



FUNDAMENTALS OF WOUND HEALING

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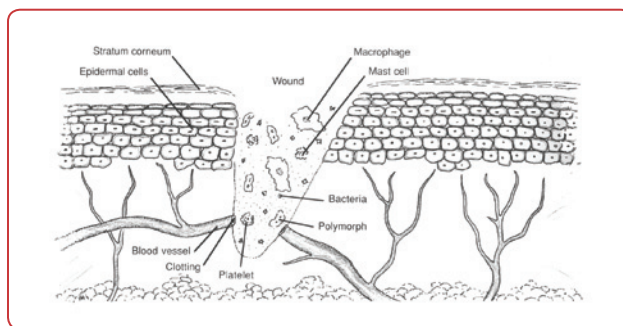
THE HEALING OF ANY WOUND FOLLOWS A NATURAL process. After a wound occurs, either by surgery or by accident, the skin initiates a process of repairing and restoring itself to near normal. This process involves both cellular and biochemical responses to the injury. The end result is a restoration of the skin's surface and a reconstitution of the dermis.

Stage 1: Inflammation (also called the vascular stage)

Immediately after a wound occurs, the repair process begins. The blood vessels that have been cut or torn now thrombose, undergoing changes that help stop the bleeding by encouraging clots to form. The biochemical changes accompanying thrombosis are profound and numerous; luckily you need to understand only a few of these. Fibrin is the release of platelets to stop the bleeding; after that, other biochemicals must be secreted to allow capillaries to dilate and bring other cells to the wounded area. Platelets, small cells responsible for much of the clotting mechanism, also must secrete substances that call other cells to the wounded area. These are called chemotactic and growth factors (platelet-derived growth factor, or PDGF, is a very important cell factor with other functions besides wound healing).

Injury and clotting set into motion a very complex series of pathways that now serve to bring into play all that is necessary to heal the wound. The injured cells, including mast cells, will secrete histamine, causing edema (swelling) and redness as the surrounding vessels dilate. Surprisingly, this is beneficial to the wound healing process. During this stage, the cellular processes start to control infection and clean up the cellular debris.

Two important types of cells arrive: polymorphonuclear cells (PMN) are the granulocytes mentioned previously, and macrophages. The polymorphonuclear cells combat bacteria, engulf foreign matter and remove clotted blood. The



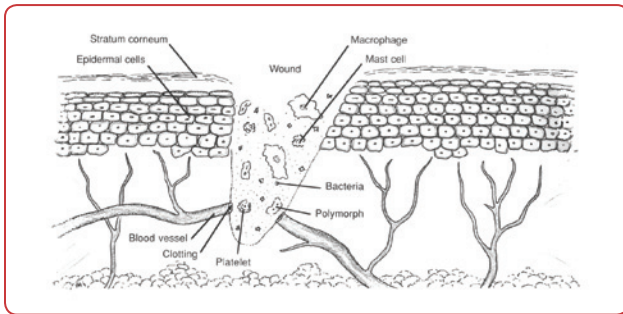
Wound healing stage 1: The blood vessels are damaged as cells are moving to defend the cut surfaces. Serum forms to impede bacteria.

macrophages serve to assist the PMN in bacterial control and removal of cellular debris, but they have an even more important function in the total process of wound healing. At the time of injury, macrophages are activated biologically to secrete many active substances that cause further wound healing. These substances include chemotactic factors, fibroblast and endothelial cell stimulating factors and a special macrophage-derived growth factor (MDGF) that is essential to the total wound healing process.

Stage 2: The re-epithelization phase

After the wound is cleaned, structural repair begins as new blood vessels and wound edge cells start to move in, much as construction workers move in after the demolition workers clear a building site. The critical step is the migration of undamaged epidermal cells from the wound edges. This event may occur hours after the wound is made and does not require increased cellular proliferation. Interestingly, right at the time of this migration, a change must occur in the basal cells; they lose their attachment to other cells by retracting the tonofilaments (intermediate filaments composed of keratin that connect epidermal cells to each other) and dissolving the intercellular desmosomes. The basal cells then develop

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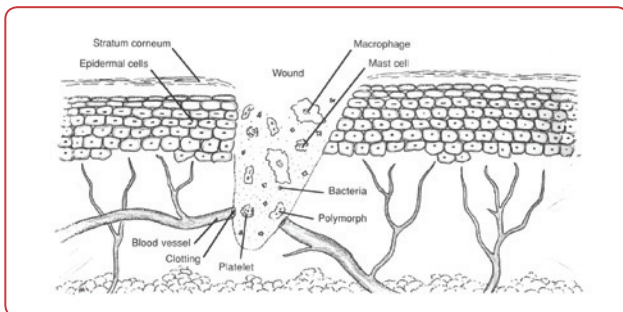
Wound healing stage 2: A clot is formed and fibroblasts start to function. Blood is stopped as the epidermis begins to migrate over the wound edges.

pseudopodia, or “migrating feet” that allow them to move about and over other cells. Once migration has started, a proliferation phase occurs and the cell division rate increases up to 17-fold. The migrating and dividing basal cells move over a specialized surface that consists of fibronectin and collagen.

Fibronectin is a glycoprotein, a combination of sugar and protein units weighing 440 kiloDaltons. Present in plasma, it appears to be produced by keratinocytes, fibroblasts and endothelial cells. Fibronectin has many functions in wound healing, including the promotion of cell migration, phagocytosis, cell adhesion and the binding of collagen to form a matrix for glycosaminoglycans (the “ground substance”). The movement of keratinocytes over the collagen/glycosaminoglycan matrix favors a route that is moist. This significantly affects the design and choice of proper wound dressings. As epithelization (formation of a new epidermis to cover the wound) occurs, and another process parallels it: the formation of granulation tissue.

Stage 3: Granulation tissue formation

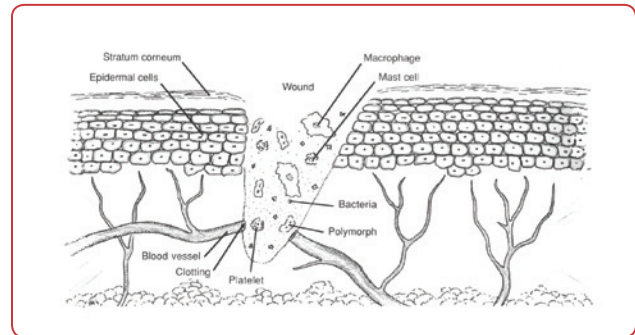
Three to five days after the wound is made, granulation tissue appears. This tissue consists of the new vasculature (blood vessels), and the new matrix of collagen, glycoproteins and glycosaminoglycans. The star of the process is the fibroblast, which produces collagen, fibronectin, elastin and the ground substances. After a few days, a new type of cell appears: the myofibroblast. This cell is unique in having



Wound healing stage 3: A scab forms and the myofibroblasts appear. Collagen strands form in the wound and the edges of the blood vessels start to repair the break. The basal layer of the epidermis actively is proliferating as the wound is roofed over.

Most wounds reach maximum strength three to four months after healing begins but never reach full normal skin strength, attaining only 75-80 percent of normal skin strength.

tiny microfilaments in its cytoplasm, allowing myofibroblasts to contract and migrate. Many substances help direct the myofibroblast's activity. Some of these have already been mentioned, such as MDGF and PDGF, but insulin and certain substances called lymphokines also affect the action of this cell. Hyaluronic acid plays a role in these early stages; dermatan sulfate and chondroitin sulfate appear later in the process. These proteoglycans seem to help regulate collagen production and placement, and provide the wound with a flexible resilient base.



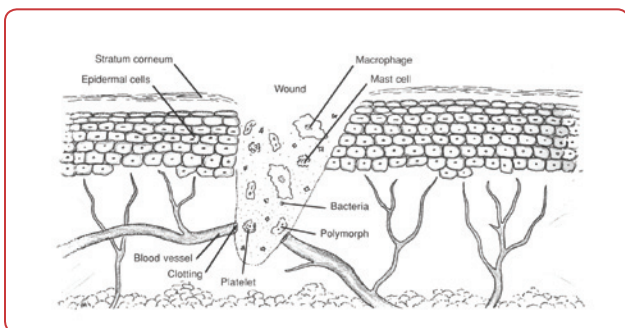
Wound healing stage 4: The wound is closed and contracting continues deep within the wound. The scab starts to fall off as it contracts. Increased fibroblasts and myofibroblasts actively strengthen the deeper layers of the wound.

The initial collagen laid down is Type III collagen, but as the wound repair progresses, Type III is replaced by Type I collagen. At this stage, the wound will begin to contract, drawing the edges together. This may occur between five and 15 days after the wound occurs, depending on the size and condition of the wound.

Stage 4: Wound contraction

The myofibroblasts play a central role in wound contraction. This complex process is believed to be controlled by a series of interconnections between the myofibroblasts and other elements of the granulation tissue. This has been termed the “fibronexus” by Singer. As the filaments in the myofibroblast contract, they pull the surrounding matrix together and force the wound to begin closing. Once this contraction has occurred, the wound is closed but not completely healed; it must now undergo matrix and collagen remodeling.

continues



Wound healing stage 5 (charts reprinted with permission from Advanced Professional Skin Care Medical Edition by Dr. Peter T. Pugliese)

Stage 5: Wound remodeling

Even in the early stages of healing, the collagen and cellular matrix is constantly being reformed and remodeled. This process of wound healing may take many months, possibly years. In some cases, it may never actually end. As the collagen increases, fibronectin is replaced and sulfated proteoglycans replace the hyaluronic acid. Water in the scar is gradually replaced by collagen bundles and the wound grows stronger. Most wounds reach maximum strength three to four months after healing begins but never reach full normal skin strength, attaining only 75 to 80 percent of normal skin strength.

The way collagen is laid down and the problems that attend scar formation are subjects of interest to both the plastic surgeon and the skin care specialist. These problems can now be approached with the information you have just learned about the physiology of wound healing. Both general systemic factors and local factors influence both the rate of wound healing and the quality of the final wound. The skin care specialist must be aware of the complications that may result from surgical wounds—including scars. ■



Peter T. Pugliese, M.D., is a family physician. Through his intensive research into skin structure and function over the last three decades, he has become the most published skin physiologist in the world. Dr. Pugliese is the author of Advanced Professional Skin Care, Medical Edition, the global educational standard for students, educators, practitioners and manufacturers in the esthetics field. For more information, contact Michael Pugliese at Michael@circadia.com.

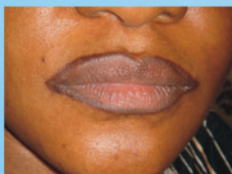


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