pressure or hypotensive episodes were recorded during the trial. Among the side effects, mild flushing (35%) and headache (2%) were the most frequent in isradipine recipients.

This study has demonstrated the favourable effects of isradipine on patients with Raynaud's phenomenon; moreover, it is the first to demonstrate a significant reduction in plasma concentrations of ET-1 during calcium antagonist treatment. The most usual manifestations of Raynaud's phenomenon are pain and numbness in the fingers, which in some subjects can be complicated by skin ulcers requiring prompt intervention. In the present study, isradipine was able to reduce the frequency, severity, and disabling nature of acute attacks of Raynaud's phenomenon; this result agrees with previous experience of the use of calcium antagonists in patients with Raynaud's phenomenon, and reflects an appropriate selection of patients to receive the treatment. In addition, the clinicoendothelial manifestations and changes in capillaroscopic findings in this series of patients, including those with type I and type II Raynaud's phenomenon, were relatively homogeneous.

The clinical improvement of our patients with Raynaud's phenomenon in response to isradipine was reflected in a reduction in plasma concentrations of ET-1. Although the pathophysiological function of ET-1 remains unknown, a number of vascular disorders are characterised by increased concentrations of this vasoactive peptide. How isradipine may influence the titres of ET-1 is difficult to explain but, because ET-1 is released in response to ischaemic stimuli, we hypothesise that isradipine is able to induce a change in ET-1 concentrations indirectly, by improving tissue perfusion. The findings of this preliminary investigation merit further investigation in larger series of patients with Raynaud's phenomenon.

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Survival after aortic dissection in giant cell arteritis

Acute aortic dissection in giant cell arteritis is rare and usually fatal. It is associated with hypertension and is commoner in females. We describe two patients who were diagnosed in life and who survived on medical treatment alone for a prolonged period: one for two years; the other still alive at three years. A third patient who required aortic grafting has survived 14 years. A literature review revealed 43 other cases, with only three survivors on medical care alone.

Case reports

PATIENT 1

A 68 year old white male presented with acute chest pain on exertion radiating to the back, accompanied by dyspnoea and sweating. He had noted weight loss of more than 7 kg over the previous months. A history of back and neck ache was ascribed to spondylosis, and should pain to a rotator cuff lesion. His pulse was 60 beats/min regular, and his blood pressure was 150/70 mm Hg. An ejection murmur was noted at the left sternal edge. Examination was otherwise unremarkable.

The patient developed intermittent pyrexia over the next three weeks, and experienced frequent left sided stabbing chest pain. The leucocyte count increased from normal to 14.5 x 10⁶/l with a neutrophilic leucocytosis, erythrocyte sedimentation rate (ESR) peaked at 94 mm/1st h and C reactive protein (CRP) at 260 mg/l. Blood and urine cultures were negative, and autoantibody screen was normal. The electrocardiogram (ECG) showed left ventricular hypertrophy, confirmed by trans-thoracic echocardiogram, which showed no other abnormality. Chest radiograph showed minor atelectasis at both lung bases. Indium labelled leucocyte scan showed some mediastinal uptake. Computed tomography (CT) of the thorax, without intravenous contrast medium (because of asthma), showed a normal mediastinum but a small rim of pleural fluid on the left. At follow up, the patient complained of right thigh claudication. Radiofemoral delay was noted. CT of the abdomen showed internal displacement of calcified atheroma in the aorta, suggestive of aortic dissection. Magnetic resonance imaging (MRI) confirmed this as distal to the origin of the left subclavian artery, extending to the aortic bifurcation. Eight weeks after discharge, he developed headaches, aching thighs and jaw claudication with temporal artery tenderness. Prednisolone 30 mg daily was begun with rapid improvement. Clinical improvement correlated with his ESR decreasing to normal and with gradual resolution of the radiofemoral delay.

PATIENT 2

A 68 year old white women presented with five days of worsening anterior pleuritic chest pain radiating to the left, sweets, nausea, and an increased temperature. She had a history of osteoarthritis, euthyroid Graves' ophthalmopathy, Raynaud's phenomenon, and long-standing hypertension. Temporal arteritis had been diagnosed on biopsy eight years previously, following a characteristic illness accompanied by an ESR of 121 mm/1st h. Prednisolone for three years had achieved remission.

On admission, the patient was pyrexial at 37.7°C. The heart rate was 110 beat/min with no radiofemoral delay, and the blood pressure was 140/90 mm Hg, equal in both arms. There was dullness to percussion at both lung bases and bronchial breathing at the left base. ESR was 86 mm/1st h, and the leucocyte count 11.4 x 10⁹/l with a neutrophilia. ECG showed left ventricular hypertrophy. Chest radiograph showed bilateral small pleural effusions and a markedly widened mediastinum. CT of the thorax and abdomen revealed a dilated thoracic aorta with a dissection in the descending portion. Transoesophageal echocardiography (TOE) initially suggested dissection commencing at the aortic root; however, repeat CT, arch aortography and MRI confirmed type B dissection. The patient was treated with a β blocker to maintain normal blood pressure, and prednisolone at a dose of 20 mg. The pain resolved and she was discharged, remaining well and normotensive for two years. Mediastinal widening then began to accelerate, MRI imaging confirming a grossly dilated dissecting descending aorta and dilatation of the ascending aorta (figure). She died soon afterwards.
Summary of cases of aortic dissection associated with giant cell arteritis reported in the literature

<table>
<thead>
<tr>
<th>Source</th>
<th>Number of cases</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Hypertension</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broch 1947</td>
<td>1</td>
<td>68</td>
<td>F</td>
<td>+</td>
<td>First recorded case (case report)</td>
</tr>
<tr>
<td>Klein 1953</td>
<td>3</td>
<td>66</td>
<td>F</td>
<td>NK</td>
<td>Series of 248 cases and 3 sudden deaths; diagnosed at postmortem examination</td>
</tr>
<tr>
<td>Larson 1984</td>
<td>2</td>
<td>NK</td>
<td>F</td>
<td>+</td>
<td>Postmortem series of 161 acute dissections; 'healed aortitis' found in 14</td>
</tr>
<tr>
<td>Nordt 1981</td>
<td>1</td>
<td>70</td>
<td>F</td>
<td>-</td>
<td>Postmortem series of 171 acute dissections</td>
</tr>
<tr>
<td>NK</td>
<td>Not known; GCA = giant cell arteritis; LHV = left ventricular hypertrophy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Discussion

Giant cell arteritis is distinguished from Takayasu's disease on clinical and pathological grounds. Medium sized arteries are usually affected, although 15% of a large series were found to have large vessel involve-
ment. Despite the characteristic histopathological findings of mononuclear cell infiltrate, disruption of the internal elastic lamina and the presence of giant cells which would be expected to reduce the elasticity and tissue strength of large arteries, aortic dissection is an unusual complication (table). The first case was reported from Norway in 1947. A postmortem study of 111 cases of dissecting aortic aneurysm from western Japan did not imply giant cell arteritis as a cause in any case, and 161 postmortem cases of dissecting aortic aneurysm from the Mayo Clinic also failed to implicate giant cell arteritis with certainty, though two had 'healed aortitis'. Klein describes three cases of aortic dissection in giant cell arteritis that were fatal, one in the descending and two in the ascending aorta. Of 125 postmortem cases seen in Manchester, England, two had evidence of giant cell arteritis. The usual cause of death is aortic rupture, though old, healed dissection may be an incidental finding. In a postmortem series of nine deaths related to giant cell arteritis, Savel-Soderbergh et al mentioned two elderly hypertensive females with aortic dissection, at least one of whom appears to have had a descending aortic lesion.

The first reported case diagnosed and who survived for some months was described in Ipswich. One previous case from our hospital did well after aortic resection and is still alive and symptom free 14 years later. In two other reports, two of 16 postmortem cases of giant cell arteritis died of aortic dissection, and two further fatal cases in women have been described from Japan.

Most of these patients showed evidence of hypertension in life or at postmortem examination. Twenty-two of 27 reported cases were described in patients with aortic dissection; the male/female ratio was 1:2:1.

Antibodies to collagen in sera from patients receiving bovine cartilage graft.

The production of autoantibodies against different collagen types has been described in association with several different experimental pathological entities. Antibodies to collagen type II have been demonstrated in relapsing polychondritis and in rheumatoid arthritis.

Cartilage grafts for tissue reconstruction have been used in many surgical specialties, especially in otorhinolaryngology and maxillofacial surgery. Autograft cartilage has the tendency to warp. For a better harvest of the material requires an additional operative procedure. Recently, we observed humoral reactivities against cartilage in patients who underwent a nasal contour reconstruction with allo-
genetic or autologous cartilage grafts. These
Survival after aortic dissection in giant cell arteritis.

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doi: 10.1136/ard.55.5.332

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