tion deformities of the fourth and fifth metatarsophalangeal joints of both feet; the proximal interphalangeal joints of the right third, fourth, and fifth toes; and also the proximal interphalangeal joints of the left fourth and fifth toes.

Case 2—A 40-year-old housewife had developed systemic lupus erythematosus at the age of 28 with arthritis, pericarditis, pleurisy, and focal proliferative glomerulonephritis. Over the past five years her hands had become progressively deformed with pronounced ulnar deviation and flexion contractures, and for two years she had had severe clawing of the toes. Radiography of the feet showed subluxation of the right third, fourth, and fifth metatarsophalangeal joints; the left third and fourth metatarsophalangeal joints; and the left first proximal interphalangeal joint. Case 3—A 29-year-old girl had systemic lupus erythematosus for 16 years which had presented with thrombocytopenic purpura, fever, pleurisy, rash, and symmetrical arthritis affecting her hands, knees, and ankles. During the previous 13 years of her disease she had remained clinically and serologically active, with average DNA binding values of 75% (upper limit 30% in the Farr technique), and the main feature of her disease had been widespread arthralgias. Her hands had become grossly deformed with severe flexion contractures, and over the past two years she had developed similar changes in her feet. Radiography of the feet showed bilateral subluxation of the interphalangeal joints of the fourth and fifth toes and the interphalangeal joint of the right hallux.

Comment
Clinical and radiological abnormalities of the hands in systemic lupus erythematosus have been well documented. The changes described here in the feet are similar to those observed in the hands, and the underlying disease is likely to be the same. The prevalence of foot deformity due to systemic lupus erythematosus is impossible to calculate accurately but is much lower than that of hand deformity. In all three patients the hands were particularly deformed and affected several years before the feet. While the patients we describe had had serologically active systemic lupus erythematosus for several years there did not appear to be a correlation between the development of arthritis and any clinical or serological pattern. The process that results in the deformity is not understood but may result from inflammatory changes in tendons or periarticular structures. Occasionally a patient with systemic lupus erythematosus is noted to have pain and erythematous tenderness along the path of tendons. Inflammation and fibrosis have been described in tendon sheaths, and this may in turn result in acute or insidious contracture of the tendon.

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Hyponatraemia in legionnaires' disease

Hyponatraemia is common in legionnaires' disease, and has been attributed to the syndrome of inappropriate secretion of antidiuretic hormone. Our results suggested, however, that an alternative explanation is more likely.

Patients, methods, and results

Between December 1976 and May 1979, 24 patients with acute pneumonia were found to have legionnaires' disease. Thirteen were hyponatraemic on admission (serum sodium concentration lower than 130 mmol(mEq)/l), and none was diabetic or receiving diuretics or corticosteroids. All had normal serum sodium concentrations (135-145 mmol/l) by the time of discharge. Before treatment five patients had measurements of sodium and osmolality, using standard methods, on simultaneous blood and urine samples.

Findings on admission in the five patients studied

<table>
<thead>
<tr>
<th>Case No</th>
<th>Serum</th>
<th>Urine</th>
<th>Serum</th>
<th>Urine</th>
<th>Hypovolaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>400</td>
<td>127</td>
<td>4</td>
<td>Present</td>
</tr>
<tr>
<td>2</td>
<td>265</td>
<td>817</td>
<td>129</td>
<td>5</td>
<td>Present</td>
</tr>
<tr>
<td>3</td>
<td>279</td>
<td>806</td>
<td>128</td>
<td>7</td>
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<tr>
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</tr>
<tr>
<td>5</td>
<td>268</td>
<td>530</td>
<td>129</td>
<td>7</td>
<td>Present</td>
</tr>
</tbody>
</table>

Conversion: SI to traditional units—Osmolality: 1 mmol/kg = 1 mosmol/kg. Sodium: 1 mmol/l = 1 mEq/l.

Patients were considered to be hypovolaemic if jugular venous pulsation became visible only with the patient lying at an angle of less than 5° and systolic blood pressure was at least 20 mm Hg greater in the supine than in the standing position. The table shows results of investigations on admission. All five patients were hypovolaemic, with high urine osmolality and urinary sodium concentrations below 8 mmol/l. In a group of 12 patients with legionnaires' disease 10 were hypovolaemic. One of the two exceptions had normal circulating...
volume clinically yet with an initial serum sodium concentration of 124 mmol/l; she had, however, been drinking large volumes of water before admission.

Comment

Since all patients with legionnaires’ disease gave a history of one or more of vomiting, watery diarrhoea, profuse sweating, and rapid weight loss, clinically detectable hypovolaemia was not surprising. In such conditions hypotension may occur by both renal and hypothalamic mechanisms, particularly if the patient is anorectic and therefore stops ingesting salt. The body’s response tends to conserve circulating volume at the expense of osmolality; though secretion of antidiuretic hormone is increased, this is entirely appropriate. In the original description of the syndrome of inappropriate secretion of antidiuretic hormone both patients were investigated in a steady state with normal circulating volume and had high urinary sodium concent-

Acute hypertension after traumatic renal artery thrombosis with high circulating concentrations of angiotensin II

Hypertension is a well-recognised complication of traumatic renal artery thrombosis. It occurs in up to half of patients who do not undergo early surgery but is rarely detected within the first few weeks of injury. A possible role for the renin-angiotensin system was suggested by Von Knorrning et al. We describe a patient who became severely hypertensive 48 hours after traumatic renal artery thrombosis and in whom plasma concentrations of renin, angiotensin II, and aldosterone were measured serially.

Case report

A 48-year-old man was admitted to hospital after a road traffic accident. He was shocked (blood pressure 70/40 mm Hg with a standard sphygmo-

Changes in plasma concentrations of active renin, angiotensin II, and aldosterone, blood pressure, serum creatinine concentrations, and urinary output after traumatic renal artery thrombosis. Intra-venous infusions of clonidine (C), hydralazine (H), and nitroprusside (Np) were administered before nephrectomy.

Comment

Although hypertension is a well-recognised complication of traumatic renal artery thrombosis, its incidence is uncertain, probably because most patients undergo early surgery and have other features that may modify blood pressure. Cass et al. found no evidence of hypertension in 31 consecutive patients with renal pedicle injuries, including seven who did not undergo nephrectomy or vascular repair, and whose blood pressure remained normal for a mean of 29 months. On the other hand, Stables et al. noted hypertension in five out of 10 patients who did not undergo surgery.

Generally, where hypertension has occurred the interval between injury and its detection varies from 13 weeks to three years; thus the