localized signs of infection, normal heart sounds and no evidence of
heart failure. However, because of a prominent tachycardia with marked malaise, two-dimensional (2-D) echocardiography was performed. This showed a mobile mass in the right atrium, intermittently prolapsing into the tricuspid valve (Figure 1).

The right atrium was surgically explored by median sternotomy and the Hickman line was removed along with two thrombi attached to its tip. The larger of the two thrombi measured 4 cm by 4 cm in diameter but no organism was grown from either thrombus or from the tip of the Hickman catheter. The patient made a good postoperative recovery and received a further 3 weeks of antibiotics. Repeat echocardiograms have been normal.

Upper extremity venous thrombosis is a well-recognized complication in patients with haematological malignancies and centrally placed indwelling silicone elastomer catheters.

However, right atrial thrombus associated with central venous catheterization is almost exclusively a complication of infancy related to prematurity, TPN and continuous catheter use. We are aware of only five adult cases of thrombosis of the right side of the heart related to central venous catheterization (right atrial in four1-3 and right ventricular in one4). All of these cases were receiving long-term TPN and all had documented infections of the central venous catheters. Both patients who survived underwent surgical removal of the right atrial thrombus.2

The remaining three cases had sudden fatal cardiovascular collapse with obstructed tricuspid valve5 and occlusion of the main pulmonary artery6 in the two patients who had autopsies.

Optimal therapy for such a complication remains open to debate. The central venous catheter should be withdrawn (unless attached to the thrombus when embolus is a potential risk) and appropriate antibiotics given. Resolution of the thrombus has occurred with heparin, thrombolytic therapy or occasionally spontaneously.7 Surgical removal is probably indicated if the thrombus is large with a risk of tricuspid valve occlusion, as in our patient, or if it is not responding to more conservative management. The possibility of right atrial thrombus should be considered in any patient with an indwelling central venous catheter with unresolving infection despite appropriate antibiotics or with evidence of cardiorespiratory compromise. In such circumstances, 2-D echocardiography is an invaluable diagnostic procedure.

References


Sudden cardiac death and the potential role of beta adrenoceptor-blocking drugs

Sir,

The article by Rayman and Kendall1 was a timely reminder of the truism that there is more to prevention of post-myocardial infarction sudden death than mere identification and abolition of ventricular arrhythmias. For the majority of patients, a useful treatment flow chart would consist of long-term low-dose aspirin for its proven value in reducing the risk of recurrent myocardial infarction and, hence, the potential risk of sudden death in silent as well as in symptomatic myocardial ischaemia,2 beta-adrenoergic blockade for those with intact left ventricular systolic function because of the benefits outlined by the authors,1 and the alternative use of angiotensin-

Figure 1 Two-dimensional echocardiography showing large thrombus (arrow) in right atrium. RV = right ventricle; RA = right atrium.
converting-enzyme inhibitors for those with symptomatic as well as asymptomatic reduction in left ventricular ejection fraction, in order to prevent recurrence of myocardial infarction. In this vulnerable subgroup consisting of patients with left ventricular ejection fractions ranging from 20% to 45%, amiodarone plays an important role in suppressing co-existing ventricular arrhythmias without the penalty of an increase in mortality rate. These arrhythmic patients, in whom beta blockade is contraindicated by co-existing left ventricular systolic dysfunction, stand to gain comparable benefits from the actions of amiodarone, such as reduction in oxygen requirements. Additional benefits include coronary as well as peripheral vasodilatation. Finally, in metaanalysis of published trials, this drug appears to confer survival benefit not only in its own right, but also in comparison with other anti-arrhythmic drugs prescribed in the post-myocardial infarction context.

O.M.P. Jolobe

Department of Medicine for the Elderly,
Tameside General Hospital,
Fountain Street,
Ashton-under-Lyme,
Lancashire O6 9RW, UK.

References

Secundum atrial septal defect repair

Sir,
I fully endorse the views expressed by Speechly-Dick and colleagues that early diagnosis and surgical repair of atrial septal defect is important to prevent long-term morbidity and to reduce mortality. However, in order to highlight the exceptional longevity and benign clinical course that can be associated with this lesion, I describe a patient who to my knowledge was the longest survivor of unrepaired atrial septal defect in the United Kingdom.

In 1986 an 89 year old man was admitted to the surgical ward of his local hospital for investigation of dysphagia. A routine chest X-ray showed cardiomegaly and prominent pulmonary vascular shadows. On further questioning he recalled that at the age of 9, he was investigated at St Bartholomew’s Hospital, London for ‘blue hands’ and a ‘hole in the heart’ was diagnosed.

On examination he was in atrial fibrillation with a slow ventricular response at a rate of 60 per minute. Blood pressure was 130/90 mmHg. Jugular venous pressure was elevated to 5 cm with a visible ‘V’ wave. Apex beat was displaced to the 6th intercostal space about 2.5 cm outside the mid-clavicular line. He had a loud pansystolic murmur audible at the apex and left sternal edge, and a mild systolic ejection murmur at the second right intercostal space. He had fixed splitting of the second heart sound. A transthoracic echocardiogram showed gross right ventricular and right atrial enlargement with mild left atrial enlargement, and large secundum atrial septal defect. He was discharged home on a diuretic. He was reviewed on his 90th birthday when he was completely asymptomatic. He died of a cerebrovascular accident when 92.

It is suggested by Rodstein et al. that a well-defined clinical picture is often present in the elderly and that such patients experience repeated attacks of bronchopneumonia, dyspnoea and angina for many years before the onset of overt heart failure. The absence of pulmonary hypertension may be an important factor in their survival. Campbell et al. observed that atrial fibrillation was nearly always the major cause of heart failure in these patients owing to production of tricuspid incompetence which in turn elevates the right atrial pressure during systole. Although this makes the interatrial shunt diastolic rather than systolic, it does not materially alter the magnitude of the left to right shunt. The limited left ventricular filling and systemic output is further diminished by the lack of atrial contribution, as seen with atrial fibrillation in any condition.

This case highlights the fact that, in patients with atrial septal defect, not only is survival possible beyond the ninth decade, but freedom from all cardiac symptoms is also possible. Owing to the lack of thorough knowledge on the rate of progression of clinical and haemodynamic worsening of such a congenital lesion, it is not easy to explain the long-term survival of such patients.

M.I. Khalid
Regional Cardiothoracic Centre,
Freeman Hospital,
Newcastle upon Tyne NE7 7DN, UK.

References
Sudden cardiac death and the potential role of beta adrenoceptor-blocking drugs.

O. M. Jolobe

doi: 10.1136/pgmj.70.825.521

Updated information and services can be found at:
http://pmj.bmj.com/content/70/825/521.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/