CHAPTER III

HEALING OF THE EPITHELIUM

Earlier studies of corneal epithelium healing have been discussed in detail by Duke-Elder and Leigh (1965) and recently reviewed by Lemp (1976). The epithelial repair involves two distinct processes. The epithelial defect is first covered by sliding of the surrounding epithelial cells (fig. III-1) accompanied by temporary cessation of mitotic activity and exfoliation. After a latent period mitotic activity increases resulting in the thickening of epithelial layer. A variable number of polymorphonuclear leukocytes appear at the wound edge after 3 hours. They originate from the conjunctival capillaries and in the case of a central corneal wound travel solely through the tear film (Robb and Kuwabara, 1962). The leukocytes are attracted probably by the release of chemotactic substances due to tissue injury, as no microbial agents have been detected in these studies. The polymorphonuclear leukocytes do not disappear until after the wound is covered by the sliding epithelium.

Sliding of cells

The phenomenon of epithelial sliding has been investigated by different techniques and in different types of corneal injury. Pfister (1975) studied 6 mm corneal erosions of rabbits by scanning electron microscopy. Five minutes after an abrasion the epithelial cells within 0.5 to 1 mm of the defect become retracted and appear to detach from one another. The tight interdigitations between adjacent cells disappear, but the desmosomes are not ruptured and the cells retain some contact with the neighboring cells (Kuwabara et al., 1976).

The surface microvilli are lost. The epithelial migration begins one hour after injury (Friedenwald and Buschke, 1944) and has been noted to reach the bottom of the wound crater in 4 to 12 hours (Robb and Kuwabara, 1962; Hanna, 1966; Bracher, 1967; Matsuda and Smelser, 1973; and Kuwabara et al., 1976) depending on the size of the wound and its depth.
In contrast to the opinion of earlier authors (Friedenwald and Buschke, 1944), Hanna (1966) by thymidine-H\(^3\) tagging of cells, conclusively demonstrated that all the cell layers participate in sliding. According to Kuwabara et al. (1976), the cells of the basal layer slide to a lesser extent. The main sliding cells are the wing cells, but the flat superficial cells regain the cytoplasmic constituents of the younger cells and slide similarly.

In our studies on the response of corneal epithelium to blunt trauma (unpublished), we found marked transformation of superficial cells adjacent to the wounded area (figs. III-2 and III-3). At the edge of abrasion the epithelial cells become flattened. The flattened and expanded portions of the cells are devoid of microvilli (Pfister, 1975). After six hours ruffling of cell surface appears in some cells. The ruffles are thin elevations of cell membrane and may be the first sign of cell movement.

Gipson and Anderson (1977), for the first time, demonstrated the presence of actin filaments along the basal membrane of the sliding cells at their advancing edges and in the cytoplasmic processes. In normal circumstances the actin filaments were seen only along the superficial plasma membranes of the superficial cells. It is likely that