

Periodontal Disease

IN CHILDREN
AND ADOLESCENTS

PAUL N. BAER, D.D.S.
SHELDON D. BENJAMIN, D.D.S.



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To
Our Wives and Children
Without Whom It All
Might Still Have Been Possible
But Not Half as Much Fun

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Preface

The idea for this book originated from a series of lectures first presented to graduate students in orthodontics and pedodontics. During this period of time, it became increasingly clear, that while textbooks on periodontology were generally in agreement that the onset of periodontal disease frequently originated during childhood or adolescence, minimal attention was given to the specific periodontal and oral problems which occurred during this most important period of life. It also became evident that systemic diseases which were reflected within the oral cavity in childhood and adolescence were frequently interrelated and many times confused with local diseases of the periodontium. Therefore, we felt it necessary to combine in a single textbook discussion of the diseases of the periodontium along with those systemic diseases which had oral manifestations.

Current knowledge on the etiology of the most common of all the destructive periodontal diseases, namely periodontitis, has established that it is largely preventable and arrestable. It is also a disease entity that is extremely well covered in all the recent textbooks on periodontology and there was little new that we could add. For these reasons juvenile periodontitis has been given minimal coverage in our book.

Periodontosis or essential periodontitis, on the other hand, which is neither a common disease nor as amenable to treatment, is given extensive coverage, since this material is not as readily available.

Each chapter is a separate unit and may be randomly selected for study without adversely affecting the whole.

In brief, this book was written to fill a void. In doing this we have summarized our personal experiences and have reviewed the pertinent literature involving investigations on the etiology and treatment of the various periodontal diseases found in children and adolescents.

The bibliographies, rather than being all inclusive, have been carefully and selectively chosen. Furthermore, it is not the purpose of our book to describe basic "how-to-do-it" techniques, but rather to give to the practicing dentist and physician insight into the oral problems which they may encounter in treating patients in this age group. For these reasons we believe this book should prove to be useful to the student who desires extra "elective" knowledge, to the general dentist, the pedodontist, the periodontist and the pediatrician.

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Acknowledgments

The chapters on the normal periodontium and the pathology of periodontal disease in children was largely the work of Dr. Henry M. Goldman and Dr. Morris P. Ruben. Dr. Frank G. Everett collaborated on the section on periodontosis. Dr. Herbert L. Tanenbaum was principally responsible for the patient with chronic heart disease. Dr. Hugh Kopel made major contributions to the treatment of the handicapped patient. Adolescent nutrition was the work of Dr. Olaf Mickelsen. Dr. Maury Massler and Dr. Corey H. Holmes contributed ma-

terial to the chapter on prevention. Dr. Gerald Lubin was principally responsible for the chapter on the psychological management of the adolescent patient.

We are deeply indebted to our contributors and wish to express our appreciation for their contributions. However, in all instances, the final version of each chapter as to wording and ideas is our own responsibility.

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1

The Normal Periodontium

GROWTH AND DEVELOPMENT

The periodontium during childhood and puberty is in a constant state of change owing to the exfoliation and eruption of the teeth. This makes a general description of the normal periodontium difficult because it varies with the age of the patient.

Zappler,¹⁸ however, has attempted a general description of the juvenile periodontium, listing its characteristics as follows:

Gingiva

1. *More reddish*, because of thinner and

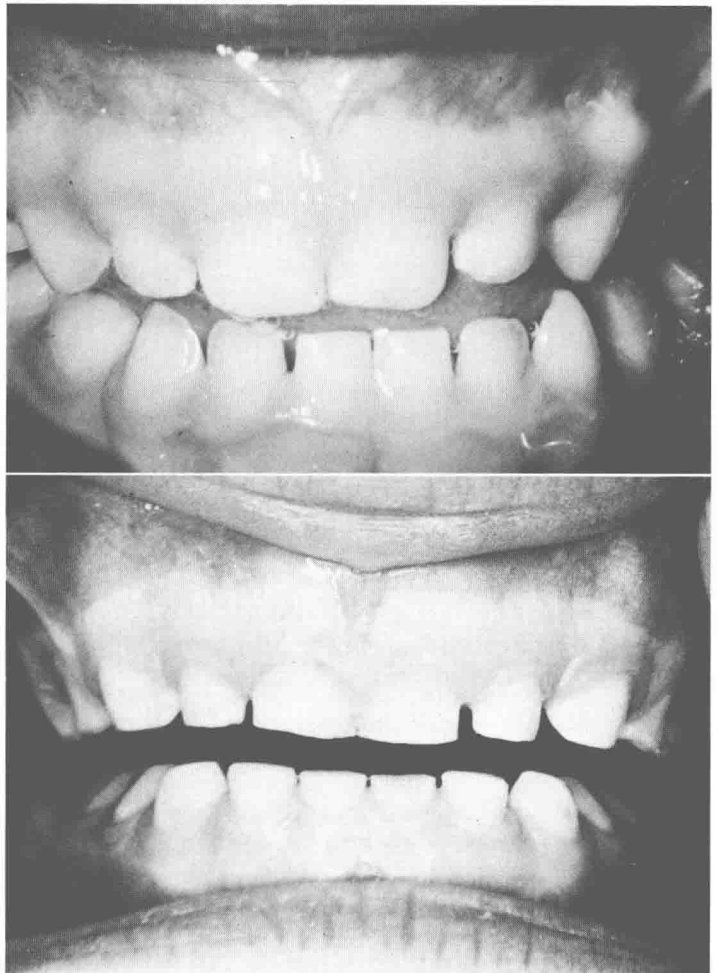


Fig. 1-1. *Top.* Normal gingiva in a 4-year-old child. The gingiva is firm and not readily retractable; there is usually a well-defined zone of attached gingiva present. *Bottom.* Normal gingiva in a 3-year-old child. Note wear facets (bruxism is common in young children).



Fig. 1-2. Diastema formation is normal in the anterior region (6-year-old child).



Fig. 1-3. Normal gingiva in a 10-year-old child. The gingiva margin is still not at its adult level. It is too high coronally. Passive eruption is generally not completed before the late teenage period.

less hornified epithelium and greater vascularity

2. *Lacks stippling*, because of the shorter and flatter connective papillae of the lamina propria

3. *Flabbier*, associated with decreased density of the connective tissue of the lamina propria

4. *Rounded and rolled margins*, related to hyperemia and edema that accompanies eruption

5. *Greater sulcular depth*, relative ease of gingival retraction

Cementum

1. Thinner
2. Less dense
3. Tendency to hyperplasia of cementoid

apical to the epithelial attachment (quoting Gottlieb)

Periodontal membrane

1. Wider
2. Fiber bundles less dense with less fibers per unit area
3. Increased hydration, greater blood and lymph supply

Alveolar bone

1. Thinner lamina dura (radiologically)
2. Fewer trabeculae
3. Larger marrow spaces
4. Decreased degree of calcification
5. Greater blood and lymph supply

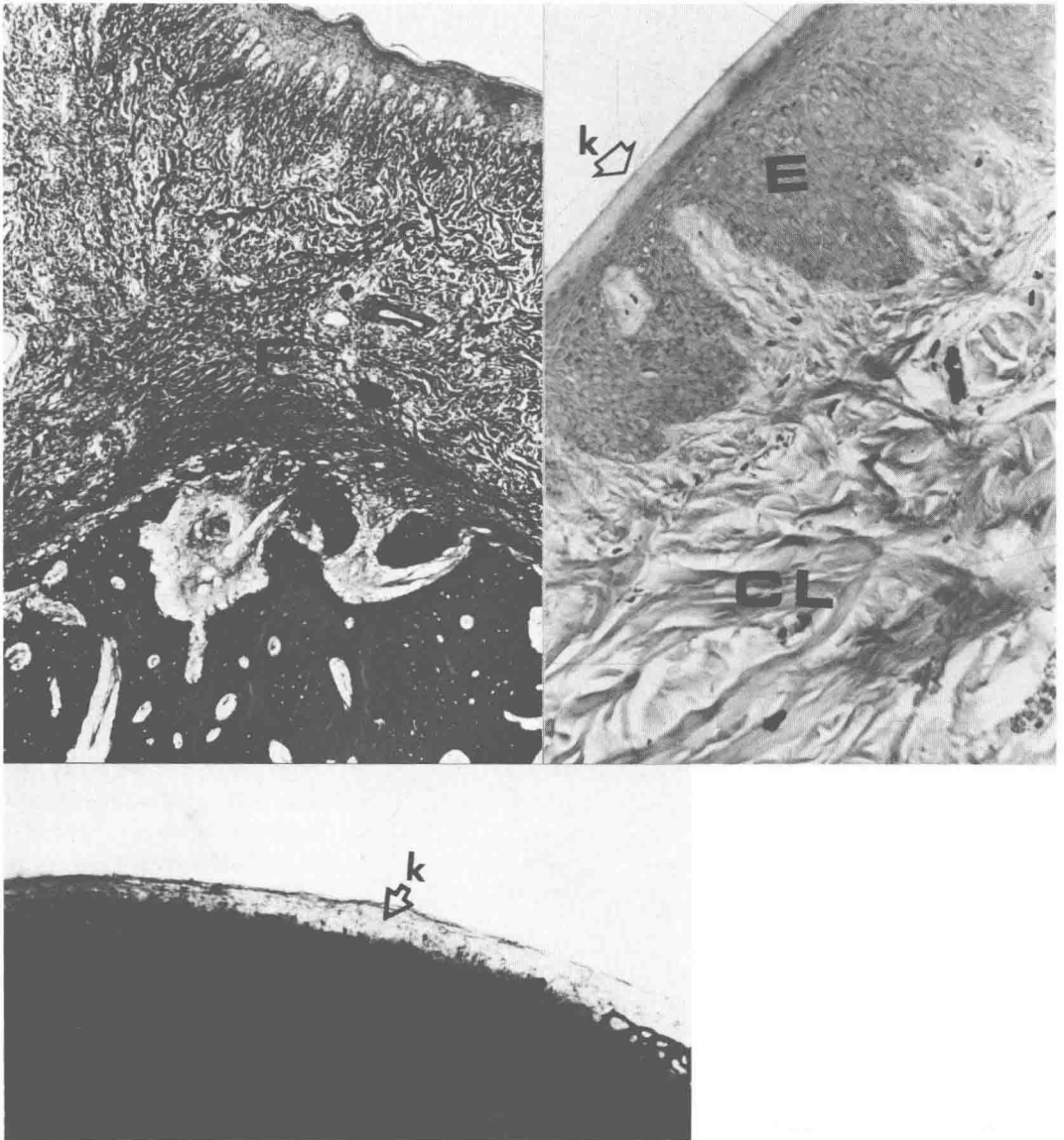


Fig. 1-4. *Top left.* Buccolingual section through interdental hard and soft tissues; an interdental diastema was present. Interdental gingiva is structured comparably to attached gingiva with dense collagenous corium and keratinizing epithelial cover. The soft tissue complex is usually tightly bound to crestal bone by means of anchoring fibers. *F* is the periosteal portion of the gingiva. *Top right.* This 100X photomicrograph reveals the quality of interdental epithelium (*E*) in the diastema zone. A definitive stratum corneum is indicated at *k*; it may be either para- or orthokeratotic. Both can be limiting to the penetration of exogenous irritants into the tissue (generally, only lipid-soluble substances may enter). The gingival corium (*CL*) is notable for stable and well-oriented collagen. *Bottom.* Perfusion specimen (dog perfused through carotid artery with patent blue violet and carbon suspension). The blue-dyed vascular transudate flows freely into the epithelium (*black band*) and is prevented from passing into the oral cavity by the stratum corneum (*k*).

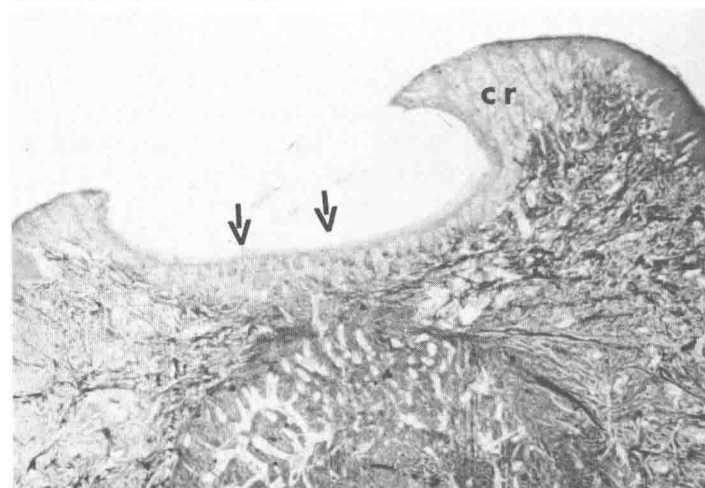
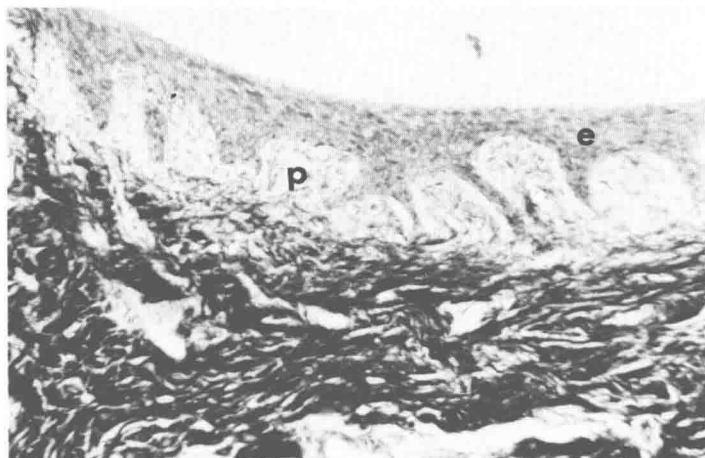
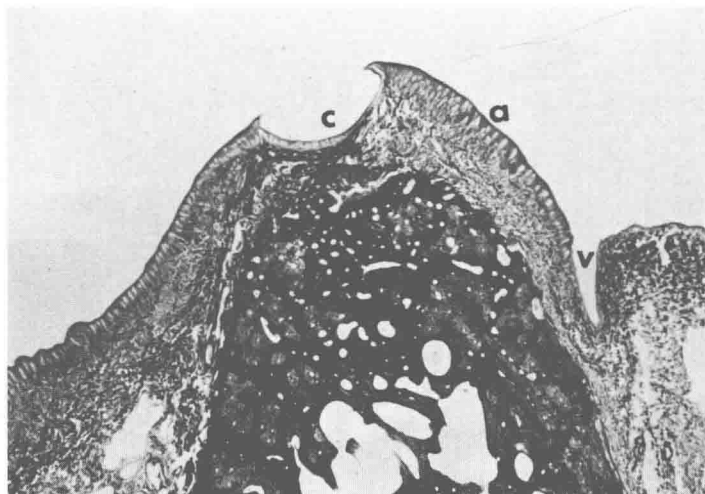


Fig. 1-5. *Top.* The bucco-lingual section (trichrome stain) through the interdental septum and contiguous soft tissues. The concavity (c) represents the col configuration of gingiva, the soft tissues conforming to the shape and dimensions of the proximal dental contact. It is lined by stratified squamous epithelium, thin and devoid of keratinization. a indicates the buccal zone of attached gingiva surmounted by keratinizing epithelium. v is the vestibular fornix. In this case the zone of attached gingiva is very broad apico-occlusally, while the dimension of alveolar mucosa is narrow. *Center.* In this 100X magnification of col epithelium and subjacent gingival corium note that the epithelium (e) is hyperplastic, an incipient response in inflammation. The area of ulceration is not evident; however, even the widening of the intercellular areas should be considered the equivalent of ulceration, since the deficiencies at the interface of epithelial cells allow bacteria and their toxins (exo- and/or endo-) to pass through the epithelial zone into the gingival lamina propria (p). There is reduction of collagen in the superficial aspect of the corium; resorption is mediated by enzyme activity. *Bottom.* In this more advanced interdental inflammation, the arrows point to inflammatory process at the col base. An inflammatory state has extended to involve both buccal (cr) and lingual walls of the col.

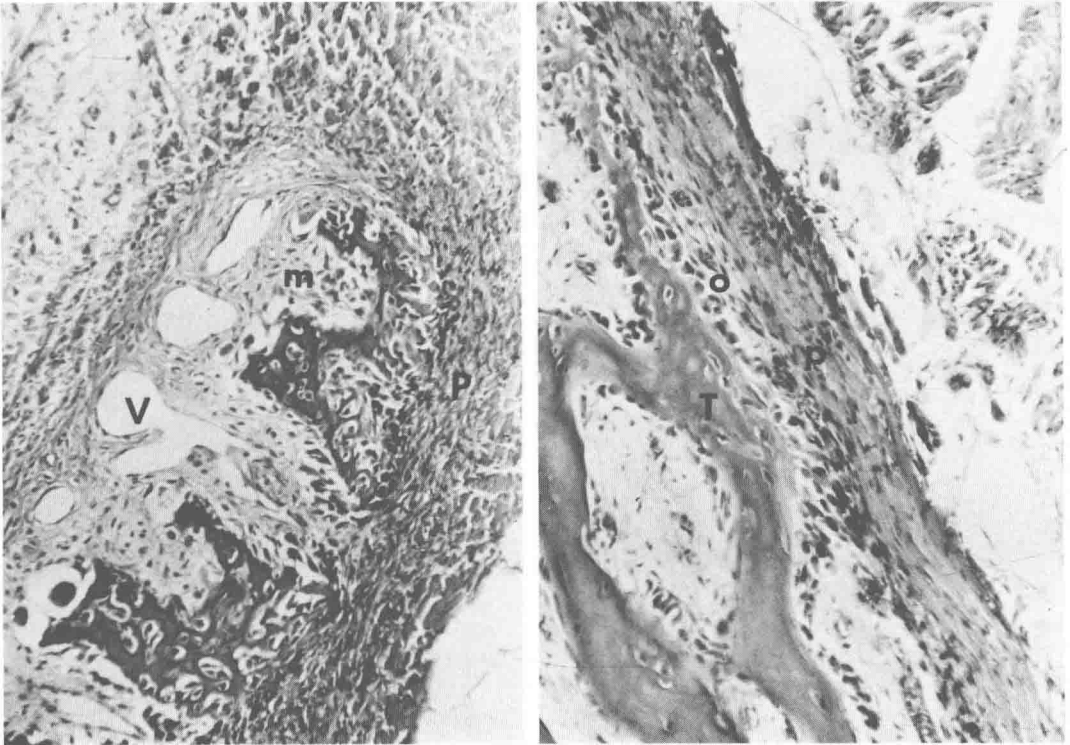


Fig. 1-6. *Left.* Developing alveolar process in fetus (about 6 months gestation). Highly cellular new bone adjacent to developing tooth. Trabecular pattern is evident without formation of buccal cortical plate. Cortices of a lamellated nature tend to be "completed" as the alveolar processes develop to their physiologic maximum in middle/late adolescence; osteogenesis becomes relatively quiescent on periosteal aspects of septa at this time. (*P*) periosteum; highly cellular adjacent to bone. *m* is the marrow area rich in osteoblasts contiguous to bone. *V* is the vascular channels. *Right.* Higher magnification of prenatal osteogenetic activity. *T* represents bony trabeculum with included osteocytes. Osteoblasts (*o*) as a part of the periosteum (*p*) engaged in elaboration of osteoid, seen here as a pale (eosinophilic) substance adjacent to osteoblasts.

6. Flatter alveolar crests associated with primary teeth

Similar comparisons are found in Finn's text and in publications by Cohen⁴ and Bradley.²

In general, the authors agree with these descriptions, noting certain modifications: The gingiva in the young child with a totally deciduous dentition is generally pink and firm, with a well-defined zone of attached gingiva; it does not appear to be either reddish or flabby, as Zappler stated. The width of the attached gingiva varies

between 1 and 6 mm. for the primary dentition and between 1 and 9 mm. for the adult dentition.^{1A} The narrowest zone of the attached gingiva is found in the region of the maxillary and mandibular first premolars or first bicuspsids. The widest area is found in the maxillary and mandibular incisor region (Fig. 1-1 *Top*). Present evidence suggests that there is an increase in the mean width of the attached gingiva from the primary to the adult dentition. After maturity, however, there is little additional change. During the period of the mixed dentition, Zappler's findings that the

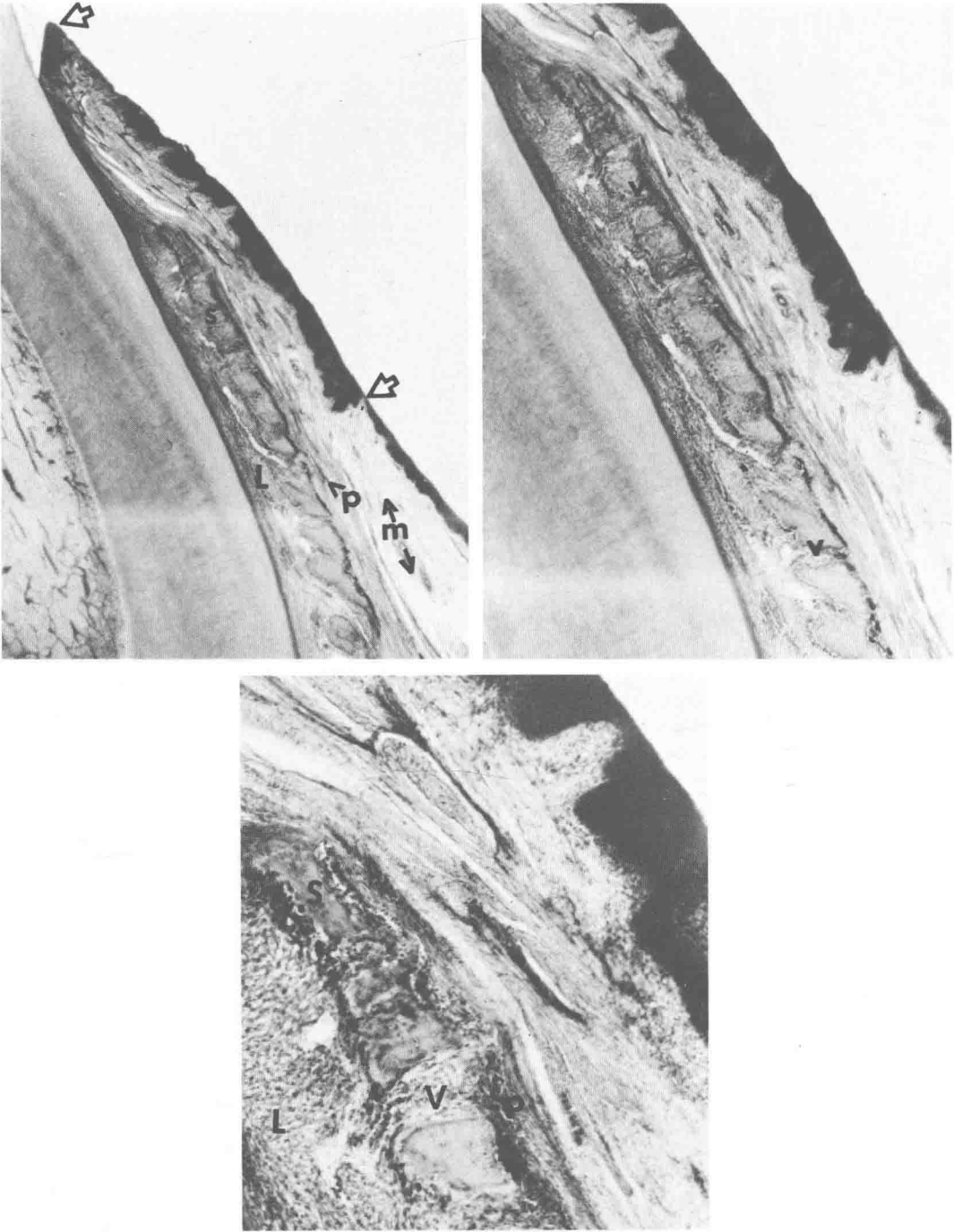


Fig. 1-7. *Top left.* The zone of attached gingiva (between *arrows*) in child tends to be inordinately wide in relationship to the width of the alveolar mucosa ($\leftarrow m \rightarrow$). Gingival connective tissue is usually denser and better organized than that of the alveolar mucosa. The epithelium of the attached gingiva is of the keratinizing type, while that of the alveolar mucosa lacks physiologic propensity for keratinization. The mucosa is resilient and pliable,