

The Angle Orthodontist

Vol. III

No. 3

*A magazine established by the co-workers of
Edward H. Angle, in his memory*

Hypertrophic Gingivitis*

JAMES R. BLAYNEY, B.S., D.D.S., M.S.

*Department of Dental Pathology and Therapeutics,
University of Illinois, College of Dentistry*

It is not necessary to make a complete review of the literature on this subject in order to perceive that the nomenclature is extremely varied. One meets such terms as "Primary Hypertrophy of the Gums," "Chronic Hyperplastic Gingivitis," "Polypus of the Gums," "Gigantism of Gums," "Elephantiasis of Gums," and "Ossified Gums." This clearly indicates the need for more careful consideration in the use of terms.

Virchow defined hypertrophy as an increase in the size of individual tissue elements while hyperplasia is an increase in the number of elements. It is quite evident that pathologists are not agreed as to the use of these terms. Beatty and Dickson¹ suggest that the term "hyperplasia" be restricted to proliferative changes in tissue—not being an actual tumor—in those regions in which it will serve no useful purpose, and that hypertrophy should indicate a disproportionate increase of an organ or some of its essential components because of an increased functional activity, i.e., hypertrophy is essentially physiologic in character.

The mechanism by which a hypertrophy may be produced is very interesting. Very briefly these factors may be described as:

1. Nervous stimulation, which is well illustrated by the enlargement of the mammary glands in some diseases of the ovary or uterus.

¹ Beatty and Dickson, *Textbook of Pathology*, 3rd Edit. 245. C. V. Mosby, 1928.

*Read before the Chicago Society of Orthodontia.

2. Improper coördination of muscular effort.
3. Effects of local stimulation, as the development of ridges on bone for the attachment of powerful muscles.
4. Excessive or abnormal nutrition which may be illustrated by the experiment of Hunter in transplanting the spur of a cock to the comb, an area which is highly vascular and also prevents the spur from being worn down. Extracts from glands may produce a like effect.
5. Closely related to local stimulation, we see work hypertrophy nicely illustrated in the myocardium of an athlete and compensatory hypertrophy, sometimes called complimentary hypertrophy, seen in the heart which compensates for a valvular insufficiency or in such bilateral organs as the kidney, following the removal of one of the units.

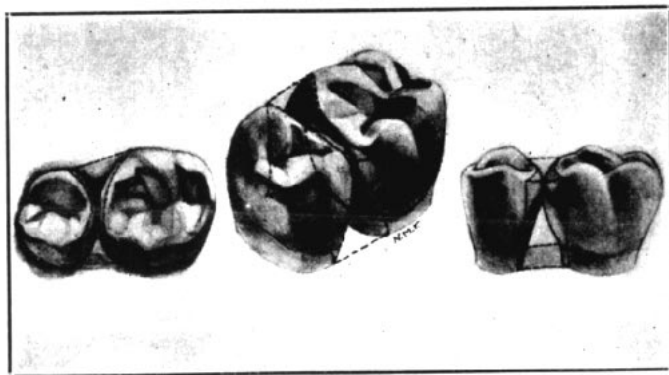


Figure 1. Shape of normal interproximal space between upper second bicuspid and first molar.

When these are applied to the oral cavity it becomes evident that the first and fourth factors must be considered in the case of a true hypertrophy while the third, i.e., local irritation, is more frequently associated with localized enlargement on the basis of a productive inflammation. This does not permit a discussion of the terms "gigantism" and "elephantiasis" except to mention that the former is usually associated with glandular dysfunction, while elephantiasis results from the blocking of the lymphatic system of the area, which first expresses itself as a localized edema which is then followed by the over production of inflammatory fibrous tissue.

Literature

Our literature contains many case reports of these conditions. They may be arranged in two classes, namely, those which may be considered as true hypertrophies, the patient seeking treatment because of a deformity; and those which are undoubtedly the result of a local irritation attended by a productive or proliferative inflammation. Also, there are certain factors

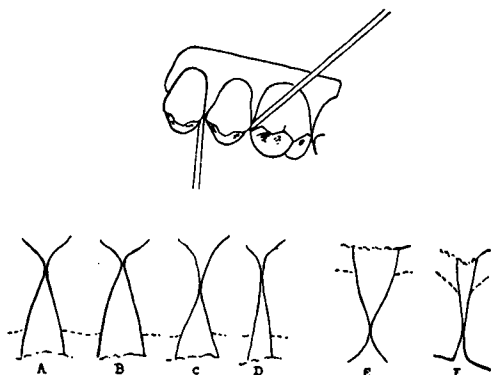


Figure 2. Drawing to illustrate shape of interproximal space in longitudinal section through contact point when viewed from buccal. (Redrawn after Black.)
A. Normal. B. Contact point too near occlusal surface. C. Contact point too near gingivae. D. Loss in mesio-distal width. E. Shape in molar region as compared to F. in anterior region.

which appear quite consistently in each group. In considering the true hypertrophies we see that both sexes are involved nearly equally. Many of the reports state that the enlargement of the gums was noticeable at birth or shortly after, and the remainder of the cases developed during childhood. There is also considerable evidence to suggest heredity. Murray¹ reports three cases in one family all under seven years of age and Ruggles² cites two brothers 15 and 17 years respectively who were both the 9th and 10th cases in the same family in three generations.

In classifying the cases reported in the literature, I find that 17 were bilateral and involved both jaws; 2 were bilateral of the maxilla; 1 was bilateral of the mandible; 1 unilateral of the maxilla and in 6 cases the description was too incomplete to permit an accurate statement. As to the age at which the hypertrophy was first noticed, I found without question

1 Erickson—Heath's Diseases of Jaws, 3rd Edit. P. 127.

2 Ruggles, S. D. Primary Hypertrophy of Gums, J. A. D. A., 11:1204.

that in six cases the process had become established at birth; 2 before two years; 2 cases before six months; 1 before one year; 1 by six years, 1 by 9 years; 5 recorded as during childhood; 8 cases in which the age was not stated; however, all patients had presented for treatment before the seventeenth year and only one case was an adult. I have emphasized this because I thought such information might be of special interest to the orthodontists.

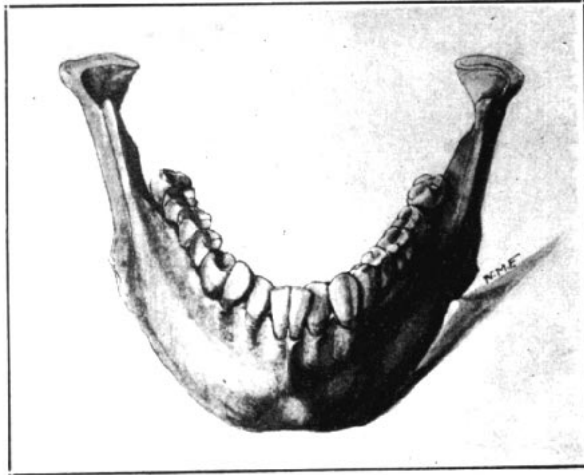


Figure 3. Typical crowding-production-loss in mesio-distal width of interproximal spaces in anterior region.

The study of the physical conditions, as presented by such cases, is most incomplete; however, the following notations have been made. In two cases it was thought that the hypertrophy was associated with a traumatic injury to the face; three were associated with neurofibromatosis (V. Ricklinghausen's disease); two with grand mal; two followed a fungus infection of the oral mucosa; two associated with amenorrhea; one with a thyroid disturbance; and one with cardiac insufficiency, producing a chronic passive congestion.

Etiology

General Factors: The preceding statement clearly indicates that the etiology of those cases which may be classed as a true hypertrophy is little understood. However, if we apply to those cases the previously mentioned factors commonly associated with hypertrophies in general, we shall see

that the majority of the cases reported fall in either the nervous stimulation or in the localized increased nutrition groups. Certainly work hypertrophy does not enter. Another factor should be borne in mind, which is the possibility of over-production of fibrous tissues (scar tissue) following the healing of either a local injury or a proliferative inflammatory reaction. It seems possible that such conditions may arise from several sources. A. Hentze¹ uses the term "frontier disease" in describing these true hyper-

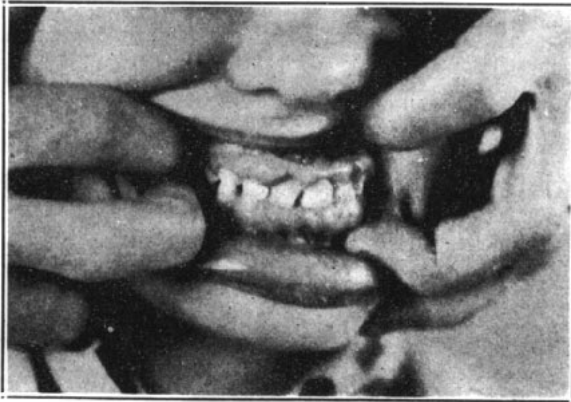


Figure 4. Band of hypertrophic tissue extending from right bicuspid region to the same area on left.

trophies, which I interpret as a "borderline disease" or, as Monier² writes, "between a chronic inflammation and a tumor of the gums; it constitutes a non-malignant degeneration of the gums."

Pregnancy is another factor in the constitutional list. Some women experience enlargements of the gingival papillae at this time. It seems to be associated with those areas which were previously inflamed because of local irritation and for this reason Garretson³ used the term "proliferative gingivitis of pregnancy." These cases which have come under my observation have tallied with this description, i.e., they were not true hypertrophies. There are many citations of such conditions being promptly relieved following delivery, without local treatment.

- 1 A. Hentze. *Fortschritte der Zahnheilkunde*, October 1928.
- 2 Leon Monier. *American Dental Surgeon*. 47:273.
- 3 Garretson, J. E. *A System of Oral Surgery*. Philadelphia. J. B. Lippincott Company, 1890.

Local Factors: The etiologic factors of localized enlargement of the gingival tissue, most frequently the septal gingivae, are indeed numerous but not so difficult to explain. In the main, all such proliferations are on the inflammatory basis and as such are not hypertrophies.

Probably the most frequently found factor in these localized swellings



Figure 5. Musculature of normal uterus.

is a deviation from the normal shape of the interproximal space. (Figure 1). I believe the most frequent deviation to be the loss in the mesio-distal width. (Figure 2.) We see such conditions where proximo-occlusal restorations have not developed normal contour; where caries has progressed slowly, permitting the area of greatest contour of one tooth to project into the developing cavity of the neighboring tooth; where failure in development of either maxilla or mandible, with the attending mal-position of the teeth, has resulted in a marked reduction in the size of the space. Sometimes the placing of orthodontic bands is sufficient irritation, added to the already existing condition, to produce such a result. With the loss in mesio-distal width there is not sufficient room to accommodate the normal bulk of septal

tissue which must, by necessity, project buccally and lingually beyond its normal position. This condition is continuously aggravated in many ways such as by injury in the excursion of food; the unevenness in contour of

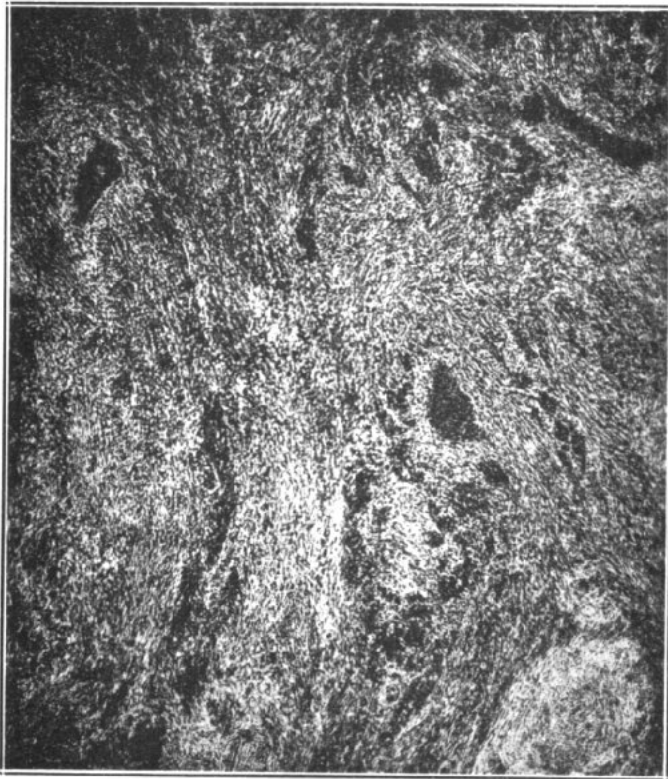


Figure 6. Musculature of an atrophic uterus.

the soft tissue increases the difficulty in keeping the part in a hygienic condition; injury inflicted by the toothbrush, etc. Added to the above factors, we must consider the mechanics of circulation in the local part. The blood supply of the septal gingivae is through the septal crest of the alveolar process and the periodontal membrane. When the mesio-distal width of the

interproximal space has been materially reduced, we often see the bony septum exhibited in the radiographic film either as a long spinous process or with considerable resorption of the crest. This change in anatomic form is accompanied by alterations in the vascular system. Under otherwise

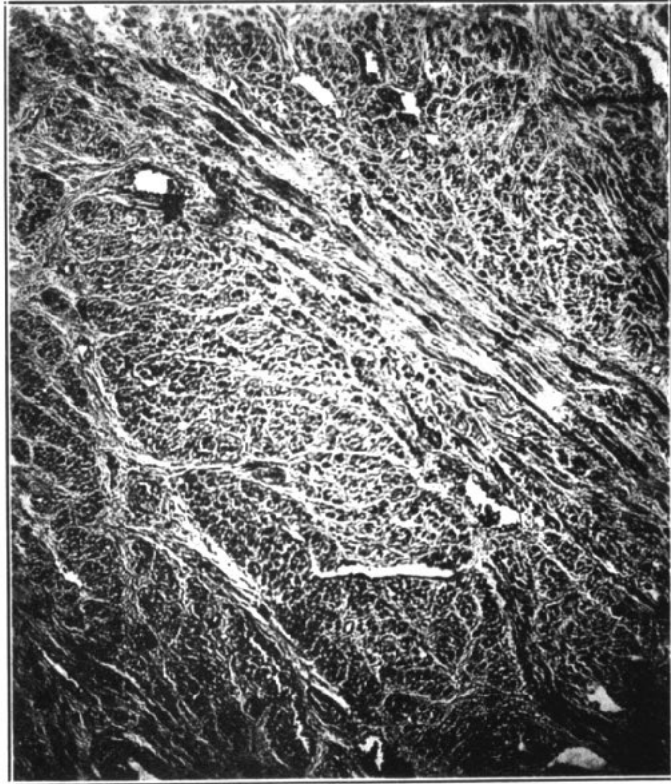


Figure 7. Musculature of a pregnant uterus.

normal conditions of the gingivae this change would not interfere with the movement of tissue fluids to a marked degree, but when it is accompanied by the previously mentioned injuries, we find that the drainage is inadequate. Thus a chronic passive congestion is grafted upon these local conditions. You see that the vicious cycle is readily established.

So far as my observations go I believe we see these localized enlargements or swellings of the septal gingivae more frequently in the anterior part of the arches. (Figure 3.) Some writers have attributed these so called hypertrophies to mouth breathing. They say that the friction pro-

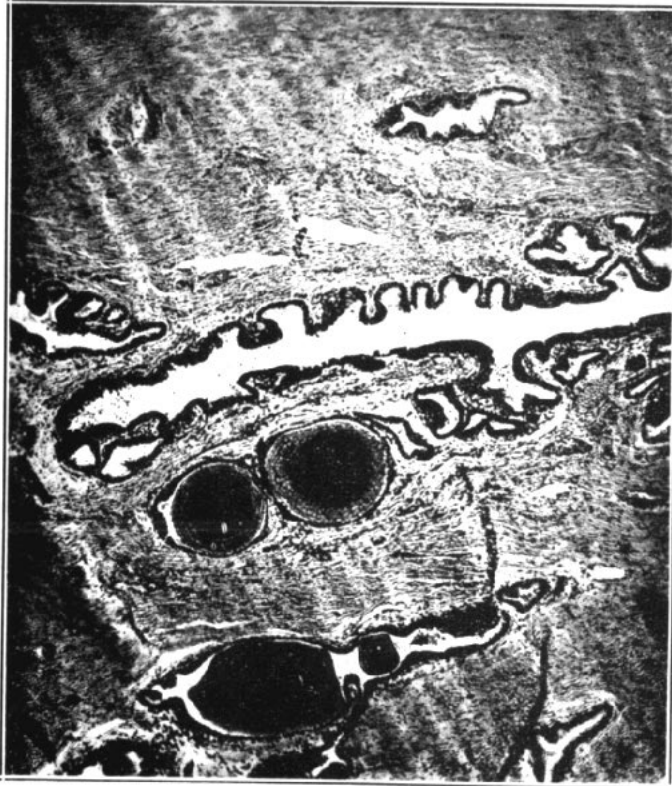


Figure 8. Hypertrophy of prostate. (Note the absence of inflammatory cells in all true hypertrophic processes.)

duced by the air passing across the mucosa is sufficiently irritating to elicit such a response.¹ This statement appears somewhat doubtful to me. Would you not expect the disturbance of occlusion, which is produced by mouth breathing, with the accompanying deviations of the interproximal spaces, to

1 Hirschfeld, Isador. Hypertrophic Gingivitis. J. A. D. A. 19:799

be of greater importance as an etiological factor than friction from the air? I hope someone will discuss this question.

We must not lose sight of the fact that certain constitutional conditions may express themselves in the oral cavity. In such cases we find the first, also the most marked symptoms, in those areas which have been subjected to local injuries. It is a well known clinical observation that mercurial stomatitis or gingivitis occurs much more readily in those areas in which the interproximal space is abnormal.

Roe,¹ also Hirschfeld,² report cases which appear to be the end result of a previous fungus infection of the oral mucosa. Roe even suggests a possible relationship between the neurofibromatosis and the yeast fungus. He further suggests that these gum hypertrophies are the result of infectious granulomata and considers the term saccharomyosis.

Pathology

True Hypertrophy: Not all of the reported cases give an accurate description of the gross pathology, not to mention a microscopic study. Holmes, Health, Whitely, Humphrey, Salter, Waterman, Hutchinson and Ruggles have described their cases so as to leave no doubt but that they were dealing with true hypertrophy and not a proliferative process. Their gross description states that the masses were hard, inelastic, not painful, semi-transparent, bled but little upon scarification or extraction of adjacent teeth and in general resembled a uterine fibroid. Due to the lack of x-ray examinations no mention was made concerning the condition of the alveolar process. In one of Salter's cases, he states that the teeth became very loose due to absorption of the alveolar process. However, there was not the slightest inflammation or pain. In the description of the treatment employed, I found only a few references to the removal of teeth and alveolar bone, along with the soft tissue. (Ruggles.) Only recently I had the opportunity of studying a case of true hypertrophy in an eleven year old girl. (Figure 4.) The greatest involvement is limited to the anterior part of the maxilla. The tissue was of normal color, firm, inelastic, not hypersensitive and showed no tendency to bleed. By a very careful x-ray examination, using direct and profile views, not the slightest change in the osseous tissue could be discovered.

1 Roe, W. J. Hypertrophy of the Gums. Dental Cosmos, 43:341.

2 Hirschfeld, Isadore. Hypertrophic Gingivitis. J. A. D. A. 19:799.

Charles Tomes described the histology of one of Heath's cases as follows. A dense stroma of interlacing fibers covered by a thin layer of mucous membrane. A true hypertrophy of fibrous tissue springing from the periosteum just within the margin of the alveoli. Waterman describes his case as purely a fibrous growth, without myeloid cells, and not likely to

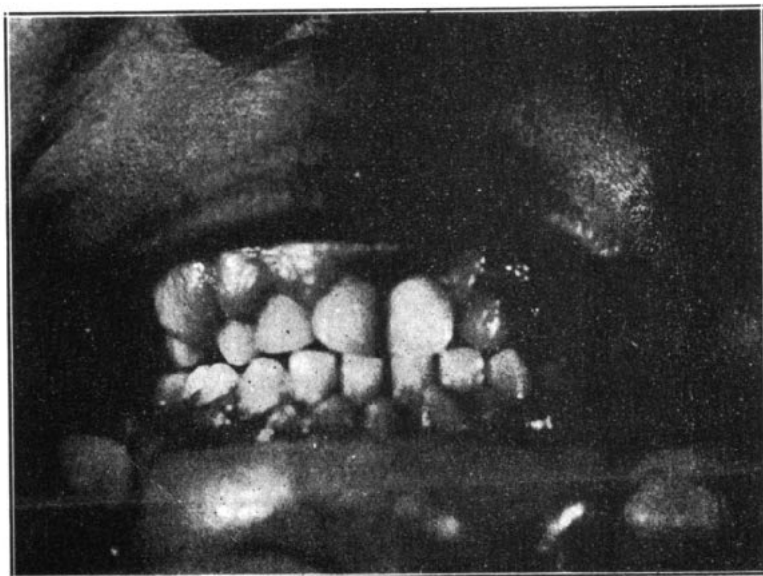


Figure 9. Condition of gingivae as case presented for treatment. (Figures 10 and 11 are sections taken of tissue removed from interproximal space between upper right first bicuspid and cuspid.)

be confounded with growth from the interdental papilla, i.e., epulis. It is evident that Tomes and Waterman recognized a clear distinction between a hypertrophy and an inflammation.

Figures 5, 6, 7 and 8 illustrate the characteristic changes which occur in hypertrophy and atrophy. The gross and microscopic findings of the true hypertrophy are in sharp contrast with the localized enlargements—polypi—of the septal gingivae.

In the latter (Figure 9) we find a tendency to hemorrhage on the slightest provocation, marked discoloration extending from a bright red to

a dark bluish red. The mass is of soft, spongy nature with varying degrees of pain. The histology agrees with the gross findings. The epithelium shows marked proliferation especially in regard to the papillary extensions. Leucocytic infiltration is marked and there may be hemorrhagic areas. The surface adjacent to the tooth, i.e., the outer boundary of the gingival crevice, may show areas of ulceration and necrosis. Figures 10 and 11 illustrate all of these changes. After studying many similar cases it becomes clear that we find very little evidence which will permit the continued use of the term "hypertrophic gingivitis" when speaking of such cases.

Cahn¹ favors the placing of such conditions within the category of neoplasms because of their tendency to return after removal; also he suggests that they be considered as precancerous. He has recommended the term "gingivoma." This is not in accord with correct terminology as the suffix "oma," meaning a morbid condition and especially referring to a tumor, is attached to the term indicating the character of tissue found in the new growth, as a fibroma, sarcoma, epithelioma, etc.

Symptoms and Diagnosis

From this hurried sketch of the pathologic processes, we see at once certain definite and rather constant symptoms.

Age: In the case of true hypertrophy, the condition is found in infants and during childhood at a time in which inflammation of the gingiva, arising from local trauma, is seldom seen.

Tendency to bleed: Areas of hypertrophy as reported by several writers may be considered as rather avascular while gingival polypii are extremely vascular and bleed upon the slightest provocation.

Consistency and Color: Hypertrophies are dense, firm, inelastic and of normal color or slightly paler than normal. Polypi are soft, boggy masses with a variety of color extending from a bright red to a reddish blue.

Pain: Areas of hypertrophy are not painful.

The areas of a proliferative gingivitis are painful, some more so than others.

Disordered Function: Most case reports of hypertrophy state that the patient presented for relief of the deformity. Some were unable to close

1. Cahn, Lester. Histopathology of Hypertrophic Gingivitis. Dental Items of Interest, 48:195.

the mouth, some experienced difficulty in swallowing, speaking or eating. I have had the opportunity to observe two cases in the college clinics. The first one, who presented a few years ago, was a colored boy about 18 years of age. The tissue over the buccal and lingual ridges of the maxilla covered approximately one half of the crowns. It was firm, unyielding, normal



Figure 10. Marked inflammatory reaction which is typical of proliferative gingivitis.

color, not painful, somewhat irregular or lobulated and not the slightest tendency to hemorrhage. In presenting this case to the students, I inadvertently mentioned about removing some of this tissue for study. The patient did not return. The second case was a young girl of eleven years who had a similar condition, more especially in the anterior region of the maxilla and there seems to be no tendency, at present, to extend incisally over the crown. Both of these cases presented for regular dental care and were not aware that such a process was taking place.

Gingival polypi cause the patient so much annoyance from bleeding and the area is so sensitive that there is no difficulty in differentiating the two. However, such is not the case in regard to giant-cell tumors as these are of a soft boggy texture, lobulated, bleed easily and may cover large portions of the crowns. These benign, giant-cell tumors most frequently are

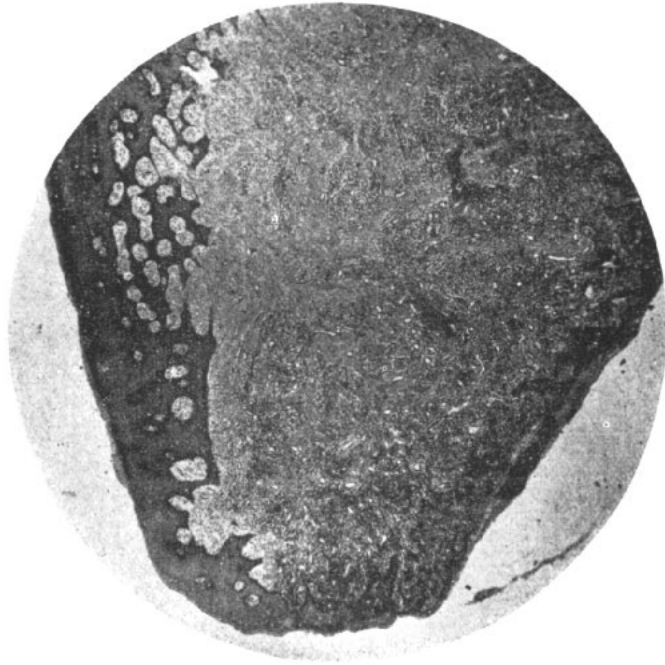


Figure 11. Showing the inflammatory reaction of proliferative gingivitis.

pedunculated, the stalk extending down to the interproximal periodontal membrane, while gingival polypi are more frequently sessile in that they are direct extensions of the gingival papillae.

Treatment: Treatment may be divided into the radical and conservative groups. Certainly, for the cases of massive hypertrophy, surgical removal is the only course open. Some of the less extensive cases may be kept under observation for a while, resorting to operative procedures when necessary.

The treatment of the proliferative cases is not so easily disposed of. Frequently the swelling of the septal gingival in neighboring interproximal spaces will produce a cleft at the point of junction. The cleft forms an

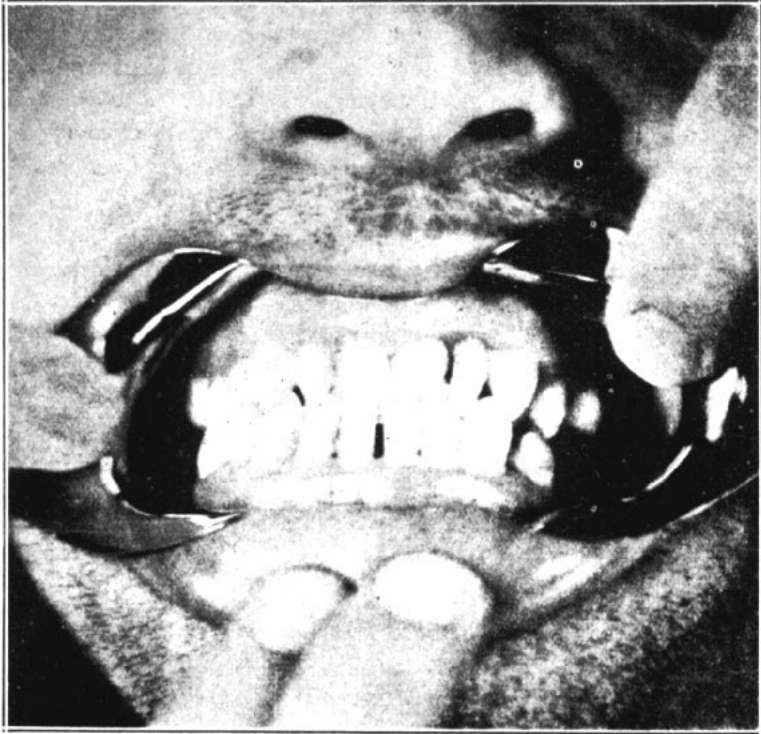


Figure 12. Marked reduction in size of gingivae after two months of treatment. (Note abnormal interproximal space and occlusion.)

excellent opportunity for the lodgment of debris and bacteria. It is not at all uncommon, in the treatment of such acute cases, to prohibit the use of the toothbrush by the patient. In so doing the dentist becomes responsible for the daily care of the tissue. As the acute symptoms subside, the care of the mouth is gradually transferred to the patient. We must take sufficient time to properly instruct the patient in regard to the selection of the proper brush and the correct manner of its use. The patient should

be made to understand that very little may be accomplished without full cooperation on his part.

All irritants, as rough margins of fillings, crowns, cavities, calculus, food debris, etc., are to be removed as quickly as possible. This may provoke profuse bleeding which is beneficial as it tends toward relieving the congestion and aids in reestablishing the circulation. The patient should be instructed to wash the mouth frequently. I have found that a warm, alkaline solution is soothing and promotes healing. A tablespoonful of lime water in a glassful of normal salt solution is excellent. Another valuable wash is a ten per cent solution of sodium perborate. As rapidly as the acute symptoms subside, the care of the mouth is left to the patient.

In chronic cases it is not as a rule necessary to take the care of the mouth out of the patient's hands. During the first sitting the patient should be very carefully instructed as to the correct technic for using the brush. I have seen remarkable results obtained in the course of one or two months. Figure 12 illustrates such a case in which the contour of the gingivae is nearly normal. I would advise against the continued use of strong astringents although such preparations are of service in cases of morbidly relaxed tissue. The best treatment is to restore the inter-proximal spaces to normal shape whenever possible. Early orthodontia treatment is a very important factor in the prevention of periodontal lesions in later life.

Conclusions

1. While I am reluctant to suggest a new term, we must clearly understand that the enlarged condition of the gums which is so frequently seen is of an inflammatory nature and not a hypertrophy. Therefore hypertrophic gingivitis is not a good term.
2. Those rare conditions in which there is a marked over-production of fibrous tissue are not limited to the gingivae but include the soft tissue coverings of the alveolar processes and the hard palate, hence the term "hypertrophy of the gums" seems to be appropriate.
3. At present, it is not possible to definitely state the etiologic factors involved in hypertrophy of the gum. However it does appear that local conditions play a secondary role.
4. Local irritations are of prime importance in the causation of gingival enlargements, which may be termed polypii.
5. The relationship between the form of the interproximal space and disease of the soft tissue which it contains has not been fully recognized.

Literature

- Anderson, B. G.—Hypertrophic Gingivitis Among Chinese.
Nat. Med. Journ. of China, 15:(1929) 453.
- Arlotta, Prof. A.—Elephantiasis of Gingivae.
Abit. J. Periodontology, July, 1932.
- Beatty and Dickson—Textbook of Pathology, 3rd Edition, 245.
C. V. Mosby and Company, St. Louis.
- Buckley, J. P.—Textbook Dental Materia Medica and Therapeutics.
Edition—Blakeston
- Cahn, Lester—Pathology of Hypertrophy of Gums.
Dental Cosmos, 66:267.
Histopathology of Hypertrophic Gingivitis.
Dental Items of Interest, 48:195.
- Evans, Henry—A Case of General Hypertrophy of the Gums Caused by Diseased Teeth.
Brit. Dent. Assn. J., 4 (1883) 506.
- Furtwaengler, A.—Zur Frage Aetiologic der Gingivitis.
Dtsch. Mensch Zhkde, 45:999
Abstract Jour. Periondontology, July 1931. 113.
- Garretson, J. E.—A System of Oral Surgery.
J. B. Lippincott Company, 1890.
- Gross—System of Surgery.
Vol. II, 1872, 503.
- Heath, Christopher—Two Cases of Hypertrophied Gums and Alveoli.
Brit. J. Dent. Sci., 22 (1879), 18-20.
- Herschfeld, Isador—The Tooth Brush. Its Use and Abuse.
Dental Items of Interest, 54:41.
Hypertrophic Gingivitis.
J. A. D. A., 19:799.
- Holmes—System of Surgery.
Vol. 4 (1864), 18-20.
- Hisey, J. W.—A Case of Excessive Hypertrophy of the Gums.
Dental Cosmos, 35 (1893), 452.
- Hutchinson, Robert—Hypertrophy of Gums.
Roy. Soc. Med. Sec. Dis. of Children, Vol. 8, pt. 1, 30-32.
- Humphrey, G. M.—Unilateral Hypertrophy of the Gums Associated with Other Abnormalities.
Ann. Surg., 3 (1886) 1-8.
- Ironside, A. E.—A Case of Hypertrophy of the Gums.
Dent. Record, 25 (1905), 461.
- Jackson, W. H.—Morbid Growth of Gums.
Dental Items of Interest, 13:(1891), 480.
- Monash, Sam—Proliferative Gingivitis of Pregnancy.
Dental Items of Interest, 48:500.
- Monier, Leon—Gingivitis Hypertrophica. Report of Two Cases.
American Dental Surg., 47:273-74.
- Read, H. G.—Hypertrophy in a Boy Four Years Old.
Brit. J. Dent. Sci., 38 (1895), 44
- Roe, W. J.—Hypertrophy of the Gums.
Dental Cosmos, 43:341.
- Ruggles, S. D.—Primary Hypertrophy of Gums.
J. A. D. A., 11:1204.
- Salter, James A.—Transparent Hypertrophy of the Gums.
Dental Cosmos, 16:91.
- Schoff, Jos.—Histopathology of Hypertrophic Gingivitis.
Dental Items of Interest, 48:574.
- Stillman, Paul R.—Histopathology of Hypertrophic Gingivitis.
Dental Items of Interest, 45:358.

- Talbot, Eugene—Interstitial Gingivitis.
The Ramson and Randolph Company, Toledo, 1913, P. 32.
- Tennis, P. C.—Hypertrophied Gum Tissue.
American Dent., Surgeon, 46:69.
- Waterman, Thos.—Hypertrophy of Gums: Partial Reaction of Superior Maxilla.
Dental Cosmos, 11:270.
- Weiss, Henri—Hypertrophy of Gums.
Brit. J. Dent. Sci, 29 (1886), 134.
- Williams, Lloyd E.—Hypertrophic Gum Tissue Attached to Palate by a Large Flat Pedicle.
Dental Record, 5 (1885) 271.
- Whitely, C.—A Case of Hypertrophied Gums.
Brit. J. Dent. Sci, 22 1879, 398-400.
- Zimmerman, Hans—Elephantiasis of Gum from Inflammatory Cause.
Dtsche. z. Wchnschr, July, 1930.
Abst. Dental Digest, 36:670.