Oral medicine

A clinical approach with basic science correlation

SECOND EDITION

口腔医学《临床与基础科学的相互关系》

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A clinical approach with basic science correlation

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Preface

For as the body is one, and hath many members, and all the members of that body, being many, are one body. . . .

This quotation from Paul aptly expresses the relationship of the oral cavity to the rest of the human body. The physical body of man is composed of parts, but it functions as a whole. Its parts are interrelated one with another and with the body as a whole. Dentistry cannot be considered separately from the other health services insofar as diagnosis and treatment of the patient are concerned. Such a separation is an oversimplification of a complex phenomenon, for in life there is no function devoid of organ integration.

Although the dentist is a specialist of the oral cavity, he must understand the patient as a biologic entity, with psychologic overlays affecting the course of disease. All the organ systems of the body play a role in the development of a clinical lesion in the mouth. The mouth serves as a mirror of systemic health and disease. Further, local lesions of the oral cavity have a direct effect on systemic health. There cannot be total health without oral health.

It is known that disease symptoms are seen by the practitioner only after the disease is well established on a cellular basis. Merely suppressing or eliminating clinical symptoms without interpreting the nature of the disease is no longer tenable for the alert diagnostician. Since the cell is the common point of origin of all diseases, reference to the basic sciences in order to explain the clinical phenomena and course of a disease becomes essential. Clinical science can advance only by delving into the basic sciences for an explanation of clinical phenomena. Comprehending the nature of a disease means not only identifying and placing a label on a group of symptoms but also understanding the underlying mechanism of the disease and its relation to the patient.

Before the clinical lesion appears, unrevealed activity on a cellular basis has taken place. For this reason, the text will stress a basic science approach to explain the clinical entity seen in the oral cavity. The hiatus between the clinical lesion and related basic science will be bridged and the material developed in a succinct manner.

The writer of a textbook of medicine or dentistry undertakes an obligation to question, evaluate, and retest the dogmas and assumptions of the past as recorded in the existing texts. It is an obligation that a new generation of professional men inherits. Too often, the printed word is regarded as the gospel, transmitted from one book to another without reexamination of the generalizations and inductions that have been promoted in the past. At the same time, one should require rigorous and unequivocal evidence before refuting the practices, beliefs, and assumptions of the past. One should adhere closely to well-documented concepts and forego any attempt to review all the ingenious laboratory and clinical investigations that are still inconclusive. The author has an obligation and responsibility to evaluate the previous works on a subject and project his own fresh viewpoint.

Existing literature on oral medicine is voluminous and not always conducive to

clarity of comprehension on the part of the clinician who reads the journals. This is perhaps to be expected in a field of biologic science where so much active research is conducted by specialists in many disciplines. Discreet selection and possibly omission of material are inevitable in a textbook on a field as vast as oral medicine. This cannot be avoided if the field of oral medicine with its related current research is to be covered in a logical, coherent, and trenchant sequence. It is the purpose of this book to attempt to reduce this profusion to the smallest possible residue of useful, practical, and sober facts.

Medicine is not an exact mathematical science. Accordingly, various opinions may explain a disease entity differently. These diversities will be carefully culled to avoid perpetuating erroneous explanations. The fables and shibboleths of the past will be identified and discarded. Where scientific data are available, they will be recounted, and where they are lacking, judicious opinion will be offered.

It is probably not possible to write a text on oral medicine without inciting some controversy. While varying opinions of others have been included, I have attempted to render my own thoughts, attitudes, and practice as clearly as possible. The lesion encountered by the dentist or physician in everyday practice will be interpreted with factual material stripped of verbosity. It will be presented against a background of a modern medical center environment and a university discipline, as it would be in a teaching hospital.

The boundless probing by research scientists has created unprecedented advances in medical and dental science during the past 10 years. These advances have created a need for a fresh approach to oral medicine. It is the purpose, then, of this text to bring up to date those advances that are enduring and meaningful and to report the technique and type of teaching that flourishes in a vigorous university teaching hospital. This should serve to sharpen the diagnostic acumen of the student or practitioner so that the digital dexterity of the operator can be better put to the test.

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When one is a member of a dynamic university-affiliated hospital team, he is in a unique position to tap expert, critical readers in other specialties. Thanks are expressed to the following colleagues at the College of Medicine, New York University, and Veterans Administration Hospital: Dr. Frank J. Lovelock (pulmonary diseases), Dr. J. C. Di John (collagen diseases), Dr. M. Dolgin (cardiovascular diseases), Dr. A. J. Marcus (hematology), Dr. H. S. Ballard (internal medicine), Dr. M. Rothchild (endocrinology), Dr. M. Goldberg (psychiatry), Dr. B. Derby (neurology), and Dr. D. P. Michaeledes (dermatology). Critical reading by Dr. William F. Harrigan, Dr. S. S. Stahl, and Dr. George Witkin, at the college of Dentistry, New York University, is gratefully acknowledged.

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My wife, Edith H. Scopp, diligently edited and proofread the manuscript. I am very grateful for her many suggestions and tireless efforts.

I. W. S.

Contents

chapter 1

Oral physiology, anatomy, and histology

To fully understand a disease entity, one must successfully bridge the gap between the basic sciences and the clinical lesion encountered in the mouth. This statement does not imply that the clinician or student should memorize part of a textbook on anatomy. Facts can be memorized fairly easily, but they may be as quickly forgotten. Anatomy texts are available as reference. Preferably, one should be conversant with the principles and methods rather than submit himself to the drudgery of memorizing all the advances since Andreas Vesalius. The scientific method and way of thought should be developed and used.

Oral physiology, anatomy, and histology provide a basis for an understanding of the normal individual in terms of bodily structure and function on a macroscopic and ultramicroscopic level. Through pathology and microbiology, alterations of structure and function resulting from disease are better understood. The diagnostic acumen of the clinician will be improved if he can develop the ability to evaluate, select, organize, and apply this information.

The clinician should have a working knowledge of the anatomic terms of structures and functions of organs and tissues in health and in disease. Without this knowledge, he would be unable to think, communicate, and reason soundly when confronted with a clinical problem. With a scientific understanding of anatomy, histology, biochemistry, and physiology, one can better cope with the pathology, diagnosis, etiology, therapy, and prognosis of oral disease.

The clinician should understand cell structure, too, more on a physiochemical basis rather than only on a morphologic basis. It is evident that *changes in structure are followed by changes in function.* Examination of cellular structure with a microscope (bright-field, dark-field, and phase-contrast) reveals a homogenous jelly. However, using more refined methods of investigation, one can see that the nucleus contains nucleoli, chromosomes, and the chromatin of resting cells. The cytoplasm contains mitochondria (power stations), centrioles (governing movement and mitosis), ribosomes ("assembly lines"), Golgi apparatus, endoplasmic reticulum, and so on. The roles of these cellular elements are gradually being exposed through the development of autoradiography, electron microscopy, spectrographic methods of analysis, cytochemical procedures, and radiation detections.

Scientists are currently making advances with the electron microscope in addition to the light microscope. Only through the dynamics of the basic sciences, properly understood and diligently applied, can one obtain a better understanding of the disease processes he may encounter in the oral cavity.

Anatomically, the oral cavity contains all the basic tissue elements found anywhere in the body. There are at least 30 different disciplines of basic science involved in

dental research. A few examples follow:

1. Immunologists intimately involved in the developing fields of immunity relating to oral disease, tumor biology, and aging and currently studying the implications of the immune response regarding antibody diversity, immunologic tolerance versus immunity for carcinogenesis, and transplantation problems

   They have sound evidence that antibodies have pathogenic potential and may be significant in the etiology of some forms of periodontal disease and recurrent oral ulcerations. The autoimmune etiology of oral disease can open the door to more effective methods of therapy.
2. Microbiologists studying the sticky extracellular polysaccharide coating of streptococci and dextran formation, intimately involved in caries and periodontal disease

   The mechanism of plaque formation and its control is being studied. There is experimental evidence to identify the water-soluble polysaccharides as dextran and levan.
3. Marine biologists working on cement-producing glands of barnacles from Puget Sound, centrifuging and distilling the cement since a lipid ingredient has been found that can serve as an adhesive dental filling
4. Biochemists developing the cross-linkage of collagen, an important ingredient of the oral mucosa
5. Metallurgists investigating an alloy of copper, nickel, and manganese that would greatly improve dentures
6. Crystallographers examining the structure of apatite crystals
7. Chemists working on enzymes, antibacterial agents, and fluorides that inhibit caries by strengthening enamel crystalline structure

The ultimate aim of these researchers, then, is to synthesize their knowledge in basic sciences and to apply it to the understanding of the clinical lesion, the prevention of oral disease, and the improving of therapeutic techniques.

DEPOSITS ON TEETH

Deposits on teeth are normally formed around all teeth in all people. These deposits are calculus (both supragingival and subgingival), materia alba, dental plaque, and organic deposits such as stained embryonal remnants and protein pellicle. The quantity and quality of the deposit vary in individuals. Despite the normality and universality of this process, deposits on teeth, particularly the dental plaque, give rise to caries and inflammation of the soft and hard tissues supporting the teeth, thus causing gingival and periodontal disease. These deposit-oriented diseases are the most frequent cause of pain and loss of teeth in the oral cavity.

Calculus

Calculus (from Latin, meaning "stone") results from calcification of bacterial and fungus accumulations that have become attached to the tooth surface (Fig. 1-1). Initiation of calculus formation is dependent upon the formation of dental plaque. It consists of mineral salts precipitated from the saliva and embedded in a bacterial and fungus matrix. These mineral salts are composed of calcium phosphate in the form of hydroxyapatite and lesser amounts of whitlockite. The microorganisms primarily involved are the filamentous higher bacterial types, the streptococci, the leptothrixes and bacterionemes, and the actinomycetes, although all bacteria in the oral cavity are participants.

Three factors assist these organisms in their role. First, they are capable of attaching themselves to the enamel or cementum. Second, their filamentous form permits them to construct a mesh, or network, that holds deposits together. Third, the filamentous organisms form crystals of tricalcium phosphate within themselves and so participate in the crystal formation. Almost all microorganisms contribute in some way to the formation of calculus by changing the local biochemical environment. Other debris such as desquamated epithelial cells, leukocytes, and food debris become incorporated. Any factor that aids plaque formation helps form calculus.

Supragingival and subgingival calculi have approximately the same composition; but the former is derived from the saliva, while the latter may be derived from the sulcular fluid. Supragingival calculus is found in both children and adults, whereas

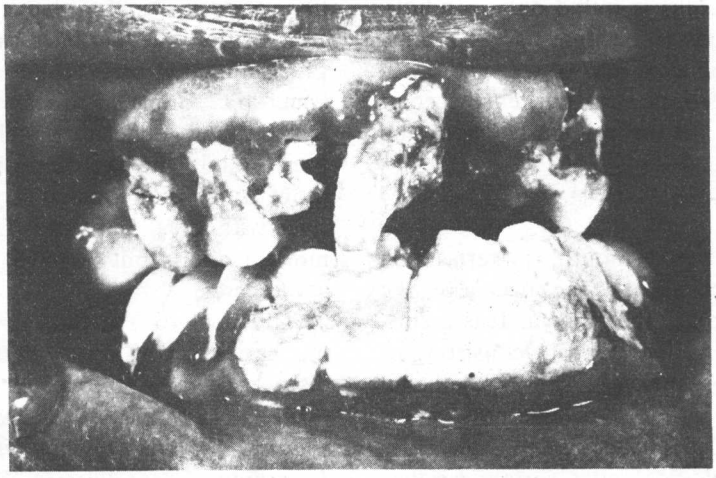

Fig. 1-1. Large formation of calculus in an unkempt mouth. A 28-year-old man was treated in the hospital for a lung abscess after having inhaled a fragment of calculus.

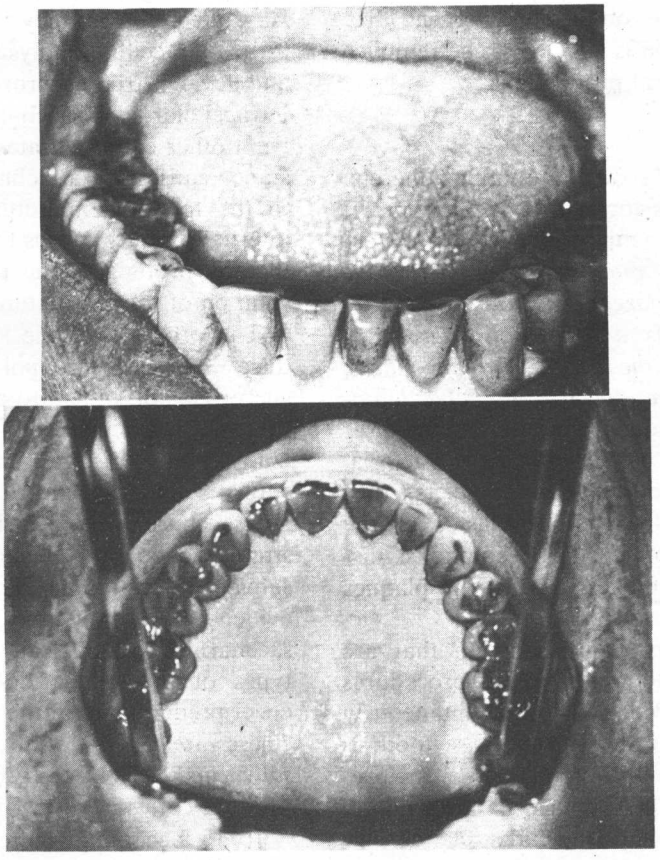

Fig. 1-2. Deposits, food, and tobacco stains around teeth. There is abrasion of the teeth from chewing and bruxism.

subgingival calculus is found only in adults. All calculus tends to become darker after it has formed. Tobacco, tea, coffee, and chromatogenic microorganisms darken the color of calculus to dark brown, dark green, and even black (Fig. 1-2).

Materia alba

In an unhygienic mouth, materia alba tends to collect around the necks of the teeth and to irritate the gingiva. It is a soft, cream-white or yellow deposit, consisting of mycotic microorganisms, bacteria, epithelial cells, and food debris. The microorganisms may lower the pH, causing the underlying enamel to decalcify. Under certain circumstances, teeth are particularly prone to the accumulation of materia alba. These are: (1) teeth that are not in occlusion so that there is an absence of function, (2) teeth that are poorly brushed, and (3) teeth of patients who are on a nondetergent diet. Since materia alba is soft, it can be removed readily with an oral prophylaxis.

Dental plaques

Dental plaques are gummy, adherent, bacterial deposits formed on teeth from the saliva and the microorganisms. *Mature plaques consist mainly of gram-positive filamentous microorganisms embedded in an amorphous matrix and a small quantity of cellular and organic debris. On the plaque surface there are cocci, rods, and leptothrixes.* The plaques may be colorless or slightly stained by chromogenic bacteria, tea, coffee, or other food products, thus imparting a dullness or lackluster to the teeth. The use of disclosing solutions, disclosing wafers, or dyes exposes the plaques by staining them.

Plaque appears on those teeth that are least accessible to oral hygiene procedures and, when removed, develops spontaneously once again. After the surface of a tooth is thoroughly cleaned, the pellicle, a structureless layer, forms before bacteria are evident. The bacteria found in early plaque are cocci; but as the plaque develops, other bacterial forms, both filamentous and rod-like, appear. As the plaque progresses, the composition of the bacteria changes from predominantly aerobic to predominantly anaerobic. Some plaques are transformed from a soft deposit to calculus, which is hard. While the soft deposit is easy to remove by brushing, calculus can be removed only with great difficulty. Calculus is common in adults but is infrequently found in adolescents.

Attached to the tooth, plaques trigger the caries process and initiate periodontal disease. *The streptococci in the plaques form extracellular polysaccharides from sucrose, which may be involved in the formation of dental caries.* When the plaques are located near the gingiva, they cause irritation and inflammation, resulting in gingivitis and periodontitis.

Specific anaerobic streptococci are capable of producing acids that cause dental caries. As stated above, they have the ability to form extracellular polysaccharides (dextran and levan) from sucrose and, in addition, intracellular polysaccharide (amylopectin) from other carbohydrates. The external substance causes the attachment of the bacteria to the tooth as microbial plaque; and the internal product causes fermentation to acid, thereby prolonging the process of acid dissolution of tooth structure.

The property of the streptococci to produce extracellular polysaccharides from sucrose enables them to adhere together and to form large colonies. In this manner, a plaque matrix is built up. The main constituents of this matrix are extracellular polysaccharides, which have been demonstrated to contain glucan and fructan, and salivary glycoproteins. There is experimental evidence to identify the water-soluble polysaccharides as dextran and levan. Three types of polysaccharide-producing streptococci predominate in the oral cavity of man. These are *S. mutans, S. sanguis,* and *S. salivarius.* All three types of streptococci produce caries in germ-free rats.

It is becoming increasingly clear that plaques vary in their pathogenic effects, chemical composition, and metabolic activ-

ities. Clinically, some plaques are associated with periodontal disease, others with caries, whereas still others form calculus. One consistent characteristic of all plaques is adhesion to the tooth surface. Research on the mechanism of adhesion should yield information that will help us to cope with periodontal disease and dental caries.

ODOR OR HALITOSIS

Depending upon a number of factors, odors from patients' breath will vary considerably. Efforts to eliminate halitosis should be directed toward determining the etiologic factors and the correction of these factors. *Use of a mouthwash is palliative and transient only and does not eliminate many of the odors.* Listed below are the causes of odors classified as local factors, systemic factors, and ingestion of food products.

Local factors

Putrefaction of food remaining between teeth
Dental caries
Periodontal disease with pocket formation
Necrotizing ulcerative gingivitis
Postnasal discharge
Excessive smoking
Deposits on teeth
Faulty dental restorations that trap food, especially under crowns and bridges
Bacterial activity without satisfactory flushing action of the saliva
Unclean dentures

Systemic factors

Diabetes
Internal hemorrhage
Necrosis
Kidney dysfunction
Gastrointestinal disease
Hepatic failure
Lung pathology

Ingestion of food products

The ingestion of certain food products such as garlic, onions, or peppermints, although they are past the oral cavity, will give the breath a foul odor, even hours later. Also, those patients who have had an excessively fat meal develop halitosis, resulting from the improper digestion of fats. This is particularly true when large quantities of milk or milk products are consumed.

Odors as an aid in diagnosis

The characteristic acetone breath odor of diabetes is associated with abnormal metabolism of foods. Kidney dysfunction diseases may generate an odor of urine. Tonsillitis and nasopharyngeal inflammation produce the odor of sour cheese. Gangrene or necrosis gives off a foul odor, as does gastrointestinal disease. There are foul odors associated with many of the infections of the oral cavity, such as cancrum oris. Granulomas or abscesses draining in the oral cavity will create a fetid odor of suppuration. Patients with a fever develop a mouth odor that is associated with the decrease in salivation and diminished action of the tongue in flushing the bacterial products from the oral cavity. Patients with hepatic failure develop a fishy odor. Those patients suffering from uremia will have a urinary or ammoniac odor. Any infection of the respiratory tract will produce a malodorous breath. The alert practitioner should consider these distinguishing mouth odors as a guide in diagnosis.

TASTE

Taste is transmitted through a rich plexus of nerves via taste buds located on the tongue. The taste buds reach the surface of the tongue through taste pores. These pores are located on the fungiform papillae, at the tip and the lateral borders of the tongue, surrounding the vallate papillae of the tongue, in the folds of the foliate papillae of the tongue, and on the epiglottis. They are thin, barrel-shaped, dark-staining cells approximately 80μ in length (Figs. 1-3 and 1-4).

Four basic types of taste sensations are recognized—sweet, salty, sour, and bitter. *Sweet taste* is experienced at the tip of the tongue and is transmitted through the taste buds to the brain by the intermediofacial nerve and the chorda tympani. *Salty taste* is picked up by the taste buds located at the lateral border of the tongue and then follows

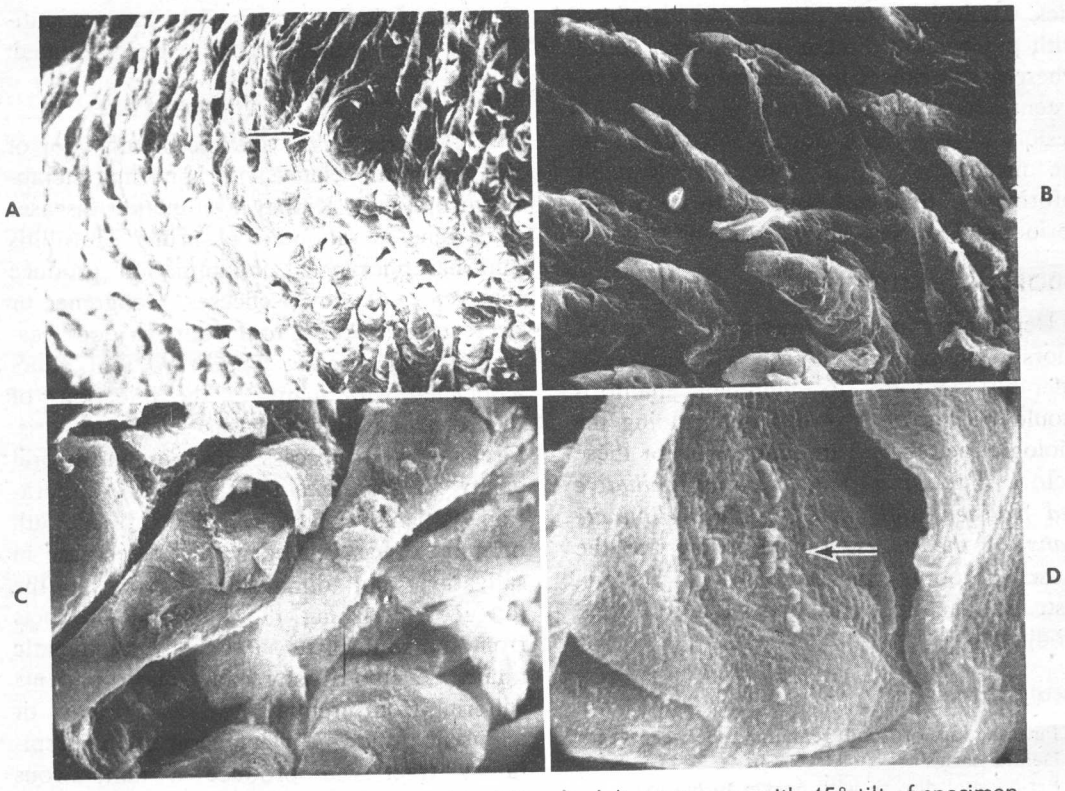

Fig. 1-3. Scanning electronmicrographs of tip of adult rat tongue with 45° tilt of specimen. **A,** 200X magnification shows filiform papillae of tongue, which are avascular and covered with keratinized epithelium. A fungiform papilla with taste pore is indicated by the arrow. **B,** 500X magnification demonstrates filiform papillae. **C,** 1,100X magnification shows debris on filiform papillae. **D,** 5,000X magnification reveals microorganisms among filiform papillae. (Courtesy Dr. Philip Person, V.A. Hospital, Brooklyn, New York, Dr. D. E. Philpott and Mr. C. Turnhill, Ames Research Center, NASA, Moffett, California.)

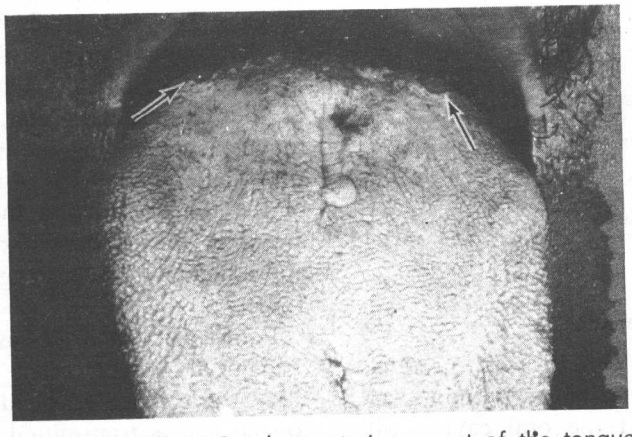

Fig. 1-4. Circumvallate papillae. On the posterior aspect of the tongue are the circumvallate papillae, 8 to 12 in number, arranged in the form of a V. The filiform papillae are tiny conical epithelial projections covered with keratin and appear over the entire dorsum of the tongue. The fungiform papillae, fewer in number, are scattered among the filiform papillae and are red because the thin layer of epithelium does not mask the capillary bed.

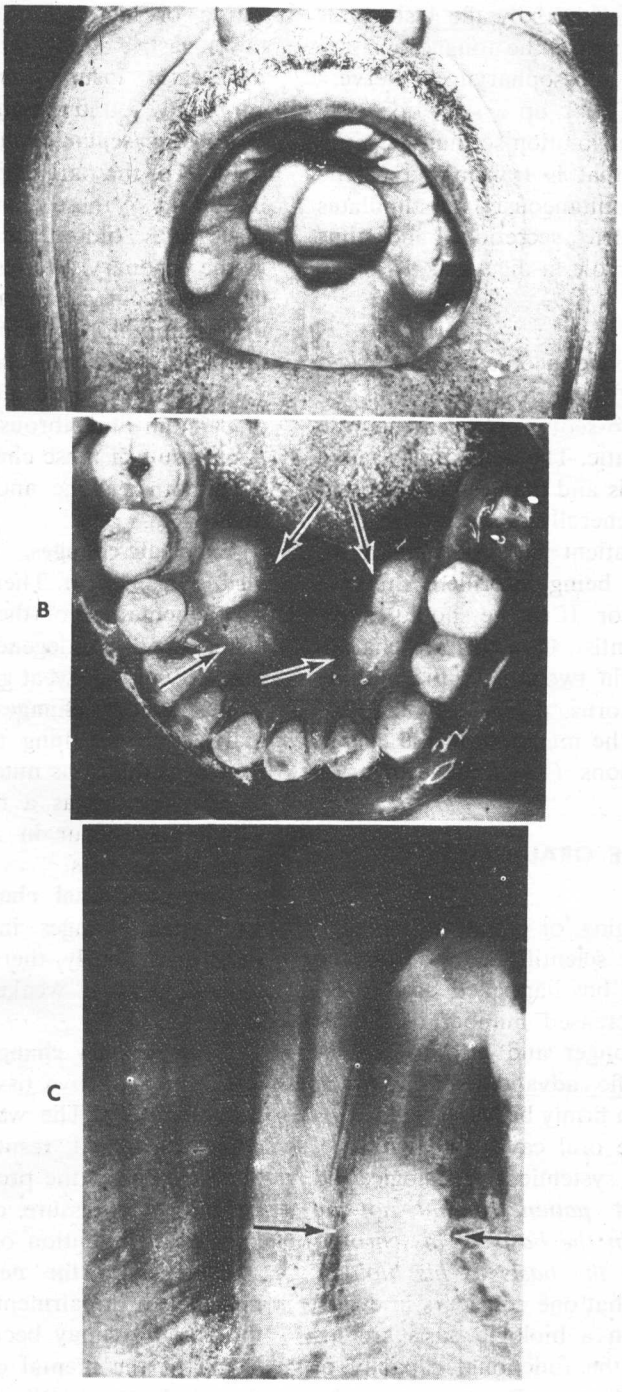

Fig. 1-5. A, Torus palatinus. Lobulated overgrowth of bone located at the midline of the palate is found in about 20% of the population. **B,** Torus mandibularis. Multiple lobulations of bone are found in the mandible in about 8% of the population. Neither the torus palatinus nor the torus mandibularis should be removed unless, as in rare instances, they interfere with the construction of a denture. **C,** Roentgenogram of a torus mandibularis. Tori appear as radiopaque areas and should not be mistaken for a pathologic condition.

the same path as the sweet taste. *Sour* and *bitter tastes* are received by the taste buds at the posterior part of the tongue and are transferred to the glossopharyngeal nerve.

Taste buds will pick up a sensation only when material is in solution so that it sets up a nerve impulse that is transmitted to the brain. Almost simultaneously, it stimulates salivary and gastric secretions and thus plays a significant role in digestion.

ORAL TORI

Oral tori are nonneoplastic, localized exostoses, or bony overgrowths, not related to stress. These osseous enlargements are always asymptomatic. The dentist may make the initial diagnosis and then tell the patient, who has been generally unaware of their existence. The patient may become a bit overanxious after being informed, thinking that he has a tumor. If so, he should be reassured by the dentist. Oral tori are characteristically found in two areas: the midline of the palate (torus palatinus) and the lingual aspect of the mandible in the cuspid and bicuspid regions (torus mandibularis) (Fig. 1-5).

CHANGES IN THE ORAL CAVITY IN AGING

The study of aging, or geriatrics, has progressed to a more scientific level during the past decade. This has happened both in response to the increased number of people who are living longer and also to the increase in scientific advances upon which geriatrics has been firmly based.

Changes in the oral cavity in aging are, in part, related to systemic, psychologic, and local changes. *A patient should not be judged as aged on the basis of his chronologic age but on the basis of his biologic age.* The factors that one considers in evaluating a patient on a biologic basis are his mental capacity, the functional capacity of various organs, his responses to stress, and his appearance.

Systemic changes in aging

Bone changes. The bone becomes atrophied, and a greater degree of osteoporosis occurs. This may be the result of poor utilization of ingested calcium resulting from lower gastric acidity or possible hepatic and pancreatic insufficiency. Osteoarthritis is commonly found among the aged.

Cardiovascular changes. There is a gradual loss of the parenchymal tissue. The most important of these changes are those taking place in the blood vessels and, in particular, in the coronary arteries. The atherosclerotic changes occurring in the blood vessels result in a diminution of the blood supply to the myocardium. There may be some degree of valvular sclerosis resulting from the deposit of calcium and fibrous tissue in the valves. As a result of these changes, the blood pressure, both systolic and diastolic, is usually high.

External changes. Aging is manifested visibly in the skin. There may be an increase in pigmentation of the skin, a decrease of water, and a degeneration and loss of elasticity of the sweat glands.

Respiratory changes. It has been estimated that the lung potential in the aged may be reduced as much as 25% as a result of fibrosis and as a result of emphysema, which may occur in a high percentage of geriatric patients.

Gastrointestinal changes. There are degenerative changes in the gastrointestinal tract; particularly, there is an atrophy of the mucosa and a weakening of the muscle layer.

Genitourinary changes. As a result of increased connective tissue, the kidneys have less efficiency. The wall of the bladder becomes atrophied, resulting in cystitis. Also, hypertrophy of the prostate may occur and, because of pressure on the urinary tract, may cause retention of urinary products.

Changes in the nervous system. There may be an impairment of the memory, and the emotions may become extremely labile. Many of the mental changes that occur in the aged may be the result of cerebral arteriosclerosis or the result of minor multiple small thrombi that have occurred in the past, resulting in impairment of the memory.

Further systemic changes of the tissues in aging are gradual tissue degeneration, re-