Assessment and Vascular Management of the High Risk Foot

Mr Jason Chuen, Director of Vascular Surgery, Austin Health
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Multifactorial Approach

Arterial Perfusion

Venous Drainage

Microbiology

Neuropathy & Pressure Care

Nutrition

Diabetes

The Unusual

Wound Healing Environment
Atherosclerosis

- Risk Factors
  - Age
  - Gender
  - Hypertension
  - Dyslipidaemia
  - Diabetes
  - Family History
  - Smoking
Unstable

- Few SMCs
- Thin fibrous cap
- Inflammatory cells
- Eroded endothelium
- Activated macrophages

Stable

- More SMCs
- Thick fibrous cap
- Lack of inflammatory cells
- Foam cells
- Intact endothelium

Why is arterial perfusion important?

- 12% of the adult population\(^1,2\)
- 20% of the population aged >70
- Associated with 6-fold increase in CV mortality\(^3\)

Natural History of Atherosclerotic Lower Extremity PAD Syndromes

PAD Population (50 Years and Older)

Initial clinical presentation

- Asymptomatic PAD 20%-50%
- Atypical leg pain 40%-50%
- Claudication 10%-35%
- Critical limb ischemia 1%-2%

1 year outcomes

- Alive with two limbs 50%
- Amputation 25%
- CV Mortality 25%

5 year outcomes

- Limb morbidity
  - Stable claudication 70%-80%
  - Worsening claudication 10%-20%
  - Critical limb ischemia 1%-2%
  - Nonfatal cardiovascular event (MI or stroke) 20%

- CV morbidity & mortality
  - Amputation (see CLI data)
  - CV causes 75%
  - Non-CV causes 25%
  - Mortality 15%-30%
Patterns of atherosclerosis

• Typically affects branch points (turbulence, shear stress)

HOWEVER

• Atherosclerosis can occur anywhere
Clinical History
Global Assessment

- Presenting complaint
- Past Medical History
- Cardiovascular Risk Factors
- Family History
- Functional assessment
- Psychosocial factors
# Symptoms

<table>
<thead>
<tr>
<th>Fontaine Stages</th>
<th>Rutherford Grades</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Asymptomatic</td>
<td>0 Asymptomatic</td>
</tr>
<tr>
<td>IIa Mild Claudication</td>
<td>I Claudication</td>
</tr>
<tr>
<td>IIb Mod-Severe Claudication</td>
<td></td>
</tr>
<tr>
<td>III Ischaemic Rest Pain</td>
<td>II Rest Pain</td>
</tr>
<tr>
<td>IV Ulceration/Gangrene</td>
<td>III Minor Tissue Loss</td>
</tr>
<tr>
<td></td>
<td>IV Ulceration/Gangrene</td>
</tr>
</tbody>
</table>
Claudication

- Latin: claudus — lame, limping
- Pain which affects the ability to walk
- Intermittent Claudication

Intermittent ischaemic claudication

- Muscular pain, ache, cramp
- Develops on walking a fixed distance / metabolic activity
- Resolves on pausing, resting (timeframe: minutes)
- Unaffected by posture or position
- Able to resume for slightly shorter distance / workload
Claudication — Differentials

- Arterial Ischaemia
- Neurogenic
  - Radiculopathy
  - Spinal Canal Stenosis
- Arthrogenic
  - Hip / Knee Arthritis
- Venous Insufficiency
- Chronic Compartment Syndrome
- Plantar Fasciitis
- Baker’s Cyst
Critical Limb Ischaemia

• “Short Distance” Claudication
• Rest Pain
• Tissue Loss

• 25% 1-year probability of amputation
• 25% 1-year cardiovascular mortality
Rest Pain

- Occurs in the most distal region of the limb
- Implies perfusion so poor that unable to maintain aerobic metabolism in skin, tissue and nerves at rest
- Exacerbated by limb elevation (often at night)
- Relieved by dependency
- NOT the same as wound pain
Clinical Examination
Clinical Examination

- Inspection
- Pulse Examination
- Thrills
- Bruits
- Capillary Refill
- Ulceration / Necrosis
- Bedside Tests
- Buerger’s Test
- Ankle Brachial Indices
Buerger’s Test
Ankle Brachial Indices / Segmental Pressures

• May not be reliable in calcified or incompressible vessels
• Global assessment not reflective of parallel tibial artery disease
• Toe Pressures + doppler vs PPG
• Transcutaneous Oxygen Measurement
The Angiosome Concept
2D Angiography

Advantages

- High quality images
- Arch assessment
- Intracranial assessment
- Temporal information
- Now digital - no film involved
- Able to Intervene

Problems

- 2D / single planar
- Subtraction artefact
MRI / MRA / CTA
3D Angiography

CT Angiography
- Volume acquisition
- IV contrast-based in (monitored) arterial phase
- Traditional CT artefacts
- Relies on Hounsfield Unit filtering / windowing
- Calcification artefact
- 2D Axial vs Volume rendered vs MIP imaging
- Allows 3D manipulation
- Allows assessment of non-target tissues

MR Angiography
- Non-contrast
- 2D Time-of-Flight (Better for slow-flow)
- 3D Time-of Flight (Sagittal / Any Plane)
- Gadolinium Contrast
- 3D Time-of-flight
- Artefacts vary with acquisition technique
- Flow voids
- Flow direction
- Venous contamination
Principles of Ultrasound

High-frequency ultrasound (usually 3-12MHz) is transmitted through soft tissue with an average speed of 1540 m/sec

**Bone transmits sound faster**

**Water transmits sound more slowly**

At acoustic boundaries some sound waves are transmitted and some are reflected

Image courtesy of Dr Jeffrey Cheng, Radiopaedia.org, rID: 55792
US Probe Construction

- Metal outer casing
- Backing block
- Electrodes apply an alternating potential difference
- Piezoelectric crystal
- Plastic ‘nose’
- Power cable
- Acoustic insulator
A (Amplitude) Mode

Received signals displayed on a graph

Y-axis = Amplitude
X-axis = Time = Distance

M (Motion) Mode imaging

A single ping is sent and echoes are listened for in one line. The louder the returned echo, the brighter the spot. This line image is projected over time to generate an M-mode image.
2D B (Brightness) Mode Imaging

Image: J Chuen, Austin Health Vascular Laboratory
Doppler Effect

Source: http://www.redchairblogs.com/starstruck/2018/03/08/finding-the-first-galaxies/
Doppler Ultrasound

The received echoes are normally at the same frequency as the sent “ping”
If the received echo is at a different frequency then:

Either from a different point source (not transducer), or

From a moving echogenic source
The velocity of the moving echogenic object can be calculated via the Doppler equation

\[ f_a = f_r - f_t = \frac{2 \cdot f_t \cdot \cos \Theta \cdot V}{c} \]
Continuous Wave Doppler

Principle of handheld doppler probe
Continous high-frequency signal sent out
Second crystal listens continuously for echoes
Subtracted frequency signal is played back in audible frequency range
Cannot assess direction of flow
Cannot control the area being sampled (gate)
Pulse Wave Doppler

Short burst of ultrasound is transmitted followed by long period of listening for echoes
Repeated many times a second
Received signals analysed for frequency change (direction, magnitude)
Selective temporal analysis of the received signal allows “gating” of the doppler signal

\[ f_r = f_i - f_d = \frac{2 \cdot f_i \cdot \cos \theta \cdot V}{c} \]
Spectral Analysis

Echoes are collected after each pulse ping (Pulse Wave Doppler) and timed to only collect from a Sample Volume (gated)

A Fast-Fourier Transformation (FFT) is applied to extract the individual return frequencies which are displayed over time

Velocities are calculated from the Doppler equation
Colour Doppler

The spectral analysis for multiple sample volumes is stratified into ranges of forward and reverse flow. This is then projected onto the 2D B-mode image as colour blocks representing estimated velocity.

Power doppler analyses the amplitude of the doppler signals, not frequency.
Spectral Changes in Stenoses

Increase in peak systolic velocity
Spectral Broadening
Loss of Spectral Window
Can be complicated!

Image: J Chuen, Austin Health Vascular Laboratory
Doppler Waveforms

Normal Triphasic Signals

Narrow frequency band

Steep systolic increase

Forward systolic flow

Rapid drop

Reverse flow in late systole / early diastole (may not see in low resistance beds)

Short forward flow in late diastole

Image: J Chuen, Austin Health Vascular Laboratory
Doppler Waveforms

Biphasic Signals

Slower systolic upstroke

Late systolic peak

Reverse diastolic flow may be preserved

steady positive flow in the diastole, or

forward flow in systole

Image: J Chuen, Austin Health Vascular Laboratory
Doppler Waveforms

Monophasic Signals

Single phase with slow acceleration / deceleration

Due to inflow restriction or hyperaemia with low outflow resistance

Can be high or low velocity

Image: J Chuen, Austin Health Vascular Laboratory
Treatment Options
Treatment Options

• Treat the cause
• Limit disease progression
• Improve arterial inflow
• Reduce metabolic demand
• Treat the symptoms / sequelae
Plaque Stabilisation

- Risk Factor Control
- Smoking
- Diabetes
- Hypertension
- Hyperlipidaemia
- Induction of collateralisation
- Exercise therapy

- Pharmacotherapy
- “Vasodilator” therapy (Antivasospastic)
- Antiplatelet therapy
Revascularisation Options
Bypass Surgery

• Inflow Vessel

• Nature of Conduit
  • Vein > 3.5mm
  • Vein < 3.5mm
  • Prosthetic

• Length of Conduit
  • Above Knee
  • Below Knee

• Tunelling

• Runoff vessels
<table>
<thead>
<tr>
<th>Outflow Procedure</th>
<th>Operative Mortality (%)</th>
<th>Expected Patency Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fem-AK popliteal vein</td>
<td>1.3–6.3</td>
<td>66 (5 yrs)</td>
</tr>
<tr>
<td>Fem-AK popliteal prosthetic</td>
<td>1.3–6.3</td>
<td>47 (5 yrs)</td>
</tr>
<tr>
<td>Fem-BK popliteal vein</td>
<td>1.3–6.3</td>
<td>66 (5 yrs)</td>
</tr>
<tr>
<td>Fem-BK popliteal prosthetic</td>
<td>1.3–6.3</td>
<td>33 (5 yrs)</td>
</tr>
<tr>
<td>Fem-Tib vein</td>
<td>1.3–6.3</td>
<td>74–80 (5 yrs)</td>
</tr>
<tr>
<td>Fem-Tib prosthetic</td>
<td>1.3–6.3</td>
<td>25 (3 yrs)</td>
</tr>
<tr>
<td>Composite sequential bypass</td>
<td>0–4</td>
<td>28–40 (5 yrs)</td>
</tr>
<tr>
<td>Fem-Tib blind segment bypass</td>
<td>2.7–3.2</td>
<td>64–67 (2 yrs)</td>
</tr>
<tr>
<td>Profundaplasty</td>
<td>0–3</td>
<td>49–50 (3 yrs)</td>
</tr>
</tbody>
</table>

AK = above the knee; BK = below the knee; Fem = femoral; Tib = tibial.
Atherectomy Devices

HOW IT WORKS

1. Catheter is fed through arteries to the blockage

- Build-up of plaque in artery
- Guide wire

2. Stainless-steel tip vibrates, breaking up fat and creating a channel through the blockage

CROSSER™ Activation
Drug elution
Tibial and Pedal disease
Pedal angioplasty
數位複合式手術室

Hybrid operating Room
Management of Tissue Loss
Tissue Loss

- Treat or control superinfection
- Salvage tissue by improving vascularity
- Remove non-viable tissue (if safe to do so)
- Be prepared to sacrifice the limb
- Some patients cannot be salvaged
Long Term Management

- Followup is paramount
- Risk factors require lifelong control
- Disease progression is inevitable
- Re-presentation with further ischaemia is likely
- Bypass Grafts and Stents have a limited life expectancy
Issues arising from change

- New treatment paradigms require a redesign of hospital organisational, physical and financial infrastructure. How do we do this?
- Can or should we push surveillance duties onto GPs, other specialties or a Vascular Surveillance Clinic?
- Does having a safer procedure mean that we can expand treatment indications?
- What happens if vascular surgeons become de-skilled in open surgery?
- Is early, high-cost intervention sustainable for our healthcare system?