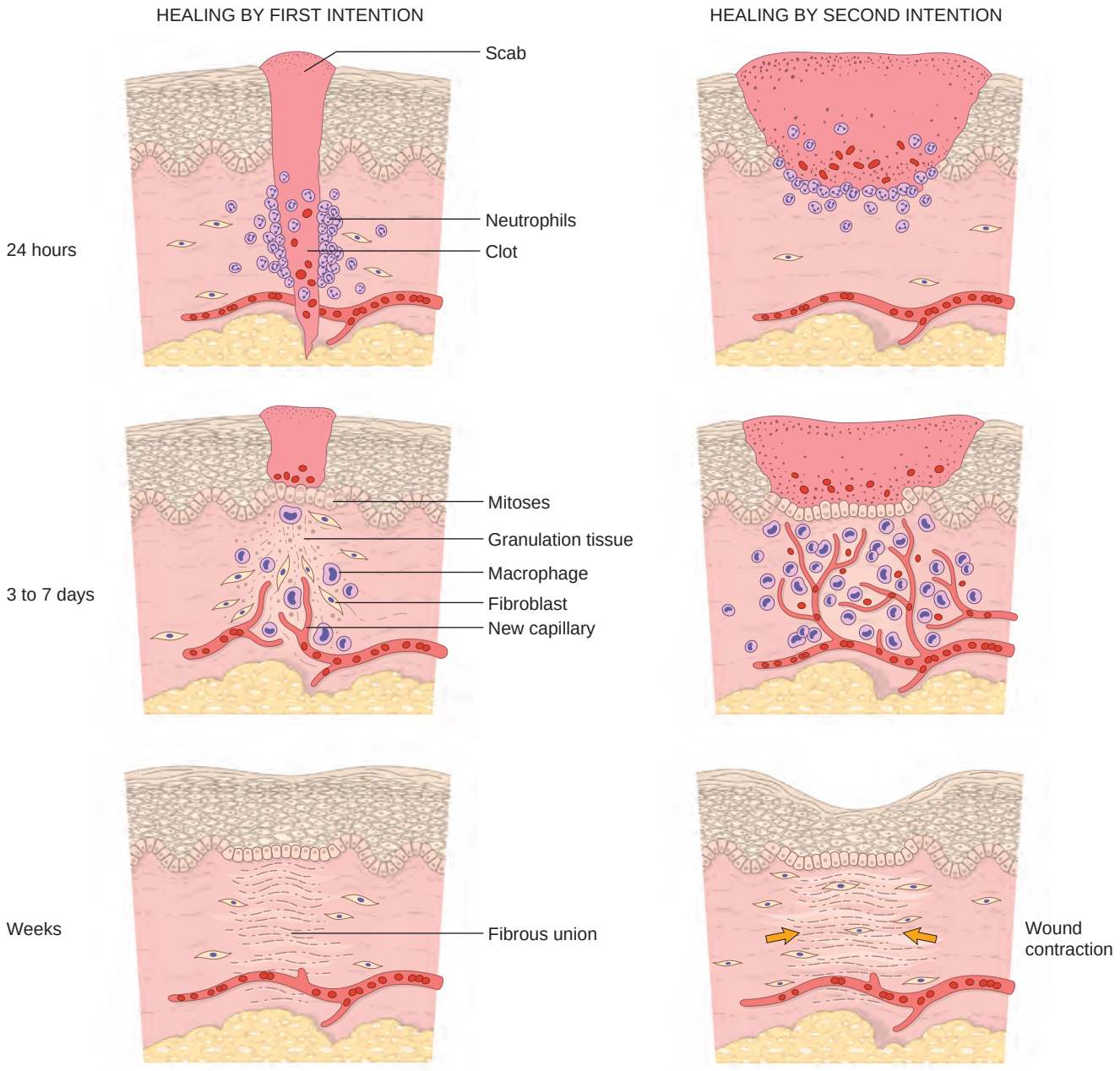

Figure 3-29 Steps in wound healing by first intention (left) and second intention (right). In the latter, note the large amount of granulation tissue and wound contraction.

TNF. Macrophages are the main source for these factors, although other inflammatory cells and platelets may also produce them. The fibroblasts produce ECM proteins, and collagen fibrils become more abundant and begin to bridge the incision. The epidermis recovers its normal thickness as differentiation of surface cells yields a mature epidermal architecture with surface keratinization.

- During the second week, there is continued collagen accumulation and fibroblast proliferation. The leukocyte infiltrate, edema, and increased vascularity are substantially diminished. The process of "blanching" begins, accomplished by increasing collagen deposition within the incisional scar and the regression of vascular channels.
- By the end of the first month, the scar comprises a cellular connective tissue largely devoid of inflammatory cells and covered by an essentially normal epidermis. However, the dermal appendages destroyed in the line of the incision are permanently lost. The tensile strength of the wound increases with time, as described later.

Healing by Second Intention

When cell or tissue loss is more extensive, such as in large wounds, abscesses, ulceration, and ischemic necrosis (infarction) in parenchymal organs, the repair process involves a combination of regeneration and scarring. In healing of skin wounds by *second intention*, also known as healing by *secondary union* (Figs. 3-29 and 3-30), the