Late arrhythmia in adults with the Mustard procedure for transposition of great arteries: a surrogate marker for right ventricular dysfunction?

M A Gatzoulis, J Walters, P R McLaughlin, N Merchant, G D Webb, P Liu

Abstract

Objective—To examine the relation between ventricular dysfunction and late clinical arrhythmia in adults who underwent the Mustard procedure for transposition of the great arteries.

Design—Observational study based on periodic outpatient assessment of biventricular function.

Setting—Tertiary referral centre.

Interventions—Analysis of data from 12 lead ECGs, echocardiography, exercise radionuclide ventriculography, and magnetic resonance imaging.

Main outcome measures—Clinical outcome and late onset clinical arrhythmia during follow up. ECG and ventricular function indices obtained before arrhythmia onset were used for analysis.

Results—51 patients (mean (SD) age 25.7 (5.0) years) fulfilled entry criteria at a mean of 23.4 (4.0) years after the Mustard procedure. Late arrhythmia occurred in 11 (22%): sustained atrial flutter/fibrillation in 10, ventricular tachycardia in one. Compared with patients who remained arrhythmia free, patients with arrhythmia had longer QRS (129 (26) vs 112 (16) ms, p = 0.01), greater QT dispersion (107 (28) vs 51 (24) ms, p < 0.001), and increased ratio of right to left ventricular end diastolic diameter (2.4 (0.9) vs 1.7 (0.7), p = 0.02), but no difference in wall thickness. Systemic ejection fraction was also reduced in the arrhythmia subgroup (at rest: 34.1 (13)% vs 47 (16)%), p = 0.04; during exercise: 37.8 (12)% vs 52 (17)%, p = 0.03). QRS duration correlated with right ventricular end diastolic diameter (r = 0.59, p < 0.001), suggesting a possible mechano-electric relation after the Mustard procedure. QT dispersion was the only predictor of clinical arrhythmia in multivariate analysis.

Conclusions—Impaired ventricular function in adults with the Mustard procedure for transposition of the great arteries relates to clinical arrhythmia. Late atrial flutter/fibrillation may be a surrogate marker for ventricular dysfunction, and these patients may also be at risk of ventricular tachycardia.

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Keywords: congenital heart disease; transposition of great vessels; arrhythmia; radionuclide ventriculography

Clinical arrhythmia and sudden cardiac death are late complications in patients with transposition of the great arteries who have undergone previous atrial switch procedures (Mustard or Senning). Systemic ventricular dysfunction has been postulated as a possible cause for both these complications. However, there are no studies specifically addressing the potential relation of clinical arrhythmia and ventricular function in this population. Furthermore, the fate of the right ventricle supporting the systemic circulation for a lifetime remains a cause of concern. Early identification of patients at risk, therefore, may allow the preservation or restoration of ventricular function by medical or surgical interventions.

The arterial switch procedure, which overcomes the shortfalls of a systemic right ventricle, has superseded the Mustard and Senning operations in most places of the world. However, even increasing numbers of patients with previous atrial switch procedures are now entering adulthood. Our aim in this study was to examine the possible relation between biventricular function and late occurrence of clinical arrhythmia in this emerging adult population.

Methods

PATIENTS

All adult patients with the Mustard procedure for transposition of great arteries who are registered with the University of Toronto Congenital Cardiac Centre for Adults (UTCCCA) and have been undergoing periodic outpatient cardiac assessment were identified from its database. The study population comprises patients who underwent full assessment of ventricular function by exercise radionuclide ventriculography and ventricular size by magnetic resonance imaging within the preceding 12 months from the onset of arrhythmia, or at the most recent follow up (for the arrhythmia-free patients) at the UTCCCA. Patients with early arrhythmia occurring before their transfer to UTCCCA were not included. Clinical assessment and standard 12 lead ECGs, 24 hour Holter recordings, chest x rays, and trans-thoracic echocardiograms routinely performed during corresponding clinic reviews were included in the analysis.

CLINICAL DATA

Demographic, clinical, and surgical details were obtained from the UTCCCA database and the patients’ records. Patients requiring

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additional surgery for ventricular septal defect closure, repair of double outlet right ventricular connection, relief of pulmonary stenosis, or combinations of these were characterised as “complex”. The arrhythmia subset comprised patients with documented sustained atrial flutter/fibrillation or ventricular tachycardia and associated symptoms during follow up at UTCCC. Patients with junctional rhythm only or non-sustained episodes of atrial or ventricular arrhythmia on Holter monitoring were not included in the arrhythmia subgroup. When functional capacity was assessed more than once, data from the most recent entry or the last full assessment before the onset of arrhythmia were used for the analysis. Finally, we did not differentiate between chronic or paroxysmal atrial tachycardia.

Periodic assessment of ventricular function with radionuclide ventriculography and magnetic resonance imaging was approved by The Toronto Hospital human ethics committee.

ELECTROCARDIOGRAPHY, RADIOGRAPHY, AND ECHOCARDIOGRAPHY
The predominant underlying rhythm and the presence of sustained atrial or ventricular tachycardia were determined from 12 lead ECGs, Holter recordings, and ECG strips. ECG measurements were made manually by a blinded single investigator from 12 lead sinus or junctional ECGs corresponding to the time of full cardiac assessment, and before the onset of arrhythmia.12 13

Cardiothoracic ratio was obtained from posteroanterior chest x rays in the standard manner. All patients had routine transthoracic echocardiograms within three months of the exercise radionuclide ventriculography and magnetic resonance imaging. Systemic ventricular dysfunction and tricuspid regurgitation were assessed (grade: I–II = absent or mild, III = moderate, IV = severe) by M mode (one dimensional) echocardiography, pulsed wave, and colour flow Doppler analysis. Available haemodynamic data from recent cardiac catheterisations were also recorded. Left ventricular hypertension was considered to be present when left ventricular systolic pressure derived from continuous wave Doppler estimation or cardiac catheterisation was \( \geq 60 \) mm Hg.

MAGNETIC RESONANCE IMAGING
Magnetic resonance imaging acquisition involved an initial coronal ECG triggered spin echo sequence to obtain the optimal angle for long axis, short axis, and transaxial “four chamber” views of the heart.14 Images were acquired as T1 weighted spin echo with an in breath hold, and TR = 1 RR interval, TE = 20 ms, FOV = 24 cm, NEX = 2, matrix 256 \( \times \) 192, and superior/inferior saturation band. Images were transferred to an independent computer work station (Advantage Windows Work Station) for analysis. Two measurements of right and left ventricular end diastolic diameters (RVEDD, LVEDD) were made at mid-ventricular level in the transaxial view, and their average was used for the analysis. Right ventricular free wall (RVWT), interventricular septal wall (IVWT), and left posterior wall thickness (LVT) were also measured at mid-ventricular level in the transaxial view in duplicate (end diastole), and their average was recorded. Interobserver and intraobserver variability was examined by repeat measurements in a subset of randomly chosen magnetic resonance imaging samples.

CARDIOPULMONARY EXERCISE TESTING
Graded exercise testing using an electronically braked cycle ergometer was performed in the semiprone position to assess cardiopulmonary status. The patient’s heart rate and rhythm were continuously monitored with a three lead ECG. Blood pressure was measured by an automated cuff at one minute intervals. Breath by breath collection of expired gases was obtained using a metabolic cart (Sensormedics 4400); with the oxygen and carbon dioxide measurement results reported as 15 second averages. After a one minute warm up phase against minimal resistance, the work load increased continuously by an average of 200 kpm every three minutes and the exercise continued until a symptom limited maximum was reached. The average duration of exercise is in the range of 6–10 minutes for this group of Mustard patients. The ventilatory anaerobic threshold was determined by analysis of the relation between ventilation and oxygen consumption, confirmed by examination of the ventilation equivalent of CO2 output over oxygen consumption (VE/VO2).

RADIONUCLIDE VENTRICULOGRAPHY
Before initial exercise testing patients received 25 mCi of technetium-99m pertechnetate for red blood cell labelling, using a standard modified in vivo technique.15 Gated radionuclide ventriculography (Axp 409 Elscint camera) was performed at rest in anterior, lateral, and oblique views. The right ventricle was studied in the projection with the most septal separation between right and left ventricles. To obtain exercise ventriculography, the patient underwent a graded exercise protocol with warm up of one minute at 200 kpm, after which the exercise load was increased to 70% of the patient’s previous maximum work load. The patients sustained exercise at this level for one minute at 200 kpm, after which the exercise load was increased to 70% of the patient’s previous maximum work load. The patients sustained exercise at this level for one minute to attain steady state and an additional two minutes for nuclear image acquisition, obtained in the previously determined best septal view. Right and left ejection fractions, using background subtraction from an adjacent pulmonary area, were then analysed by two experienced independent observers with commercially available software, after which the results were averaged. The average duration of exercise in the range of 6–10 minutes for this group of Mustard patients.

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The 51 patients reported in this study are a subset of a larger cohort of 122 Mustard patients who are registered with the UTC-CCA. There was no difference between the two groups for sex (73 male, 49 female, p = 0.23), age at Mustard procedure (2.13 (2.36) years, p = 0.44), length of follow up (23.79 (4.2) years, p = 0.54), or prevalence of late arrhythmia (19.7%, p = 0.63), suggesting that there was no selection bias.

**Electrocardiography and echocardiography**

Functional rhythm was the predominant rhythm in 11 patients. Four patients had received a pacemaker for sick sinus syndrome (n = 3) and complete atrioventricular block (n = 1); none of these had a history of tachyarrhythmia. These four patients, and the nine patients with junctional rhythm who remained free of atrial flutter/fibrillation and ventricular tachycardia, were assigned to the non-arrhythmia subgroup for the purpose of statistical analysis. Electrocardiographic, radiographic, and echocardiographic data are shown in table 1. Maximum QRS duration and QT dispersion was significantly greater in the arrhythmia subgroup than in the arrhythmia-free subgroup (fig 1).

Eight patients had one or more residual haemodynamic lesions: intra-atrial baffle leak (n = 3), left ventricular hypertension (systolic pressure ≥ 60 mm Hg because of left ventricular outflow tract obstruction (n = 3), pulmonary hypertension (n = 2)), and small residual ventricular septal defect (n = 1).

**Magnetic resonance imaging**

Magnetic resonance imaging data from the 51 Mustard patients are shown in table 1. The ratio of right to left ventricular end diastolic diameter was significantly greater in the arrhythmia patients when compared with the remainder, suggesting relative right ventricular enlargement (fig 1). By contrast, no difference was seen in right to left ventricular wall thickness between the arrhythmia subset and the remainder of the Mustard patients. When absolute measurements and ratios of biventricular diameter and wall thickness were tested against ECG conduction parameters for correlates, there was a significant correlation between maximum QRS duration and right ventricular end diastolic dimension (r = 0.59, p < 0.001, fig 2), but not right ventricular wall thickness.

Repeated measurements of right and left ventricular end diastolic dimension and wall thickness in six samples randomly chosen from the Mustard cohort were performed twice by two blinded observers. The percentage differences ranged from 0–7% for within observer variability, and 1–11% for between observer variability.

**Radionuclide ventriculography and cardiopulmonary exercise testing**

Radionuclide ventriculography and cardiopulmonary testing was performed in all 51 subjects. Forty three patients ended the exercise test because of fatigue, the remainder...
because of exertional dyspnoea. Systemic (right ventricular) function in the Mustard patients was significantly reduced compared with normal healthy controls (ejection fraction at rest 44.5 (1.0)% vs 58 (7)%, p = 0.027, and during exercise 49.3 (1.0)% vs 69.5 (8.0)%, p = 0.002). Heart rate response to exercise was blunted in the Mustard patients (140 (26) vs 178 (19) beats/min in the controls, p < 0.001). Cardiopulmonary exercise performance was also impaired in the Mustard cohort compared with the healthy controls (anaerobic threshold 11.1 (4.0) ml/kg/min, p < 0.001; maximum oxygen uptake 17.5 (6.0) ml/kg/min, p < 0.001, respectively). Within the Mustard patients, those who subsequently developed clinical arrhythmia had significantly reduced right ventricular ejection fraction, both at rest and during exercise, compared with the arrhythmia-free patients by the end of the study (fig 1).

ARRHYTHMIA

Eleven patients (22%) had documented, sustained atrial flutter/fibrillation (n = 10) and ventricular tachycardia (n = 1) during their follow up at the UTCCC at a mean age of 25.6 (4.8) years. Five of them presented with palpitations and exertional dyspnoea, four with presyncope or syncope, and two with congestive heart failure. The demographic, surgical, and haemodynamic details of these 11 patients with clinical arrhythmia are shown in table 2 and compared with the remainder of the Mustard cohort. All but two patients required electrical cardioversion. Three of them have been considered for the double switch conversion. One patient with ventricular tachycardia was too unstable for left ventricular retraining with pulmonary arterial banding and died during heart transplantation (myocardial standstill). The remaining eight patients have been managed medically with antiarrhythmic drugs or angiotensin converting enzyme (ACE) inhibition.

PREDICTORS OF LATE CLINICAL ARRHYTHMIA

Univariate analysis of potential predictors of arrhythmia is shown in table 3. In this cohort of adults with Mustard procedure for transposition of great arteries, multivariate analysis showed QT dispersion to be the only independent predictor of late clinical arrhythmia.
RV dysfunction and arrhythmia after Mustard procedure

Table 2 Comparison between patients with clinical arrhythmia versus the remainder (Mustard patients)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Arrhythmia (n=11)</th>
<th>No arrhythmia (n=40)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, repair (years)</td>
<td>2.16 (0.96)</td>
<td>2.5 (2.2)</td>
<td>0.6</td>
</tr>
<tr>
<td>Male/female</td>
<td>7/4</td>
<td>28/12</td>
<td>0.7</td>
</tr>
<tr>
<td>“Complex” repair</td>
<td>4</td>
<td>11</td>
<td>0.7</td>
</tr>
<tr>
<td>FU length (years)</td>
<td>23.6 (4.5)</td>
<td>23.3 (3.7)</td>
<td>0.8</td>
</tr>
<tr>
<td>NYHA &gt; III</td>
<td>3</td>
<td>4</td>
<td>0.1</td>
</tr>
</tbody>
</table>

ECG

<table>
<thead>
<tr>
<th>Variable</th>
<th>Arrhythmia (n=11)</th>
<th>No arrhythmia (n=40)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional rhythm</td>
<td>2</td>
<td>9</td>
<td>0.9</td>
</tr>
<tr>
<td>Superior QRS axis</td>
<td>5</td>
<td>6</td>
<td>0.03</td>
</tr>
<tr>
<td>RR interval (ms)</td>
<td>985 (319)</td>
<td>912 (238)</td>
<td>0.4</td>
</tr>
<tr>
<td>QRS duration max (ms)</td>
<td>129 (26)</td>
<td>112 (16)</td>
<td>0.01</td>
</tr>
<tr>
<td>QT interval (ms)</td>
<td>519 (59)</td>
<td>458 (47)</td>
<td>0.001</td>
</tr>
<tr>
<td>QT dispersion (ms)</td>
<td>107 (28)</td>
<td>51 (24)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>U waves</td>
<td>4</td>
<td>4</td>
<td>0.04</td>
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</table>

Echocardiography

<table>
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<th>No arrhythmia (n=40)</th>
<th>p Value</th>
</tr>
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<tbody>
<tr>
<td>Right ventricular dysfunction</td>
<td>7</td>
<td>21</td>
<td>0.5</td>
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<tr>
<td>TR moderate</td>
<td>5</td>
<td>10</td>
<td>0.2</td>
</tr>
<tr>
<td>RVEF &gt; III and TR mod</td>
<td>5</td>
<td>6</td>
<td>0.03</td>
</tr>
<tr>
<td>LV hypertrophy</td>
<td>3</td>
<td>2</td>
<td>0.06</td>
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Magnetic resonance imaging

<table>
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<tr>
<th>Variable</th>
<th>Arrhythmia (n=11)</th>
<th>No arrhythmia (n=40)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVEDD (mm)</td>
<td>43.6 (9.6)</td>
<td>40.5 (7.6)</td>
<td>0.3</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>20.3 (6.8)</td>
<td>27.8 (8.8)</td>
<td>0.03</td>
</tr>
<tr>
<td>LV systolic, rest (mm Hg)</td>
<td>116 (17)</td>
<td>119 (14)</td>
<td>0.30</td>
</tr>
<tr>
<td>RV free wall thickness (mm)</td>
<td>11.9 (2.1)</td>
<td>13 (3.5)</td>
<td>0.4</td>
</tr>
<tr>
<td>LV wall thickness (mm)</td>
<td>8.2 (2.0)</td>
<td>8.6 (2.4)</td>
<td>0.7</td>
</tr>
</tbody>
</table>

Discussion

We have shown that late clinical arrhythmia in patients with the Mustard procedure for transposition of great arteries relates to impaired system right ventricular function.

Our radionuclide angiographic data reinforce previous studies showing systemic right ventricular dysfunction in patients undergoing the Mustard procedure for transposition of great arteries. Many of our adult patients were asymptomatic at follow up. However, during exercise stress, their cardiac limitations became apparent, as manifested by their decreased maximum workload, diminished anaerobic threshold and VO2max, and lower systemic ventricular ejection fraction. Maximum oxygen uptake of 17.5 ml/kg/min at a mean of 23.4 years after the Mustard procedure in our adult cohort was significantly lower than the mean of 27 ml/kg/min at 10.3 years after Mustard operation in a report on young adolescents. Right ventricular ejection fraction from the present series, both at rest (44.5%) and during exercise (49.3%), was also lower than in a previous report by Hochreiter and colleagues (right ventricular ejection fraction 52% at rest and 56% during exercise) at a mean of 17.8 years after the Mustard procedure. These differences in systemic ventricular function and cardiopulmonary performance may reflect the longer period of follow up after the Mustard procedure in our older patients.

Late clinical arrhythmia in this study (21.5%) consisted predominantly of atrial flutter/fibrillation and was more common than in two recent reports by Gewillig and colleagues (16%) and Gelatt and associates (14%). Again, the longer follow up time in our study may account for this difference (mean follow up 23.4 years after the Mustard procedure). These differences in systemic ventricular function and cardiopulmonary performance may reflect the longer period of follow up after the Mustard procedure in our older patients. Late clinical arrhythmia in this study (21.5%) consisted predominantly of atrial flutter/fibrillation and was more common than in two recent reports by Gewillig and colleagues (16%) and Gelatt and associates (14%). Again, the longer follow up time in our study may account for this difference (mean follow up 23.4 years after the Mustard procedure). These differences in systemic ventricular function and cardiopulmonary performance may reflect the longer period of follow up after the Mustard procedure in our older patients. Whereas cardiac enlargement on chest radiography was seen in the overall population, there was no significant difference in right ventricular end diastolic dimension between the patients with arrhythmia and the remainder. However, left ventricular end diastolic dimension was significantly smaller in the arrhythmia subgroup; this, combined with the normal left ventricular function in both subgroups and the higher ratio of RVEDD/LVEDD in the arrhythmia subset, is consistent with a ventriculo-ventricular interaction and may suggest relative right ventricular enlargement. The longer QRS duration in the arrhythmia subgroup, which in turn correlated with right ventricular end diastolic dimension on magnetic resonance imaging, would also support this theory. There was no difference between absolute or relative (to the left) right ventricular free wall thickness between the arrhythmia patients and the remainder. This suggests that right ventricular hypertrophic response to chronic pressure overload—or its absence—may not be responsible for arrhythmogenesis in these patients, at least in isolation. This concurs with the earlier report by Lorenz and colleagues, in which “inadequate” right ventricular hypertrophy did.
not appear to be the cause of late dysfunction in 22 patients assessed by magnetic resonance imaging after atrial switch procedures. The severity of tricuspid regurgitation assessed echocardiographically was not significantly greater in the arrhythmia subgroup. Likewise, closure of ventricular septal defect or relief of left ventricular outflow tract obstruction at the time of the Mustard procedure was not an independent predictor of late arrhythmia. The combination of tricuspid regurgitation with right ventricular dysfunction, however, was more common in the arrhythmia patients. This finding contrasts with the recent report by Gelatt and colleagues, in which damage to the tricuspid valve during ventricular septal defect closure was postulated as a causative mechanism of atrial flutter and sudden death. Our data suggest that tricuspid regurgitation may rather be the result of progressive right ventricular dysfunction and ensuing annular dilatation, and not secondary to iatrogenic factors. Different lengths of follow up between the two studies may explain this discrepancy.

Despite the common arrhythmogenic substrate, namely previous intra-atrial surgery and the presence of intra-atrial baffles, not all patients after the Mustard procedure develop late clinical arrhythmia. An additional factor, or factors, seems to be required for arrhythmogenesis. Our data suggest that a greater degree of right ventricular dysfunction, as expressed by the lower right ventricular ejection fraction in the arrhythmia subset (compared with the arrhythmia-free patients), is a contributory factor in initiating or sustaining the arrhythmia, and in turn leads to haemodynamic compromise. This is purely speculative, but it needs to be determined whether ACE inhibitors have a positive effect on right ventricular function and in turn on the incidence of late arrhythmia in this population.

QT dispersion on the surface ECG, a marker of ventricular inhomogeneity more frequently reported in groups susceptible to ventricular tachycardia, was also significantly greater in the arrhythmia subgroup and predictive of late arrhythmia. This pronounced QT dispersion, seen in patients who went on to develop predominantly atrial flutter/fibrillation, can be taken as further evidence that ventricular dysfunction is a contributory factor in this population predisposed to atrial arrhythmia. Furthermore, it suggests that these patients may also be at risk of ventricular tachycardia. This was indeed the case with one of our patients. Although sustained ventricular tachycardia has not been widely reported in Mustard cohorts, we speculate that with increasing length of follow up and the anticipated progression of right ventricular dysfunction in a subset of adult patients, this will become an increasing problem in the future.

Sudden death, presumably arrhythmic, has been reported in all large series. As with atrial flutter, it has been postulated that sudden death relates to right ventricular dysfunction. Complete atioventricular block, atrial flutter with one to one atioventricular conduction, and sustained ventricular tachycardia degener-ating to ventricular fibrillation are all potential causes. Its precise mechanism, however, remains unknown. Only one of our patients died. This was the patient with ventricular tachycardia and end stage heart failure, who died from myocardial standstill during heart transplantation. There is a clear need to identify patients at risk of sudden death. Our study cannot answer this question, as none of the patients with atrial flutter/fibrillation died. Nevertheless it provides evidence of the presence of a mechano-electric interaction in patients with Mustard procedure for transposition of great arteries. Impaired systemic ventricular function in these adult patients is associated with late clinical arrhythmia. At a mean of 23 years after the Mustard procedure this arrhythmia was predominantly atrial flutter/fibrillation.

LIMITATIONS

We have not specifically addressed the diastolic function of the systemic and pulmonary ventricles, which may be equally important, both in terms of cardiopulmonary exercise performance and arrhythmogenesis. Volumetric data derived from cine magnetic resonance imaging were not always available and hence were not included in the analysis. Additional predictors of late arrhythmia may exist and could be identified with a larger patient sample and a longer period of observation in future studies.

CONCLUSIONS

We conclude that impaired systemic ventricular function in adult patients late after the Mustard procedure for transposition of great arteries relates to clinical arrhythmia. Atrial flutter/fibrillation may be a surrogate marker for ventricular dysfunction in this population. Adult patients presenting with late atrial tachyarrhythmia after the Mustard procedure may also be at risk of ventricular tachycardia.

We thank our colleagues from The Hospital for Sick Children for their continuing support of the University of Toronto Congenital Cardiac Centre for Adults programme. MAG was supported in part by a 1997 University of Toronto, Department of Medicine, postgraduate fellowship award. PL is a recipient of a research chair of the Heart and Stroke Foundation of Ontario and was supported in part by a grant from the Heart and Stroke Foundation of Ontario.

A 54 year old woman was admitted to our hospital because of severe chest pain and progressive dyspnoea. Physical examination revealed hypotension, elevated central venous pressure, and a loud triphasic myocardial friction rub. Echocardiography showed a large amount of pericardial fluid compressing the heart and a round structure freely floating in the pericardial fluid. The apex of the heart moved the mass like the nose of a seal that plays a ball. Because of haemodynamic failure lateral thoracotomy with partial pericardiectomy was performed. About 700 ml of fluid and the unidentified pericardial object were removed. Histopathological examination revealed signs of an aspecific chronic fibrinous exudative pericarditis, and the structure was pure fibrin in origin. Cultures and serological study yielded no evidence of a causative organism. The patient made a quick and full recovery and returned to work.