be due to thrombo-emboli (90%), air, fat, amniotic fluid, nitrogen (the bends), medical equipment or tumour cells

**Deep Vein Thrombosis** – Thrombus in leg due to inactivity most commonly; leads to increased venous pressure, fluid leaks out causing oedema and a painful, unilaterally swollen calf that is warm to the touch

**Disseminated Intravascular Coagulation** – Formation of small blood clots throughout the body which consumes coagulation proteins & platelets. This means normal coagulation is disrupted and there is abnormal bleeding; can be acute or chronic leading to multi-organ failure

**Infarction** – Necrosis due to ischaemia that can result in gangrene.
- **White Infarct** – Follows occlusion of end artery; supplied tissue dies and appears pale/white due to lack of blood (e.g. heart, brain, spleen, kidney)
- **Red Infarct** – Occurs when there is extensive haemorrhage into dead tissue. Can occur in tissues with dual blood supply (infarct in main supply, secondary supply can’t rescue tissue but allows blood to enter dead tissue e.g. lung) or rich anastomoses (capillary beds merge & haemorrhagic necrosis occurs e.g. intestines)

**Atheroma**

In response to endothelial injury LDL accumulates in artery wall & is oxidised in the intima; this initiates an inflammatory process in which monocytes migrate into artery wall & become macrophages. These engulf oxidised LDL to become foam cells; when they die lipid is released to form a lipid core within the vessel. A fibrous cap (mostly collagen) is formed by smooth muscle cells as an attempt of tissue repair to contains the lipid core; this is triggered by secretion of growth factors by platelets, injured endothelium, macrophages & smooth muscle cells.

A stable plaque reducing lumen size to <70% presents with stable angina and plaque rupture with thrombus formation can cause acute coronary syndrome following complete or incomplete occlusion. The latter may occur as erosion of the fibrous cap exposes blood to thrombogenic material, resulting in a platelet clot followed by fibrin thrombus.

Endothelial injury ⇔ LDL to intima ⇔ LDL oxidised to foam cells ⇔ macrophages eat foam cells ⇔ macrophages die ⇒ become lipid core ⇒ fibrous cap forms ⇒ atheroma

**Atheroma** – Accumulation of intracellular & extracellular lipid in the intima & media of large & medium sized arteries (NOT veins)

**Atherosclerosis** – Thickening & hardening of arterial walls as a consequence of atheroma

**Arteriosclerosis** – Thickening of the walls of arteries & arterioles as a consequence of disease (usually hypertension or diabetes)

**Aneurysm** – atheroma causes vessel to lose elasticity and becomes permanently weaker & dilated (high risk of spontaneous rupture when >6cm in diameter)

**Cells Involved**
- **Endothelial Cells** – Altered permeability to lipoproteins, secretion of collagen and stimulation of proliferation & migration of smooth muscle cells
- **Platelets** – Stimulate proliferation & migration of smooth muscle cells (PDGF)
- **Smooth Muscle Cells** – Take up LDL & other lipid to become foam cells and synthesise collagen & proteoglycans contributing to abnormal extracellular matrix
- **Macrophages** – Oxidise LDL, take up lipids to become foam cells, secrete proteases which modify matrix and stimulate proliferation & migration of smooth muscle cells
- **Lymphocytes** – TNF can affect lipoprotein metabolism; also stimulate proliferation & migration of smooth muscle cells
- **Neutrophils** – Secrete proteases leading to continued local damage & inflammation

**Risk Factors:**
- **Increasing Age**
- **Gender** – Women relatively protected before menopause