Infarction

- Tissue necrosis due to ischaemia
  - vascular insufficiency of any cause
  - usually arterial occlusion due to thrombosis/embolism

- Mainly due to oxygen deficiency, but toxin accumulation & reperfusion injury may contribute

- Number of determining factors
  - Size of vessel and size of vascular territory
  - Partial / total vascular occlusion
  - Duration of ischaemia
Appearance of Infarct

- Artery
- Occlusion
- Normal tissue
- Infarcted tissue
- Surface fibrinous exudate
- Ill-defined infarct borders

Preview from Notesale.co.uk
Event Sequence

1. Coagulative necrosis
2. Infiltration by neutrophils
3. Infiltration by macrophages
4. Phagocytosis of debris
5. Granulation tissue formation
6. Scar formation
<table>
<thead>
<tr>
<th>Time</th>
<th>Microscopic Features</th>
<th>Gross Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 4 hr</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>4 – 12 hr</td>
<td>Early coagulation necrosis (nucleus: pyknosis, cytoplasm: eosinophilia)</td>
<td>None</td>
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<tr>
<td>12 – 24 hr</td>
<td>Further necrosis, haemorrhage, early neutrophil infiltrate</td>
<td>Dark mottling</td>
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<td>1 – 3 days</td>
<td>Marked neutrophil infiltrate and necrosis</td>
<td>Mottled with yellow-tan necrotic centre</td>
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<tr>
<td>3 – 7 days</td>
<td>Early phagocytosis of dead cells by macrophages (at border)</td>
<td>Hyperaemic border, central yellow-tan softening</td>
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<td>7 – 10 days</td>
<td>Well-developed phagocytosis, early granulation tissue formation</td>
<td>Maximal yellow-tan softening, depressed red-tan margins</td>
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<tr>
<td>10 – 14 days</td>
<td>Well-developed granulation tissue, early collagen deposition</td>
<td>Red-gray depressed infarct borders</td>
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<tr>
<td>2 – 8 wk</td>
<td>Increased collagen deposition, decreased cellularity</td>
<td>Grey-white scar progresses from border toward centre</td>
</tr>
<tr>
<td>&gt; 2 months</td>
<td>Acellular collagenous scar</td>
<td>Dense gray scar</td>
</tr>
</tbody>
</table>
Infarct, day 10

Granulation tissue after macrophage phagocytosis of infarcted cells
Reperfusion Injury

Possible effects of re-establishing blood flow:
- prevention of all necrosis
- salvage of reversibly injured cells
- accentuation of damage to irreversibly injured cells
- new cellular damage

Latter two constitute reperfusion injury
- Accentuated or new damage due to re-establishing blood flow
- Many effects of ischaemic injury only seen when perfusion re-established
Septic Shock

- Usually due to lipopolysaccharide (LPS/endotoxin) in walls of gram negative bacteria
- LPS consists of fatty acid core and complex carbohydrate coat
- Similar molecules in walls of gram positive bacteria or fungi
- results in
  - endothelial damage
  - complement activation
  - activation of macrophages with cytokine release