrombotic therapy with aspirin, if not contraindicated, may be considered in young patients with recurrent episodes who are considered low risk for stroke.

Investigation

Class I

1. Echocardiography to rule out cardiomyopathy and/or structural heart disease is recommended in patients with newly-presenting atrial flutter and fibrillation.
2. Holter monitoring and exercise testing should be performed in young patients with chronic atrial arrhythmia because of the increased occurrence of one-to-one conduction.

(All recommendations Level of evidence: C).

Atrial fibrillation in children without congenital heart disease is relatively uncommon. It is usually associated with structural heart disease or other arrhythmia substrates. Therefore, in children presenting with AF, careful evaluation for underlying heart disease should be performed. Children with WPW syndrome may develop AF secondary to supraventricular tachycardia and may be amenable to curative ablation therapy. In the setting of dilated or hypertrophic cardiomyopathy AF occurs relatively frequently and may portend a poor prognosis in the latter. Therapies for maintenance of sinus rhythm are favoured in this setting. In general, acute and chronic treatment of AF in children does not differ from adults.

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Chapter 10: Management of Atrial Fibrillation in the Emergency Department and Following Acute Myocardial Infarction

BRETT HEILBRON, GEORGE J. KLEIN, MARIO TALAJIC, PETER G. GUERRA

RECOMMENDATIONS FOR THE MANAGEMENT OF AF IN THE EMERGENCY DEPARTMENT:

Class I

1. In stable patients with duration of AF > 48 hours or of uncertain duration in whom a decision has been made to attempt cardioversion, optimize rate control and anticoagulate to an INR of 2.0 – 3.0 for 3 weeks prior to cardioversion. (Level of Evidence: C)
2. In patients with AF of duration > 48 hours or of uncertain duration who are highly symptomatic after efforts to achieve adequate rate control, transesophageal echo to exclude atrial thrombus can be considered prior to cardioversion. (Level of Evidence: B)
3. Select a strategy of rate control or rhythm control based on symptoms and clinical variables (see text. Level of Evidence: B)
4. When a decision is made to cardiovert patients with AF < 48 hours, synchronized DC cardioversion or pharmacological cardioversion may be utilized. See chapter 3. (Level of Evidence: C)
5. When DC cardioversion is chosen, use biphasic waveform when available to increase success and reduce cardioversion energy. (Level of Evidence: B)
6. After acute conversion of an episode of atrial fibrillation or atrial flutter, long-term antithrombotic therapy should be prescribed based on thromboembolic risk and bleeding risk from antithrombotic therapy (see Chapter 12) (Level of Evidence: A)
7. In patients with AF and pre-excitation, perform urgent cardioversion if hemodynamically unstable. If stable, consider using Class 1 (e.g. procainamide) or Class 3 (e.g. ibutilide) antiarrhythmic agents. (Level of Evidence: C)
8. Hospital admission can be limited to highly symptomatic patients, those with structural heart disease, those who have had an embolic event or those at high risk for thromboembolism, and those with failure of rate control in the emergency department. (Level of Evidence: C)

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Class IIa

1. Anticoagulation should be considered for most patients presenting to the ER with AF with either unfractionated heparin or low molecular weight heparin. Exceptions include those already on warfarin with an INR > 2.0 or those in whom the short term risk of bleeding on anticoagulant therapy is believed to exceed the risk of thromboembolism. (Level of Evidence: C)
2. After conversion to sinus rhythm has been achieved, decide whether antiarrhythmic drug therapy is indicated based on the estimated probability of recurrence and the symptoms during AF. (Level of Evidence: C)

CLASS III

1. Do not administer digoxin, calcium channel blocking agents or beta-blocking agents alone to patients with pre-excitation during AF. (Level of Evidence: B)
2. Do not administer adenosine to attempt rate control or cardioversion during AF. (Level of Evidence: B)

RECOMMENDATIONS FOR MANAGEMENT OF PATIENTS WITH ATRIAL FIBRILLATION AND ACUTE MYOCARDIAL INFARCTION:

Class I

1. Use electrical cardioversion for patients requiring urgent restoration of sinus rhythm for hemodynamic reasons. (Level of Evidence: C)
2. Administer beta-blockers to slow a rapid ventricular response in patients without contraindication to beta-blockers. Diltiazem may be used as an alternative. These agents may be given orally or intravenously, depending on the urgency. (Level of Evidence: C)
3. Administer intravenous digitalis or amiodarone to slow a rapid ventricular response in patients with impaired LV function. (Level of Evidence: C)
4. Administer heparin for patients with AF and acute MI, unless contraindicated. (Level of Evidence: C)

Class III

1. Do not administer type IC antiarrhythmic drugs to patients with AF in the setting of acute MI. (Level of Evidence: C)

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Atrial fibrillation is the most common arrhythmia managed by emergency physicians—and there is increasing evidence that selected patients with acute AF can be safely managed in the emergency department without need for hospital admission. The principles of management are identification and treatment of precipitating or underlying causes, hemodynamic stabilization / rate control, reduction of thromboembolism risk, and conversion / maintenance of sinus rhythm.

Identification of precipitating or underlying etiology should be considered as in Chapter 1. A strategy of rate or rhythm control should be chosen, based on the patient's clinical status, the duration of AF, the experience of the treating physician, and the status of anticoagulation. Rate control can usually be achieved with beta blocking or calcium channel blocking agents. Adenosine and digoxin play little or no effective role in controlling rate in the emergency room setting.

Prior to either electric or pharmacologic cardioversion, anticoagulation should be considered. Most patients should be given heparin or low molecular weight heparin while preparing for cardioversion. Patients with AF of over 48 hours duration or of unknown duration should be anticoagulated with warfarin for 3 weeks before elective cardioversion, with pharmacologic rate control in the interim. In patients with AF of duration > 48 hours or of uncertain duration who are highly symptomatic after efforts to achieve adequate rate control, transesophageal echo to exclude atrial thrombus can be considered prior to cardioversion.

All patients should be considered for long term anticoagulation based on thromboembolic risk and bleeding risk from antithrombotic therapy (see Chapter 12). Following restoration of sinus rhythm, a decision regarding use of antiarrhythmic drugs should be made based on the estimated frequency of recurrence and degree of symptoms. Hospitalization and electrocardiographic monitoring may be required in those with severe symptoms, heart failure, or associated thromboembolic events or in those who may be at risk while initiating antiarrhythmic or rate slowing drugs.

In the setting of acute myocardial infarction, beta blockers should be given whenever possible. If beta blockers are contraindicated, the rate can be slowed with digoxin or amiodarone. Cardioversion should be performed if hemodynamically unstable. Class 1C antiarrhythmic drugs should not be administered in this setting.

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Chapter 11: Atrial Flutter – Similarities and Differences

CHRISTOPHER S. SIMPSON, L. BRENT MITCHELL, GEORGE J. KLEIN

RECOMMENDATIONS:

Pharmacologic management

Class I

1. When pharmacologic management for patients with atrial flutter is selected, either the rate-control strategy or the rhythm-control strategy is appropriate (Level of Evidence: C).
2. The pharmacologic agents used for rate-control and for rhythm-control for patients with atrial flutter are the same as those used for patients with atrial fibrillation (Level of Evidence: C).
3. When a Class IC or 1A agent is chosen to treat a patient with atrial flutter, an AV node blocking agent should generally be used concurrently (Level of Evidence: C).

Thromboembolic risk management

Class I

1. As with AF, atrial flutter patients at high risk for systemic emboli should receive chronic oral anticoagulation (Level of Evidence: B).
2. Patients should have therapeutic INR measurements on warfarin for at least 3 weeks before and at least three weeks following the restoration of sinus rhythm, whether by pharmacologic therapy, DC cardioversion, or catheter ablation. Alternatively, cardioversion may be accomplished without prior long-term anticoagulation if the atria have been cleared by low risk findings on a transesophageal echocardiogram (TEE). Following a TEE-guided strategy, patients should be subsequently anticoagulated for at least 3-4 weeks. (Level of Evidence: C).

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Catheter ablation

Class 1:

- 1. Curative catheter ablation for symptomatic atrial flutter may be offered as a first line therapy; presented as a reasonable alternative to pharmacologic therapy. (Level of Evidence: B).**
- 2. Atrioventricular node ablation with permanent pacing should be reserved for patients with symptomatic atrial flutter despite optimal medical therapy when curative ablation is not feasible. (Level of Evidence: C).**

The electrocardiographic pattern of atrial flutter (AFI), regular atrial activation signals at rates of 240 to 340 per minute usually with no intervening isoelectric periods, distinguishes AFI from AF. Nevertheless, there is a strong clinical relationship between AFI and AF. A single patient may have, at different times, paroxysms of both AFI and AF.

As with AF, the pharmacologic treatment of AFI can be directed at rate control (rate-control strategy) or at restoration and maintenance of sinus rhythm (rhythm-control strategy). Because rate control can be more difficult to achieve in patients with AFI than in patients with AF, the rhythm control strategy is often the primary approach to therapy. Class III antiarrhythmic drugs appear more effective in preventing AFI as they prolong atrial refractoriness (sotalol, amiodarone, dofetilide), Class Ic agents may slow the atrial rate and permit 1:1 AV nodal conduction and concomitant administration of AV nodal blocking drugs is recommended.

Atrial flutter appears to have the same risk of thromboembolism as AF. Therefore, the strategy for risk stratification and antithrombotic therapy should be the same for AFI as AF (Chapter 12).

Because AFI is usually due to an anatomically defined re-entrant circuit in the right atrium, catheter ablation has been shown to be highly effective and can be considered a first line therapy along with medical therapy.

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Chapter 12: Therapies for the Prevention of Stroke and Other Vascular Events in Atrial Fibrillation and Flutter

STUART J. CONNOLLY, ANNE M. GILLIS

RECOMMENDATIONS:

Class I:

1. All patients with AF or atrial flutter should be stratified for risk of stroke and vascular events, and for risk of bleeding with anticoagulation therapy. (Level of Evidence: A)
2. Patients with AF or atrial flutter at high risk of stroke should receive oral anticoagulation unless there is an excessive risk of hemorrhage. (Level of Evidence: A)
6. Patients with AF or atrial flutter at intermediate risk should receive either oral anticoagulation or aspirin (75-325 mg per day) and low risk patients may receive ASA, unless there is excessive risk of bleeding (Level of Evidence: B)
7. Patients undergoing DC cardioversion for AF or atrial flutter should receive therapeutic oral anticoagulation for 3-4 weeks prior to, and after, the procedure. Low risk patients may be cardioverted without oral anticoagulation if done within 48 hours of arrhythmia onset. Transesophageal echocardiography guided cardioversion (ACUTE Trial Protocol) is an acceptable alternative to oral anticoagulation. (Level of Evidence: C)
8. When reversal of oral anticoagulation is required (such as for surgery), therapy should be discontinued 5-6 days beforehand. Consideration should be given to use of heparin or LMH during this period in higher risk patients. (Level of Evidence C)

The overall risk of stroke in patients with AF is 4.5 % per year. This risk, however, varies according to the clinical factors associated with AF. In healthy young AF patients with no other clinical conditions, the risk of stroke and other vascular events is <1% per year. On the other hand, in elderly patients with multiple risk factors the risk of vascular events exceeds 10% per year. Therefore risk stratification should guide therapy. The 1998 American College of Chest Physicians' risk stratification is widely accepted, comprehensive and is recommended. The tables below outline the factors used to stratify risk and the antithrombotic therapy recommended for each risk group.

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RISK FACTOR STRATIFICATION

HIGH-RISK FACTORS

- ▶ History of stroke/TIA
- ▶ Hypertension
- ▶ Reduced LV function
- ▶ Age > 75 years
- ▶ Mitral stenosis
- ▶ Prosthetic heart valve

MODERATE-RISK FACTORS

- ▶ Age 65 – 75 years
- ▶ Diabetes
- ▶ Coronary artery disease without LV dysfunction

ANTITHROMBOTIC THERAPY BY RISK GROUPS

RISK FACTORS

- ▶ Any high-risk factor or more than one moderate-risk factor
- ▶ One moderate-risk factor
- ▶ No high-risk factors and no moderate-risk factors

RECOMMENDED THERAPY

- Warfarin
(target INR 2.5, range 2.0–3.0)
- Aspirin 75-325 mg/day or warfarin
(target INR 2.5 range 2.0–3.0)
- Aspirin 75-325 mg/day

Intermittent AF should be considered as an equivalent risk to permanent AF. Atrial flutter should also be managed with the same criteria as AF. In all patients the risk of thromboembolism should be balanced against the risk of hemorrhage. High risk patients should be anticoagulated to maintain an INR from 2.0 – 3.0. Intermediate risk patients may be treated with either ASA (75-325 mg daily) or anticoagulation. Low risk patients may be treated with ASA if there is little risk of bleeding.

In patients with AF of greater than 48 hours' duration, adequate anticoagulation should be given for 3-4 weeks before and after cardioversion. If cardioversion is required more urgently, transesophageal echocardiography, to rule out left atrial thrombus, should be performed prior to cardioversion and anticoagulation given for 3-4 weeks following.

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