

The fibrin clot acts as a wound matrix in which the platelet produces growth factors that are important for wound healing [24].

In the inflammation phase, an influx of leucocytes at the wound bed is significant. Cells such as the monocytes, macrophages, neutrophils are useful in the inflammation phase. The wound is cleansed of infections and debris. Proinflammatory cytokines and growth factors activate and recruit fibroblast and epithelial cells. Neutrophils express reactive oxygen species (ROS), proinflammatory cytokines and proteases at the surface of the wound. Neutrophils recruit cells that clean-up the wound surface [24, 25]. In the proliferation phase, the inflammatory and migratory cells induce cellular proliferation resulting in the formation of granulation tissue and epithelization. A good blood supply suitable for the delivery of nutrient, gas, and metabolite exchange is important at this phase resulting in the proliferation of dermal and epidermal cells within the wound bed [23,26]. Angiogenesis also occurs at this phase resulting in the formation of new blood vessels. The wound is totally covered with epithelium with the formation of granulating tissues [25].

In the final wound-healing phase, there is a restoration of the integrity of the epidermal. The proliferation of the fibroblasts within the wound with the production of ECM-forming granulation tissue and new blood vessels are visible in this phase. The contraction of the wound resulting from fibroblast motility with a re-organization of the matrix takes place. The formation of scar tissue is also visible at this phase characterized by a tensile strength which is comparable with the unwounded skin. An increased number of apoptotic cells in the final phase of wound healing results from TGF- α (a tumor necrosis factor) and FGF-2 (a stimulator of cell proliferation). The formation of scars resulting from wound healing such as hypertrophic scar and keloid formation has been reported to be caused by the inability of myofibroblasts to undergo timely apoptosis [24, 25].

3. WOUND DRESSINGS

Wound dressings are classified as traditional, bioactive, biologics, and interactive wound dressings (Fig. 2) [25] (Internet source: Wound Dressing). The traditional wound dressings are designed primarily to cover the wound surface such as gauze and gauze cotton composites. However, their use is limited by their poor permeation of vapor and ability to cause bleeding, leakage of exudates that promotes bacterial infections and damage to the newly formed epithelium [25,27] (Internet source: Wound Dressing). Interactive wound dressings such as foams, films, gels, and composites are prepared from biopolymers and synthetic polymers. They exhibit distinct features such as good moist environment for improved wound healing, promote debridement, and enhance re-epithelialization [28].

Biologics-based wound dressings are classified as allografts, stem cell therapies, skin equivalents, tissue derivatives, and xenografts [25]. They are used for the treatment of chronic wounds. However, their use is limited by the risk of transmission of diseases and infections, they are expensive, the body may reject them due to immune reactions [25,29]. Biologic wound-healing therapies also involve the use of bioactive agents which can be obtained from plants. These bioactive agents exhibit biological properties such as anti-inflammatory, antioxidant, and antimicrobial activities [30]. Monoterpene is a family of naturally occurring terpene-based compounds common in essential oils. Their level of toxicity is low making them useful in wound dressing. Biologic dressings induce autolytic debridement and granular wound bed [30]. Skin substitutes are used to replace the function of the skin and they are often used in chronic wounds. They accelerate the rate of healing with reduced side effects. They are classified as either acellular or cellular. Acellular skin substitutes are composed of a scaffold of fibronectin, hyaluronic acid, or collagen. The cellular skin substitute is composed of cells such as keratinocytes and fibroblasts [30].

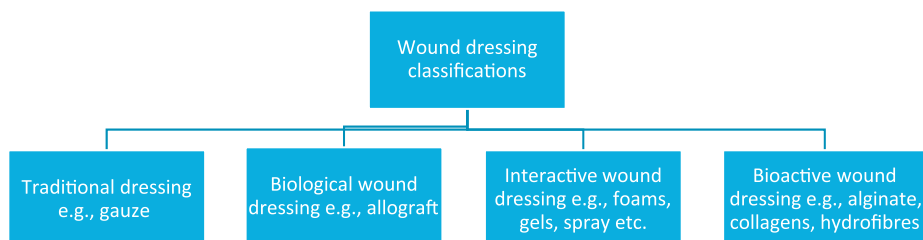


FIG. 2 Classification of wound dressings.