Raised pulmonary artery pressures measured with Doppler echocardiography in rheumatoid arthritis patients

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Abstract

Objective. To study the prevalence of echocardiographic abnormality and pulmonary hypertension in an unselected population of patients with rheumatoid arthritis (RA).

Method. One hundred and forty-six RA patients, irrespective of cardiopulmonary symptoms, were assessed clinically and by echocardiography, including pulmonary artery pressure measurement, ECG, pulmonary function tests and high-resolution computed tomography scanning of the thorax.

Results. Two-dimensional echocardiography demonstrated significant cardiac disease in the form of reduced left ventricular ejection fraction (<64%) in 9% of patients, moderate mitral regurgitation in 4%, aortic stenosis in 4%, aortic regurgitation in 3% and Valsalva sinus rupture in 0.7%. In addition, 1% had detectable pericardial effusions. Thirty-one per cent of the RA patients had an estimated pulmonary artery systolic pressure of 30 mmHg or more, and 21% of all the RA patients had pulmonary hypertension without significant cardiac disease or lung disease evident on pulmonary function testing.

Conclusions. A wide and frequent variety of echocardiographic cardiac abnormalities may be found in an unselected population of patients with RA. Using Doppler echocardiography, we have found pulmonary hypertension secondary to lung disease in 6% of the population and a larger than expected prevalence of mild primary pulmonary hypertension in patients with RA. The latter observation may be relevant to the high incidence of cardiovascular-related deaths observed in patients with RA.

Key words: Rheumatoid arthritis, Doppler echocardiography, Pulmonary artery, Systolic pressure.

Rheumatoid arthritis (RA) is a common condition affecting 1% of the white population and over 5% of people over the age of 65 yr. Amongst many other extra-articular features, several forms of cardiac involvement have been described in RA, most commonly pericarditis and pericardial effusion. Pulmonary hypertension has also been described in RA patients. This is usually a result of RA-associated lung disease [1]. Isolated case reports of primary pulmonary hypertension have been published [2]. Primary pulmonary hypertension is often clinically silent until well advanced. It has a very poor prognosis and a median survival of only 2–3 yr [3]. Secondary pulmonary hypertension progresses more slowly than the primary form. Treatment initially needs to be directed at the underlying cause. The presence of pulmonary hypertension secondary to lung disease implies a poor prognosis.

Doppler echocardiography has been proven to be a reliable non-invasive method for detecting pulmonary hypertension in chronic lung disease [4, 5] and systemic sclerosis [6]. The aim of this study was to determine the prevalence of echocardiographic abnormalities and raised pulmonary artery pressure in a district general hospital population of patients with RA. All patients were evaluated with full pulmonary function tests, chest radiographs and high-resolution computed tomography (HRCT) to determine whether raised pulmonary artery pressure is secondary to lung disease.

Methods

One hundred and forty-six consecutive patients attending the rheumatology outpatient departments of St Helens and Knowsley Hospitals NHS Trust with an established diagnosis of RA, as defined by the American Rheumatism Association 1987 criteria, were enrolled irrespective of chest symptoms or signs, after they had provided informed consent. In view of the radiation...
exposure involved in the study, patients were excluded if they were pregnant or planning a pregnancy.

Clinical assessment
A questionnaire on each patient was filled in by JKD. This noted the duration of RA, extra-articular complications, the use of current and previous disease-modifying drugs, corticosteroid use, early morning joint stiffness, and a global patient assessment of disease activity. Each patient filled in the Modified Stanford Health Assessment Questionnaire to assess functional impairment. Questions were asked relating to previous chest disease, cough, dyspnoea, sputum production, chest pain, weight loss and risk factors for respiratory disease, including smoking, medications, domestic pets and occupation. Cigarette consumption was evaluated in pack years (1 pack yr = 20 cigarettes/day for 1 yr). Details are given in Table 1.

A detailed clinical examination was performed. All patients had venous blood taken for full blood count, plasma viscosity, renal and liver function, C-reactive protein and plasma proteins. Immunological investigations included immunoglobulins, rheumatoid factor and antinuclear antibodies. All patients underwent echocardiography, ECG, chest radiography, HRCT and full pulmonary function testing.

The research ethics committee of St Helens and Knowsley Hospitals approved the study.

Echocardiography
M-mode and cross-sectional echocardiography were performed with the patient in the left lateral position, using a Hewlett-Packard echocardiogram (Sonos 1000, HP77025A ultrasound imaging). Non-imaging, continuous-wave Doppler signals were recorded with a Doptek 2.5 MHz transducer. One senior technician performed the echocardiography. Whenever possible, this technician, who was blinded to clinical details, determined pulmonary artery pressure and identified structural abnormalities.

Tricuspid regurgitation was identified in continuous-wave mode at the apex. The peak instantaneous drop in systolic pressure from the right ventricle to the atrium was calculated from the peak signal velocity of the tricuspid regurgitant signal by the simplified Bernoulli equation, \( AP = 4v^2 \), where \( AP \) is the trans-tricuspid gradient and \( v \) is the peak velocity measured.

The final estimate of the pulmonary artery systolic pressure was obtained by adding the patient’s jugular venous pressure to the estimate of the trans-tricuspid gradient [7].

The ejection fraction was calculated as follows [8]:

\[
\frac{\text{left ventricle diastolic volume} - \text{left ventricle systolic volume}}{\text{left ventricle diastolic volume}} \times 100
\]

Pulmonary function tests
Pulmonary function tests comprised spirometry, static lung volume, gas transfer factor and flow loops. They were performed in the cardiorespiratory department at Whiston Hospital by one senior technician. In all cases the pulmonary function tests were performed on the same day as the echocardiogram.

High-resolution computed tomography
The RA study population was screened for interstitial lung disease by chest HRCT scanning. This was performed with a Siemens Somatom hiQ scanner. Scanning time was 1.3 s. Supine and prone views were taken. The serial slices were 2 mm in width and 10 mm apart.

Statistical analysis
The \( \chi^2 \) test with Yates’ correction was used to compare frequencies. The Mann–Whitney \( U \)-test was used to compare quantitative data.

Definitions
Pulmonary hypertension. The gold standard for pulmonary artery pressure measurement is invasive right-heart catheterization. Pulmonary hypertension, defined by right-heart catheterization of the pulmonary artery is a pressure of 20 mmHg or greater at rest and at least 30 mmHg during exercise [9].

Echocardiography has now been used widely in patients with cardiac disease. Reported correlations between Doppler and catheter measurements range from 0.89 to 0.97; the average standard error for systolic pressure from the right ventricle to the atrium was calculated from the peak signal velocity of the tricuspid regurgitant signal by the simplified Bernoulli equation, \( AP = 4v^2 \), where \( AP \) is the trans-tricuspid gradient and \( v \) is the peak velocity measured.

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Echocardiography has now been used widely in patients with cardiac disease. Reported correlations between Doppler and catheter measurements range from 0.89 to 0.97; the average standard error for systolic pulmonary artery pressure ranges from 5 to 9 mmHg, and interobserver variability is <3% [10–12]. Denton et al. [6] found in scleroderma patients that the mean absolute difference between Doppler echocardiography and right-heart catheterization values of pulmonary systolic artery pressure (PASP) was 9.8 mmHg. We have taken Denton et al.’s definition of pulmonary hypertension on Doppler echocardiography as an estimated PASP of 30 mmHg or greater.

Significant lung disease. Significant lung disease that could be causing pulmonary hypertension was defined as pulmonary function measurements outside the normal range: a forced expiratory volume in 1 s/forced vital capacity (FEV/FVC) ratio of less than 65% or a vital capacity lung volume of less than 80% of the predicted value [13, 14].

Significant cardiac disease. Cardiac causes of secondary pulmonary hypertension are known to include heart disease resulting in elevated left atrial and end-diastolic left ventricular pressures [15]. Thus, patients with moderate mitral regurgitation, mitral stenosis or a left ventricular ejection fraction below 64% were considered to have a cardiac cause for their PASP.

ECGs were interpreted by JKD. Abnormal traces

### Table 1. Clinical details of the 146 RA patients studied

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Range 30–83, sd 10.3, mean 58.6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>101 (69%) females, 45 (31%) males</td>
</tr>
<tr>
<td>Disease duration (yr)</td>
<td>Range 0.5–35, sd 7.99, mean 12.7</td>
</tr>
<tr>
<td>Rheumatoid factor (latex test)</td>
<td>110 (76%) patients positive</td>
</tr>
<tr>
<td>Health Assessment Score</td>
<td>Range 0–3, sd 0.75, mean 1.7</td>
</tr>
<tr>
<td>Smoking status</td>
<td>57 (39%) current smokers; 47 (32%) never smoked</td>
</tr>
<tr>
<td>Average pack yr of smoking</td>
<td>17</td>
</tr>
</tbody>
</table>
were based on the criteria defined in the Oxford Textbook of Medicine [16].

**Pulmonary artery pressure control group**

For the normal population, limited data are available on pulmonary artery pressure estimated by Doppler echocardiography. A study of 20 normal healthy adults by Vachiery et al. [17] using Doppler echocardiography found that the maximum estimated pulmonary artery pressure was 24 mmHg.

To assess the diagnostic validity of the results of the echocardiogram, a control group for echocardiogram readings was incorporated into the study. One hundred and forty-three echocardiography examinations performed in the cardiorespiratory department of St Helens and Knowsley Trust Hospitals were recorded prospectively. The patients were adults (48% female, age range 16–88 yr, average age 62 yr) referred for assessment of potential or known heart disease. Patients with RA were excluded. Echocardiography was undertaken with the same Hewlett-Packard echocardiogram by the same senior technician who performed the echocardiography in the patients with RA.

**Results**

**All RA patients**

One hundred and forty-six RA patients underwent all the investigations. One hundred and eleven (76%) patients had sufficient tricuspid regurgitation visible for their pulmonary artery pressures to be assessed by Doppler echocardiography. No patients had a visibly elevated jugular venous pressure. Five patients had calcification of the mitral valve but no patients had mitral stenosis. No patients had right ventricular dilatation or hypertrophy. The structural findings for the RA patients are listed in Table 2.

**RA patients with raised pulmonary artery pressure**

Forty-five RA patients had a pulmonary artery pressure of 30 mmHg or greater. No patients with a history of pulmonary embolus had pulmonary hypertension. Three RA patients had impaired left ventricular function, as assessed by left ventricular ejection fraction, one patient having concomitant obstructive lung disease. One patient had moderate mitral regurgitation and obstructive lung disease. Chest HRCT showed that 13 patients had emphysematous bullae, five had interstitial lung disease with a fibrosing alveolitis pattern, and three had emphysematous bullae and fibrosing alveolitis. In nine of these patients the lung disease was sufficiently severe to cause significant volume loss (as defined above, under Significant lung disease) on pulmonary function testing. Clinically, 6% of the RA population studied had pulmonary hypertension that was secondary to lung disease.

The pulmonary artery pressures are shown in Table 3, and the ECG findings that were found most frequently in patients with pulmonary hypertension are shown in Table 4.

The clinical features of the patients with primary pulmonary hypertension were compared with those of RA patients who had a pulmonary artery pressure below 30 mmHg. The findings are shown in Table 5. No significant difference was found between the two RA groups when sex, disease duration, disease severity, smoking and corticosteroid treatment were compared. The acute-phase response, as assessed by plasma viscosity and C-reactive protein measured at the time of clinical evaluation, did not differ significantly between the groups. All the patients with primary pulmonary hypertension were recorded as taking NSAIDs, and 94% of the patients with RA whose PASP was below 30 mmHg were taking NSAIDs; again this was not statistically significant.

We subsequently investigated the patients with primary pulmonary hypertension for the presence of antiphospholipid antibodies, but none of the patients tested was found to have a significantly elevated concentration (normal range, IgG 0–9 GPL U/ml, IgM 0–4 MPL U/ml).

**Pulmonary artery pressure in the control group (Table 6)**

Fifty-five out of 144 patients had a pulmonary artery pressure of 30 mmHg or greater (range 31–82). Cardiac causes of pulmonary hypertension were found in 38 patients. Fourteen patients had an elevated PASP without a known cardiac cause. This is significantly less \((P = 0.025)\) than that found in the rheumatoid population.

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**Table 2. Structural findings found on echocardiography of patients with RA**

<table>
<thead>
<tr>
<th>Finding</th>
<th>No. patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic stenosis</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
<td>5 (3)</td>
</tr>
<tr>
<td>Aortic root dilatation</td>
<td>12 (8)</td>
</tr>
<tr>
<td>Left atrial enlargement</td>
<td>33 (23)</td>
</tr>
<tr>
<td>Minimal mitral regurgitation (Doppler flow detected)</td>
<td>25 (17)</td>
</tr>
<tr>
<td>Moderate mitral regurgitation</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Reduced left ventricular function (&lt;64%)</td>
<td>13 (9)</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Tricuspid regurgitation (Doppler flow detected)</td>
<td>113 (77)</td>
</tr>
<tr>
<td>Valsalva sinus rupture</td>
<td>1 (0.7)</td>
</tr>
</tbody>
</table>

**Table 3. Pulmonary artery pressure in RA patients**

<table>
<thead>
<tr>
<th>PASP</th>
<th>No. patients*</th>
<th>No. patients with PPH*</th>
</tr>
</thead>
<tbody>
<tr>
<td>30–39 mmHg</td>
<td>39 (27%)</td>
<td>29 (20%)</td>
</tr>
<tr>
<td>40–50 mmHg</td>
<td>4 (3%)</td>
<td>1 (0.7%)</td>
</tr>
<tr>
<td>&gt; 50 mmHg</td>
<td>2 (1%)</td>
<td>0</td>
</tr>
</tbody>
</table>

PPH, primary pulmonary hypertension.

*Numbers in parentheses are percentages of the 146 RA patients who had echocardiograms.
Raised pulmonary artery pressure in RA

<table>
<thead>
<tr>
<th>ECG findings</th>
<th>No. patients</th>
<th>PFT/HRCT findings</th>
<th>Echocardiography findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-wave enlargement</td>
<td>6</td>
<td>3 obstructive lung disease, 2 obstructive and interstitial lung disease, 1 interstitial lung disease</td>
<td>PASP 24, 25, 26, 29, 30 mmHg and 1 unable to obtain reading</td>
</tr>
<tr>
<td>RAD</td>
<td>1</td>
<td>Obstructive</td>
<td>26 mmHg</td>
</tr>
<tr>
<td>P wave enlargement, RAD and right bundle branch block</td>
<td>1</td>
<td>Obstructive</td>
<td>33 mmHg</td>
</tr>
<tr>
<td>Right bundle branch block</td>
<td>2</td>
<td>Obstructive</td>
<td>39 and 44 mmHg</td>
</tr>
</tbody>
</table>

Obstructive lung disease was defined by pulmonary function testing (PFT); interstitial lung disease with a fibrosing alveolitis pattern was diagnosed by thoracic HRCT.

RAD, right axis deviation.

Discussion

We have taken a cross-section of routinely reviewed district general hospital RA patients and have screened them for cardiac and lung disease. Particular attention was paid to the presence of raised pulmonary artery pressure. The diagnosis of primary pulmonary hypertension has a very poor prognosis. The identification of pulmonary hypertension secondary to lung disease both alters management and conveys a poor prognosis. The RA patients were carefully assessed for lung disease, as this is thought to be the most common cause of pulmonary hypertension in RA.

Pulmonary artery pressure was also recorded in 143 patients with known or suspected heart disease in order to ascertain the importance of raised pulmonary artery pressure. We found that 10% of cardiology patients had mildly elevated pulmonary artery pressure without a well-established cardiac cause of pulmonary hypertension.

Echocardiography identified a number of structural abnormalities in the RA population. Previous studies have reported similar findings in RA patients when compared with a normal population. RA patients have been found to have an increased prevalence of pericardial effusion, mitral valve abnormalities and impaired left ventricular function [18, 19]. Aortic root enlargement was also found more frequently in patients with RA than controls [18].

Although researchers agree on the structural cardiac changes that are increased in RA, consistency between echocardiographic studies in RA patients is not found. The reported prevalence of pericardial effusion ranges from 4% [19] to 44% [20] and that of mitral valve abnormalities from 6% [21] to 30% [22].

This study has demonstrated the new finding that 21% of district general hospital RA patients have mild to moderate primary pulmonary hypertension, as defined by Doppler echocardiography. By the use of full pulmonary function testing at the time of echocardiography, we excluded any significant lung disease. Chest HRCT was also performed on these patients, and it identified early fibrosing alveolitis in an additional six patients in the pulmonary hypertension group. Without volume loss on pulmonary function testing, the fibrosing alveolitis is unlikely to be at a stage to be causing

Table 4: ECG findings in RA patients

<table>
<thead>
<tr>
<th>ECG findings</th>
<th>No. patients</th>
<th>PFT/HRCT findings</th>
<th>Echocardiography findings</th>
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RAD, right axis deviation.

Table 5: Characteristics of RA patients according to pulmonary artery pressure

<table>
<thead>
<tr>
<th>Variable</th>
<th>Primary pulmonary hypertension (mean)</th>
<th>Pulmonary artery pressure &lt; 30 mmHg (mean)</th>
<th>P value (Mann–Whitney U-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>56.9 ± 7.4</td>
<td>58.4 ± 10.5</td>
<td>0.55</td>
</tr>
<tr>
<td>RA duration (yr)</td>
<td>11.2 ± 8.3</td>
<td>13.6 ± 8.6</td>
<td>0.26</td>
</tr>
<tr>
<td>Cigarette pack yr</td>
<td>10.1 ± 11.7</td>
<td>17.1 ± 19.4</td>
<td>0.21</td>
</tr>
<tr>
<td>HAQ score</td>
<td>1.73 ± 0.72</td>
<td>1.59 ± 0.79</td>
<td>0.33</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>40.4 ± 2.3</td>
<td>40.6 ± 3.4</td>
<td>1.0</td>
</tr>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>12.9 ± 1.2</td>
<td>14.0 ± 4.3</td>
<td>0.18</td>
</tr>
<tr>
<td>C-reactive protein (mg/l)</td>
<td>30.6 ± 29.1</td>
<td>30.9 ± 31.6</td>
<td>0.98</td>
</tr>
<tr>
<td>Plasma viscosity (cps)</td>
<td>1.72 ± 0.10</td>
<td>1.77 ± 0.38</td>
<td>0.84</td>
</tr>
</tbody>
</table>

Table 6: Pulmonary artery pressure in the control group

<table>
<thead>
<tr>
<th>PASP</th>
<th>Total number</th>
<th>Without cardiac cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>30–39 mmHg</td>
<td>39 (27)</td>
<td>14 (10)</td>
</tr>
<tr>
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<td>12 (8)</td>
<td>0</td>
</tr>
<tr>
<td>&gt; 50 mmHg</td>
<td>4 (3)</td>
<td>0</td>
</tr>
</tbody>
</table>

Numbers in parentheses are percentages of all control patients who had an echocardiogram.
secondary pulmonary hypertension. Whether or not fibrosing alveolitis on HRCT predicts the rate at which pulmonary hypertension progresses is yet to be determined.

Doppler echocardiography is both sensitive and specific for the diagnosis of pulmonary hypertension [6, 18]. By taking a definition of pulmonary hypertension as a PASP of 30 mmHg or greater, we will have underestimated the number of patients with pulmonary hypertension. This is supported by the observation that patients with right-heart strain patterns on ECG had estimated pulmonary artery pressures of at least 24 mmHg.

The relative lack of ECG changes has been noted in studies with right-heart catheterization. This occurs particularly in secondary pulmonary hypertension [23]. P-wave changes are found in 20% of patients with pulmonary hypertension and right-sided strain patterns in 70% [24]. In the two patients with severe pulmonary hypertension, ECGs showed left bundle branch block patterns masking the typical changes of right-sided heart predominance.

It has been noted recently that RA patients have an increased number of abnormalities related to the function of the left ventricle [19, 25]. The frequency of these abnormalities is significantly higher than that in control populations when patients with diabetes mellitus, myocardial infarction and hypertension are excluded.

Impairment of left ventricular function can cause elevation of pressure on the left atrium and raise pulmonary artery pressure. It would seem unlikely that this is the cause of the raised pulmonary artery pressure, as patients with a reduced left ventricular ejection fraction have been excluded.

When using a PASP of 30 mmHg as the cut-off point to compare the features of RA patients, we have not found an explanation for the raised pulmonary artery pressures. This may be because we have used a high threshold to diagnose pulmonary hypertension with echocardiography; a PASP below 24 mmHg may well be more suitable for comparisons. The study would certainly need to be extended to have sufficient power to ensure that a type II statistical error does not arise. To be certain that the degree of acute-phase response is not related to the development of pulmonary hypertension, serial measurements would be superior to a single reading at the time of assessment.

The only RA study that has included pulmonary artery pressure, by Wislowska et al. [19], did not record a PASP above 35 mmHg. The paper is not focused on pulmonary hypertension and does not give the method of determining PASP, the number of patients who had PASP readings and the reason for using a threshold above 35 mmHg. We found that 19% of all the RA patients had PASP within the range 30–35 mmHg. It is possible that this mild but definite pulmonary hypertension was recorded by Wislowska et al. but not published because of the threshold used. It may be that only a small number of their patients had PASP estimates made, thus reducing the chance of identifying those with pulmonary hypertension. It certainly suggests that the left-sided heart abnormalities they found in their population were not causing significant cardiac disease. It is also interesting to postulate that if they had no patients with PASP above 30 mmHg, the selection bias between their study group and ours may explain the cause of pulmonary hypertension in our patients.

Our control population represents pulmonary artery pressure readings in a non-rheumatoid population undergoing echocardiography on the same equipment by the same technician. Obviously, these readings do not represent the normal population and referral bias explains the high prevalence of cardiac disease. We will have potentially overestimated the number of patients with primary pulmonary hypertension, as the control group was not screened for lung disease or other known causes of pulmonary hypertension. Despite these factors, the rheumatoid population still had a significantly higher prevalence of raised pulmonary artery pressure.

A limitation of our study is that we did not assess pulmonary artery pressure by echocardiography in normal age- and sex-matched controls. A study of 20 normal healthy adults by Vachiery et al. [17] with Doppler echocardiography found that the maximum estimated pulmonary artery pressure was 24 mmHg.

As Doppler echocardiography and cardiac catheterization have been reported to have a correlation of between 0.89 and 0.97 in cardiac causes of pulmonary hypertension [10, 11], we have not undertaken catheterization of our RA patients. In 1998 the first case report appeared in which echocardiographic estimation of pulmonary artery pressure was found to be incorrect at the time of right-heart catheterization [26]. It would seem unlikely that, in a large study of RA patients that has identified 45 patients with raised PASP pressure, this is an aberrant finding.

We have identified mild pulmonary hypertension in RA patients. In these patients no significant symptoms, signs, ECG or structural echocardiographic changes were apparent. Of course, this may be because we detected the disease at an early stage, but right-sided ECG abnormalities were detected at pulmonary artery pressures of 24 mmHg and above in patients with severe lung disease. Bach et al. [4] also found that pulmonary hypertension (>35 mmHg) secondary to emphysema was associated with right-sided structural abnormalities visible on echocardiography. It is likely that an explanation of these differences in the cardiac response to raised pulmonary artery pressure will be forthcoming only when we understand the pathogenesis of pulmonary hypertension in RA patients.

Due to the physical limitations arising from arthritis, RA patients present with shortness of breath as a result of cardiorespiratory disease at a later stage than the normal population. Deaths that have been assumed previously to be due to ischaemic heart disease, because the patients presented with chest pain or cardiac arrest, may really have been due to primary pulmonary hypertension.

PASP can be identified non-invasively by Doppler
echocardiography. This is dependent on a trace of tricuspid regurgitation being present and a chest wall physique that allows suitable views to be obtained. We found, with a dedicated technician, that up to 75% of RA patients could have PASP estimates with Doppler echocardiography, and so, should pulmonary hypertension progression be inexorable, then treatment trials could easily be undertaken on RA populations with echocardiographic assessment of outcome.

This study raises an important issue—we have found that 31% of unselected hospital RA patients have pulmonary hypertension on echocardiography. In 6% of our RA patients this was due to lung disease and in a further 4% it was due to cardiac disease. Mild to moderate primary pulmonary hypertension is not a rare finding in the RA population. Further research into the pathogenesis and progression of raised pulmonary artery pressure in RA patients is needed.

Acknowledgements

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References