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Peripheral Oedema and Venous Disorders

The Institute of Chiropodists and Podiatrists March 2010

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Peter Roberts
By the end of this session you will be able to:

• understand the causes of Peripheral Oedema
• differentially diagnose between local oedema and systemic causes
• recognise the clinical signs and symptoms of DVT, thrombophlebitis, and other causes of peripheral oedema
• Appreciate the podiatric implications and management strategies for patients with peripheral oedema
Peripheral Oedema

- Balance between extracellular and intracellular fluid is disturbed
- Changes of extracellular fluid volume
  - 50% of body weight
    - 75 kg man 42 litres 2/3 intracellular 1/3 extracellular
- Interstitial fluid & blood plasma
- Na Cl extracellular
Systemic Causes

- Heart failure
- Kidney impairment / failure
- Liver cirrhosis / failure
- Hypoalbuminaemia
- Idiopathic oedema women

- Localised oedema
Causes

**Lifestyle**

- Immobility
- Hot weather
- Exposure to high altitudes
- Burns
- Menstruation
- Pregnancy,
- Medication such as high blood pressure medicine and corticosteroids
- High Salt intake

**Systemic**

- kidney disease
- heart failure
- chronic lung disease
- thyroid disease
- liver disease
- malnutrition.
• Cellulitis
• Ischaemia
• Muscle rupture compartment syndrome
Peripheral Arterial Disease

Venous Disorders

Lymphatic Disorders
Peripheral Arterial Disease

Supply

Venous Disorders

Drainage

Lymphatic Disorders

Drainage
What would you expect to occur if drainage was impaired?
Drainage/Removal of waste products
Lymphatic System

• The lymphatic system is a network of closed but permeable vessels containing valves
• The function of the lymphatic system is to clear interstitial fluid from tissues
Lymph Vessels

Semilunar valve
Drainage of interstitial fluid

Travels to the sub-clavian vein

To the lymph nodes for filtration and activation of lymphocytes

Returns to the arterial system

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Lymphatic Pathology

- Fluid accumulation can arise due to the absence, damage to, or obstruction of lymphatic vessels affecting the transporting capacity of the system.

- When fluid formation is increased to a point that it overwhelms lymphatic absorption oedema occurs.
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Lymphoedema

- Can present as unilateral or bilateral
- Is local or systemic in origin
- Soft and pitting or firm and woody
- Oedema presents primarily on the dorsum of the foot and disappears overnight
- As the condition becomes chronic oedema extends proximally, in severe cases to the thigh
Lymphoedema
Classifying Lymphoedema

- Primary (congenital)
- Secondary (acquired)
• Congenital Lymphoedema (autosomal dominant trait)

• Characterised by chronic lymphatic obstruction
Secondary Lymphoedema

Acquired

- **Intrinsic**
  - malignancy
  - radiotherapy
  - surgery
  - infection
    - congenital conditions

- **Extrinsic**
  - trauma
  - plaster cast
  - obesity
Presentation

- Initial soft swelling of the foot and leg
- Over time this becomes firmer and heavier
- Skin becomes thickened and keratinised, may see lichenification
- May weep oedematous fluid
- May see secondary superficial bacterial infections and cellulitis
Clinical Diagnosis

• Depends on history of symptoms and on characteristic skin changes termed “elephantiasis”
• Speed of onset
• Differential diagnoses include:
  – Venous oedema
  – “armchair legs”
  – Lipoedema
Grades of Lymphoedema

- **Grade 1** swelling will pit if the skin is pressed and is reduced if the limb is elevated
- **Grade 2** has much more excess fibrous tissue, so it does not pit, and is not much reduced by elevation (e.g. in bed overnight or for 2-3 days). The limb gradually 'hardens' because of the excess fibrous (scar) tissue due to the chronic inflammation
- **Grade 3** *Elephantiasis* has gross changes to the skin
Kaposi-Stemmer Sign

- Skin becomes thicker, demonstrated by an inability to pinch a fold of skin at the base of the 2\textsuperscript{nd} toe
Papillomatosis

- Skin creases become enhanced
- A warty texture develops
Treatment of Lymphoedema

- Swelling is treated by elevation and compression (hosiery or multi-layer bandaging)
- Manual lymphatic drainage - massage
- Meticulous skin care required
- If secondary, then need to treat underlying cause
- Severe cases surgical bypass
Diuretics

- Loop
- Thiazide
- Potassium sparing
- Carbonic anhydrase inhibitors
Refer on

- GP
- Lymphoedema clinic
- Lymphodema support groups
Podiatric Implications

- Psychosocial factors- body image
- Quality of life implications
- Reduced mobility
- Altered gait
- Footwear implications
Podiatric Implications

- Reduced tissue viability
- Increased risk of foot ulceration
- Poor wound healing
- Severe oedema can reduce arterial perfusion and sensation
Anatomy of a Vein (and arteries)

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Venous Return

- Venous return (blood flowing back to the heart from the veins) depends on pressure differences from venules (90 mmHg) to the right atrium (0 mmHg)
- Venous return is assisted by contraction of the heart, skeletal muscle pumps and respiration
Calf muscle pump

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Respiratory Pump

- During inhalation the diaphragm moves inferiorly, this results in compression of abdominal veins and blood flows upwards to the right atrium.
- Pressures reverse during exhalation but valves within the veins prevent backflow.
Pathologies

Pathologies develop when venous return is impaired thus leading to chronic venous hypertension. (The back pressure of blood in the veins is elevated)

Common pathologies include:

- Varicose veins
- Superficial thrombophlebitis
- Deep vein thrombosis
- Venous Ulceration
Chronic Venous Hypertension
(From Valvular Incompetence)

• Perforator Incompetence
• Superficial venous incompetence
• Deep venous incompetence
• A combination of these three
Chronic Venous Hypertension

- Congenital defect
- DVT, Surgery, Trauma, Ageing, Pregnancy
- Pregnancy, Obesity

Valvular Incompetence

Backflow of blood from deep to superficial veins

Chronic Venous hypertension
Normal capillary fluid movement
Capillary fluid movement and venous hypertension
Venous pathology flow chart

1. Valvular Incompetence
2. Backflow of blood from deep to superficial veins
3. Vessel Dilation
4. Leakage into Tissues and Skin
5. PATHOLOGY
Varicose Veins

- It is estimated that over six million people in the UK suffer from varicose veins - five million of which are women.
- Onset usually reported between 20 - 40 years.
- Varicose veins are not regarded as a particularly dangerous problem, but they can be very painful and unsightly.
- However severe varicose veins can compromise tissue viability.
What are Varicose Veins?

- Dilated tortuous veins of the lower limb
- Arise due to incompetent venous valves
- Veins become enlarged and congested with blood
- Can range from minor dilatations to large grape like structures
Varicose Veins

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Causes of varicose veins

- Pregnancy: 70-80% of pregnant women develop varicose veins during the first trimester
- Pregnancy causes an increase in hormone levels and blood volume which can enlarge veins
Environmental factors

- Occupational - Standing or sitting in one position for too long, inactivity
- Diet - diet high in fat, refined sugar and low fibre is thought to be a contributory factor
- Injury - Valves can be damaged by knocks
- More frequent with advancing age but can occur at any age.
Symptoms of Varicose Veins

• Symptoms are exacerbated by long periods of standing
• Feelings of fatigue, heaviness, aching, burning, throbbing, itching, cramping and restlessness of the legs
Venous Insufficiency
- Clinical Observations

- Ankle oedema
- Haemosiderosis
- Teleangectasia
- Varicose eczema
- Atrophie Blanche
- Venous ulceration
Teleangectasia
(Dermal flare/ spider veins)
Venous Ulceration: common causes

- Trauma - supermarket trolleys
- Varicose eczema - itching
- Reduced tissue viability
Varicose Eczema

• In the early stages the main changes are orange-brown discolouration of the skin. Later the skin surface becomes scaly, itchy and develops other features of eczema.

• Weeping and infection are common which can result in venous ulceration.
Varicose Eczema

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Eczema or Cellulitis?

- Eczema = Itchy
- Cellulitis = Pain
Infected Varicose Eczema
Venous Ulceration
Atrophie Blanche/
Lipodermatosclerosis

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Management of Varicose Veins

- Exercise
- Avoid standing for long periods
- Elevate legs when resting
- Use support/elastic stockings
- Avoid constrictive clothing/crossing legs
- Surgery (Ligation, Stripping, Sclerosing)
Compression Hosiery

- Compression hosiery helps counteract stasis by applying a firm continuous graduated pressure to the muscles and veins in the leg.
- More pressure is exerted at the ankle to assist venous return.
Sclerotherapy

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Surgery

• Ligation: Tying off of a varicosity with a suture or wire ligament
• Stripping: Avulsion of the varicosity (usually indicated in severe cases)
Stripping a Varicose Vein
Venous Thromboses

- Presence of a thrombus in a vein
- Superficial thrombophlebitis
- Deep vein thrombosis
## Arterial and Venous thrombi compared

<table>
<thead>
<tr>
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<th>ARTERIAL</th>
<th>VENOUS</th>
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<tbody>
<tr>
<td><strong>Clinical setting</strong></td>
<td>Person with atheroma</td>
<td>Immobile person</td>
</tr>
<tr>
<td><strong>Pathogenesis</strong></td>
<td>Turbulent flow</td>
<td>Stasis</td>
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<td></td>
<td>Damaged Endothelium</td>
<td>Hypercoaguable Blood</td>
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<td><strong>Symptoms</strong></td>
<td>Sudden onset</td>
<td>Slow onset</td>
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<tr>
<td><strong>Complications/ Fate</strong></td>
<td>Infarction Arterial embolism</td>
<td>Pulmonary embolism via the heart</td>
</tr>
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Composition of Venous Thrombi

- Erythrocytes
- Fibrin mesh
- Few platelets

Pathogenesis is stasis, increased blood coagulability and vascular wall damage - ‘Virchow’s triad’
## Virchow’s Triad

<table>
<thead>
<tr>
<th>Altered Blood</th>
<th>Increased cells</th>
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<tbody>
<tr>
<td></td>
<td>Increased platelets</td>
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<tr>
<td></td>
<td>Increased protein</td>
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<tr>
<td></td>
<td>Decreased fluid</td>
</tr>
<tr>
<td>Altered vessel wall</td>
<td>Endothelial loss</td>
</tr>
<tr>
<td></td>
<td>Endothelial damage</td>
</tr>
<tr>
<td>Altered flow</td>
<td>Stasis</td>
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<td></td>
<td>Turbulence</td>
</tr>
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</table>
Risk Factors

• Vein wall alterations
• Stasis (immobility, long periods of sitting/standing)
• Coagulopathies (e.g. polycythemia)
• Inherited hypercoagulability
• Obesity
• Varicose veins
• Use of oestrogen-based oral contraceptives
Superficial Thrombophlebitis

- Inflammation of superficial vein with associated thrombotic occlusion
- Pain, tenderness, erythema, warmth
- Vein may be raised, indurated and cord like
- Treat with:
  - NSAIDs
  - Rest
  - Elevation
  - Compression
  - Antibiotics if evidence of infection
Thrombophlebitis
Deep Vein Thrombosis (DVT)

- May be asymptomatic
- May be variable combinations of:
  - tenderness
  - pain
  - oedema
  - warmth
  - bluish skin discoloration
  - prominent superficial veins
  - mild fever
  - Homan’s sign (but don’t do it)
DVT Treatment

- Prevention of pulmonary embolism and chronic venous insufficiency
- Require hospitalisation
- Anticoagulant therapy
- Thrombolytic therapy
Leg post DVT
PBL – Picture task

- Diagnosis
- Aetiology
- Risk factors leading to the pathology (i.e. factors you would reasonably expect to find in a patient's history)
- How and why you would assess the vasculature and what results you might expect.
- What your podiatric management would be (remember this includes appropriate referral)
- What health promotion strategies might you employ
Venous Reflux Testing

- With the Doppler it is possible to test for the presence/absence of venous reflux (an abnormal backward flow)
- Testing should be performed with the patient standing, knee slightly flexed
- Locate the Popliteal vein with the Doppler
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Venous Reflux Testing

- Compress calf for 3 seconds to cause a surge of blood
- You will hear forward flow on the Doppler
- When the valves are competent they will close and prevent any backward flow
- No further noise should be detected
Summary

- Need good plumbing and waste removal systems
Thank you for listening

Any Questions???

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