Figure 1.1.1. Sequence of plaque development in atherosclerosis. Mechanisms of occlusion via continued lesion growth or thrombus development are identical for intimal hyperplasia, but on an accelerated timeline (from Salunke and Topoleski, 1997).

Figure 1.1.2. (a) Occluded femoral artery and other vessels; (b) typical below-knee femoropopliteal bypass (in red) to restore blood flow to the lower vasculature (Lamberth and Doty, 1984).
Figure 1.1.3. Illustration of a conventional end-to-side distal anastomosis. Arrows indicate direction of blood flow and shading depicts typical sites of hyperplasia development (Sottiurai, 1999).
Figure 1.1.4. Flow events, biological processes and methodology governing improved graft-end designs.
Figure 1.3.1. The natural history of atherosclerosis. Similar clinical symptoms develop for intimal thickening (from Salunke and Topoleski, 1997).
Figure 1.3.2. The updated response to injury hypothesis of Ross (1986).
Figure 1.3.3a. Plaque fissuring and thrombus in human coronary artery. The thrombus (T) is comma shaped with a portion inside the plaque and a portion occluding the lumen. The torn edge of the plaque cap is indicated by the arrow (from Davies, 1990).
Figure 1.3.3b. Arterial thrombus including red blood cells, activated platelets, and fibrin mesh (provided by ML Longest).
Figure 1.3.4. Simplified schematic of the currently understood mechanisms of arterial thrombosis.

**Adhesion**
- Triggered when a thrombogenic surface is exposed to blood
- Platelets adhere to proteins on the surface via GPs

**Coagulation Cascade**
- Predominately driven by thrombin

**Activation**
- Platelet functions triggered by chemical or physical stimuli
- Chemical stimuli: ADP and thrombin
- Physical stimuli: shear stress
- Other stimuli: biomaterials
- Activation produces positive feedback for activation of other platelets:

**Aggregation**
- Leads to formation of platelet plug
- Incoming platelets may be chemically activated by passing through an agonist cloud produced by the adherent thrombus
- Unactivated platelets may be bound and then activated
Figure 1.3.5. Possible outcomes following plaque cap rupture (from Davies, 1994).
Figure 1.5.1. Length scales associated with blood particle deposition.
Figure 1.5.2. The recruitment and migration of leukocytes (from O’Brien and Chait, 1994).
Figure 1.6.1. Shear stress and exposure time required for the activation of platelets in vitro as compiled by Hellums (1994).
Figure 1.6.2. Observations of platelet deposition in an *ex vivo* collagen coated stenosis with a 4 mm upstream diameter from Markou et al. (1993).
Figure 1.6.3. Time course of platelet accumulation on collagen-coated tubes in the *ex vivo* experiment of Markou et al. (1993). Phases of thrombosis (I-III) suggested by Wootton and Ku (1999).
Figure 1.6.4. Average platelet accumulation rates from popular *ex vivo* experiments. Data taken from the sources listed in the legend and complied by Wootton and Ku (1999).
Figure 1.7.1. Empirical correlation of Aarts et al (1986) for the diffusivity of platelets at various mean hematocrits.