clot, which is visible to the naked eye (Austin 2013; Baum and Arpey 2006). The fibrin and platelets are the main components of the clot matrix formed, which represents an indispensable scaffold for all the cells that are recruited to the wound during the first steps of the healing process (Gurtner et al. 2008; Baum and Arpey 2006).

Inflammation

In order for the healing process to take place the fibrin clot is destroyed (through a process called fibrinolysis), facilitating cell migration to the wound site. Fluid movement to the wound is promoted through local vasodilation triggered by growth factors released from platelets, and enhancement in the permeability of capillaries in the area (Baranoski and Ayello 2004). The complement system (which involves a variety of different plasma proteins) is then activated: this complicated enzyme-triggered cascade is responsible for opsonization, phagocytosis and ultimately, the destruction of bacterial cells (Charles et al. 2001). Chemotactic substances (such as chemotaxin) are released attracting polymorphonuclear (PMN) neutrophils and monocytes, which will differentiate into macrophages (Mulder et al. 2002). Neutrophils produce important inflammatory factors such as interleukin-1 (IL-1) and tumor necrosis factor alpha (TNF-α), and proteases which are able to break down damaged extracellular matrix (ECM) components. Macrophages play many roles, including phagocytizing bacteria, as well as producing collagenases, elastase, other growth factors and many types of cytokines. Evident symptoms of inflammation are: erythema, swelling and an increase of the temperature in the injured area (Granick and Teot 2012).

Proliferation

This phase is characterized by two events that are crucial for the progress of healing: angiogenesis, which is defined as the process of formation of new blood vessels or repairing of pre-exiting ones allowing blood supply to the site of injury, and the production of new ECM (Flanagan 2013; Tonnesen et al. 2000). Formation of capillary buds from vascular endothelial cells is promoted by angiogenesis factors, such as vascular endothelial growth factor (VEGF) and fibroblast growth factor-2 (FGF-2). Fibroblasts migrate to the wound in response to the transforming growth factor-β (TGF-β) which is released from macrophages, proliferate and start the production of collagen fibres and other connective tissue proteins. As a result, so-called 'granulation tissue' is formed: newly formed blood vessels within the ECM appear red and granular (Huether and McCance 2013). Myofibroblasts (differentiated fibroblasts, similar to smooth muscle cells due to the presence of parallel fibres in their cytoplasm) present in the granulation tissue are responsible for wound contraction, which usually starts 6 to 7 days post-injury (Hinz et al. 2007; Huether and McCance 2013). The choice of the initial dressing in this phase is crucial and it has to consider that any mechanical stress could lead to renewed bleeding, special care has to be taken to manage the wound and encourage the best healing outcome (Davey and Ince 1999).