

CME Article

Managing ventricular ectopics: are ventricular ectopic beats just an annoyance?

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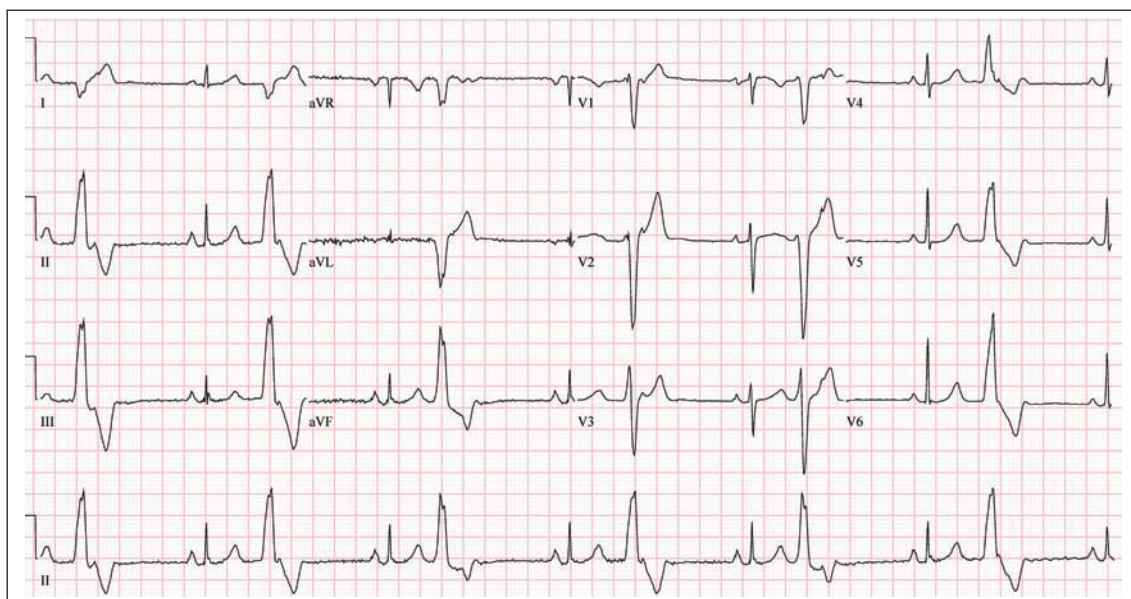


Fig. 1 ECG shows ventricular bigeminy.

CLINICAL PRESENTATION

A 40-year-old woman was referred for an evaluation of an incidental finding of frequent ventricular ectopic beats found in the resting 12-lead electrocardiogram (ECG). She had occasional palpitations but no syncope or a change in her effort tolerance. The patient had a past history of “a

small hole in the heart”. There was no significant family history of heart disease or sudden cardiac death. Physical examination was unremarkable apart from a systolic murmur that was audible over the left sternal edge. Based on the 12-lead ECG (Fig 1), she was treated with metoprolol 25 mg twice daily. What does Fig. 1 show?

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ECG INTERPRETATION

Fig. 1 shows ventricular bigeminy. The premature ventricular complex (PVC) has a left bundle branch block morphology and inferior axis, suggesting that it originates from the right ventricular outflow tract (RVOT).

CLINICAL COURSE

The patient underwent 24-hour ECG monitoring, which showed 28,351 isolated PVCs with frequent runs of ventricular bigeminy, ventricular couplet and non-sustained ventricular tachycardia (NSVT), with the longest run at 25 consecutive beats at a ventricular rate of 177 beats per minute (bpm). Transthoracic echocardiogram was normal except for a small restrictive perimembranous ventricular septal defect. The coronary angiogram was also normal. The beta blocker therapy did not significantly reduce the PVC load and severity. The high PVC load (> 20,000/24 hours) and the presence of closely coupled PVCs increased our concern, as the possibility of a malignant variant of RVOT ventricular arrhythmia is associated with a high risk of sudden cardiac death (SCD). A successful RVOT radiofrequency catheter ablation was performed, with complete abolishment of the PVCs.

DISCUSSION

Sir James Mackenzie was the first to describe extrasystoles in 1890 when he noticed that chambers of the heart could beat outside of their correct order.⁽¹⁾ Today, ventricular extrasystoles are also called premature ventricular complexes or contractions, ventricular premature beats or ventricular ectopics. They are caused by the spontaneous firing of ectopic foci in the ventricle(s). Hence, on surface ECGs, they are reflected by QRS complexes that are wide (> 120 ms) and not preceded by P waves.

Most patients with PVCs are asymptomatic, although a small minority may experience palpitations or dizziness. PVCs are one of the most common arrhythmias and can occur in patients with or without underlying heart disease. The prognostic importance of PVCs is variable. However, most subjects, even with frequent PVCs, have excellent outcome if there is no underlying structural heart disease or congenital channelopathies such as the Brugada syndrome or prolonged QT syndrome. On the other hand, PVCs have been associated with an increased risk of sudden death in some patients. The presence and severity of the underlying structural heart disease is the most important determinant of a poorer prognosis, followed by the frequency and characteristics of the PVCs. Frequent, multimorphic PVCs, particularly in the

context of underlying poor myocardial function, connote a poorer prognosis.

Prevalence

A population study showed that PVCs were recorded on standard 12-lead ECGs in 0.8% of subjects in a healthy military population, with a prevalence of 0.5% in those under the age of 20 years and 2.2% in those over 50 years of age.⁽²⁾ In the Framingham Heart Study, the prevalence of patients having \geq one PVC per hour in a one-hour ambulatory ECG monitoring was 33% in men and 32% in women without coronary artery disease (CAD). Among patients with established CAD, the prevalence was 58% in men and 49% in women.⁽³⁾ The wide variability of data reported is likely related to the different durations of monitoring for PVC prevalence, which correlates with increasing age, the presence of heart disease, hypertension, faster sinus rates, African-American ethnicity, male gender, lower educational attainment and lower serum magnesium or potassium levels.⁽⁴⁾

Pathophysiology

The probable mechanisms for PVCs include enhanced automaticity, re-entry and triggered activity. In enhanced automaticity, an ectopic ventricular focus fires spontaneously. This may occur in response to a heightened adrenergic state, myocardial ischaemia or electrolyte imbalance. Early after-depolarisations triggered by a preceding impulse can lead to premature ventricular ectopics if the cell membrane activation threshold is exceeded. This mechanism is often responsible for bradycardia-associated PVCs, but is also implicated in PVCs induced by ischaemia, electrolyte abnormalities and digoxin toxicity. Re-entrant PVCs typically originate in areas of differential conduction due to scarring or ischaemia.

PVC in structurally normal heart

What is the significance of PVCs in a structurally normal heart? Kennedy et al and Engstrom et al found no correlation between frequent/complex PVCs and mortality or cardiac events in men without CAD.^(5,6) However, there were conflicting findings in other studies.^(7,8) In the Framingham Heart Study, men with frequent PVCs had a two-fold increase in the five-year mortality risk and cardiac event rate (after correction of other cardiovascular risk factors) compared to those without.⁽⁷⁾ Also, in the Multiple Risk Factor Intervention Trial (MRFIT), PVCs were associated with a significantly higher risk of SCD with an adjusted risk ratio (RR) of 3.0, but were not associated with any significant increase in the risk of

deaths from CAD. Frequent or complex PVCs (multiform, pairs, runs, R-on-T) led to a significantly higher risk of SCD (adjusted RR 4.2).⁽⁸⁾ Both the Framingham and MRFIT studies followed up their subjects for up to six and seven years, respectively. Despite the conflicting data, in general, PVCs in healthy individuals without any structural heart disease do not usually pose any risk of SCD.

PVC in heart disease

The incidence, frequency and complexity of ventricular arrhythmias are greater in the presence of known or suspected heart disease. Intuitively, the management of PVCs centres around the exclusion of structural and coronary heart diseases.

Post myocardial infarction

The incidence of spontaneous ventricular arrhythmias in the post-infarction period can be as high as 85% for PVCs and 11% for NSVT.⁽⁹⁾ In the GISSI 2 study, the presence of more than ten PVCs per hour prior to discharge was associated with 6% mortality. Frequent PVCs itself was also an independent risk factor for total cardiac death and SCD in the first six months following the infarction.⁽¹⁰⁾ In addition, polymorphic PVCs connote a poorer prognosis than uniform morphologic PVCs. The presence of complex PVCs (defined as R-on-T, runs of two or more, multiform or bigeminal complexes) conferred a 25% risk of cardiac death, compared with 6% in men who were free of PVCs by five years.⁽¹¹⁾ More importantly, a large multicentre study concluded that PVCs and left ventricular dysfunction were independently related to mortality risk.⁽¹²⁾ Left ventricular ejection fraction (LVEF) below 30% was found to be a better predictor of early mortality (less than six months) and the presence of ventricular arrhythmias was a better predictor of late mortality (after six months).⁽¹²⁾ The rate of PVCs per hour measured one year after the index infarction has also been shown to be highly predictive of subsequent death.⁽¹³⁾ Therefore, it is reasonable to conclude that the presence of PVCs after acute myocardial infarction is associated with an increased total mortality in some patients, particularly those with impaired left ventricular function. Its significance is enhanced with the complexity and rate of its occurrence.

Stable coronary artery disease (CAD)

Califf et al evaluated the significance of ventricular arrhythmias using 24-hour ambulatory monitoring in patients with CAD. They found that ventricular arrhythmias were independently associated with

subsequent mortality, and were strongly related to poor left ventricular function (i.e. LVEF).⁽¹⁴⁾ However, after adjustment was made for LVEF, no significant association remained between ventricular arrhythmias and mortality.

Cardiomyopathy

It has been estimated that 70%–95% of patients with cardiomyopathy and congestive heart failure have frequent PVCs, and 40%–80% manifest runs of NSVT, which is associated with higher mortality.⁽¹⁵⁾

Exercise-induced PVCs

Numerous studies have reported the association of PVCs during exercise with all-cause mortality irrespective of the presence of cardiopulmonary disease or exercise-induced ischaemia.^(16,17) Its predictive value was also shown to be independent of resting PVCs.⁽¹⁶⁾ More importantly, PVCs predicted cardiovascular mortality to the same degree as a positive ischaemic response, and its predictive value was independent of the presence or absence of ischaemic changes on the exercise ECG.⁽¹⁷⁾ Another study of 29,244 patients over five years demonstrated that frequent PVCs during the recovery phase of the exercise stress test was a stronger predictor of an increased risk of mortality.⁽¹⁸⁾ However, a few other studies have failed to establish this relationship. The association between exercise-induced PVCs and cardiovascular mortality is plausible, but the extent of its clinical implication in asymptomatic individuals with structurally normal hearts is as yet uncertain. Most of the studies to date were merely designed to determine the predictive value of PVCs and did not consider its mechanism during exercise.

PVC in the elderly

The prevalence of PVCs increases with age, and their occurrence has been associated with an increase in total and cardiovascular mortality. An epidemiological study demonstrated that PVCs in subjects with structurally normal hearts carried no adverse prognostic significance under the age of 30 years, but in those older than 30 years, the presence of PVCs began to influence the risk of CAD.⁽¹⁹⁾ Another study in the Netherlands also suggested that the presence of PVCs and a fast baseline heart rate (which are markers of sympathetic dominance) were predictive of mortality in old age.⁽²⁰⁾ This, however, remains a contentious issue, with studies on healthy geriatric individuals countering this finding.⁽²¹⁾ From these conflicting results, it is reasonable to conclude that the prognostic significance of PVCs depends primarily on the underlying substrate of structural heart disease, the prevalence of which tends to increase with advancing age,

thus leading to the observation of a correlation between age and prevalence of PVCs.

PVC in special groups

Pregnancy

PVCs may increase in frequency during pregnancy in otherwise healthy women. Fortunately, most of these are benign and do not require specific treatment. Most antiarrhythmic drugs are safe except for amiodarone, which has been associated with significant foetal abnormalities. Nevertheless, reassurance is the optimal management if no reversible cause is found.

Hypertrophic obstructive cardiomyopathy (HCM)

To date, there is limited data suggesting that frequent PVCs are, by themselves, indicative of an increased risk of sustained ventricular arrhythmia. It has a low positive and relatively high negative predictive value for sudden death despite its frequent occurrence in this population. In a 24-hour Holter monitoring study, 88% of patients with HCM had PVCs, including 12% with > 500 PVCs, 42% with couplets, 37% with SVT, and 31% with NSVT.⁽²²⁾ Treatment of PVCs is warranted only in symptomatic patients. The 2006 American Heart Association/American College of Cardiology/European Society of Cardiology (AHA/ACC/ESC) consensus statement on the management of HCM recommended beta-blockers or verapamil, if indicated.⁽²³⁾

Idiopathic ventricular outflow tract ventricular tachycardia

Idiopathic VT is a fairly common type of VT, with a structurally normal heart seen in young to middle aged individuals. The characteristic ECG pattern for these arrhythmias is a left bundle-branch block pattern with inferior-axis morphology if it is of an RVOT origin. Although the prognosis is generally good, there is a malignant variant characterised by very frequent PVCs (defined as > 20,000 PVCs per day) and PVCs with a short coupling interval in addition to a history of syncope.⁽²⁴⁾ Exclusion of right ventricular dysplasia is important, especially if there are VTs of different morphologies or a family history of sudden death. PVCs arising from the left ventricular outflow tract have right bundle branch block morphology and appear to have a similar prognosis as those originating from the RVOT.

Congenital heart disease

A meta-analysis of 39 studies, including 4,627 patients with corrected congenital heart disease, showed that the combination of ventricular dysfunction and complex PVCs correlates with late SCD.⁽²⁵⁾ However, in the absence

of ventricular dysfunction or symptoms, isolated PVC has minimal prognostic significance in these individuals. Antiarrhythmic therapy is not indicated for asymptomatic patients with isolated PVCs.

Athletes

It is well known that competitive athletes are at a higher risk of SCD compared to the general age-matched population. The major causes of SCD in athletes are HCM (36%), coronary artery anomalies (19%), arrhythmogenic right ventricular dysplasia and myocarditis.⁽²³⁾ The presence of PVCs in athletes warrants careful evaluation, as they may point to underlying structural cardiac anomalies. The 2005 36th Bethesda Conference recommends that athletes without structural heart disease who have PVCs at rest as well as during exercise and exercise testing (comparable to the sport in which they compete) can participate in all competitive sports. However, if the athletes experience symptomatic PVCs during exercise, they can only participate in sports such as golf, billiards, bowling, cricket, curling and riflery. Athletes with structural heart disease, who are deemed to be at high-risk and who have PVCs, can only participate competitively in low-intensity sports.⁽²⁶⁾

Evaluation of PVCs

A detailed history, including drug and family history, helps to identify high-risk patients requiring further risk stratification. The prognosis and management are individualised according to symptom burden and severity of underlying heart disease, in addition to the clinical presentation. Therefore sudden episodes of collapse with loss of consciousness may constitute a high risk when compared to a presentation of palpitations. Physical examination is often unrevealing in such patients who present with PVCs. The main focus of clinical examination is toward evaluating underlying structural heart disease.

A standard 12-lead resting ECG with a rhythm strip allows the physician to characterise and localise the PVCs. Importantly, it allows for identification of related important electrical abnormalities such as long or short QT syndrome, Brugada syndrome, arrhythmogenic right ventricular dysplasia or electrolyte disturbances. Also, it may suggest underlying structural or ischaemic heart disease, which explains the presence of the PVCs. In addition, QRS duration and repolarisation abnormalities are both independent predictors of SCD. In some studies, ST segment depression or T-wave abnormalities are associated with up to four times increased risk of cardiovascular death and SCD.⁽²⁷⁾ A prolonged QTc interval is also an independent predictor of SCD, with

QTc > 440 ms significantly predicting cardiovascular death with an adjusted relative risk of 2.1.⁽²⁸⁾ A QTc < 300 ms, which is often used to define the short QT syndrome, is an independent predictor of SCD.⁽²⁹⁾

Blood sampling for electrolytes is important, looking in particular for hypokalaemia and hypomagnesaemia. Plasma levels of pro-arrhythmic medications or the presence of suspected illicit drugs may also be checked. Conventional Holter ambulatory monitoring is useful if the symptoms are suggestive and 12-lead ECG is unrevealing. Such techniques can be very helpful not only in diagnosing a suspected case but importantly, also in establishing PVC frequency and complexity, while relating the symptoms to the presence of the arrhythmia. While a 24- to 48-hour continuous Holter recording is appropriate whenever the arrhythmia is known or suspected to occur at least once a day, conventional event monitors are more appropriate for sporadic episodes because of their extended recording capability. Recently, an implantable loop recorder (which allows recording of ECGs for up to three years) has been shown to be extremely useful in diagnosing serious tachyarrhythmias and bradyarrhythmias in patients with life-threatening symptoms such as syncope.

The readily available, relatively inexpensive echocardiography performs an important role in diagnosing the aetiology of and in prognosticating PVCs. Therefore it should be performed in patients with suspected structural heart disease and in the subset of patients at high risk for development of serious ventricular arrhythmias or SCD, such as acute myocardial infarction survivors. In general, PVCs in healthy individuals without any structural heart disease do not usually pose any risk of SCD.

The ACC/AHA/ESC 2006 guidelines for the management of patients with ventricular arrhythmias recommended the exercise stress test as a means of unmasking ventricular arrhythmias due to ischaemia or suspected catecholamine-induced ventricular arrhythmias. It may be considered in the investigation of isolated PVCs in middle-aged or older patients without other evidence of CAD.⁽²³⁾ Exercise testing may provide prognostic information in patients with catecholamine-induced arrhythmias, given that the presence of exercise-induced ventricular ectopic increases mortality at 12 months by three-fold relative to patients with PVCs at rest only.⁽³⁰⁾ However, exercise-induced PVCs in apparently normal individuals should not be used to dictate therapy unless associated with documented ischaemia or sustained VT.

Electrophysiology (EP) testing has been used for arrhythmia assessment and risk stratification for SCD since its introduction in the early 1970s. However, EP testing yield varies, fundamentally dependent on the kind

and severity of the underlying heart disease, the presence or absence of spontaneous VT, concomitant drug therapy, the stimulation protocol and the site of stimulation. The highest induction rates and reproducibility are observed in patients after myocardial infarction.⁽³¹⁾ The presence of PVCs, particularly frequent complex PVCs in post-myocardial infarction patients or those at higher risk of sudden death warrants further evaluation in order to determine future arrhythmic risk. EP testing is probably useful in the evaluation of patients with remote myocardial infarction and symptoms suggestive of ventricular tachyarrhythmia, such as palpitations, presyncope and syncope.⁽²³⁾ While EP testing is not required in recent trials such as MADIT II and Sudden Cardiac Death in Heart Failure (SCD-HeFT), in the earlier MADIT trial, inducibility of VT in patients with NSVT on Holter monitoring identified a population at high risk for VT/ventricular fibrillation.⁽³²⁾ However, EP testing may only play a minor role in non-ischaemic dilated cardiomyopathy due to its low inducibility and low reproducibility as well as the low predictive value of induced VT.⁽²³⁾ In ventricular outflow tract ventricular tachyarrhythmia, EP testing for the evaluation of outflow tract VT is basically similar to that for other VT entities whose aim is to establish precise diagnosis to guide curative catheter ablation.⁽³³⁾ EP testing remains unclear or controversial in arrhythmogenic right ventricular cardiomyopathy and HCM.

ECG techniques such as signal-averaged ECG (SAECG), T Wave Alternant (TWA) testing, heart rate variability, baroflex sensitivity and heart rate turbulence may be useful for improving the diagnosis and risk stratification of patients with ventricular arrhythmias or those who are at risk of developing life-threatening ventricular arrhythmias. Currently, only SAECG and TWA testing have been approved by the US Food and Drug Administration. In the era of fibrinolysis or angioplasty to infarct artery, the usefulness of SAECG is limited to being a high negative predictive value of 89%–99% to exclude a wide-complex tachycardia as a cause of unexplained syncope.⁽³⁴⁾ Similarly, TWA testing appears to have a very high negative predictive accuracy,⁽³⁵⁾ thus improving the diagnosis and risk stratification of high risk patients with ventricular arrhythmias.⁽²³⁾

Treatment for PVCs

Routine treatment with antiarrhythmics is not warranted. Most patients with PVCs are asymptomatic, and reassurance is the only therapy required if there is no evidence of structural heart disease. For symptomatic patients, avoidance of precipitating factors such as smoking, excessive alcohol and caffeine intake may

reduce the frequency of PVCs. Of greater importance is the exclusion of underlying structural heart disease or other conditions that may precipitate PVCs, such as electrolyte imbalance or drug toxicity. After correctable factors have been addressed, medications such as beta-blockers can be used in the setting of a hyperadrenergic state or myocardial ischaemia if there is a need to suppress the PVCs. Lignocaine may be used during the peri-infarct period.

In general, exercise-induced PVCs should not be treated routinely unless there is documented ischaemia or sustained VT. With the exception of beta-blockers, the use of antiarrhythmic drugs to suppress exercise-induced PVCs in otherwise normal individuals has not been shown to be effective in reducing SCD. Suppression of asymptomatic PVCs is no longer considered a therapeutic aim for prevention of death in post-infarction or cardiomyopathy patients. Some antiarrhythmic drugs (such as flecainide and encainide) have been associated with increased mortality due to their proarrhythmic properties in patients with previous myocardial infarction or underlying CAD.

If an antiarrhythmic is indicated, beta-blockers are the first-line for suppression of symptomatic PVCs. They have been conclusively demonstrated to reduce mortality in post-infarction and heart failure patients, and thus should be part of the standard therapy. The role of amiodarone as a second-line antiarrhythmic in this setting is supported by findings in the Basel Antiarrhythmic Study of Infarct Survival, which suggested that low-dose amiodarone (i.e. 200 mg/day) in patients with persisting asymptomatic complex arrhythmias after myocardial infarction decreases mortality in the first year after myocardial infarction.⁽³⁶⁾ This finding was corroborated by a later study, the Canadian Amiodarone Myocardial Infarction Arrhythmia Trial, in which there was 48.5% relative risk reduction of arrhythmic death or resuscitated ventricular fibrillation, although no significant difference in all-cause or cardiac mortality was noted.⁽³⁷⁾ A meta-analysis involving 6,500 post-myocardial infarction and heart failure patients with a median frequency of PVCs at 18 per hour demonstrated that amiodarone results in overall reduction of 13% in total mortality.⁽³⁸⁾ To date, the therapy that brings about the greatest reduction in sudden death in patients with low ejection fraction is implantation of a cardioverter-defibrillator.⁽³⁹⁾

Radiofrequency ablation is now a well-recognised, non-pharmacological technique for the elimination of frequent symptomatic ventricular ectopic beats when pharmacological treatment has failed or is not preferred. The 2006 ACC/AHA/ESC guidelines gave this a Class IIa indication for patients who are otherwise at low risk for

SCD and who have frequent symptomatic, predominantly monomorphic PVCs that are drug resistant, or for those who are drug intolerant or do not wish to have long-term drug therapy. In addition, ablation of asymptomatic PVCs may also be considered when the PVCs are very frequent, so as to avoid or treat tachycardia-induced cardiomyopathy (Class IIb).⁽²³⁾

ABSTRACT

How important are PVCs and what should we do about them? PVCs are not a disease in themselves, but a marker of possible underlying conditions that may increase the risk of cardiac death. They serve as a flag to alert us to exclude structural heart disease, the presence of which is the strongest predictor of adverse events. However, it is important to know that PVCs are common in people with no structural heart disease. In this situation, the prognosis is generally excellent. Suppression of PVCs with antiarrhythmic medication is not indicated routinely, unless the patient is symptomatic or at risk of tachycardia-induced cardiomyopathy owing to the very high frequency of PVCs. Where pharmacological therapy has failed, there is now the option of radiofrequency ablation for elimination of frequent symptomatic PVCs. The ECG is a simple yet useful tool to improve risk assessment, especially in those with known cardiovascular disease.

Keywords: premature ventricular complex, sudden cardiac death, syncope

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

Multiple Choice Questions (Code SMJ 20110A)

	True	False
Question 1. Regarding premature ventricular complex (PVC):		
(a) PVCs are one of the most common arrhythmias and can occur in patients with or without underlying heart disease.	<input type="checkbox"/>	<input type="checkbox"/>
(b) The presence and severity of underlying structural heart disease is not the most important determinant of its prognostic significance.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Recent large population-based study by Massing et al showed healthy patients with PVCs were more than two times as likely to die due to coronary heart disease than those without PVCs.	<input type="checkbox"/>	<input type="checkbox"/>
(d) PVC can be a sign of established coronary artery disease, with a prevalence of 58% in men.	<input type="checkbox"/>	<input type="checkbox"/>
Question 2. Regarding PVC in post myocardial infarction:		
(a) The presence of more than ten PVCs an hour prior to discharge after myocardial infarction was associated with 6% mortality.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Polymorphic PVCs connote a poorer prognosis than uniform morphologic PVCs.	<input type="checkbox"/>	<input type="checkbox"/>
(c) The presence of complex PVCs (defined as "R on T", runs of two or more, multiform or bigeminal complexes) conferred a risk of cardiac death.	<input type="checkbox"/>	<input type="checkbox"/>
(d) The presence of PVCs after acute myocardial infarction is not associated with increased total mortality, particularly in those with impaired left ventricular function.	<input type="checkbox"/>	<input type="checkbox"/>
Question 3. Regarding exercise-induced PVCs:		
(a) The probable mechanisms for PVCs include enhanced automaticity, re-entry and triggered activity.	<input type="checkbox"/>	<input type="checkbox"/>
(b) PVCs during exercise are associated with an increase in all-cause mortality irrespective of the presence of cardiopulmonary disease or exercise-induced ischaemia.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Frequent PVCs during the recovery phase of the exercise stress test was a stronger predictor of an increased risk of mortality.	<input type="checkbox"/>	<input type="checkbox"/>
(d) There is an association between exercise-induced PVCs and cardiovascular mortality, but the extent of its clinical implication in asymptomatic individuals with structurally normal hearts is as yet uncertain.	<input type="checkbox"/>	<input type="checkbox"/>
Question 4. Regarding management of PVCs:		
(a) The major determinants of risk of sudden cardiac death (SCD) are related more to the type and severity of associated cardiac disease and less to the frequency or classification of ventricular arrhythmia.	<input type="checkbox"/>	<input type="checkbox"/>
(b) A positive family history of SCD is not a strong independent predictor of susceptibility to SCD.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Echocardiography is useful for determining the presence or absence of structural heart disease as well to assess cardiac function.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Exercise stress test as a means of unmasking ventricular arrhythmias due to ischaemia or suspected catecholamine induces ventricular arrhythmias.	<input type="checkbox"/>	<input type="checkbox"/>
Question 5. Regarding treatment of PVCs:		
(a) Routine treatment with antiarrhythmic drug is warranted.	<input type="checkbox"/>	<input type="checkbox"/>
(b) In symptomatic patients, avoidance of precipitating factors such as smoking, excessive alcohol and caffeine intake helps.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Antiarrhythmic drugs (such as flecainide, encainide) have been associated with increased mortality due to their proarrhythmic properties in patients with underlying coronary artery disease.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Radiofrequency ablation is now a well-recognised, non-pharmacological technique for the elimination of frequent symptomatic ventricular ectopic beats when pharmacological treatment has failed or is not preferred.	<input type="checkbox"/>	<input type="checkbox"/>

Doctor's particulars:

Name in full: _____
MCR number: _____ Specialty: _____
Email address: _____

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 December 2011 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 18 November 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: (October 2011 SMJ 3B CME programme): 12 noon, 11 November 2011.