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CME Article

Narrow QRS complex tachycardia presenting as palpitation

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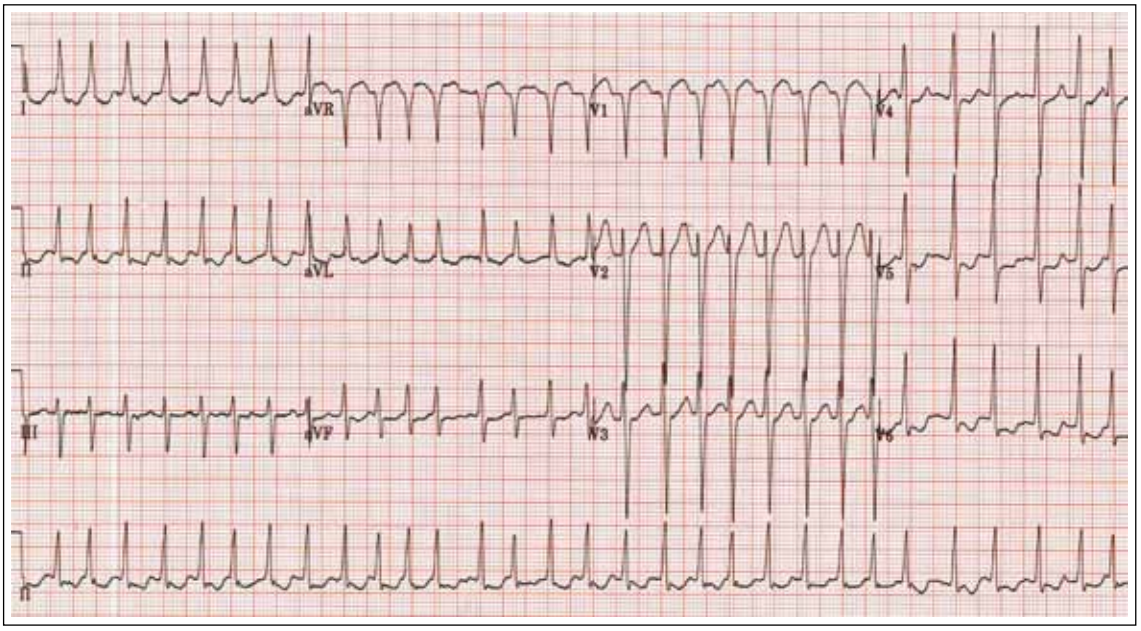

Fig. 1 ECG shows irregular narrow QRS complex tachycardia without any discernable P waves, i.e. atrial fibrillation.

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CASE 1

CLINICAL PRESENTATION

A 79-year-old Chinese woman presented with a history of palpitations for five days. It was continuous and associated with intermittent giddiness. She did not have any syncope, fall or history of chest pain or shortness of breath. There was a past history of hypertension, diabetes mellitus and transient ischaemic attacks. She had a permanent pacemaker implanted in November 2009 for symptomatic sick sinus syndrome. At presentation, the patient's heart rate was 180 beats per minute (bpm). Her pulse was irregular and the blood pressure was 140/80 mmHg. She did not have any clinical features of heart failure. Examination of the nervous system was unremarkable. An electrocardiogram (ECG) was done (Fig. 1). Based on the ECG interpretation, she was given intravenous digoxin infusion (250 mcg) over one hour, and her heart rate slowed down to 120 bpm. She was also started on oral metoprolol 25 mg bid and hospitalised for further management.

ECG INTERPRETATION

Fig. 1 shows a narrow complex tachycardia at a heart rate of 189 bpm. The RR interval is irregularly irregular, with no discernable P waves. This is consistent with atrial fibrillation with a rapid ventricular response.

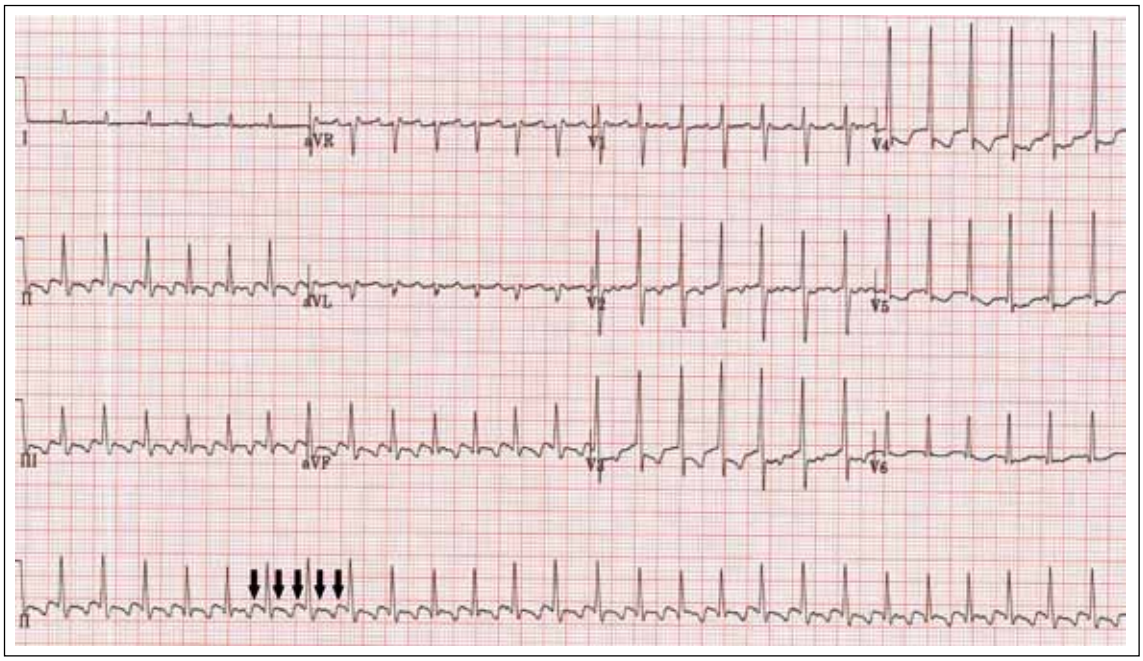

Fig. 2 ECG shows regular narrow QRS complex tachycardia with flutter waves (arrows) seen in leads II, III and aVF. Alternate flutter wave is not conducted, i.e. 2:1 AV conduction.

CLINICAL COURSE

This was the first documented presentation of paroxysmal atrial fibrillation in the patient. Thyroid function test was normal. Echocardiogram showed normal chamber sizes and normal left ventricular systolic function. She was given intravenous digoxin for rate control in the emergency department. This was followed by oral digoxin and oral metoprolol in the ward. The patient spontaneously reverted to sinus rhythm. Her CHADS2 score was 5. In view of the high CHADS2 score, she was started on oral warfarin for prevention of thrombo-embolism with a target international normalised ratio (INR) of 2–3. The patient was discharged with oral metoprolol and warfarin in addition to her other previous medications.

CASE 2

CLINICAL PRESENTATION

A 65-year-old Chinese woman presented to the emergency department with intermittent episodes of palpitations for the past two weeks. The latest episode lasted for about 24 hours and was associated with nausea. She did not have any giddiness, syncope or loss of consciousness. The patient had a past history of ischaemic heart disease and had coronary artery bypass grafting done two years ago. Her cardiovascular risk factors were hypertension and dyslipidaemia. On arrival at the emergency department, her heart rate was 160 bpm and blood pressure was 120/75 mmHg. She did not have any features of heart failure. What does the ECG show (Fig. 2)?

ECG INTERPRETATION

Fig. 2 shows a regular narrow complex tachycardia at a heart rate of 165 bpm. 'Saw-tooth' flutter (F) waves (arrows) are seen in leads II, III and aVF. The rate of the flutter waves is about 320 bpm with a 2:1 atrioventricular (AV) block, giving it a ventricular rate of about 165 bpm.

CLINICAL COURSE

Based on the ECG interpretation, 6 mg of intravenous adenosine was administered to the patient as a fast bolus. This was followed by 150 mg intravenous amiodarone infusion administered over 30 minutes. Repeat ECG showed that she was pharmacologically converted to sinus rhythm, with a heart rate of 94 bpm. She was admitted for observation but remained asymptomatic during her hospital stay. Echocardiogram showed normal left ventricular systolic function, dilated left atrium and left ventricular hypertrophy. She was offered electrophysiology study and radiofrequency ablation for the atrial flutter to prevent recurrences. However, the patient preferred medical therapy. She was started on oral amiodarone for maintenance of sinus rhythm. She was also started on warfarin therapy with a target INR of 2–3 for stroke prevention.

DISCUSSION

Atrial fibrillation

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. It is more prevalent in men and with increasing age.[1] Adverse consequences of AF are due to a reduction in cardiac output and left atrial or atrial appendage thrombus formation, which may lead to systemic embolisation.[2] The most important risk factors (besides increasing age) for atrial fibrillation are: (1) hypertension; (2) heart failure; (3) ischaemic heart disease; (4) mitral valve disease; and (5) thyrotoxicosis.

The electrocardiographic features of AF include: absent P waves; presence of fibrillatory or 'f' waves at a rate of 350–600 bpm, with 'f' waves varying in amplitude, morphology and intervals; unsually narrow QRS complexes (unless there is pre-existing bundle branch block, rate-related aberrant conduction or pre-excitation); ventricular rates that vary from 90 to 170 bpm (with ventricular rates in excess of 200 bpm considered to be unusual and suggestive of catecholamine excess or conduction through an accessory pathway); and irregular rhythm.

The American College of Cardiology/American Heart Association/European Society of Cardiology (ACC/AHA/ESC) proposed the following classifications for AF:[3] (1) Paroxysmal: AF is classified as paroxysmal if the episode terminates spontaneously in less than seven days (usually 24 hours); (2) Persistent: AF is classified as persistent if it fails to self-terminate within seven days; (3) Permanent: Permanent AF is considered to be present if the arrhythmia lasts for more than one year and cardioversion has either not been attempted or has failed; and (4) Lone: Lone AF describes paroxysmal, persistent or permanent AF in individuals without structural heart disease.

History-taking, physical examination as well as specific laboratory and cardiac testings are all part of the evaluation of AF. The minimum evaluation suggested by the ACC/AHA/ESC guidelines is as follows: history-taking and physical examination to define symptoms of AF; clinical pattern or classification; frequency and duration of AF episodes; any precipitating causes and modes of termination of AF; and response to drug therapy. However, episodes of AF may be asymptomatic.[4] ECG, chest radiography, echocardiogram and assessment of thyroid function are the four minimum investigations required for evaluation of AF. Additional tests, such as Holter monitoring, exercise testing, event recorders and electro-physiologic studies, may be required in certain settings.

Treatment

In the treatment of AF, the issues of rhythm control (i.e. reversion to normal sinus rhythm followed by maintenance of sinus rhythm) vs. rate control (i.e. administration of medications to control the ventricular rate in patients with chronic AF) as well as prevention of systemic embolisation need to be addressed.

In the past, many physicians preferred rhythm control. However, the results of two large AF trials (AFFIRM and RACE) have changed the management strategy toward rate control.[5,6] These trials concluded that rate control has similar outcomes as rhythm control and that it is the preferred initial approach in the management of AF. The three primary settings in which rhythm control strategy using antiarrhythmic drugs to maintain sinus rhythm should be considered are persistent symptoms (palpitations, dyspnoea, lightheadedness, angina, presyncope and heart failure) despite adequate rate control, inability to attain adequate rate control and patient preference.[7] Rate control in AF can be achieved by slowing down AV nodal conduction with a beta blocker, a non-dihydropyridine calcium channel blocker (diltiazem or verapamil), or in patients with heart failure or hypotension, digoxin. Rate control should be assessed both at rest and on exertion.

There are two standard approaches for converting AF to sinus rhythm – synchronised external cardioversion and pharmacologic cardioversion. Electrical cardioversion is preferred due to greater efficacy and a low risk of proarrhythmia. The overall success of electrical cardioversion is 75%–93% and is inversely related both to the duration of AF and the left atrial size.[8] DC cardioversion is also preferred in patients who are haemodynamically unstable. Drugs used for pharmacologic cardioversion include flecainide, propafenone and amiodarone.[9] However, only 20%–30% of patients who are successfully cardioverted maintain sinus rhythm for more than one year without chronic antiarrhythmic therapy.[2] As recommended by the 2006 ACC/AHA/ESC guidelines, the choice of drugs for maintenance of sinus rhythm varies with the clinical settings. Flecainide or propafenone is preferred in patients with no or minimal structural heart disease, while amiodarone is preferred in patients with reduced left ventricular ejection fraction or heart failure.[3] The 2010 ESC guidelines and the 2011 ACC/AHA/HRS focused update on AF management have added dronedarone as another alternative first-line agent.[10,11]

Surgical ablation and radiofrequency catheter ablation can be used for maintenance of sinus rhythm. Pulmonary vein isolation using radiofrequency catheter ablation is increasingly performed in AF patients. The

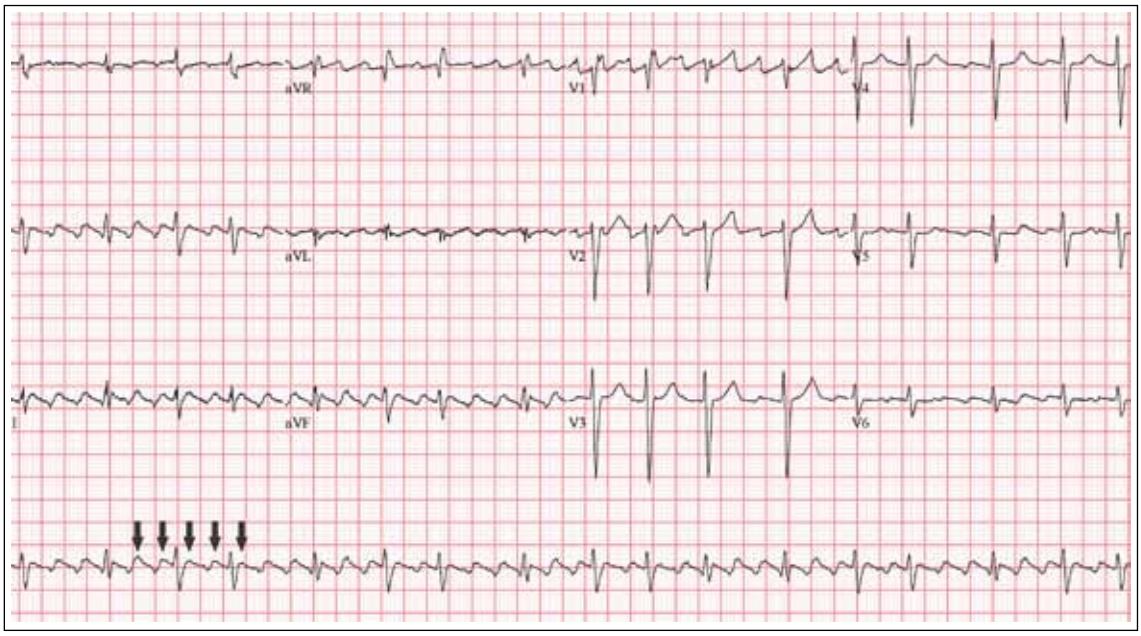

Fig. 3 ECG shows atrial flutter with variable AV conduction. 'Saw tooth' flutter waves (arrows) are seen in leads II, III and aVF.

detailed description and indication of catheter ablation is beyond the scope of this article. Radiofrequency AV nodal-His bundle ablation with permanent pacemaker implantation and AV nodal conduction modification are non-pharmacologic therapies for achieving rate control in patients who do not respond to pharmacologic therapy.

Anticoagulation in AF

Anticoagulation in AF is required in two settings – during cardioversion to sinus rhythm and during long-term management in AF. In non-valvular AF of more than 48 hours' duration, the guidelines strongly recommend warfarin for 3–4 weeks prior to and after cardioversion.[2,12] The recommended target INR is 2–3. Alternatively, prior to cardioversion, screening transoesophageal echocardiogram to document the absence of atrial thrombi can be performed. After cardioversion, it is recommended that warfarin therapy should be continued for four weeks.[7,9]

The incidence of stroke associated with AF is 3%–5% per year in the absence of anticoagulation. AF significantly increases the risk of stroke (relative risk of 2.4 in men and 3.0 in women).[13,14] However, the risk varies markedly among patients. The choice of therapy, i.e. anticoagulant (warfarin/dabigatran) vs. antiplatelet (aspirin/plavix) varies with the estimated risk of ischaemic stroke or peripheral embolisation. A number of risk stratification models are available for patients with AF, but CHADS2 score is the most widely used and validated model.[15]

In CHADS2, 'C' stands for congestive heart failure (any history), 'H' for hypertension, 'A' for age > 75 years, 'D' for diabetes mellitus, and 'S' for secondary prevention in patients with prior ischaemic stroke/transient ischaemic stroke (TIA). Each risk factor in the CHADS2 score is given one point, except for stroke/TIA history, which is given 2 points. Therefore, the CHADS2 score can range from 0 to 6. Patients with a CHADS2 score of 0 are at low risk for ischaemic stroke or peripheral embolisation (0.5% per year) and can be managed without any anticoagulation. Patients with CHADS2 score ≥ 2 are at high risk (> 4% per year) and should be treated with anticoagulant therapy (warfarin/dabigatran). Patients with a CHADS2 score of 1 are at intermediate risk (1.5%–2.5% per year). The choice of therapy in this group depends on many factors, including patient preference. Aspirin alone is a reasonable option in patients with a CHADS2 score of 1. A target INR of 2–3 is recommended for most patients with AF who are on warfarin. Advanced age (> 75 years) is an independent risk factor for bleeding during anticoagulation, and some experts have argued that a lower INR target, i.e. 1.8–2.5 is a reasonable compromise between toxicity and efficacy for some patients in this age group.[3]

The choice of whether to start warfarin alone or in combination with unfractionated heparin or low-molecular-weight heparin is based on an assessment of the risk of a thrombus developing within the next several days vs. the risk of bleeding complications. In most patients with non-valvular AF, the risk of stroke during those few days typically required to reach therapeutic INR is very low. Therefore, it is reasonable to administer warfarin on

an outpatient basis without bridging. For patients with non-valvular AF deemed to be at high risk of thrombus formation/thromboembolism (such as a history of prior cerebrovascular event/TIA or the presence of intracardiac thrombus) and low risk of intracranial bleeding, initiation of warfarin with a heparin bridging regimen is reasonable. Despite the compelling evidence that anticoagulation with warfarin reduces the risk of stroke in most patients with AF, warfarin therapy continues to be underutilised.[16] Another problem with warfarin is that maintenance of the target INR, which is often not achieved, and the failure to maintain a therapeutic INR are associated with worse outcomes [17] Until recently, warfarin was the most effective drug available for prevention of systemic embolisation in patients with AF. However, dabigatran (a reversible direct thrombin inhibitor), an alternative oral anticoagulant, has demonstrated superiority to adjusted dose of warfarin in a randomised control trial.[18] Dabigatran has an advantage over warfarin in terms of efficacy and safety, and it does not require monitoring of the INR. Its disadvantages include twice-daily dosing, higher pharmaceutical cost, the lack of an antidote/reversing agent and the potential need for dose adjustment in patient with chronic kidney disease (mild to moderate severity).

Atrial Flutter

Atrial flutter is a reentrant arrhythmia. It is characterised by rapid regular atrial depolarisations at a characteristic rate of approximately 300 (range 240–340) bpm. Although many issues related to atrial flutter (e.g. restoration to sinus rhythm, maintenance of sinus rhythm, rate control and prevention of systemic embolism) are similar to those of AF, it is, however, a fairly distinct arrhythmia.

ECG features of the common type of atrial flutter are the presence of 'saw tooth' flutter waves, typically seen in inferior leads II, III and aVF. Flutter waves in these leads are fairly regular, with constant amplitude, duration, morphology and reproducibility throughout the cardiac cycle. However, in lead V1, the flutter waves are often upright, mimicking discrete P waves. In untreated patients, the ventricular response is usually one-half of the atrial rate (i.e. 2:1 AV nodal conduction with a ventricular rate of approximately 150 bpm). A diagnosis of atrial flutter should always be considered in regular narrow QRS complex tachycardia whenever the ventricular rate is around 150 bpm. The QRS complex is narrow, unless there is functional aberration or pre-existing bundle branch.[19] One of the flutter waves may be obscured by the QRS complex or the ST-T wave in patients with 2:1 AV nodal conduction. In this setting, atrial flutter may be misdiagnosed as sinus tachycardia or a paroxysmal supraventricular tachycardia with a rate of 150 bpm. The rhythm is regular in atrial flutter if there is a constant AV nodal conduction (e.g. 2:1). However, if the AV conduction is variable, the rhythm will be irregular (Fig. 3).

The evaluation of atrial flutter is similar to that for AF. History-taking and physical examination are essential parts of evaluation. Treatment issues are also similar to those of AF, and include rhythm control (conversion to sinus rhythm and maintenance of sinus rhythm) vs. rate control (administration of AV node blocking agent to slow down the ventricular rate in atrial flutter) and the prevention of systemic embolisation.

For rhythm control, the standard approach for converting to sinus rhythm is synchronised internal or external DC cardioversion or pharmacologic cardioversion with Class 1A (procainamide, disopyramide), Class 1C (flecainide, propafenone) or Class III antiarrhythmic agents (amiodarone, sotalol). DC cardioversion is performed in haemodynamically unstable patients, while both DC and pharmacologic cardioversions can be performed in haemodynamically stable patients. The rate of recurrence of atrial flutter is about 50% at one year in the absence of antiarrhythmic therapy for maintenance of sinus rhythm.[20] As with AF, the pharmacologic strategy for maintenance of sinus rhythm requires Class 1A, 1C or III antiarrhythmic drugs. However, because of the high rate of recurrence and proarrhythmic effects of drugs, radiofrequency ablation is increasingly preferred over long-term pharmacologic therapy in patients with typical atrial flutter. The efficacy and safety of radiofrequency ablation was illustrated in a report from the NASPE Prospective Catheter Ablation Registry. Acute success was achieved in 86% of cases, with a long-term recurrence rate of 15%.[21]

Rate control involves the administration of calcium channel blocker (verapamil, diltiazem) or beta blockers. Digoxin is used less frequently, as its rate-lowering effect is offset during exertion. However, the main indication of digoxin use is in patients of heart failure with impaired left ventricular systolic function and atrial flutter or AF. The risk of systemic embolisation in atrial flutter is perceived to be similar to that in AF. Therefore, the choice between warfarin and aspirin is based on perceived embolic risk, as in the case of AF.

ABSTRACT

Atrial fibrillation is the most common sustained cardiac arrhythmia. The rhythm in atrial fibrillation is irregular. Correct interpretation of the electrocardiogram (ECG) is essential. Atrial

flutter can present as regular or irregular narrow QRS complex tachycardia. Knowledge of the ECG features of atrial flutter will help to differentiate it from paroxysmal supraventricular tachycardia. The treatment strategy in atrial fibrillation should focus on rhythm control vs. rate control, and anticoagulation should be started based on the calculated risk of systemic embolisation. Atrial flutter is a unique arrhythmia that has similar management strategies to those of atrial fibrillation; however, radiofrequency ablation is increasingly preferred due to its higher rate of efficacy and safety compared to pharmacological therapy.

Keywords: anticoagulation, atrial fibrillation, atrial flutter, narrow QRS tachycardia, palpitations, rate control, rhythm control

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REFERENCES

1. Go AS, Hylek EM, Phillips KA, et al. Prevalence of diagnosed atrial fibrillation in adults: national implications for rhythm management and stroke prevention: the AnTicoagulation and Risk Factors in Atrial Fibrillation (ATRIA) Study. JAMA 2001; 285:2370-5.
2. Atrial fibrillation: current understandings and research imperatives. The National Heart, Lung, and Blood Institute Working Group on Atrial Fibrillation. J Am Coll Cardiol 1993; 22:1830-4.
3. European Heart Rhythm Association, Heart Rhythem Society, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation). J Am Coll Cardiol 2006; 48:854-906.
4. Israel CW, Grönefeld G, Ehrlich JR, Li YG, Hohnloser SH. Long-term risk of recurrent atrial fibrillation as documented by an implantable monitoring device: implications for optimal patient care. J Am Coll Cardiol 2004; 43:47-52.
5. Wyse DG, Waldo AL, DiMarco JP, et al. A comparison of rate control and rhythm control in patients with atrial fibrillation. N Engl J Med 2002; 347:1825-33.
6. Van Gelder IC, Hagens VE, Bosker HA, et al. A comparison of rate control and rhythm control in patients with recurrent persistent atrial fibrillation. N Engl J Med 2002; 347:1834-40.
7. Snow V, Weiss KB, LeFevre M, et al. Management of newly detected atrial fibrillation: a clinical practice guideline from the American Academy of Family Physicians and the American College of Physicians. Ann Intern Med 2003; 139:1009-17.
8. Gallagher MM, Guo XH, Poloniecki JD, et al. Initial energy setting, outcome and efficiency in direct current cardioversion of atrial fibrillation and flutter. J Am Coll Cardiol 2001; 38:1498-504.
9. McNamara RL, Tamariz LJ, Segal JB, Bass EB. Management of atrial fibrillation: a review of the evidence for the role of pharmacologic therapy, electrical cardioversion, and echocardiography. Ann Intern Med 2003; 139:1018-33.
10. European Heart Rhythm Association, European Association for Cardio-Thoracic Surgery, Camm AJ, et al. Guidelines for the management of atrial fibrillation: the Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). Europace 2010; 12:1360-420.
11. Wann LS, Curtis AB, January CT, et al. 2011 ACCF/AHA/HRS focused update on the management of patients with atrial fibrillation (Updating the 2006 Guideline): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Heart Rhythm 2011; 8:157-76.
12. Singer DE, Albers GW, Dalen JE, et al. Antithrombotic therapy in atrial fibrillation: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest 2008; 133:546S-592S.
13. Wolf PA, Kannel WB, McGee DL, et al. Duration of atrial fibrillation and imminence of stroke: the Framingham study. Stroke 1983; 14:664-7.
14. Frost L, Engholm G, Johnsen S, Møller H, Husted S. Incident stroke after discharge from the hospital with a diagnosis of atrial fibrillation. Am J Med 2000; 108:36-40.
15. Gage BF, Waterman AD, Shannon W, et al. Validation of clinical classification schemes for predicting stroke: results from the National Registry of Atrial Fibrillation. JAMA 2001; 285:2864-70.
16. Go AS, Hylek EM, Borowsky LH, et al. Warfarin use among ambulatory patients with nonvalvular atrial fibrillation: the anticoagulation and risk factors in atrial fibrillation (ATRIA) study. Ann Intern Med 1999; 131:927-34.
17. Reynolds MW, Fahrbach K, Hauch O, et al. Warfarin anticoagulation and outcomes in patients with atrial fibrillation: a systematic review and metaanalysis. Chest 2004; 126:1938-45.
18. Connolly SJ, Ezekowitz MD, Yusuf S, et al. Dabigatran versus warfarin in patients with atrial fibrillation. N Engl J Med 2009; 361:1139-51.
19. Singh D, Teo SG, Poh KK. Regular narrow complex tachycardia. Singapore Med J 2011; 52:146-9.
20. Crijns HJ, Van Gelder IC, Tieleman RG, et al. Long-term outcome of electrical cardioversion in patients with chronic atrial flutter. Heart 1997; 77:56-61.
21. Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. Pacing Clin Electrophysiol 2000; 23:1020-8.

SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 201107A)

	True	False
Question 1. Regarding atrial fibrillation:		
(a) Its prevalence increases with age.	☐	☐
(b) It increases the risk of stroke.	☐	☐
(c) It is more common in men.	☐	☐
(d) The pulse is regular on examination.	☐	☐
Question 2. ECG in atrial fibrillation shows:		
(a) Regular R-R interval.	☐	☐
(b) Absence of P waves.	☐	☐
(c) Ventricular rate of 90–170 bpm.	☐	☐
(d) QRS complexes that are usually wide.	☐	☐
Question 3. In CHADS2 score:		
(a) 'A' stand for age > 60 years.	☐	☐
(b) 'D' stands for diabetes mellitus.	☐	☐
(c) 'H' stands for hypertension.	☐	☐
(d) A previous history of stroke or TIA is assigned 2 points.	☐	☐
Question 4. Regarding ECG in atrial flutter:		
(a) It shows regular 'saw tooth' flutter waves in leads II, III and aVF.	☐	☐
(b) Discrete flutter waves in lead V1 may mimic P waves.	☐	☐
(c) R-R interval is always irregular.	☐	☐
(d) In untreated patients, the typical ventricular rate in atrial flutter with 2:1 AV conduction is around 150 bpm.	☐	☐
Question 5. A 76-year-old man with a history of hypertension and diabetes mellitus presented with newly diagnosed asymptomatic atrial fibrillation on routine ECG screening. In this patient:		
(a) The CHADS2 score is 1.	☐	☐
(b) Anticoagulation should be commenced.	☐	☐
(c) Rhythm control is preferred over rate control.	☐	☐
(d) Echocardiogram should be done.	☐	☐

Doctor's particulars:

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MCR number: _____ Specialty: _____

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SUBMISSION INSTRUCTIONS:
(1) Log on at the SMJ website: http://www.sma.org.sg/cme/smj and select the appropriate set of questions. (2) Select your answers and provide your name, email address and MCR number. Click on "Submit answers" to submit.

RESULTS:
(1) Answers will be published in the SMJ September 2011 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 02 September 2011. (3) All online submissions will receive an automatic email acknowledgment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.

Deadline for submission: (July 2011 SMJ 3B CME programme): 12 noon, 26 August 2011.