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THE EFFECT OF ALTERED FUNCTION ON GINGIVAL TISSUE

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WHEN a fixed bridge which had a pontic in contact with the epithelium was removed, it appeared that the tissue under the pontic was inflamed; but it soon became indistinguishable from that of the adjacent tissues. This observation suggested that when oral epithelium is covered by a pontic certain gross and reversible changes occur in the underlying tissue.

In this preliminary report the microscopic structure of the tissue under a pontic is described and reasons for the changes seen are discussed.

History

The maxillary central incisors were extracted 14 years previously. After 10 years, during which a removable partial denture replaced the missing teeth, a fixed bridge was inserted. At that time the gingival tissue on which the pontics rested was healthy and no different from the adjacent tissue. The bridge remained functional for four years. It eventually was removed for aesthetic reasons; and the epithelial tissue adjacent to and under one pontic was at once excised and prepared for histological examination.

Results

The histological examination showed that the epithelium which clinically appeared normal can be divided into two zones (Fig. I). The outer zone consists of normal keratinized epithelium. The inner zone, immediately adjacent to the epithelium covered by the pontic, is thickened and, curiously, lacks rete pegs. The transition from the inner zone of exposed epithelium to a non-keratinized epithelium under the pontic is abrupt. In some areas this non-keratinized epithelium consists of only two or three layers of flattened cells (Fig. II). During the clinical examination the altered epithelium appeared to be intact. Histologically, however, breaks in epithelium are evident. These may be artifacts or they may be real. No definite opinion can be expressed at this stage.

The appearance of the underlying connective tissue is equally curious. Directly below the covered epithelium the tissue is hyperaemic and infiltrated with lymphocytes and, to a lesser extent, plasma cells. The vascularity and cellular infiltration are most marked under the epithelium covered by the pontic, less evident under the inner zone, and absent under the first zone of clinically healthy epithelium (Fig. III). Below the cellular infiltration the tissue appeared to be oedematous and, by contrast with the more superficial layers, almost acellular.



FIG. I. —A low power view of a section of the epithelium adjacent to and under a bridge pontic showing its alteration under the pontic and the distribution of the cellular infiltration. (Picro-Mallory.)

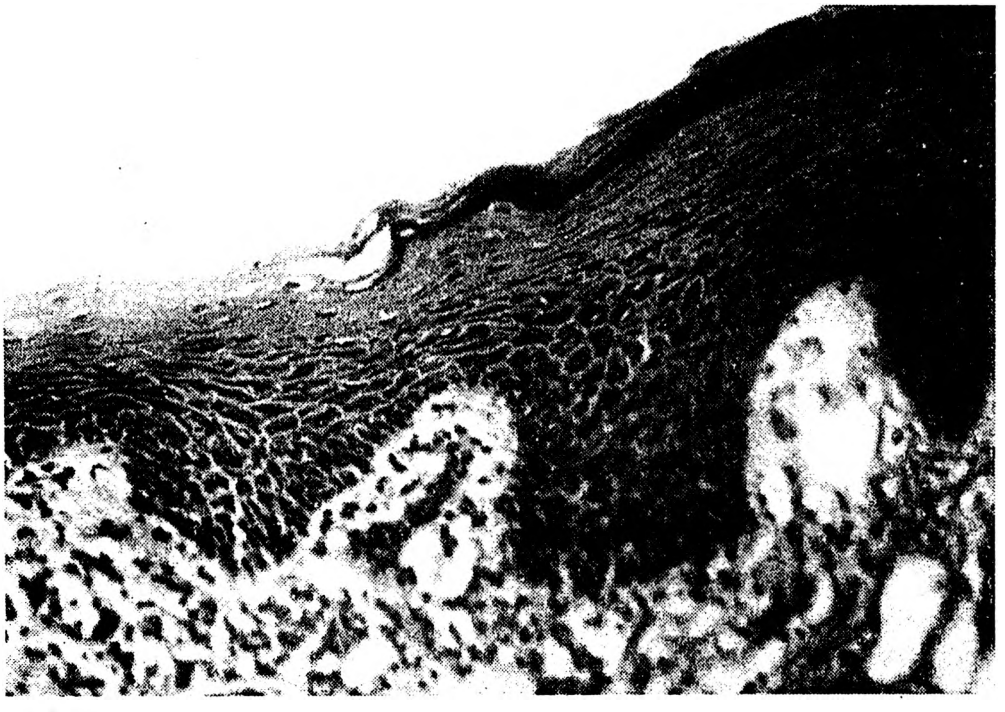


FIG. II.—Higher magnification of the same section as seen in Fig. I showing the transition from keratinized to non-keratinized epithelium.

DISCUSSION

For cosmetic reasons the construction of a certain type of fixed bridge is such that the pontic of the completed appliance is in firm contact with the underlying epithelium. Unless the continuous growth of the epithelium under pontics, especially that covered by a number of pontics, altered after a bridge has been placed, this proliferation of epithelium could cause displacement of the pontic. There is no clinical evidence of such displacement, however, and the histological appearance of the epithelium here described suggests that, once it is covered by a pontic, proliferation of epithelium is considerably reduced.

The change which occurred in the epithelium may have been caused by the friction on it produced by the pontic during masticatory function. Such movement, if marked, would tend to cause a breach in the epithelium. Although the appearance of the underlying tissue is suggestive of a chronic inflammation, this appearance is not necessarily due to ulceration of the epithelium. If the frictional effect was of a milder character, thickening rather than thinning of the epithelium could be expected.

The appearance of the epithelium under the pontic may be ascribed to degenerative changes due to loss of function. Hence this degeneration of the covered epithelium must proceed until it reaches a steady state. This steady state must thus be maintained for the entire period that the pontic remains in position, and can only revert to normal after removal of the pontic.

It is suggested, however, that the alteration in the epithelium is not due to traumatic stimuli from the pontic nor degenerative as a result of loss of function. It is an adaptive change to alter the function of this epithelium.

The placement of a pontic will lead to the accumulation of dead cells, bacteria, and degradation products of food in a stagnation area. If the underlying tissues do not withdraw to re-establish a non-stagnating area, this area could be cleansed effectively if it was flushed with a fluid—but the covered keratinizing stratified squamous epithelium does not favour the passage of fluids through it. The thin



FIG. III.—Higher power view of a frozen section of the epithelium to show the thinness of the epithelium in certain sites under the bridge pontic. (Toluidine blue.)

epithelium present under a pontic does, however, provide circumstances favouring the passage of fluid through this surface.

If the altered oral epithelium allows fluid exchange between the plasma and saliva, the difference in osmolarity between these fluids favours the flow of water and certain electrolytes from the saliva to the blood. Similar circumstances in the kidney are overcome by regulation of the hydrostatic pressure. Regulation of the hydrostatic pressure in the vascular underlying tissues would promote the flow of fluid through the epithelium into the stagnating area and hence into the saliva.

If the stagnating area under a pontic is cleansed by this mechanism an alteration of the metabolic processes to produce a secretory epithelium is not required.

If the altered epithelium allows for the passage of fluid from the plasma, this increased permeability would also allow the passage of substances in the opposite direction. Such epithelium, although protected by the overlying pontic, would also be more vulnerable to trauma. The tissue reaction below the altered epithelium may thus be due to either the permeability and/or the friability of the altered epithelium.

The histological appearance of the gingival tissue underneath the pontic which is described is in some respects similar to that described by Cohen[1] in the col. The question, whether the similarities between these entities are confined only to the histological features, is being investigated.

ACKNOWLEDGMENTS

We wish to thank Professor A. E. Dodds for reading the manuscript and Miss Moore for taking the photographs.

REFERENCE

1. COHEN, B. The pathology of the interdental tissues. D. Pract., **9** : 167-173, 1959.

IATROGENIC DISORDERS IN DENTISTRY

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INTRODUCTION

The meaning given by different authors to the term "iatrogenic" varies. Literally translated, it means "that which is produced by the healer" (Durocher and Novak). Harrison (1958) applies it to ". . . not only the direct injuries that may result from therapeutic and diagnostic measures, but also to the hurt that can be inflicted by words or actions".

This paper is restricted to the untoward effects caused by drugs in common use in dentistry.

ANTIBIOTICS

Welch, (quoted by Alling and Pulaski, 1959), in a survey of one-third of the general hospitals in the United States of America for a three-year period ending in 1957, revealed that approximately one thousand cases of life-threatening complications following antibiotic therapy were encountered. To quote Goulding (1957), "The attitude constantly should be not that it *might* help to give just a little antibiotic, but is this dangerous drug absolutely necessary?"

The untoward reactions arising from the administration of antibiotics fall into three categories:

1. Direct toxic effects of the antibiotic.
2. Reactions encountered in sensitized patients or due to patient idiosyncrasies.
3. Superimposed infections caused by antibiotic-resistant organisms.

1. PENICILLIN

While penicillin is the least toxic of the antibiotics, it is also the most allergenic;

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