Oedema (‘an abnormal build-up of fluid in the tissues’) can be a presenting feature of many serious medical conditions, notably congestive heart failure, liver failure, malnutrition and the nephrotic syndrome. Peripheral oedema can also result from venous or lymphatic obstruction or from excessive administration of salt and water. Agents such as non-steroidal anti-inflammatory drugs (NSAIDs) and calcium channel blockers can also produce peripheral oedema.

Presentation
Patients present complaining of swelling of the legs. In severe cases oedema extends to cause abdominal swelling (from ascites), sacral oedema, pleural effusions, pulmonary oedema and even facial swelling. Oedema is often, although not always, posturally dependent, and in bed-bound individuals it may be confined to the sacrum.

Diagnosis
Accurate history taking is vital. Symptoms and signs of cardiac, liver and renal disease should be sought. Two questions are the key to the diagnosis. Is the oedema unilateral or bilateral? Is the venous pressure raised or not? It is also important to determine whether oedema is present in other sites. Oedema diffusely affecting the whole body suggests a low serum albumin, or ‘leaky’ capillaries, rather than heart failure.

Bilateral leg oedema
In bilateral leg oedema, the diagnosis rests in determining whether the venous pressure is elevated and whether there are signs of liver disease, severe immobility or malnourishment.

- **Heart failure**: leg oedema occurs from right-sided heart failure and is always associated with a high jugular venous pressure (JVP) (Table 5.1). Hepatomegaly is often seen, as are signs of underlying cardiac pathology. If the oedema is mild in the legs, but severe in the abdomen, pericardial constriction should be considered.
- **Liver failure**: leg oedema is caused by a low serum albumin (usually < 20 g/dL). There may be signs of chronic liver disease, such as spider naevi, leuconychia, gynaecomastia, dilated abdominal veins indicating portal hypertension, and bruises (impaired liver synthetic function). The JVP is not elevated. In severe chronic liver disease (e.g. cirrhosis), liver enzyme tests may be only mildly disturbed, although the pro-
to determine blood flow and computed tomography (CT), and occurs:

by ultrasonographic studies of the abdomen, using colour flow Doppler
compression on the inferior vena cava (IVC). This can be diagnosed
• IVC compression

• Serum albumin and cause leg oedema.

• Malnutrition

• General immobility: the patient is usually elderly, and obviously
immobile from general infirmity or cerebrovascular disease. The JVP
down, and there are no signs of liver or renal disease.

• Malnutrition: any chronic illness may be associated with a catabolic
state and a degree of malnutrition that can be severe enough to depress
serum albumin (usually < 30 g/dL), urinary protein (usually
> 4 g/24 h) and serum creatinine and urea) or an inability to excrete
fluid (nephritic syndrome, associated with hypertension and low urine
output).

• General immobility: the patient is usually elderly, and obviously
imobile from general infirmity or cerebrovascular disease. The JVP
is down, and there are no signs of liver or renal disease.

• Malnutrition: any chronic illness may be associated with a catabolic
state and a degree of malnutrition that can be severe enough to depress
serum albumin and cause leg oedema.

• IVC compression: rarely, bilateral leg oedema can be caused by
compression on the inferior vena cava (IVC). This can be diagnosed
by ultrasonographic studies of the abdomen, using colour flow Doppler
to determine blood flow and computed tomography (CT), and occurs:
• in extreme obesity

• in severe (tense) ascites from whatever cause

• with extensive venous thrombosis in the IVC, such as occurs in
malignancy, or as a complication of the nephrotic syndrome.

Unilateral leg oedema
One-sided leg swelling is likely to have a local underlying cause, such as:

• A deep venous thrombosis (DVT) in the leg causes a slow onset
(more than a few hours) of unilateral leg pain, swelling, with skin
warmth, and possibly tenderness in the calves and along the veins,
particularly the great saphenous vein. As symptoms/signs are unreli-
able for diagnosis, all patients with suspected DVTs should undergo
definitive investigations (vein ultrasonography or venography) and be
examined for complicating pulmonary embolisms (PEs) (see Chapter 7).

• Ruptured Baker’s cyst: a Baker’s cyst is a knee joint bursa that juts
into the popliteal fossa and usually occurs in rheumatoid arthritis. It
may rupture and cause sudden-onset leg pain and calf swelling. Ultra-
sonography is diagnostic.

• Cellulitis: consists of an intense spreading skin erythema, sometimes
well demarcated, occasionally tracking up the line of the lymphatics. It
is often very painful and is associated with a temperature, and raised
erthrocyte sedimentation rate (ESR), C-reactive protein and white
count. The organism is usually one of the staphylococci or streptococci
species, and is occasionally grown from blood cultures, although rarely
from skin swabs.

• Lymphatic obstruction results in a ‘woody’ form of unilateral
oedema, sometimes described as ‘non-pitting’. It is very rare in the
West, and when found is usually the result of carcinomatous invasion
and obliteration of the draining lymph nodes, e.g. in metastatic
melanoma. In Africa lymph obstruction is very common, often bilat-
eral, and caused by filarial infestation.

• Pelvic tumours can unilaterally compress veins, causing unilateral
oedema.

• Localized immobility can cause unilateral leg oedema, e.g. long-
standing hemiparesis.

 Investigations
These vary depending on the features established by history and exam-
ination but determination of serum albumin, urinary protein loss, liver
function tests, creatinine, ECG, chest X-ray and echocardiography are
often appropriate.

 Treatment
Therapy is directed at correcting the underlying cause. In bilateral
oedema diuretics are often used to promote salt and water excretion,
although their use should be balanced against the risk of hypovolaemia
and worsening renal function, postural hypotension and falls. Several
different classes of diuretic agent are used (see Table 5.2). The use of a
loop diuretic in combination with a thiazide can produce a pronounced
diuretic effect that is useful in resistant oedema. Spironolactone, a
competitive aldosterone antagonist, produces a mild natriuresis and
potassium retention, and is utilized in conditions with secondary hyper-
aldosteronism such as liver cirrhosis with ascites. Spironolactone and
amiloride are ‘potassium-sparing’ diuretics, in contrast to the loop and
thiazide diuretics which promote potassium depletion.

<table>
<thead>
<tr>
<th>Class</th>
<th>Example</th>
<th>Diuretic potency</th>
<th>Na/K+ lowering potential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiazide</td>
<td>Bendrofluamethiazide</td>
<td>+</td>
<td>+/+/</td>
</tr>
<tr>
<td>Loop</td>
<td>Frusemide</td>
<td>++/++</td>
<td>+/+++</td>
</tr>
<tr>
<td>K+ sparing</td>
<td>Amiloride</td>
<td>±</td>
<td>±/±/</td>
</tr>
<tr>
<td></td>
<td>Spironolactone</td>
<td>±±</td>
<td>±/±/</td>
</tr>
</tbody>
</table>