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THE HISTOLOGY OF AUTOLOGOUS SKIN GRAFTS IN THE HUMAN ORAL CAVITY

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Investigations have been made in order to follow histologically the healing process of split skin grafted to the oral cavity. The material was obtained from 28 female and 4 male patients having received preprosthetic surgery due to an atrophic lower alveolar process. Biopsies were taken from the junctional area between the graft and the oral mucosa 10 days, 1, 3, 6, and 12 months postoperatively. After 10 days there was a distinct hyperplasia of the grafted epidermis. The junction between the epidermis and the oral epithelium were clearly defined as a change from orthokeratosis in the epidermis to slight parakeratosis in the oral epithelium. The hyperplasia were in most cases still present in the 12 months specimens. Some, however, showed a normal or even atrophic epidermis after 6 months. The junction between the oral epithelium and grafted epidermis could be clearly demonstrated 12 months postoperatively. In the connective tissue only a moderate inflammatory reaction were present throughout the period examined. No elastic fibres were formed in the junctional area making the transition between graft and oral mucosa easy identifyable. In 6 patients infection with candida albicans developed in the graft probably caused by the antibiotic treatment. Biopsies showed marked epithelial hyperplasia with parakeratosis and intraepithelial accumulations of leucocytes around candida hyphae. These changes are identical to those seen in oral leucoplakias with superimposed candida infection.

Skin grafts have been used in reconstructive oral surgery since the beginning of this century (*Esser*, 1916; *Moszkowicz*, 1917; *Weiser*, 1918; *Dorrance*, 1920; *Gillies*, 1920). In preprosthetic surgery it was introduced by *Pickerill* (1918) with the purpose of establishing a proper buccal sulcus in patients with marked atrophy of the alveolar process. During the last decades the use of skin grafts in preprosthetic surgery has increased following the introduction of new surgical methods (*Schuchardt*, 1952; *Trauner*, 1952; *Rehrmann*, 1953; *Obwegeser*, 1963). The advantage of using skin grafts in sulcus extensions is the stable results with only a slight degree of relapse of the created vestibular depth.

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The epidermis of the skin graft is subjected to a profound change in environment when transplanted to the oral cavity. There has been but few reports concerning the histological changes in the epidermis and the dermis after transplantation to the oral cavity. *Umeda* (1967) has reported on the healing process of full thickness skin grafted to the oral cavity of dogs. *Pini* (1950), *Matras* (1967), and *Schwenzer & Wüstenfeld* (1970) have described the histology of intraoral split skin grafts in man. The findings of the latter authors seem to be mutually contradictionary on some aspects.

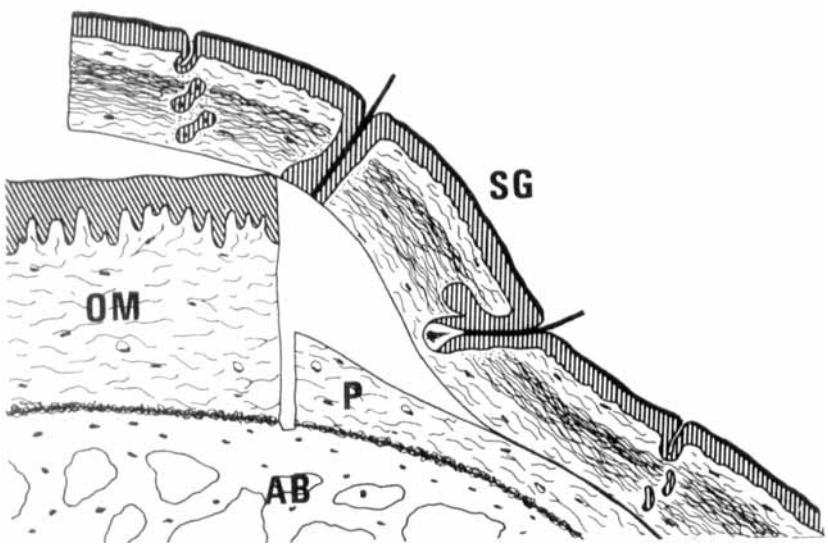
The purpose of the present study has been to follow histologically the healing process of split skin grafted to the oral cavity in humans, and to evaluate the long term effect of the oral environment on the graft.

MATERIAL AND METHODS

The material was obtained from 28 females and 4 male patients having received preprosthetic surgery due to an extremely atrophic lower alveolar process. The age range was 30—72 years, the mean age being 59 years.

The surgical procedure used has been described by *Trauner* (1952), *Rehrmann* (1953), and *Obwegeser* (1963). A sulcus extension was made by cutting the buccinator and mental muscles on the buccal side and the mylohyoid muscle and part of the genioglossal muscle on the lingual side leaving a narrow strip of oral mucosa on top of the alveolar process. The periosteum was left intact. The wound edges of the oral mucosa were sutured near the base of the mandible. The periosteum and remaining mucous membrane covered by a split skin graft (Plate I), which was fixed with a splint for 10 days. The grafts were taken under sterile conditions from the flexor side of the thigh, using a Stryker® dermatome adjusted to 0.010 inches. During the fixative period parenteral penicillin treatment was administered. Within three months all patients were supplied with new dentures.

A part of the graft was prepared for histological examination, serving as a control specimen. Oral biopsies were taken 10 days, 1, 3, 6, and 12 months postoperatively. The oral biopsies consisted of the junctional area between the graft and the oral mucosa as demonstrated in Fig. 1. All specimens were fixed in a 10 per cent aquous formalin solution and embedded in paraffin. 7 μm sections were cut at right angles to the epithelial surface and stained with haematoxylin-eosin, periodic acid-Schiff (PAS) with and without diastase pretreatment, van Gieson-Hansen's connective tissue stain, Gomori's aldehyde-fuchsin stain for elastic fibres, and the Fontana Masson stain for melanin pigment.



I

Plate I. Schematic diagram showing the relation between split skin graft and oral mucosa at the time of transplantation. For further explanation, see text. The skin graft (SG) contains hair follicles and sweat gland ducts. Oral mucosa (OM), periosteum (P), and alveolar bone (AB).

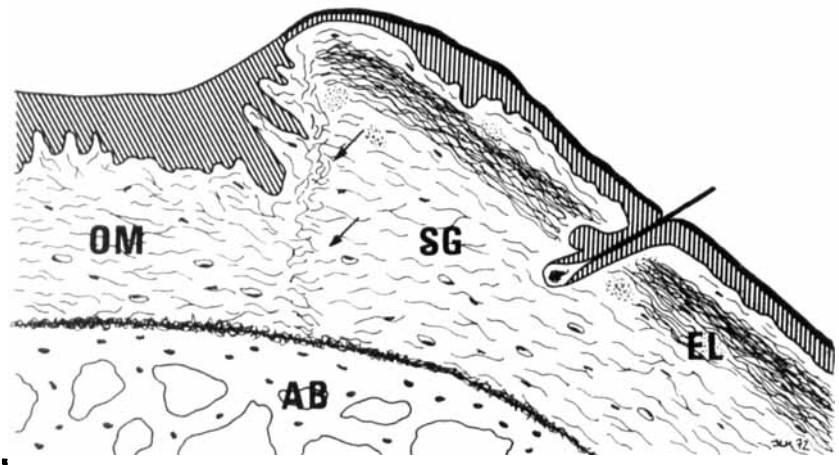


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Plate II. Schematic diagram showing crista formation in the junctional area 10 days after transplantation. Line of junction (arrows) between the skin graft (SG) and the oral mucosa (OM). Elastic fibres of the skin (EL).

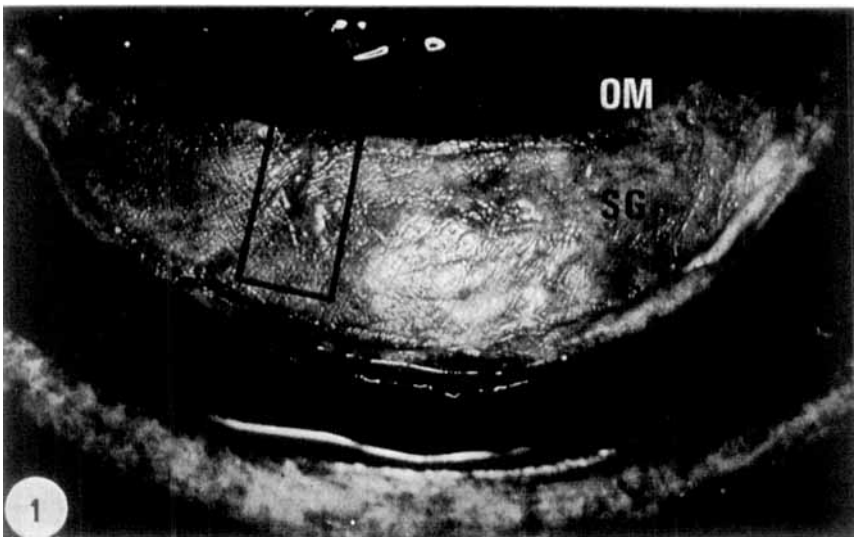


Fig. 1. Intraoral view showing the lower alveolar process covered by a split skin graft (SG). Oral biopsies were taken from the junctional area between the oral mucosa (OM) and the graft (framed).

RESULTS

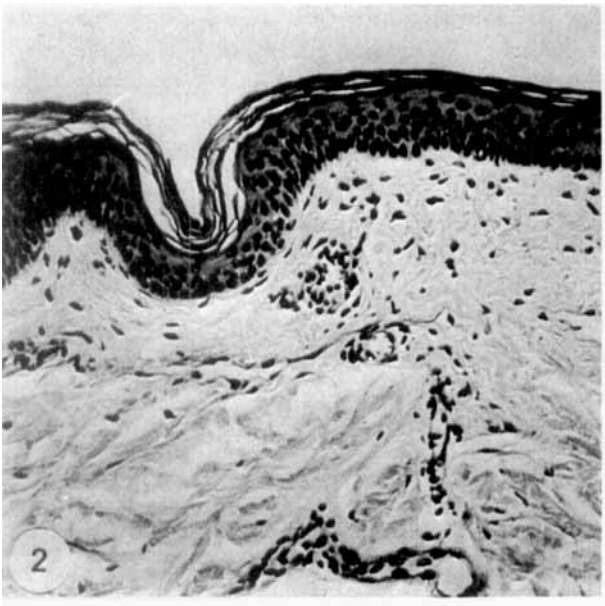
The graft (Figs. 2, 3) consisted of the epidermis and the upper part of the dermis. The epidermis was six to eight cell layers thick including one or two distinct granular cell layers, three to four rows of spinous cells and a single layer of basal cells containing melanin pigment (Fig. 3). The intercellular spaces could not be clearly distinguished, and intercellular bridges were only discernable. A few rete pegs were present. The epidermis contained no PAS-positive material.

In the dermis delicate collagenous fibres were present close to the epithelium, whereas coarse fibre bundles were found in the deeper layers with a course parallel to the surface. Elastic fibres formed a fine juxtaepithelial plexus. In the deeper layers the fibres were thicker and intermingle with the

Fig. 2. Histology of the split skin graft prior to transplantation. Haematoxylin-eosin, × 180.

Fig. 3. Skin graft showing melanin pigment in the basal cells of the epidermis. Fontana-Masson, × 310.

Fig. 4. Junctional area between graft and oral mucosa 10 days after transplantation. Note the abrupt change in histology (arrow-head) from epidermis of the graft (SG) to oral epithelium of the mucosa (OM). A modrate juxtaepithelial infiltration of infimmatory cells is seen in the transition zone. Haematoxylin-eosin, × 180.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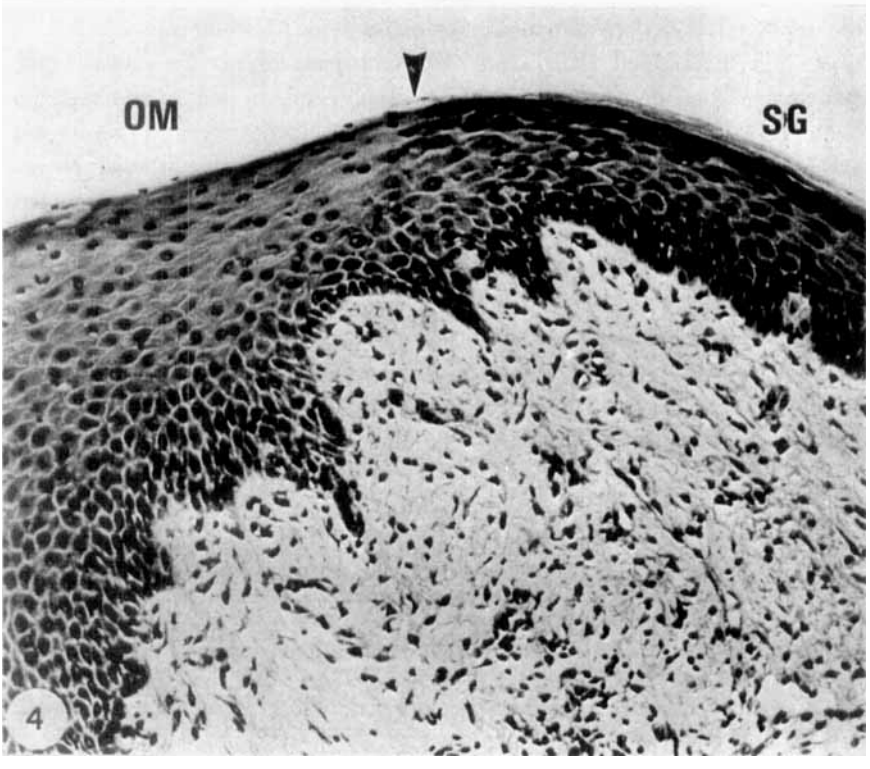

OM
SG
4

collagenous fibres. A few hair follicles with attached smooth muscle cells and sebaceous glands were found. No inflammation was present.

10 days postoperatively. 10 days after transplantation a hyperplasia of the epidermis with numerous broad rete pegs was present. The epidermis consisted of approximately 15 cell layers. Close to the junction between the skin graft and the oral mucosa the hyperplasia was less pronounced. The junction was clearly defined as a change from slight parakeratosis in the oral epithelium to orthokeratosis in the epidermis (Fig. 4). In the spinous cell layers the intercellular spaces were widened with clearly defined intercellular bridges. In the basal cell layer the content of melanin pigment had decreased. However, a slight amount of melanin pigment was scattered in the spinous cell layer. A PAS-positive diastase-resistent, cytoplasmic reaction was found in the spinous and granular cell layers. The oral epithelium showed a marked hyperplasia with acanthotic rete pegs in the junctional area (Fig. 4 and Plate II).

Due to the surgical procedure a small crista was formed at the transition zone between the skin graft and the mucous membrane (Figs. 1, 5). In the transition area a moderate infiltration of polymorphonuclear leucocytes was found juxtaepithelially, and in the recipient bed. Most of the collagenous fibres had maintained their main direction parallel to the surface. The connective tissue between the graft and the recipient bed was formed by granulation tissue and immature collagen. The elastic fibres in the deeper layers of the dermis appeared normal whereas the juxtaepithelial plexus could not be demonstrated. In the zone between the graft and the oral mucosa no elastic fibres were found. There were no changes in the structure of the blood vessels. The epidermal appendices were unchanged.

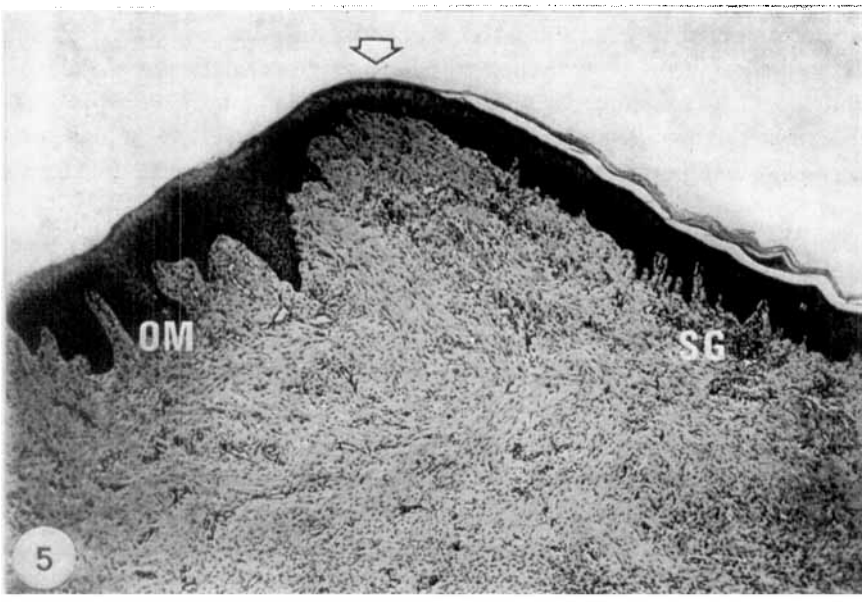
One and three months postoperatively. No histological differences were noted, between the one and the three months specimens. The hyperplasia of the epidermis was still present being less pronounced at the junction area.

In most specimens the junction between the oral mucosa and the skin graft was distinct through all layers of the epithelium (Fig. 7). In the re-

Fig. 5. Crista formation at the junction (arrow-head) between skin graft (SG) and oral mucosa (OM) 10 days after transplantation (compare with Plates I & II). Epithelial hyperplasia is seen on either side of the transition zone. In the connective tissue between the graft and the recipient bed granulation tissue and immature collagen is seen. Haematoxylineosin, × 37.

Fig. 6. Melanin pigment in the graft epidermis one month after transplantation. Note the even distribution in all cell layers. Fontana-Masson, × 310.

Fig. 7. Junctional area one month after transplantation. Distinct border (arrow) between epidermis of the graft (SG) and epithelium of the oral mucosa (OM). In the connective tissue a moderate perivascular, lymphocyte infiltration is demonstrated. Haematoxylin-eosin, × 180.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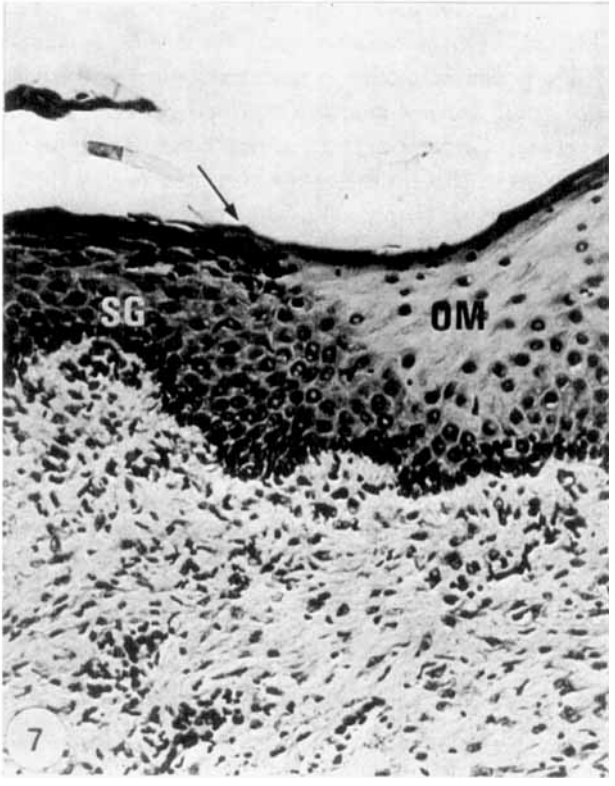
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maining specimens the junction was masked by inflammatory cells migrating through the epithelium. Orthokeratinization, an increased number of granular cell layers, and distinct intercellular spaces were found in the epidermis. The PAS-positive reaction in the spinous cells was still present. Melanin pigment was observed evenly distributed in all cell layers of the epidermis (Fig. 6).

In the connective tissue lymphocytes were found in the transition zone with accumulations close to the epithelial junction. In the dermis a moderate perivascular, lymphocytic infiltration was demonstrated. A maturation of the granulation tissue in the transition zone had occurred with no formation of elastic fibres. Occasionally normal structured epidermal appendices were found.

Six to twelve months postoperatively. The six and twelve months specimens demonstrated the same histological changes with two different patterns of reaction in the epidermis. In most specimens the hyperplasia of the epidermis persisted (Fig. 8), and the histological and cytological findings were in accordance with the observations in the three months specimens. However, in some specimens the epidermis showed normal thickness or was atrophic (Fig. 9). The melanin pigment was mainly located to the basal cell layer. The junction between the epidermis and the epithelium of the oral mucosa was in all specimens clearly defined (Figs. 10, 11). In the keratinized cell layers an abrupt change from parakeratosis in the oral epithelium to orthokeratosis in the graft was demonstrated (Fig. 11). A granular cell layer was seen in the epidermis only. The spinous cell layer of the epidermis took up stain more heavily than the corresponding cell layers in the oral epithelium. Furthermore a difference was found between the stainability of the intercellular spaces showing no PAS-positive reaction in the graft versus a PAS-positive reaction especially in the upper cell layers of the oral epithelium.

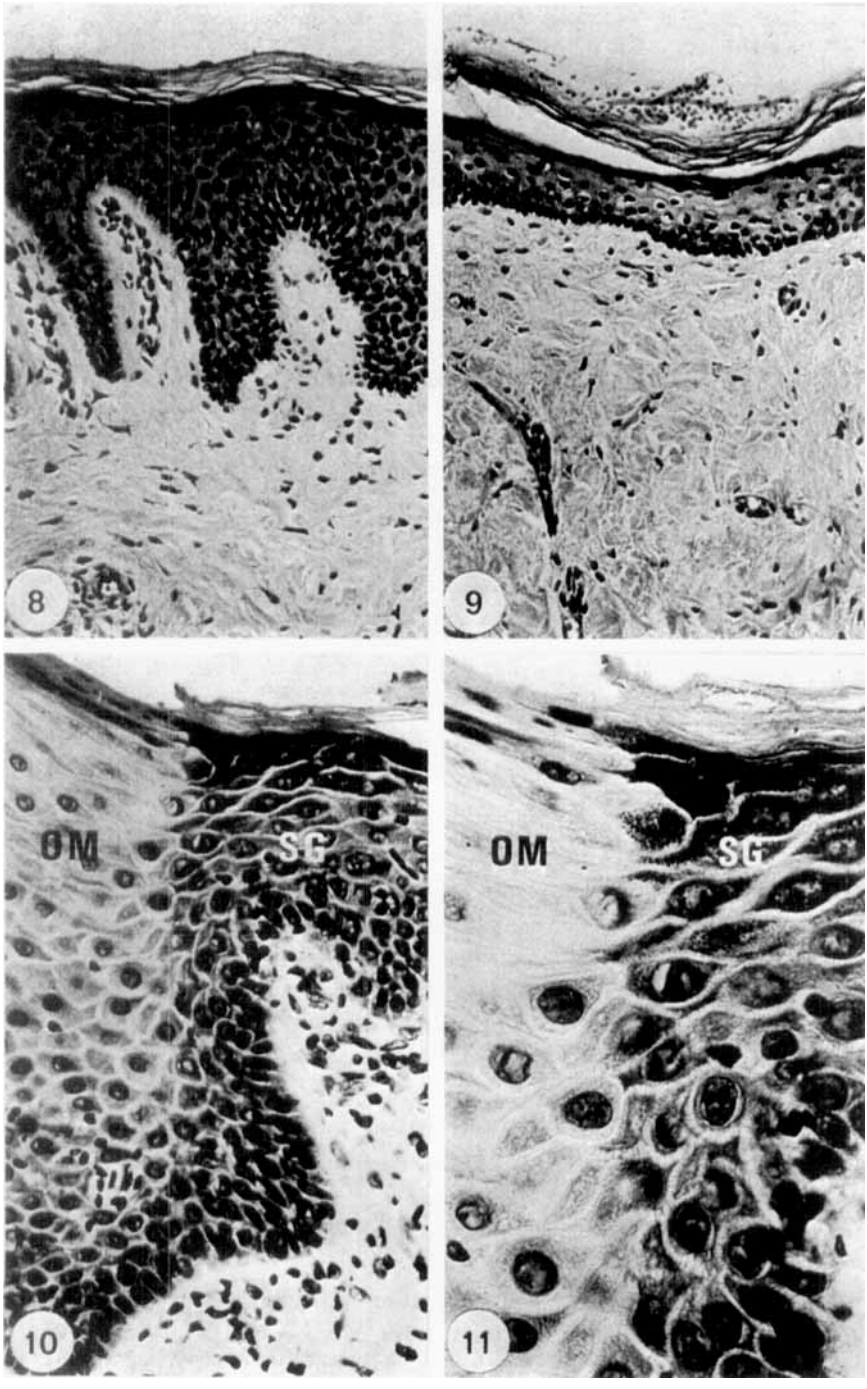
Accumulations of lymphocytes were seen perivascularly in the dermis, and juxtaepithelially in the transition zone. Here, inflammatory cells were

Fig. 8. Skin graft 12 months postoperatively showing persistance of epidermal hyperplasia. Moderate perivascular lymphocyte infiltration. Haematoxylin-eosin, × 180.

Fig. 9. Skin graft 12 months postoperatively showing epidermal atrophy. No inflammation present. Haematoxylin-eosin, × 180.

Fig. 10. Junctional area between graft (SG) and oral mucosa (OM) 12 months postoperatively. Inflammatory cells are found in the juxtaepithelial connective tissue and intraepithelially. Haematoxylin-eosin, × 300.

Fig. 11. The junctional zone in the epithelium shown in Fig. 10 at a higher magnification. Note the abrupt changes in cytology between the epidermis of the graft (SG) and the epithelium of the oral mucosa (OM). The tissue specificity of the epidermis and the oral epithelium has been preserved. Haematoxylin-eosin, × 730.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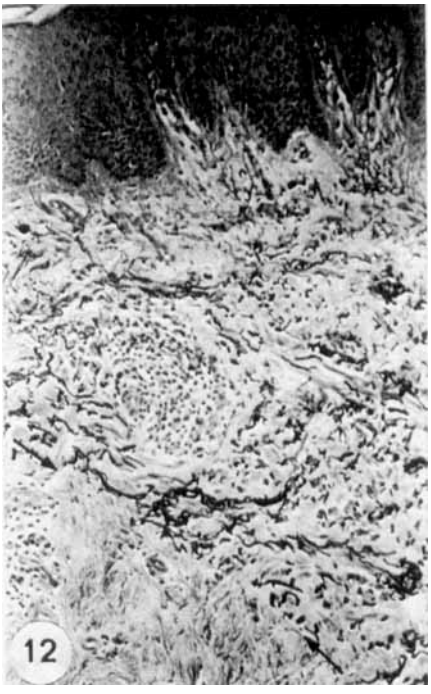

Fig. 12. Coarse elastic fibres in skin graft 12 months after transplantation. The border between graft and recipient bed is clearly demonstrated (arrows). Weigert's resorcin fuchsin, × 110.

often seen migrating through the epithelium (Fig. 10). In the specimens showing epithelial hyperplasia inflammatory cells were found in the subepithelial connective tissue. The connective tissue of the graft can clearly be identified when staining for elastic fibres had been used, because the coarse elastic fibres were only observed in the graft and not in the recipient bed (Fig. 12). Furthermore single hairs, smooth muscle cells, and sebaceous glands of normal structure could be seen.

Infection with candida albicans. In six patients an infection with candida albicans developed in the graft during the first three months probably due to the antibiotic treatment. The diagnosis was confirmed by agar-plate cultivation and by direct microscopic examination of oral smears (Fig. 14).

The biopsy specimens from these patients showed areas with marked epithelial hyperplasia (Fig. 13). alternating with epithelial atrophy in the graft. The superficial cell layers were parakeratotic, and in these layers accumulations of leucocytes were found. Many mitotic figures were observed

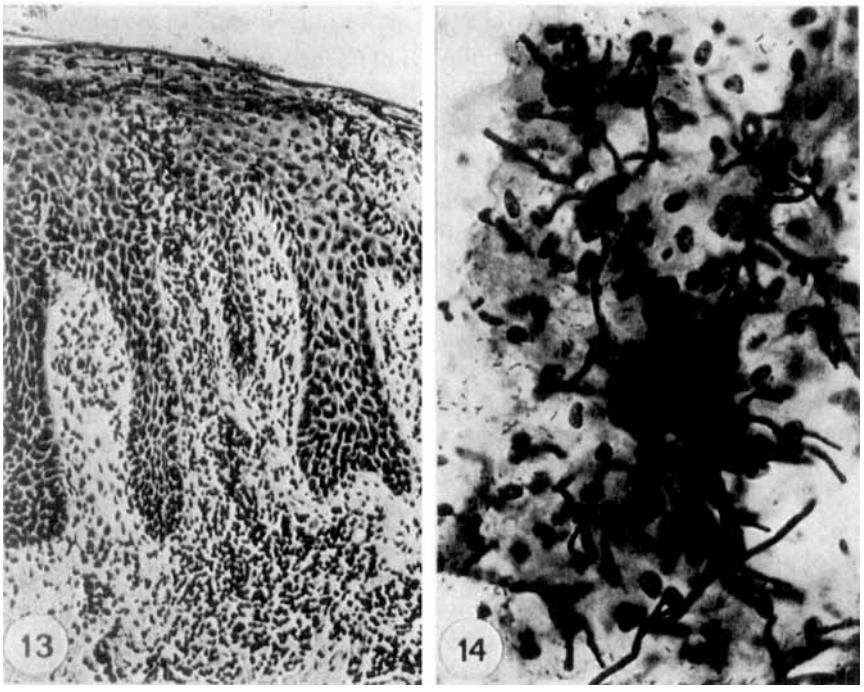

Fig. 13. Biopsy from oral skin graft infected by candida albicans. Note the marked epidermal hyperplasia. The epidermis shows parakeratosis and intercellular accumulations of leucocytes. In the subepithelial connective tissue a moderate chronic inflammatory reaction is seen. Haematoxylin-eosin, × 180.

Fig. 14. Smear from oral skin graft infected by candida albicans. Desquamated epithelial cells are covered by colonies of candida hyphae. PAS, × 310.

in the slender rete pegs. In the connective tissue there was a moderate to heavy chronic inflammatory reaction.

DISCUSSION

The present investigation has shown that split skin grafted to the oral cavity retains the structure characteristic for skin, in spite of the marked changes in the surrounding milieu. Numerous investigations have shown that the differentiation and maintainance of epithelial specificity depends directly on the influence from the underlying mesenchymal tissue (*Hardy*, 1951; *McLoughlin*, 1961; *Wessels*, 1962; *Billingham & Silvers*, 1967, 1968;

McLoughlin, 1968). *In vitro* investigations of human skin have shown that the dermal tissue is necessary for the maintainance of the epidermal characteristics (*Briggaman & Wheeler*, 1968).

The investigation shows that even one year after the transplantation it was possible to determine the line of epithelial contact between the graft and oral mucous membrane. This in spite of the fact that the transition zone in the connective tissue was a newly formed connective tissue probably derived from both the oral connective tissue and from the dermis.

Although the epidermis retained its specificity, the investigation has shown vital cellular changes in the grafted epidermis throughout the period studied. After ten days a simple hyperplasia was found. This is in contrast to the findings of *Umeda* (1967), who in full-thickness autotransplants transferred to the buccal mucosa in dogs found epithelial necrosis 10 days postoperatively. In the present investigation, however, split skin grafts have been used, in which only small amounts of connective tissue were transplanted. This difference in the epithelial reaction may be due to a better nutrition of the transplanted epithelium in the split skin grafts than in the full-thickness grafts. Corresponding differences have been described between split skin and full-thickness autografts in skin (*Hinshaw & Miller*, 1965).

The epithelial hyperplasia and acanthosis has not only been found after transplantation of skin to the oral cavity, but a similar hyperplasia has been described after orthotopic transplantation (*Gillman et al.*, 1955; *Hinshaw & Miller*, 1965). In the present study, however, the hyperplasia persisted in some specimens one year after transplantation, whereas in other specimens the thickness of the epithelium was normalized, or even an atrophic epithelium was found. There seems to be a correlation between the hyperplasia and the presence of inflammatory cells in the connective tissue. Where a normal or atrophic epidermis was found no inflammatory reaction was seen juxtaepithelially. How an inflammatory process affects the squamous epithelium is unknown, but the studies of *Menkin* (1960) suggest that during the inflammation substances are liberated which stimulate the mitotic activity. Provided, that the amount of desquamated epithelial cells per time unit is unchanged, an increase in the mitotic activity will lead to a hyperplasia.

The epidermis at the junction to the oral epithelium is, however, not hyperplastic. This may be due to the formation of the crista in this area caused by the operative technique used (Plates I & II). It is well known that squamous epithelium covering tumours or abscesses is often flattened. The marked hyperplasia of the oral epithelium along the junction, may be explained by the fact that the epithelium migrates down along the cut surface

through the connective tissue. This direction of the epithelial migratory activity is well known from studies on wound healing in other mucous membranes (*Fejerskov*, 1972).

It is not known why in a series of specimens an inflammatory reaction persists in the corium one year after transplantation. This reaction does not seem to be caused by the presence of dentures, as *Nedelman, Gamer & Bernick* (1970) have shown that inflammatory changes are normally not found in oral mucous membrane covered by dentures.

Besides these changes, changes have been found in the single cell layers of the epithelium. The marked dilated intercellular spaces, specially in the stratum spinosum with clearly defined intercellular bridges correspond to the findings of *Matras* (1967) in skin autotransplants to the oral cavity. These changes were seen in the hyperplastic epithelium, and may be caused by the inflammatory reactions in the underlying connective tissue. This is supported by the fact that the intercellular spaces were not dilated when the epidermis had normal thickness without underlying inflammatory changes.

In the normal epidermis glycogen has not been found (*Wislocki, Fawcett & Dempsey*, 1951; *Montagna, Chase & Lobitz*, 1952), but after transplantation glycogen accumulation has been seen intracellularly. Corresponding findings have been described in orthotopic skin autografts by *Scothorne & Scothorne* (1953), and in wound healing investigations in skin (*Lobitz & Holyoke*, 1954; *Lobitz, et al.*, 1962; *Trott & Peikoff*, 1963). The presence of glycogen has been directly related to the keratinization process. Thus, *Trott & Peikoff* (1963) have stated that the glycogen accumulated in the spinous cell layers, if there is a temporary decrease in »the maturation and keratin formation». In the present material, however, no changes in the normal keratinization process were found, the epidermis being orthokeratinized in all specimens. According to *Schultz-Haudt & From* (1961) the two most reliable hypotheses concerning glycogen accumulation in squamous epithelium are: 1) either because the cells are damaged with reduced activity, or 2) there is an increased supply of glucose from the connective tissue due to an inflammatory reaction. The increase in the amount of glucose cannot be transformed in the metabolism of the cells and is therefore stored in the cytoplasm as glycogen.

The hyperplasia of the squamous epithelium in the present study, however, did not seem to reflect a reduced cell activity, and histochemical investigations on skin autotransplants to the oral cavity in dogs (*Umeda*, 1968) have shown that there is an increase in the amount of oxydative enzymes in the graft epidermis. Thus, the accumulation of glycogen may hardly be related to the reduced cell activity, but rather to the pathological changes in the

connective tissue. The present investigation, however, does not permit further conclusions.

Apart from the perivascular inflammatory cells in the connective tissue, morphological changes in the dermis were very few. The coarse bundles of collagen in the graft persisted. Furthermore, it was possible to find the coarse bundles of elastic fibres in the graft, whereas it was not possible one year after transplantation to find elastic fibres in the transition zone between the graft and the oral mucosa. Thus, the elastic fibres can be used as markers of the connective tissue in the graft, as demonstrated in orthotopic mucosal grafts in monkeys (*Karring, Östergaard & Löe*, 1971).

The present study, therefore, indicates that even though split skin autografts are exposed to marked changes in the surrounding milieu — saliva, bacteria and pressure from dentures — only minor changes take place in the graft. Furthermore, these changes seem to be almost the same as those found in orthotopic transplants.

The marked changes in the grafted epidermis in patients showing an oral infection with candida albicans were remarkable. The parakeratinized, hyperplastic epithelium alternating with atrophic areas and the marked penetration of inflammatory cells through the epithelium was similar to the changes observed in oral leucoplakias with superimposed candida infection, the so-called »speckled leucoplakias» (*Jepsen & Winther*, 1965; *Pindborg et al.*, 1963). The present study, therefore, showed that when exposed to the oral milieu the epidermis may react similarly to candida infection as to hyperkeratinized oral epithelia.

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