Surgical pathology of the diseases of the mouth and jaws / by Arthur E. Hertzler.

HERTZLER'S MONOGRAPHS ON SURGICAL PATHOLOGY

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SURGICAL PATHOLOGY OF THE SKIN, BLOODVESSELS, MUSCLES AND NERVES
SURGICAL PATHOLOGY OF THE GENITO-URINARY ORGANS
SURGICAL PATHOLOGY OF THE FEMALE GENERATIVE ORGANS
SURGICAL PATHOLOGY OF THE MAMMARY GLAND
SURGICAL PATHOLOGY OF THE PERITONEUM
SURGICAL PATHOLOGY OF THE GASTRO-INTESTINAL TRACT
SURGICAL PATHOLOGY OF THE THYROID GLAND
SURGICAL PATHOLOGY OF THE DISEASES OF THE NECK
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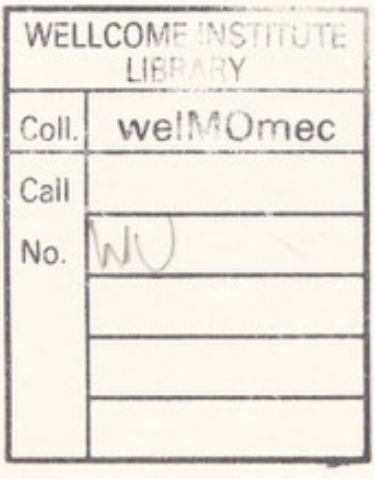

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PREFACE

WITH this volume my little series of ten volumes on surgical pathology is ended. It has been an arduous but withal a fascinating task. My patients have told me what to write and I have put it down to the best of my ability, slowly, painfully, in the wee hours of the morning.

This volume has had its particular handicaps, two of them. In the first place this field has moved away from me, a country doctor, to the specialists in the upper respiratory passages and the dentists. The result has been that I have seen fewer patients. Even so there are occasions when the general surgeon must be thoroughly at home in the entire field because there are conditions when the specialists become timid and call for the general surgeon. In the second place the mouth and upper respiratory passages offer special difficulties for the photographer. It has been impossible to secure photographs of many important and interesting lesions which have come to me.

I have always found it impossible to write of things that I have not seen. Therefore many of the things described in other books, I have not mentioned because I have not seen them. I have traveled a long ways so that I conclude that what I have not seen must be rare and it is fair to hope that likely the majority of surgeons will live a lifetime without seeing them.

It is difficult to collect a literature after a book has been written. One has something of the feeling one has when he pays income tax on money long ago expended. I have always held the idea that one had no right to quote literature which he had not read in the original. I have been sorely tempted, in this final round, to go into the market and buy citations at four cents a piece, the current rate. However I have adopted the more respected practice of having my secretary copy them out of the Cumulative Index. Therefore if the papers cited are not good, do not blame me, I have not read them.

It is a pleasant task at the end to pay tribute to my secretary, Ruth Rose. She has the uncanny faculty of reading my wriggly notes and keeping things together. In truth I believe if she saw a *Spirochaete* under the microscope she could type off a rounded sentence without a moment's hesitation. I need not pay tribute to my photographer for twenty-seven years, the incomparable Jim Barlow. The photographs, both gross and microscopic, have been complimented by reviewers the world over, and

reviewers are sometimes right. Without Jim there could not have been any books. It would have been impossible for any patients to tell their story.

So after thirty-five years of writing I shall trade my pen for a lollipop. But one can never tell, habit is a terrible thing. Geriatrists advise against trying to break the aged of their long cherished habits.

A. E. H.

November 28, 1938

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CHAPTER I

General Consideration of Surgical Affections of the Mouth and Jaws

THE surgical affections of the mouth and its accessory cavities present a complicated picture not only because of the multiplicity of the diseases which occur here, but even more so because many of the lesions are modified by the constant presence of moisture complicated and abetted by bacteria and digestive fluids. No other region of the body has these complicating factors in equal degree. Because of these associated factors we are constantly confronted with the necessity of differentiating between the two or estimating the influence that one has on the other. Lesions which would rapidly heal, if not so complicated, remain as chronic lesions possibly ultimately to develop malignant ulcers. It is the presence of inflammation, benign ulcer, and a malignant lesion, which make the diagnostic problems particularly difficult.

In including in one volume the diseases of the nasopharynx along with those of the oral cavity one commits a questionable act. This must be explained by the fact that the general surgeon is unappreciative of the niceties of the limitations of the specialists. No doubt the laryngologists are more closely related to the general surgeon than are the dentists; that is the former have the more surgical tools.

Be this as it may, the surgeon's relation to the lesions of the oral cavity and the nasopharynx has changed materially in the last several decades. Time was that when artery forceps were required in an operation the job was turned over to the general surgeon. Dentistry has now advanced from a tooth pulling and cavity filling vocation to a surgical specialty, which is as it should be. The result is that many of the infections and minor lesions, which formerly came to the surgeon, are now managed by them. Some even manage the major operation, so that they have really become surgeons with the advantage of special training of the dentist.

The laryngologists have approached even more nearly the operative achievements of the general surgeon. Nowadays most laryngologists have their own artery forceps and perform all major operations connected with the respiratory apparatus.

This means that the general surgeon's activities in these fields have become restricted. His activities are now largely limited to unusual con-

ditions, in which the diagnosis is obscure and the technic extra hazardous, and in consequence the prognosis bad. Lesions in this region are a plenty which belong to neither of the specialists above mentioned and rightfully belong to the general surgeon. In some cases, of course, nobody knows what it is all about. Therefore, a collaboration of the several specialists combining the detailed knowledge of the lesions of this region is required, which may demand the experience of the general surgeon in major operative procedures. That the technic of the general surgeon is always perfect must here, of course, be assumed.

In addition to the natural history of the disease incident to these organs the picture is often confused by preceding irradiation or other treatment, with or without accurate diagnosis, leading perhaps to a temporary disappearance of the lesion, so that what the surgeon finally comes to see is a combination of disease and treatment, of original disease, and recurrence.

It is obvious, therefore, that the general surgeon does well if he acquaints himself with the diseases of this region, so that he may at any time be prepared to combat diseases that come to him, and also, it may be added, that he may know when a given lesion had best be referred to the dentist or the laryngologist.

When a lesion of the mouth is approached by the general surgeon the fundamental question in most cases is to decide whether it is inflammatory or neoplastic in nature; whether primary, recurrent or metastatic.

In approaching a lesion of the mouth and the accessory cavities it is usually well to interrogate the neck. If an inflammatory or a metastatic lesion is found here one can with accuracy proceed to the examination of the region where the primary lesion must lie. Transmission of either or both of these diseases, by way of the lymphatics, to the neck frequently produces lesions so complicated that it is difficult to say which is primary and which secondary, as the primary may be overlooked and attention fixed on the secondary lesion. For instance, a small cancer in the mouth may produce a metastasis in the neck, but this may be masked by a huge inflammatory induration. The discovery of the mouth lesion may save an ill-advised incision in the neck induration.

In the volume on the diseases of the neck it was noted that it is necessary constantly to refer to the oral cavity as sources of secondary disease of the neck, which may closely resemble primary lesions of that region. In the studies of the diseases of the oral cavity the neck must be considered just as the mouth had to be kept in mind in the study of neck affections. In the surgical sense the two regions are inseparable. It is the broad knowledge of the diseases of the neck, which every surgeon possesses,

which enables him at once to properly evaluate the various phenomena which the disease presents.

Since the greatest aid in diagnosis of any disease is to be able to say what a lesion might be, that is to say what diseases occur in any region, it seems worthwhile in the beginning to take a bird's-eye view of the whole field in anticipation of a more extended discussion of each in the chapters which are to follow. In doing so the domain of the dentist and the laryngologist are best considered separately, the mouth and jaws as a distinct field from the upper respiratory tract.

In such a presentation the tongue and floor of the mouth remain in large measure neutral ground to which the general surgeon is welcomed because it belongs to neither of the other specialties. Formerly this region was claimed by the surgeon but recent tendencies favor the radiologist, a tendency entirely satisfactory to operators. As is usually the case when other specialists reach the end of their rope the radiologist, and the neurologist, are welcomed.

CLASSIFICATION OF THE DISEASES OF THE ORAL CAVITY

It is advantageous to classify disease as to cause if possible, which it seldom is, at least until the diagnosis has been made. Therefore the clinical features of the disease must serve as a basis for classification in most cases.

In determining the clinical diagnosis of the diseases of the mouth as above implied, many factors must be considered not necessary in most clinical problems. The topography often furnishes our first clue. What disease may occur in a certain region must first be considered. In practical surgery when the objective findings are obscure one asks what lesions are commonest in the affected region, for obviously if one has reduced the probabilities to a small number one naturally fixes on the more common until the exact diagnosis can finally be determined. We should not overlook the fact that here, as elsewhere, the surgeon rarely advances beyond extreme probabilities in his diagnosis when he begins his operation. He must recognize this fact for sooner or later, as he proceeds, the truth will out and he must be agile enough to anticipate this event. He buries his blunders in most instances only after the facts have intruded themselves into the picture.

After separating the lesions one from the other, according to their clinical manifestations and topography, one must consider associated conditions affecting the lesion. As above noted, one always considers how or if reactive forces, due to infection or irritation, are affecting the original lesion. What the influence of previous treatment may have had on the

lesion must also be considered. In lesions so exposed as the diseases of the mouth there is a temptation for the doctor to dope them with a variety of chemical substance. One might assume such treatment would be revealed in the history. If such treatment has been applied by some irregular practitioner, the patient may deny any treatment because of mortification at having allowed himself to be so treated. More often the patient is ignorant as to what treatment has been employed, even if he has been told.

Because of the complicating factors it is well first to determine one's diagnosis by a study of the objective findings and then make such use of what he learns from the history as may fit in his picture. What he sees and feels must be his guide, rather than what he has been told.

The clinical nature of the disease is often influenced by treatment that has been employed. It has seemed to me that epithelial malignancies grew particularly rapidly while the patient was being vigorously treated under a mistaken diagnosis. This is particularly so, I am sure, when mercurials are used to combat an imaginary syphilis. This is particularly so when epithelioma of the gums is treated as syphilis. The progress of the disease before the time of observation may, therefore, be complicated quite apart from the age and constitution of the patient, which naturally all clinicians understand. In final summary, the conclusion is based chiefly on the clinical experience of the surgeon. Here, as in dermatology, one learns to see only by seeing.

Having determined the nature of the lesion we search to see if we can determine what may have been its cause. The pathologist finds his chief satisfaction in learning the causation of a disease. This determined he has fulfilled the requirement of his specialty. The surgeon must frequently supplement such knowledge with local considerations. For instance, tuberculosis of the fauces or cheek has quite a different import than when the disease is located on the tongue. The same is true of syphilis and epithelial malignancies. Form and location are important after the disease has been correctly named.

In some cases the diagnosis is but little more than a name, notably when the diagnosis is made from a frozen section. It becomes useful only when supplemented by information the surgeon must supply for himself. For instance, the pathologist diagnoses an epulis which he can do with exactitude. He may consider the disease in the abstract, without reference to the variation of lesion dependent on its location. In fact he has little opportunity to study the disease, as it is influenced by variation in location. The surgeon, on the other hand, deals with lesions one at a time and must pay attention to influences in which topography plays a part. One

of these lesions in one location may be something wholly different when encountered in another location, matters not revealed by the microscope.

Taking it by and large, the diseases of the mouth and jaws may be divided into two groups: the inflammations and the tumors. If the surgeon can make this division with certainty he has gone a long way toward making a diagnosis. If a tumor is uncomplicated this may be easy. Each of these may take on unusual forms because of the situation in which we find them, which requires detailed consideration. These details become the problem of the several chapters to follow.

For purposes of simplicity we may consider separately the inflammations and the tumors. The inflammations may be classed as follows:

I. Inflammations of the Oral Cavity
   - Inflammations of the Mucosa
   - Submucosal Infections

II. Specific Ulcers of the Mucosa
   - Tuberculosis
   - Syphilis

III. Inflammation of the Bones
   - Local Infections
   - Bone Necrosis
   - Osteomyelitis of the Jaw

INFLAMMATIONS OF THE ORAL CAVITY

Under the general heading of inflammations of the oral cavity may be included all lesions attended by reaction, whether they be attended by loss of surface or not. Lesions of accessory structure must be included because of the part they play in producing or simulating inflammations within the mouth.

Inflammations of the Mucosa. Inflammations of the mucosa may attend specific lesions such as tonsillitis and diphtheria and most of the exanthematous, and in many other infectious diseases. The nature of the infection may be determined by the diagnosis of the primary disease. Specific infections, as Vincent's infection, the early stages of tuberculosis and syphilis may present first a diffuse inflammation. Here the causative factor is first revealed by microscopic examination. The mouth involvement in digestive disturbance may here be ignored. Inflammations of the mouth in the old days were usually caused by the use of calomel and this possibility was the first to enter the doctor's mind. Even so one occasionally encounters such cases because other mercurials may offend the mucosa.

Infections of the Submucosa. Infections of the submucosa are usually the result of some trauma. Some foreign body taken into the mouth may produce this. A sialolith may transmit the infection or a broken tooth or a defective denture may furnish the point of entrance of the bacteria. The characteristic feature is the limitation of the area of invasion and the obvious submucosal edema.

SPECIFIC INFECTIONS OF THE MUCOSA

The first question to be answered, when confronted by an ulcerous lesion in the mouth, is: Is the ulcerous lesion a neoplasm, a specific infection or an inflammation? Von Bergmann once said in his thunderous tones: "There are but three common diseases in the mouth: tuberculosis, cancer and syphilis. In comparison to these all others are rarities." Like most trite sayings this has but limited application. In this short grass country both syphilis and tuberculosis are rarities, rarity of the first being due to high moral standards, the other to the high general state of nutrition.

By all odds the common ulcers in these parts are traumatic. One of the most common antecedents of ulcers of the tongue and cheeks is irritation caused by a broken tooth, and of the gums due to pressure of a plate. Usually the relation of ulcer to trauma is at once obvious by simple inspection. The problem is complicated by the fact that the most common precursor of malignancy is just such an ulcer. History avails but little, for at most instances it only records the periods of its recognition by the patient, and this may be wholly misleading. It suffices here to say that these ulcers quite commonly result in malignancy and the recognition of the borderline must reside in the touch of the surgeon's finger. A simple ulcer has sharply defined borders without infiltration while the edge of malignancies has the cancer hardness. The attempt to solve this problem by biopsy has resulted in many notable errors. The surgeon had better depend on his sense of touch.

Tuberculosis. Tuberculous ulcers are usually located on the tongue, larynx, and fauces. The borders of the ulcers are soft, undermined with a dirty gray color. This may be confusing when marked induration, even to tumor hardness if there is marked inflammatory induration. Here the constitution of the patient is worthy of consideration but it may be very misleading. Most tuberculous ulcers with marked induration have behind them a lesion of the lung. Conversely a lesion of the tongue may be the first intimation of the presence of a lung lesion. On the contrary tuberculous ulcer of the fauces and buccal mucosa usually indicate a preceding state of malnutrition.

Syphilis. Tertiary luetic ulcers are most commonly found in the fauces, less commonly on the tongue. They have the common bean shape, the border is undermined and they develop acutely, commonly with the history of a preceding nodule, the gumma. Here also the patient must be diagnosed. Acute lesions may be diagnosed with the microscope.

INFLAMMATIONS OF BONE

Inflammations of bone fortunately usually involve only localized processes incident to the infected tooth. This may result in an abscess in the soft parts which obscures the bone lesion. Local destruction of bone is less common and represents an extension of infection from a tooth of considerable extent and of some duration. Necrosis of a large part of the bone is expressive of an acute osteomyelitis and may affect either jaw. Fortunately, this is very rare.

Local Infections. When a tooth becomes infected, the infection finds a way about the socket and, extending toward the surface, produces the familiar gum boil. This is usually not attended by macroscopic bone destruction and subsides when the tooth infection is relieved.

Bone Necrosis. As a result of root infection, more or less of the bone becomes necrotic and requires surgical removal. The least acute form results in a local periosteal abscess which may find exit in any region in the mouth or cheek with but molecular destruction of bone. In exceptional cases the area of necrosis may involve a part of the alveolar border, particularly if there is added a local injury attending extraction of a tooth or by a fracture of the jaw.

Osteomyelitis of the Jaw. Total necrosis of a part or all of a jawbone may follow extensive infections. In the upper jaw the whole alveolar border may become involved as an initial process and may be cast off in its entirety. In the lower jaw the extension is usually step-wise, more and more of the jaw becoming involved.

TUMORS OF THE ORAL CAVITY

A great variety of tumors occur in the mouth. The commonest are of course epithelial in origin and may be either ulcerous, as above noted, or fungating, forming protruding tumors. Mixed tumors, the epulides and the dentigerous tumors are common. The jaws produce tumors either endosteal or exostoses, simple or mixed. With the tumors of the oral cavity must be reckoned those of the accessory sinuses which may be infected or not.

The tumors of the mouth may be classed as follows:

I. Epithelial Malignancies
   Ulcerous Malignancies
   Fungating Malignancies
II. Mixed Tumors
III. Tumors of Bone

Under these general heads it will be profitable to catalogue some of the more common lesions in order that we may gain, at the outset, a perspective of the fundamental differences between the infections and the neoplastic diseases.

EPITHELIAL MALIGNANCIES

Two important types of epithelial malignancy must be recognized, the ulcerous and fungating. The former, little inclined to early emphasize their presence, like all ulcerating malignancies, tend to early metastasis. The fungating form are more likely to attract attention because of the mechanical interference they cause but because of their inverting tendencies are less malignant.

Ulcerating Malignancies. Most cases of cancer of the tongue, cheeks, and floor of the mouth begin as ulcer. Many begin from some sort of mechanical injury, such as a plate or a jagged tooth, and therefore they are frequently preceded by a simple ulcer, but the majority occur in regions in which there has been no trauma. A malignant ulcer develops unheralded and attracts attention only because of the pain it causes.

The ulcer is usually superficial, with a reddened border because of the infection complicating the malignancy. The edge of the malignant ulcer has a cancer hardness when felt, let it be emphasized, with the bare ungloved finger. When favorably situated, pressure on the ulcer with a slide may make the cancer nests visible to the naked eye. This test is more often applicable when the lesion is located on the lip. It must be differentiated from tuberculous and syphilitic ulcers as well as simple inflammatory ulcers.

Fungiform Ulcers. These are most commonly situated in the alveolar border, often after teeth have been extracted. Instead of a scooped out lesion, it protrudes above the surface of the surrounding tissues. The fungating tumor is hard and it bleeds on manipulation. This is characteristic. Granulomatous tumors develop about teeth still present, are soft and withstand a considerable degree of manifestation before bleeding is produced.

Mixed Tumors. Under this head may be classed, for clinical convenience, a variety of tumors differing much in form but all have the com-

mon factor. They are relatively benign but all tending toward malignancies. This view-point may be rank heterodoxy but it certainly helps to a clinical understanding of this group of diseases.

Beginning with the epulides we have tumors which may be localized on the gums and when destroyed the story is ended. But sometimes the story does not end there. There may be a more deeply lying tumor, the epulis forming but the crown. A more serious lesion may lie beneath.

The endogenous tumors are cysts and adamantinomas. The cysts may be simple and when destroyed end the process but there may be areas which develop again and a new and more significant tumor develop. The elemental tissue is of enamel structure, therefore epithelial. This structure may be associated with more elemental tissues, chiefly fibrous or some modification of it. If neglected or inadequately treated these tumors become malignant. When they do so, it is the adamantine structure that invades. It is the common practice to call these tumors adamantine carcinomas. This is unfortunate because they do not possess the malignancy of carcinoma of surface epithelial origin. The better term is malignant adamantinoma. Even when they invade the adjacent tissues they are little prone to cause metastases and if completely removed, even after invasion, they are usually cured. In their clinical behavior they are parallel in a general way with the mixed tumors of the parotid gland.

Tumors of Bone. Most true tumors of bone are periosteal and declare their presence by a thickening, localized or diffuse. Exostoses are not rare but are simple and clinically unimportant, being benign.

Malignant tumors of bone are rare. Periosteal sarcomas are very rare tumors and are likely to be confused with the much more common condition in which a tumor of the soft parts becomes attached to the bone.

Tumors of Accessory Origin. Tumors arising within the nasal fossae or the antra may expand these cavities and protrude into the mouth producing bleeding masses.

DISEASE OF THE RHINOPHARYNX

Time was when it seemed that those subject to fatigue headaches selected the "nose and throat" as a specialty. Nowadays the province of the general surgeon is quite generally taken over by capable specialists. The field as it looks to the general surgeon is divisible into the nasopharynx and the larynx.

These may be outlined as follows:

I. Diseases of the Nasopharynx
   Hypertrophies

Polyps
Malignancies

II. Diseases of the Larynx
Inflammations
Benign Tumors
Malignant Tumors

III. Diseases of the Antra
Infections
Tumors

DISEASES OF THE NASOPHARYNX

The diseases above listed do not comprise by any means all the subjects which occupy specialists in this field but they do include most of the conditions which may concern the general surgeon.

Hypertrophies. The turbinates formerly occupied the entire field and were common objects of surgical attack, to the everlasting sorrow of the patient, it may be added. Now they are relegated to objects of differential diagnosis.

Polyps. These exaggerated hypertrophies fill the nasal cavity most commonly but they also form tumors protruding into the pharynx. These larger polyps were formerly the property of the general surgeon but nowadays he sees them only by courtesy of the specialists, or in remote regions where specialists do not reign, if there is any such place remaining.

Malignancies. They may be epithelial or sarcomatous or what is now called lympho-epithelioma. The malignancies which invade this region are, by common consent of both the specialist and the surgeon, handed over to the radiologist because of the tacit recognition that they are at best incurable.

DISEASES OF THE LARYNX

The major lesions of the larynx generally speaking are still best handled by those accustomed to manage operations of magnitude. This is not denying that many specialists have approached the skill in this regard generally possessed by general surgeons. But three groups need to be recognized: the inflammations, the benign and the malignant tumors.

Inflammations. The inflammations of the larynx, which but lead to the discomfort of the patient and to those about him, do not concern the surgeon. When a degree of thickening is reached so that respiration is interfered with, operative procedures may be required. These hyper-

trophic processes may be due to unknown causes or specific lesions such as tuberculosis and syphilis. Transmitted edemas may attend a great variety of unrelated diseases. The papillomas furnish the great majority of such lesions. They are benign growths which cause annoyance rather than obstructed breathing. They are usually villiform but may be rounded. That they likely sometimes become malignant is a matter to be kept in mind.

Malignant Tumors. Carcinomas comprise most members of this group. They tend first of all to occlude the larynx and, as a terminal stage, produce metastases in the regional lymph glands. They are problems in major surgery, whether they be tackled by specialists or general operators. Except for problems in diagnosis the procedures are anatomical.

DISEASES OF THE ANTRA

Time was when specialists called the general surgeons in to do their major operations on the accessory cavities. They required them to do many things to the antra which they do not do now that the responsibility devolves on them. This is well, for it seemed that when one once began to operate he had a permanent patient.

Literature

In the volume on the diseases of the neck, I had to complain that there was no comprehensive work covering the whole region. On the contrary in this volume there are a number of works which cover more or less the whole region of the mouth.

In the first place Spencer and Cade, Diseases of the Tongue, ed. 3 of Butlin's, Blakiston, Phila., 1931, have brought the excellent old treatise up to date, for that part of the subject which it covers has never been equalled. Bloodgood's article (Chap. V, Vol. IV, D. Lewis' Practice of Surgery, Prior Co., Hagerstown, Me., 1929) contains many keen observations which are exemplary in the details presented. Blair's treatise, Surgery of the Mouth and Jaws, ed. 2, Mosby, St. Louis, 1916, contains many observations which indicate the master clinician. Sir Kenneth Goadby's Diseases of the Gums and Oral Mucous Membrane, Oxford Med. Pub., London, 1923, has merit. It has been my policy to quote no foreign language work but Perthes and Borcher's Verletzungen und Krankheiten der Kiefer, Band 53, Neue Deutsche Chir., F. Enke, Stuttgart, 1932, presents such a clear review of the entire subject and contains such a comprehensive bibliography that it demands inclusion here. Padgett's Surgical Diseases of the Mouth and Jaws, Saunders Co., Philadelphia, 1938, is a very competent addition to this field.

CHAPTER II

Nonmalignant Disease of the Lips

IT IS a question whether the lips should be considered a part of the face or as belonging to the oral cavity. The young swain no doubt regards them as part of the face. Generally speaking, however, their most salutory function is to act as a portcullis for the oral cavity and to prevent the emission of sounds the unguarded vocal cords may feel impelled to emit. But the surgeon, particularly the mature surgeon, may well regard them as the initial end of the digestive cavity. Since the nature of their diseases is so like those of the mouth it is convenient to discuss them here.

The first problem with any disease of the lip is to exclude the possibility of malignancy. So many lesions of the lip are now treated by nonsurgical means as malignant without adequate proof of their nature that many misconceptions as to the efficiency of such treatment are being propagated. Needless to say that when cure of a malignancy is claimed microscopic proof of the correctness of the diagnosis should be presented. A clinical diagnosis may be sufficient in most cases for practical, but not for statistical, purposes. At the same time it must be emphasized that many nonmalignant lesions tend toward a malignant stage but the cure of these is not the cure of a malignancy and should not be so recorded. Their successful treatment is an achievement but the surgeon should know just what he has cured. This proof can be secured only by removing it and subjecting it to a microscopic examination. Excising a part of a lesion for diagnostic purposes and then subjecting the remaining part to irradiation, as is sometimes done in very scientific institutions, approaches the ridiculous.

The nonmalignant diseases of the lip may be divided into three general classes: those which resemble carcinomas and may terminate as such, those which resemble carcinoma but which have no tendency to become cancerous; and those which do not resemble carcinoma and differ but little from like lesions of cutaneous and mucous surfaces. Finally the lips are subject to inflammations as any other soft tissue. In days gone by they were subject to direct sudden trauma when they emitted uncomplimentary expletives, but today the automobile windshield is the common object producing injury.

To the first group belong keratoses, leukoplakias, cutaneous horns,

ulcers, and fissures. Each of these may present a precancerous lesion and as such ofttimes it is difficult even with the microscope to say when the border-line has been passed. All these require removal but must be subject to microscopic examination, otherwise the surgeon will remain as ignorant as before.

Among the lesions which resemble carcinoma but do not tend to become such may be mentioned the granulomas due to pus infections, tuberculosis, and syphilis.

Those lesions which neither resemble cancer nor tend to become such are benign lesions which do not differ from like lesions elsewhere—cysts, papillomas, and angiomas.

The inflammations too present themselves for consideration. The traumas and the common acute infections have no laboratory interest but some become chronic and produce an indurated area which may be mistaken for a carcinoma. Acute infections of the lip have a great surgical interest because of the disposition to extend to inaccessible regions.

These various lesions may be catalogued as follows:

I. Precancerous Lesions of the Lips
   - Keratoses
   - Leukoplakia
   - Ulcers
   - Fissures
   - Cutaneous Horns
   - Epithelial Warts

II. Granulomas of the Lip
   - Granuloma Pyogenicum
   - Tuberculosis
   - Syphilis

III. Benign Tumors of the Lip
   - Cysts
   - Adenomas
   - Lymphangiomas
   - Hemangiomas

IV. Inflammations of the Lip
   - Localized
   - Spreading

If the student knows what lesions may occur in a certain region the diagnosis is half made. It is being confronted with a lesion when one does not know what it might be that causes confusion. Nowhere is this more true than in the lesions of the lip.

The more important lesions are those closely associated with malignant changes. Frank malignancies are easily recognized in the clinic, not so the border-line cases. If one knows nothing about the one word "cancer," naturally all diseases will appear to be such and so diagnosed. Therefore to know cancer it is essential to know all about lesions that are not cancer. With this knowledge thoroughly mastered the border-line lesions are more easily comprehended. Even so lesions are now and then encountered which can be diagnosed only with the aid of the microscope. Therefore eternal vigilance must be the watchword and all lesions no matter how insignificant they may be, or how certain the clinical diagnosis may seem, must be verified by a slide. Only by the exercise of such vigilance can error be reduced to a minimum.

PRECANCEROUS LESIONS OF THE LIPS

A precancerous lesion is one which in its normal course will ultimately become cancerous. That is its proper meaning. Often the term is used when the surgeon is not sure of his diagnosis. Therefore in its proper usage it expresses knowledge, in the improper use it expresses ignorance. Here, of course, it will be used in its proper sense as follows: a precancerous lesion of the lip is one which tends to terminate in malignancy but certainly is not yet malignant. Usually this can be established in the clinic. Sometimes a microscopic examination is necessary and one finds instances in which doubt remains even when all these resources are concentrated on the problem. Then one turns to the patient: what will the after-course disclose?

History is of but little aid because it is not what they have been but what they are at the time of observation that interests both the patient and the surgeon. Although situated on the most exposed part of the body, lip lesions if not painful are sometimes unobserved for a surprisingly long period of time. This applies of course to the unromantic period of life. For instance a staid business man presents a lesion first discovered last week. Obviously it was at least a month old. He admitted his newly hired secretary discovered it. Such chance relationships may be responsible for tales of sudden growth which obviously cannot be correlated with the facts. If the truth of rapid growth can be established, it may be accepted as evidence of malignancy. It is worth while in establishing this fact to inquire into the circumstances which led to its discovery. It seems incredible that obvious lesions of the lip may remain undiscovered. Of course in most cases the existence of a lesion was known but was regarded as of no moment.

This is a convenient place to renew the tirade against treating undiagnosed lesions. Much confusion has been caused by the statistics of the

curability of lip cancer when treated by nonsurgical measures because the diagnosis is unverified by microscopic examination. The benign are not separated from the malignant. All lesions take their place in statistical tables not according to what they were but according to what the compiler thought they were. A precancerous lesion is not a cancer and curing such is not curing cancer. It cannot be too often repeated that he who claims to have cured a malignant disease should have microscopic evidence that his diagnosis was correct. Furthermore the disappearance of the local lesion does not constitute a cure. This applies particularly to carcinoma of the lip. Exhibiting lesions as cured, no matter what the measures employed, after six months or a year is certain to confuse the issue. Unhappily for the surgeon who treats only actually verified cancer, the results are not so favorable as when nonmalignant lesions are treated by nonoperative measures. The nonoperative therapeutist has licked up the gravy and has left the dishes for the surgeon to wash. The surgeon does not like this for his statistics, being accurate, make rather sad reading. We rail at the women to come in early with their breast lesions so that we may cure them. Yet lips exposed to constant inspection do not warrant our promise to cure even if we see the lesion early.

Keratosis of the Lips. This is one of the most common lesions of the lip. It is most usually seen in those exposed to the winds in dry weather. While it is not uncommon in earlier life, the well developed form is usually seen in those beyond middle life. In young persons the lesion may heal spontaneously, as with change of occupation, or with the use of mild local treatment. In those of more advanced years the lesion usually remains unchanged for years but the tendency is to terminate in a malignant lesion. It is essentially a classical precancerous lesion.

Pathogenesis. Though due usually to exposure, an acute disease such as herpes sometimes marks the beginning of the disturbance. The superficial epithelial cells remain in position producing a roughened scaly lesion. It may be confined to a part of the lip or extend across the greater part of it. It may remain unchanged indefinitely but often the intensity varies, sometimes better, now worse, depending usually on the season of the year or other changes in climatic conditions. Usually the most extensive changes are found near the center of the lip shading off gradually to each angle of the mouth. Sometimes a circumscribed area is involved. These are more likely than the diffuse to become malignant. The upper lip is rarely involved.

In time the affected area thickens and occupies more of the surface of the lip. Fissure may occur to complicate the lesion particularly near the angle of the mouth.

Sooner or later a certain area of the keratotic lesion thickens. This change is perceptible to touch as hard plate-like resistance below the keratotic surface, suggesting somewhat the feel of an early chancre. This is the end of the precancerous stage. The changes usually take place in one part of the lesion only, leaving the less affected parts for comparison.

Pathology. The superficial epithelial layers become thickened but remain in position producing a roughened area resembling a psoriasis. In

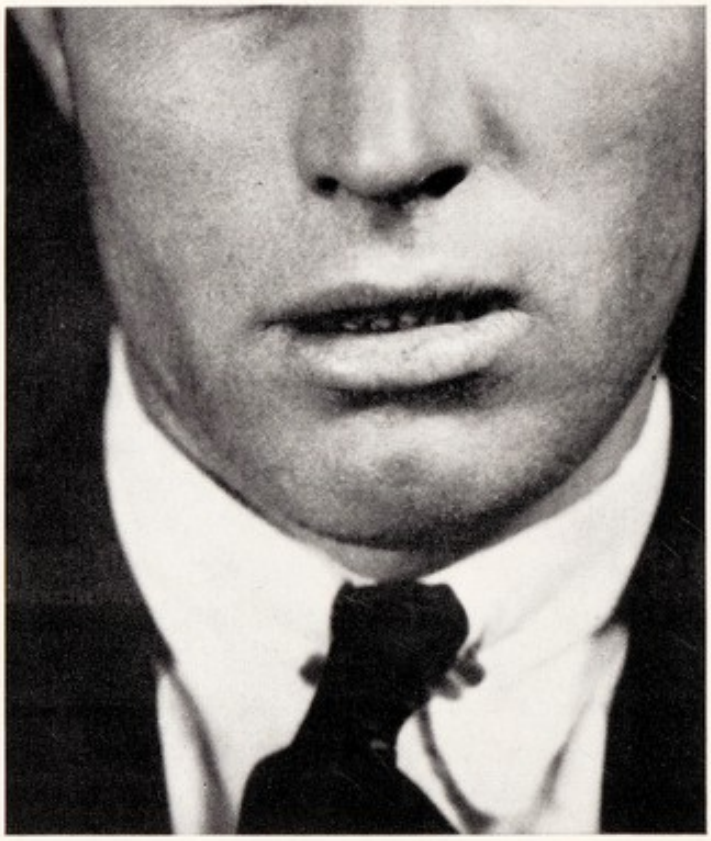

FIG. 1. Keratosis of the lip in a young man. The epithelial thickening is like scales of bran.

the beginning the fine flaky scales are scarcely perceptible and form only a narrow line (Fig. 1). When more fully developed the lesion extends across the greater part of the lip and is nearly as broad as the lip is thick. Instead of the covering being flake-like, as it is in the early stages, the epithelium makes more or less a heavy covering. Such surfaces cannot be picked off without causing bleeding. These are truly premalignant conditions. Sometimes the lesions present local areas of thickening, forming tiny wart-like elevations. Several such areas may form (Fig. 2). Sometimes this thickening may show a true horn-like elevation (Fig. 3).

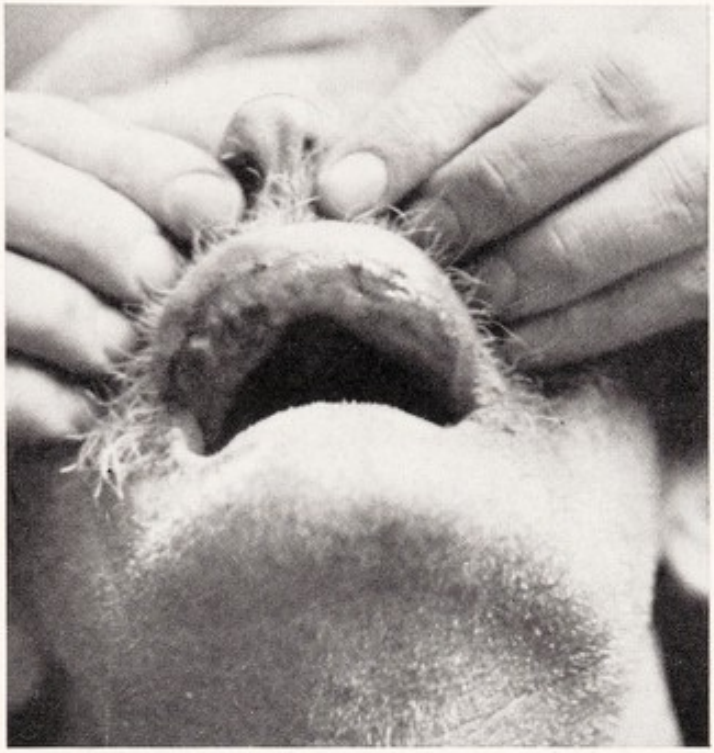

FIG. 2. Localized thickened areas are shown on the upper lip.

On the contrary, instead of a thickening, ulceration may begin. These should be especially carefully examined for beginning malignancy and should be so regarded until proven innocent. This means that they should be removed and examined microscopically. When malignancy forms, only one point becomes so involved. Development of malignancy

FIG. 3. Long existing keratosis of the lower lip. At the left is beginning ulceration, on the right is beginning horn formation.

at several points, so far as I know, does not happen. Even so it is not always possible to say in the clinic just which point is most likely to show beginning malignant changes. Therefore, when in doubt the whole area must be subjected to microscopic examination. When fissures form in keratotic lesions, particularly if they bleed and refuse to heal, malignant change likely has already begun. At least this must be assumed until the slide proves the contrary. These, it should be remarked, sometimes prove malignant when the slide fails clearly to reveal it.

As in keratotic lesions on the face, when the superficial layers of cells are easily removed, the lesion is likely benign. If bleeding is caused, it is

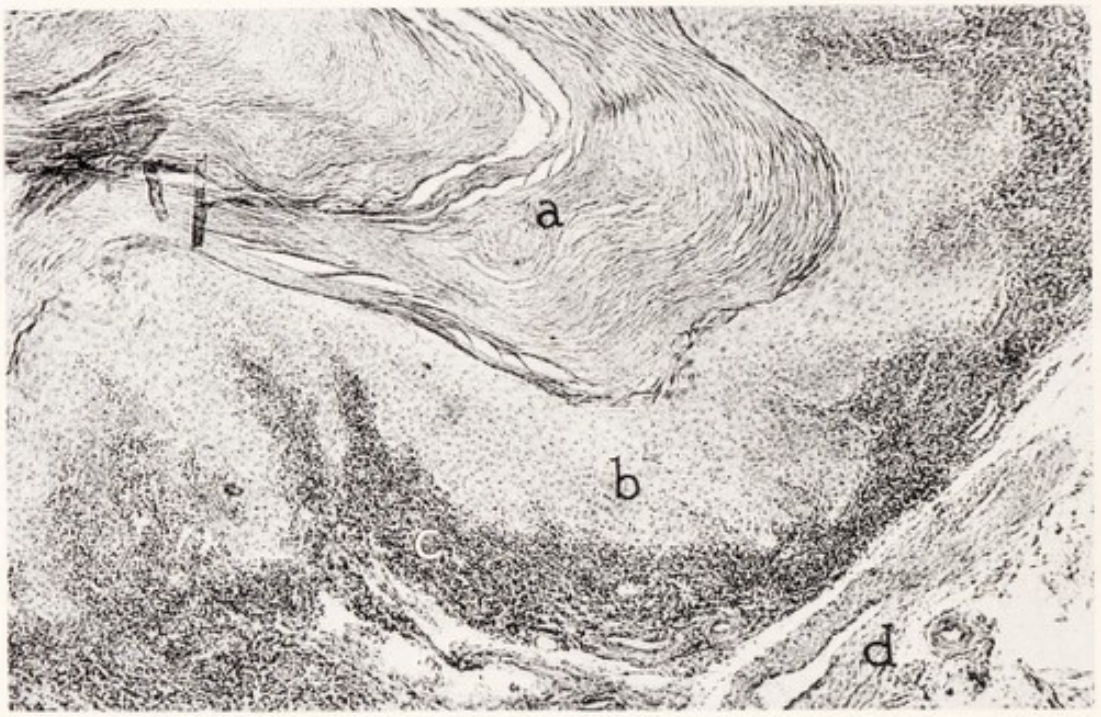

FIG. 4. Keratosis of the lip: *a*, the mass of keratotic epithelium is teased off. It is made up of degenerated cells of which only the nuclei remain; *b*, beneath is a viable layer of epithelium over which are deeply staining cells; *c*, submucosa infiltrated with round cells.

in a transitional stage. Picked up between thumb and finger if benign there is no induration nor is pain caused by this maneuver unless it is associated with inflammation. The presence of inflammation is in itself a warning sign. A reactive process beneath is a stimulus to epithelial proliferation. Some thickening is compatible with benignancy but is always a signal for caution.

Histology. The chief microscopic change in keratosis of the lip consists in a great increase of the horny layers. In these cases, usually of long standing, there is a heavy plate of epithelium which when forcibly

removed leaves the surface denuded of all epithelium (Fig. 4). The basal layers are less markedly affected but usually show some increase. In cases in which there is thickening of the basal layer tending to extend into the underlying tissue, malignancy is being approached (Fig. 5). When the lesion is benign there is but slight round cell infiltration. If such are present and arranged around advancing epithelium, the lesion is suspicious and when the epithelial cells penetrate the subjacent tissues, the border-line has been passed. The superficial cells in old lesions may be degenerated, particularly near a fissure. Exudate may form on the surface producing a hard dry scab giving the impression of malignancy.

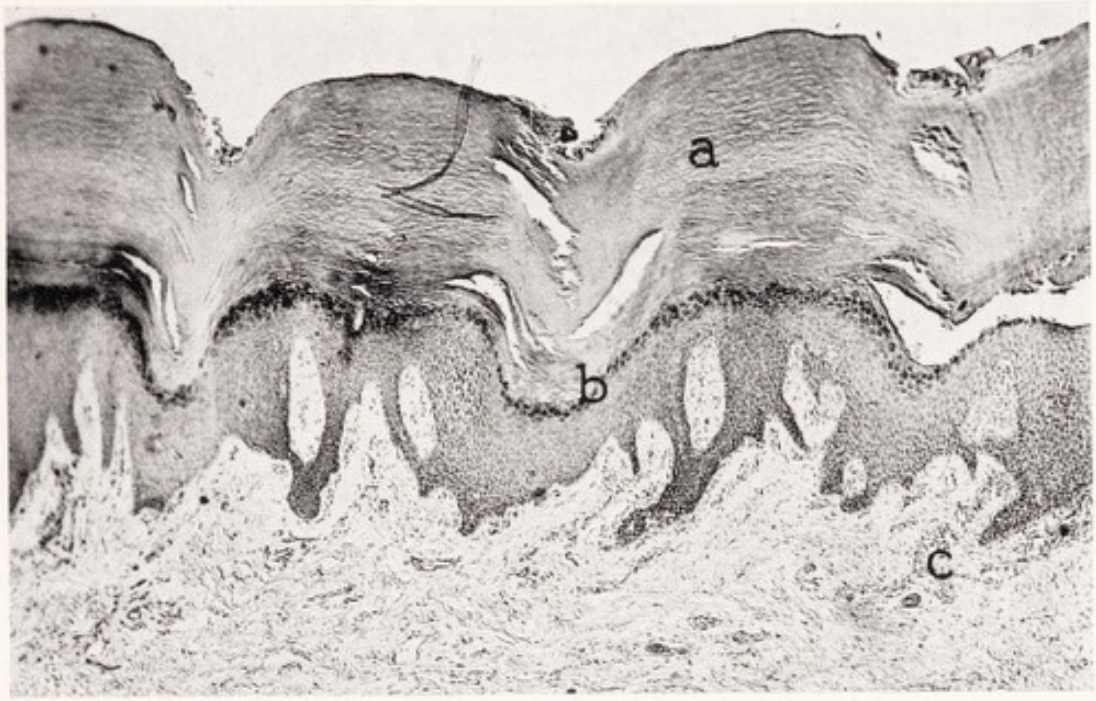

FIG. 5. Keratosis of the lip. Marked thickening of both superficial and deep layers: *a*, keratotic superficial cells; *b*, deep cells proliferated and at *c* there is slight round celled infiltration around the advancing column of epithelial cells which show some changes in cell type.

Such exudates are coagulated serum and contain no notable number of cells.

Leukoplakia. This lesion is due to a piling up of the epithelial cells much as in keratosis and has much the same clinical significance. Why the scales are white is not known. The mouth is the common site of leukoplakia and when the lip is involved it usually is associated with like lesion of the buccal cavity but is always discontinuous. However, the lip may be the only region involved.

Pathogenesis. The cause of the lesion is not known. The attempts to

convict the spirochaeta have been unsuccessful. Smoking is blamed, but most men smoke, and nonsmokers are by no means immune. These implications make it evident that the lesion is confined chiefly to the male sex.

The surface epithelium piles up and assumes a silvery white color. At first the epithelial layer is faintly white, sort of suffused, but as the lesion becomes more intense a heavy white surface forms so that it seems to form a layer which could be wiped off, but this is a delusion. Lesions confined to the vermilion border of the lip usually terminate diffusely, ending gradually in the normal mucosa (*A*, Fig. 6), but may be circum-

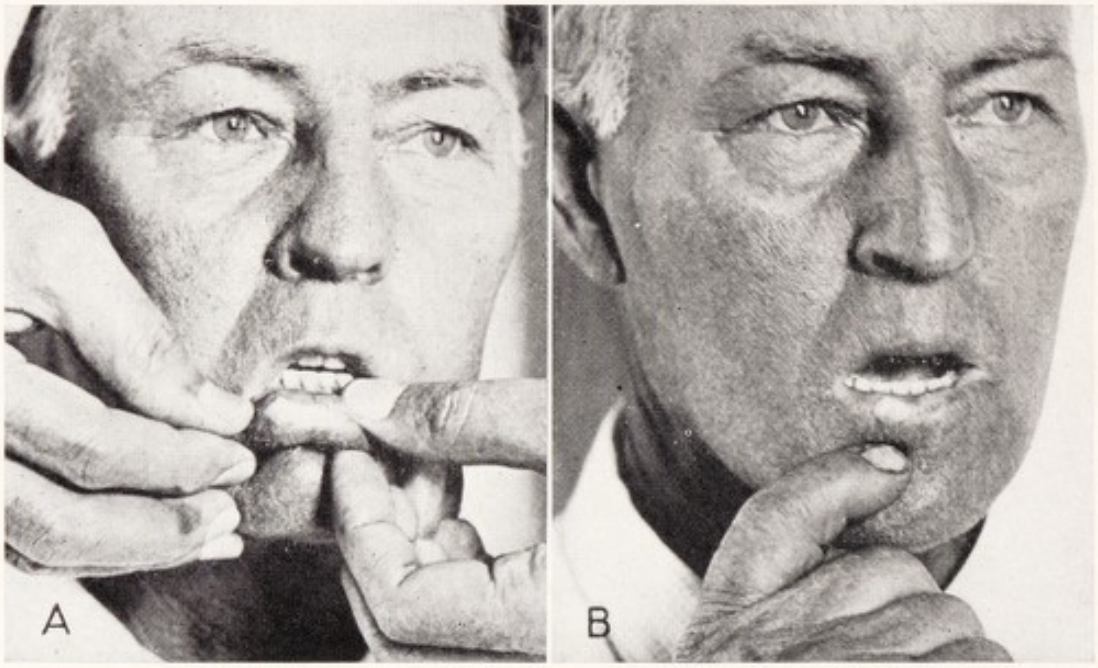

FIG. 6. Leukoplakia of the lower lip. *A*, The lesion terminates gradually into the normal mucosa. *B*, The lesion is sharply circumscribed.

scribed and definitely elevated (*B*, Fig. 6). The usual course is to remain stationary for years. When the border-line of malignancy is approached the lesion thickens. Even when definitely malignant it is of a mild nature and seems definitely curable when confined to the lip. I have not encountered an exception.

Pathology. Leukoplakia is compatible with a soft mucosa and though it looks firm is surprisingly pliable; considerable manipulation does not disturb the surface sufficiently to cause bleeding. When there is thickening perceptible to the touch, particularly when the surface epithelium is felt to be rough, it is suspicious and when manipulation causes bleeding the border-line has been passed.

Histology. The microscopic picture of leukoplakia parallels the keratotic lesions. Usually in the process of preparation the silvery scales are lost but there remains a layer of thin palely staining epithelium (Fig. 7). The epithelial layer is thickened with but little or no round cell infiltration. On the contrary the surface epithelium may be thinned due to the casting off of most of the silvery layer, and the lower layer of cells tend to invade the adjacent tissue. The round cells may be abundant (Fig. 8). If the round cells are abundant about the penetrated epithelium the

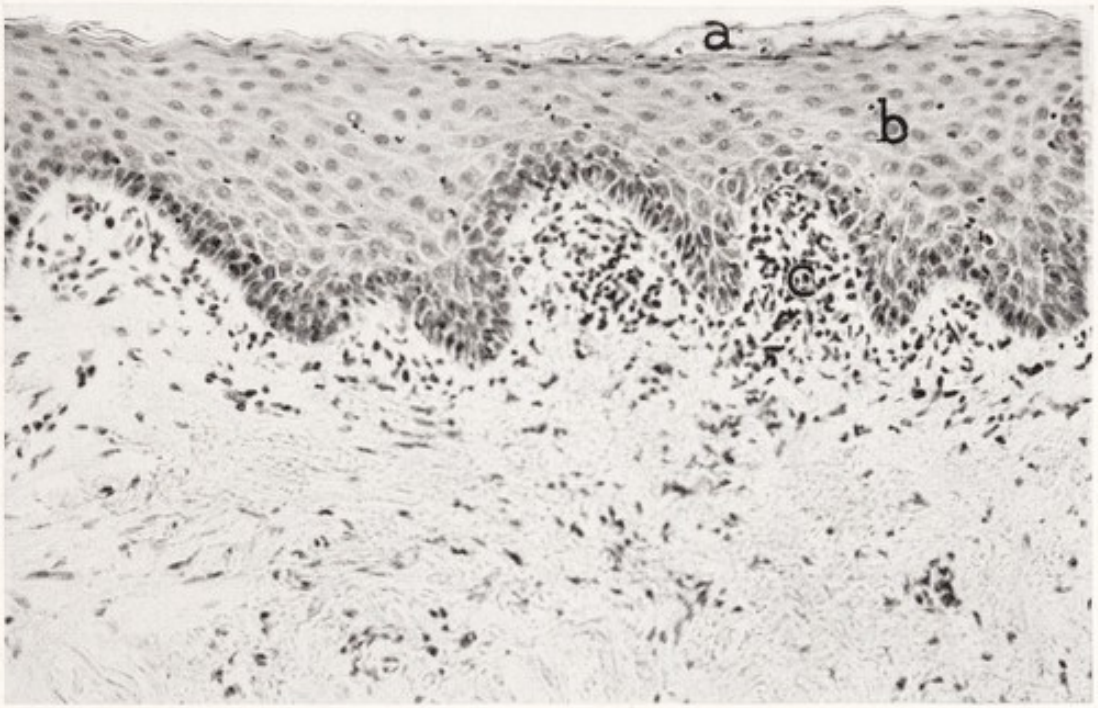

FIG. 7. Leukoplakia of the lip: *a*, the few silvery scales remaining are palely staining; *b*, epithelium thickened, the deeper layers deeply staining; *c*, slight round cell infiltration below the epithelial layer. No epithelial proliferation, and there is no change in cell type.

border-line to malignancy is approached and if separate nests are found the lesion is definitely malignant.

Benign Ulcers of the Lip. The term "ulcer" here as elsewhere implies a defect of the epithelium. These are relatively rare lesions. Benign ulcers are not produced by a single or repeated trauma but may be associated with continuous irritation. Some cases begin as a labial herpes, a fever blister attending some acute disease, which fails to heal. Usually a cause is not in evidence. They are as apt to occur on the upper lip as on the lower. As an advance stage of keratosis an ulcer may be produced. These may be benign but should be regarded as suspicious lesions. So long as the borders are soft they are yet benign. They differ from pyo-

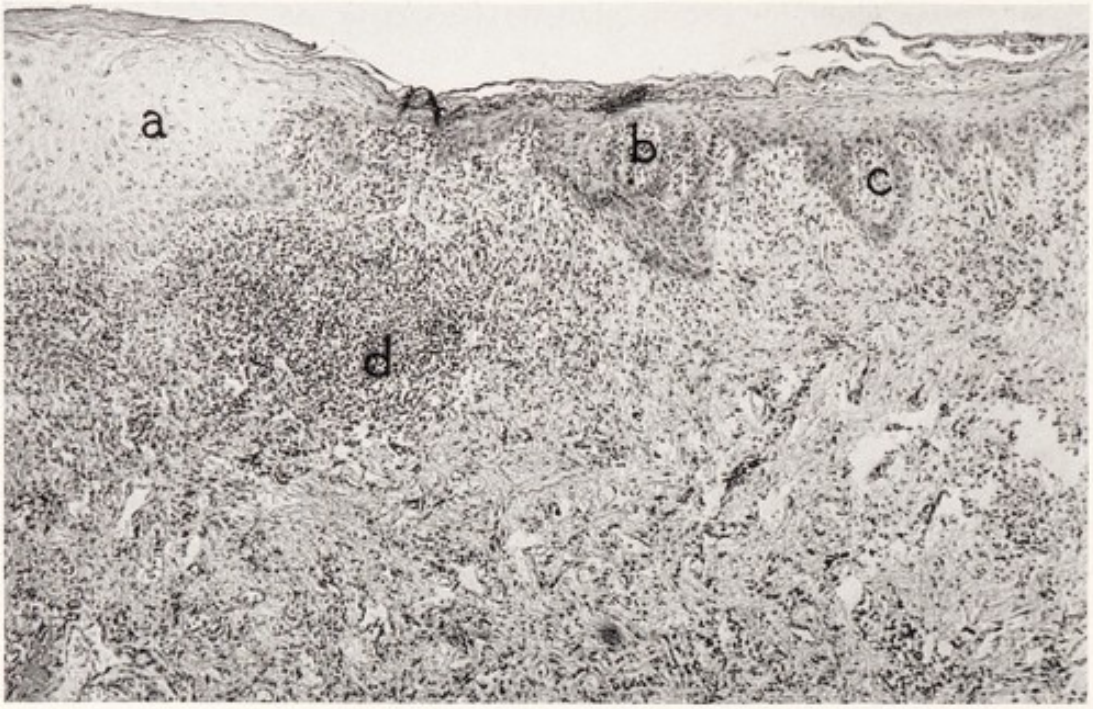

FIG. 8. Leukoplakia of the lip approaching malignancy: *a*, normal epithelium; *b*, epithelium thinned at the leukoplakic border, the leukoplakic cells having been cast off; *c*, epithelium hyperplastic but without definite nest formation; *d*, round cell infiltration.

genic granulomas in that there is no injury and no formation of exuberant granulations. They begin as simple chronic ulcers except as above noted and continue as such.

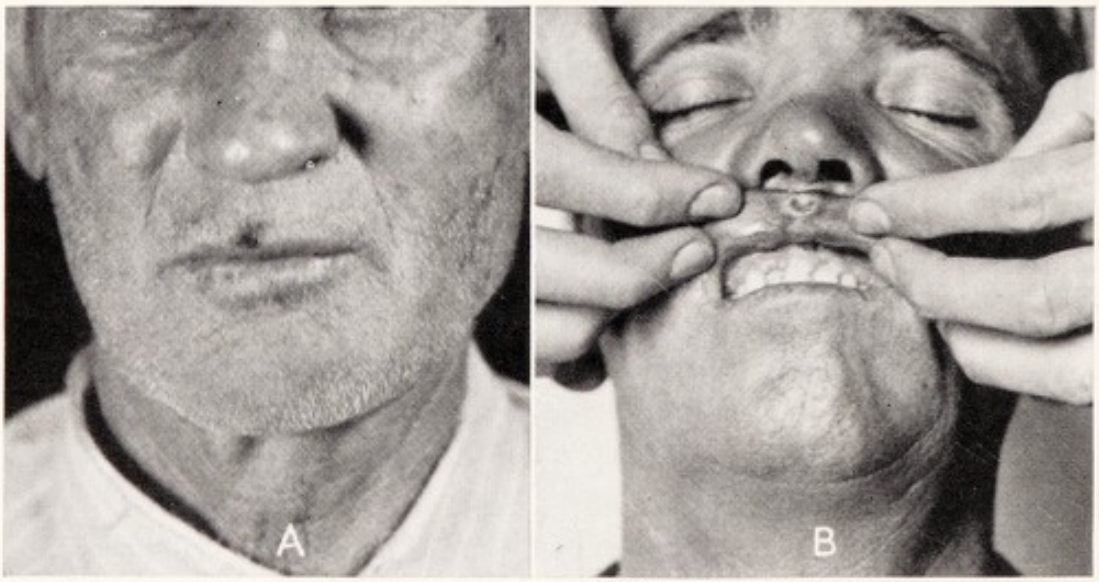

FIG. 9. Benign ulcers of the lip: *A*, the defect of the epithelium of the upper lip is irregular and the lesion superficial; *B*, the lesion is deep and the epithelium about it shows some proliferation but no invasion.

Pathogenesis. The spontaneous ulcers usually show a defect, a hollowed out area. Such lesions may continue for months, even years, without notable changes. Sometimes they heal only to recur. These recurrent lesions usually end in malignancy. The defect may be a loss of continuity of the epithelium without any proliferation (*A*, Fig. 9) or the epithelium may be obviously in a state of proliferation producing a piling up about the border (*B*, Fig. 9). On the lower lip they may occupy the site of the common habitat of the pipe stem or a broken tooth. Now that the days of the old clay pipe are gone they still occur with equal

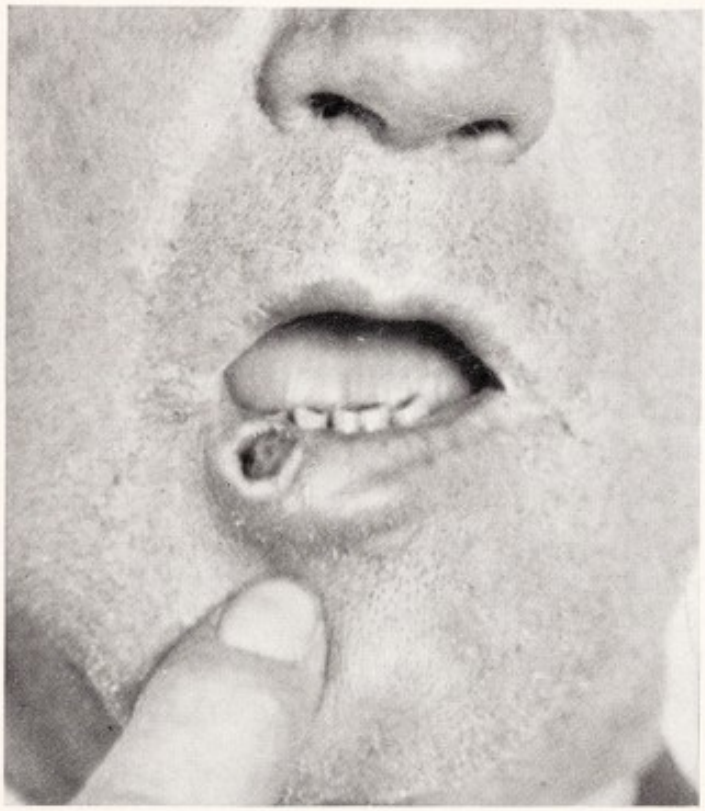

FIG. 10. Chronic ulcer of the lip. The lesion was soft with overhanging borders. Began 6 years before, following herpes labialis. Metastases formed without notable change in the ulcer.

frequency. This is another evidence of male delinquency gone to pot, going to prove that the diseases commonest in males are not necessarily evidence of their sins but they occur just because they are males. We should not mix our social quarrels with our ideas of pathogenesis. Malignancy if it follows usually does not do so for a period of years. Chronic ulcers may acquire a thin epithelial covering and persist for years. These ulcers may remain stationary indefinitely. In rare cases though the local lesion remains unchanged so far as palpatory changes are concerned, a metastatic lesion may develop (Fig. 10).

Pathology. The ulcer is superficial and soft but occasionally a deep ulcer in a state of reaction may show considerable induration which may suggest malignancy and yet be benign. On the contrary they may feel soft and yet show epithelial cell invasion. They do not bleed when manipulated and lack cancer hardness. Naturally as in the case of any transition there is a period of doubt. On cross-section they are superficial without infiltrated border.

Histology. The essential feature of the lesion is a round cell infiltration with but slight proliferation of the bordering epithelium. Even these

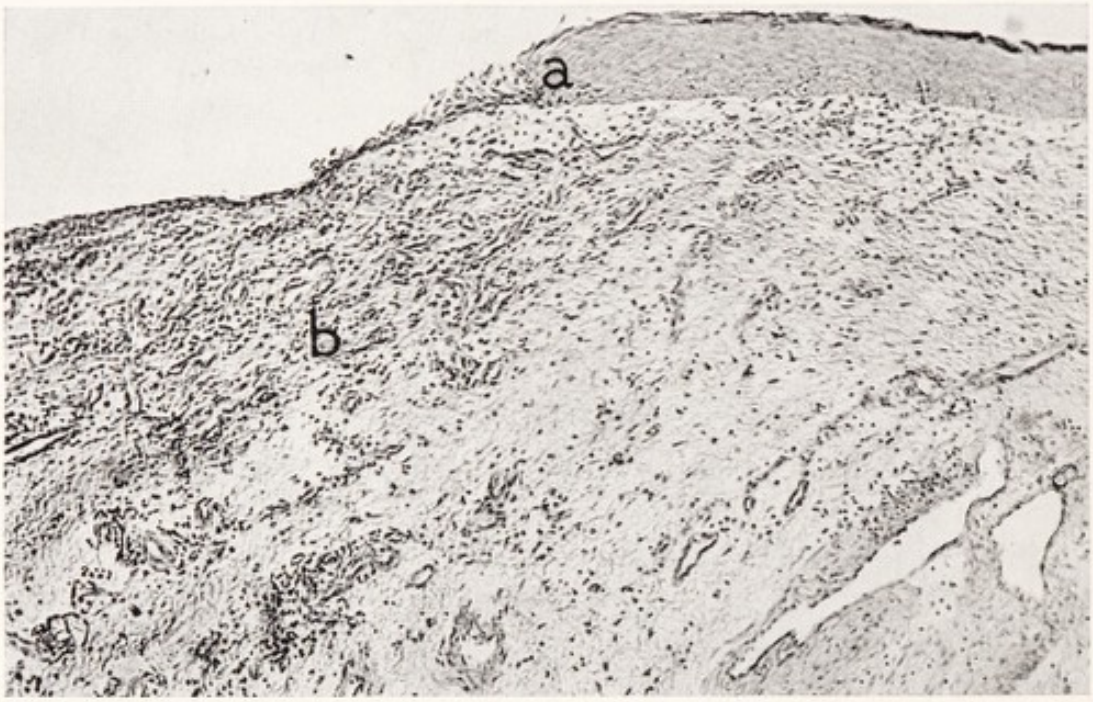

FIG. 11. Benign ulcer of the lip of many years' duration. The epithelium about the border is unchanged and ends abruptly at the border of the ulcer, *a*. The base of the ulcer is made up of unchanged connective tissue with but little round cell proliferation, *b*.

changes may be but little in evidence (Fig. 11). On the contrary the tissue reactions may be marked by pronounced round cell infiltration. Epithelial cells may project out over the surface of the ulcer and even into the granulation tissue. Experience has shown that lesions of this degree usually remain cured after local excision but pathologically they crowd the border-line very closely. This is particularly true if there are detached epithelial cells. In fact, though there may be no notable change in the ulcer, metastasis may place it definitely in the malignant clan (Fig. 12). Even if there is no evidence of malignancy, the patient should be followed for some years. The local lesion may remain cured yet metastases may become evident in the lymph glands.

Fissures of the Lip. Fissures are but a corollary to ulcers and may precede them. They follow drying of the mucosa and consequently are seen chiefly in persons exposed to the elements. Sometimes they follow trauma. Less often they remain after a healed herpes. I once operated on such a lesion and was amazed to find trichina in it. The patient was a butcher and had the habit of holding his knife in his mouth when neither was otherwise engaged.

Pathogenesis. The fissures form, may heal and form again, without manifesting any malignant tendency. These may remain for years with-

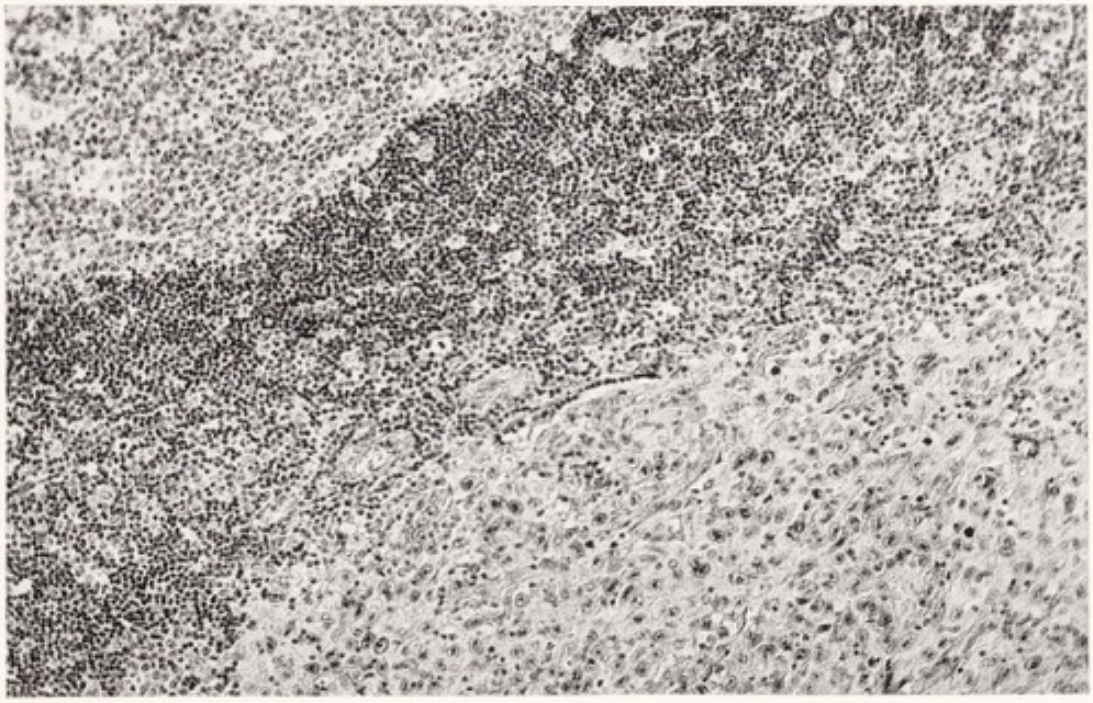

FIG. 12. Slide from a lymph gland of the neck. The lip lesion appeared to be benign both on gross and microscopic examination. The slide shows definite epithelial metastasis adjacent to which is lymphoid tissue. The gland was the size of a pea.

out notable change (Fig. 13). If the borders become thick or they bleed when irritated they are border-land or beyond.

Pathology. The simple fissure is lined with low granulation tissue and the edges are soft but may be painful to pressure without its being a sign of malignancy. Complaint of pain may be registered while the patient is in the act of acquiring for himself a chew of tobacco. This is especially likely to be true when the fissure is located exactly in the angle, the usual site. The approach toward malignancy is signalized by a hardening of the border of the fissure. The cross-section shows a simple separation of the surface epithelium without obvious thickening.

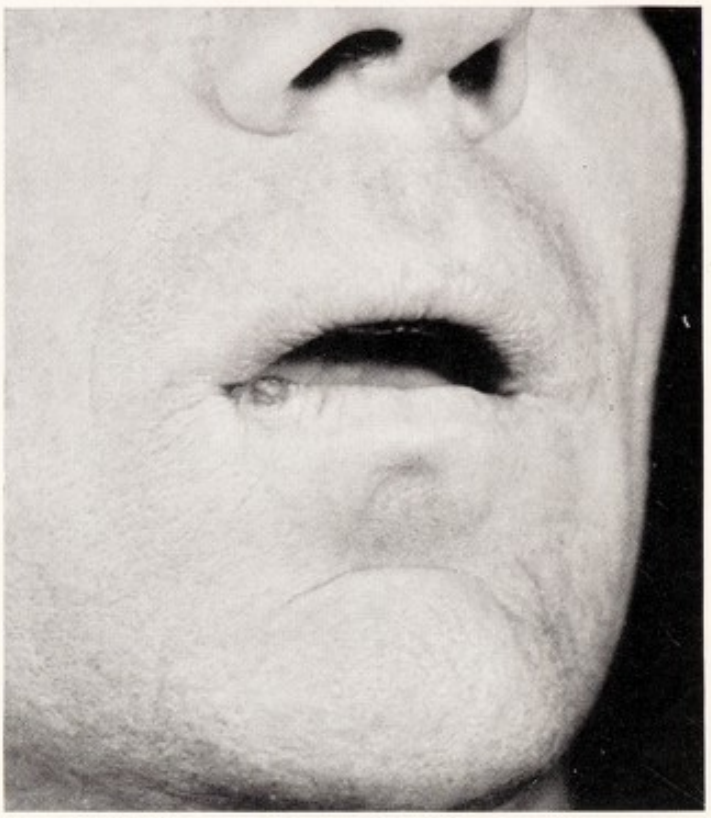

FIG. 13. Fissure at the angle of the mouth. Beside it is a small horn. Both followed a herpes.

Histology. Pronounced round cell infiltration forms the picture. There may or may not be proliferation of the adjacent epithelium. The cellular changes exactly parallel those of simple ulcer.

Cutaneous Horns of the Lip. Cutaneous horns form one of the most common lesions of the lip. They do not differ from like lesions of other

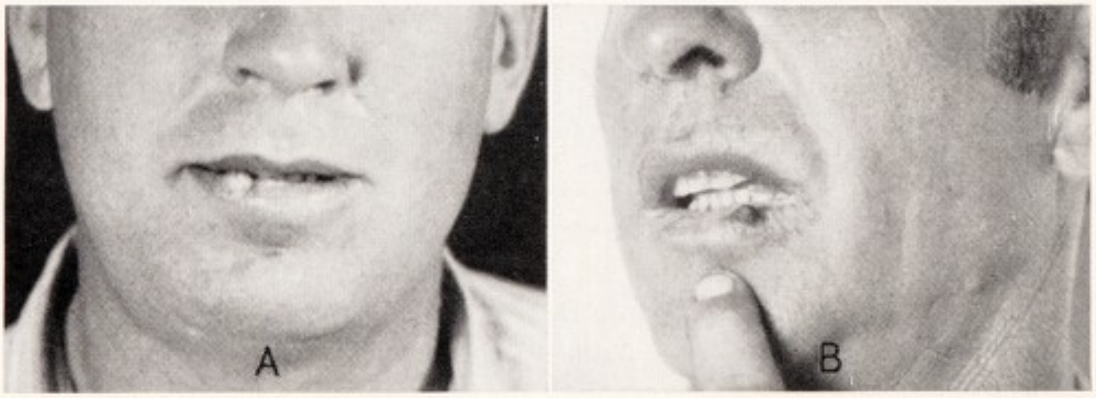

FIG. 14. Cutaneous horns. *A*, The tumor is not completely hornified and the skin is indurated as well as hyperemic. The outer border still showed an ulceration. *B*, Early well defined horn. It seemed fairly to sit on the epithelium so superficial was it.

parts of the body but they are more prone to become malignant in this situation. They may do this with but little gross evidence of the change. They are quite common in women, therefore are not due to clay pipes or any other semimoral dereliction. I was impressed with the difficulty of evaluating the slight microscopic changes in these lesions by the following occurrence: An eminent pathologist after examining a slide declared the lesion benign. As an after-thought he called after me: "Doctor, if there is a recurrence, let me know."

Pathogenesis. The first appearance is as a small elevation, hard to the touch, with a sharply defined base. Some of them, at least, develop on

FIG. 15. Cutaneous horn of the lip. The lesion was hard and sharply circumscribed, the adjacent skin was indurated.

the base of an ulcer or granuloma (*A*, Fig. 14). One part may show cornification while the remainder is still granulomatous. Such cases seem to be a cross between a leukoplakia and a horn. On the other hand the lesion may, though small, be exceedingly hard (*B*, Fig. 14). They grow in height rather than in diameter so that often they are several times as high as broad (Fig. 15). It is astonishing how long patients will tolerate such lesions before seeking relief. Such neglect declares their social standing. Those which grow in breadth and become fissured are more prone to become malignant. Though these lesions are necessarily subject to trauma they remain unchanged for many years. They may become inflamed about their base because of irritation from trauma which may spontaneously subside but this even should always excite suspicion that

the border-line is being approached. The first gross sign of beginning malignancy is a lessened mobility of the base.

Pathology. These structures form pyramid-like elevations on the labial mucosa. They may be very superficial and the adjacent mucosa be wholly free from any change. At the other extreme they may be markedly indurated about the base and the growth no longer freely movable. When the stage of malignancy is reached there is definite cancer induration about the base. One is sometimes in doubt in the clinic as to which side of the line they should be placed. The growth is

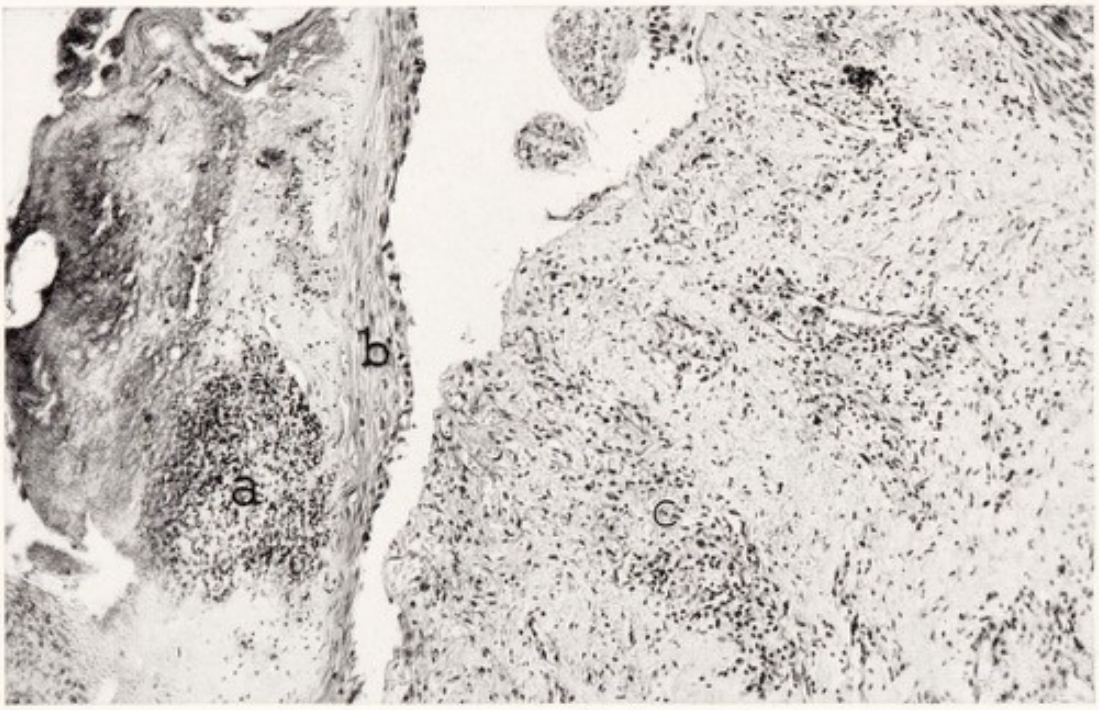

FIG. 16. Cutaneous horn of the lip: *a*, complete cornification; *b*, epithelial proliferation about the base; *c*, layers of round cell infiltration; *d*, connective tissue showing but little change.

so dense it cannot be cut with a knife, being formed of cornified epithelium so that the term "horn" is wholly justified. The base is slightly wider than the upper parts of the tumor but the soft parts are not infiltrated so that the tumor can be tilted from side to side without movement of the deeper parts and without causing any pain.

The cross-section shows a dense cornified mass sharply defined at the point of attachment with the underlying soft parts. If the line is not sharply defined, the case must be regarded as a border-line one.

Histology. The soft parts at the base which can be cut show sharp definition from the connective tissue beneath. Round cell infiltration is

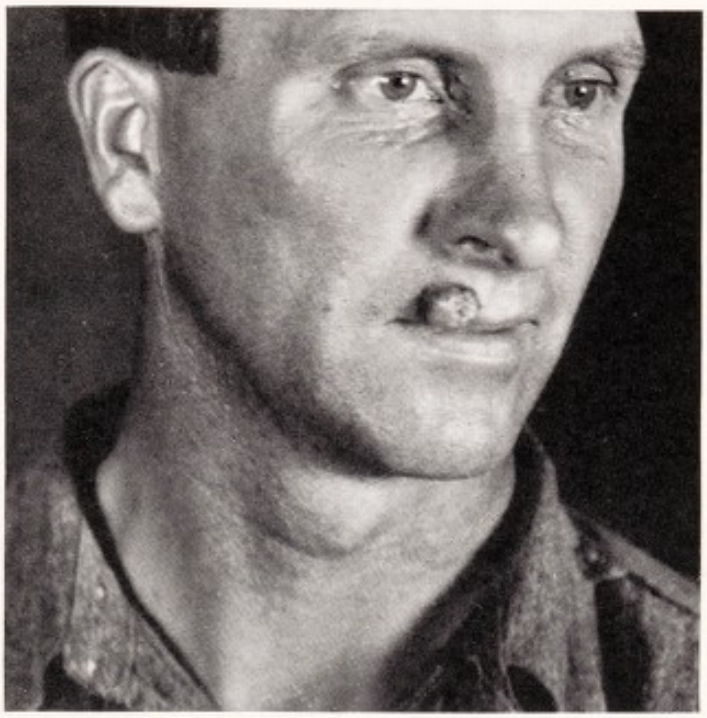

FIG. 17. Benign papilloma of the lip. The ovoid tumor is sharply defined against the normal skin.

absent unless there is restlessness on the part of the epithelium. Such cases are truly border-line (Fig. 16).

Epithelial Papillomas. As a corollary to the horns the epithelial warts may be mentioned. They are the exact counterparts of epithelial warts occurring elsewhere. In fact they have their origin in the skin at the vermilion border and gradually encroach on it.

Pathogenesis. These lesions begin as any other wart but tend to gain some size. They become elevated above the normal skin and mucosa about them (Fig. 17). They do not tend to become malignant.

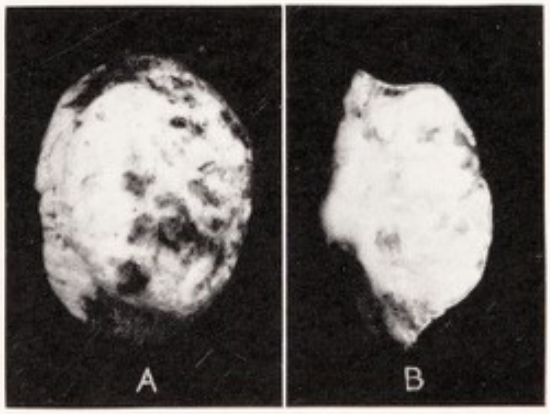

FIG. 18. Hard papilloma of the lip: *A*, the surface is irregular; *B*, it is sharply defined from the adjacent tissue.

Pathology. Their sharp definition and hard elevated surface is characteristic. They terminate sharply (*A*, Fig. 18). This is emphasized on cross-section (*B*, Fig. 18).

Histology. The histology is that of the horns but the keratinization is less marked (Fig. 19).

GRANULOMAS OF THE LIPS

Granulomas differ from ulcers in that exuberant granulations form, producing a tumor which is elevated above the surrounding normal epi-

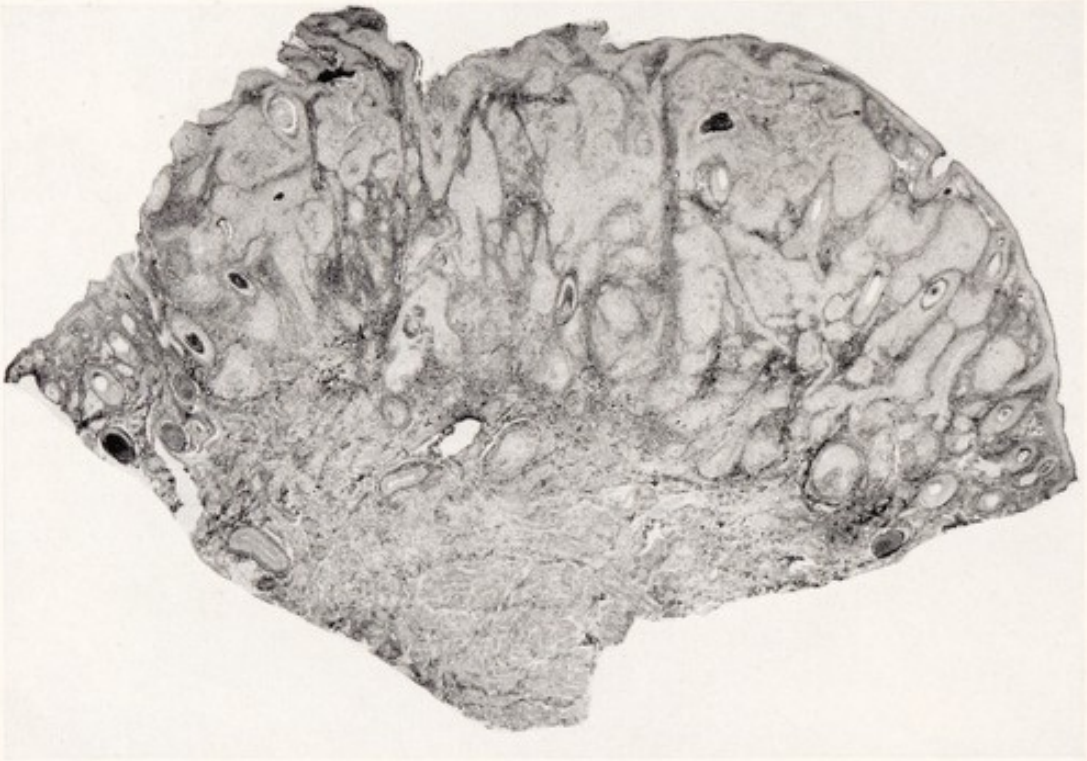

FIG. 19. Hard papilloma of the lip. The bulk of the tumor is made up of hyperplastic epithelium. The connective tissue base retains its acidophilic character and there is no round cell infiltration.

thelium. Pyogenic granulomas are not an uncommon end-result of trauma. Specific granulomas, tuberculosis and syphilis are much less common. To these may be added the rare blastomycosis.

Pyogenic Granuloma of the Lip. Granulomas of the lip are the equivalent of like lesions elsewhere, signifying a loss of epithelium and the presence of exuberant granulations which prevent the epithelialization of the surface.

Pathogenesis. In many cases a definite trauma is recognized but sometimes there is no history of injury. The injury may be a direct blow

or an irritation from some object such as a wheat beard. The lesion may heal spontaneously after a time or may persist for many months or even several years. Those which remain long unhealed may become fibrous, even keloidal. They form more or less circumscribed lesions usually elevated above the surrounding surface (Fig. 20). They may form wart-like elevations which endure indefinitely. I once had one which formed following an injury at 3 years of age and was surrendered to me at the age of 27. They are usually reddish in color.

Pathology. The lesion is usually elevated but may be no higher than the surrounding structures. Their surface is soft to the touch, the degree

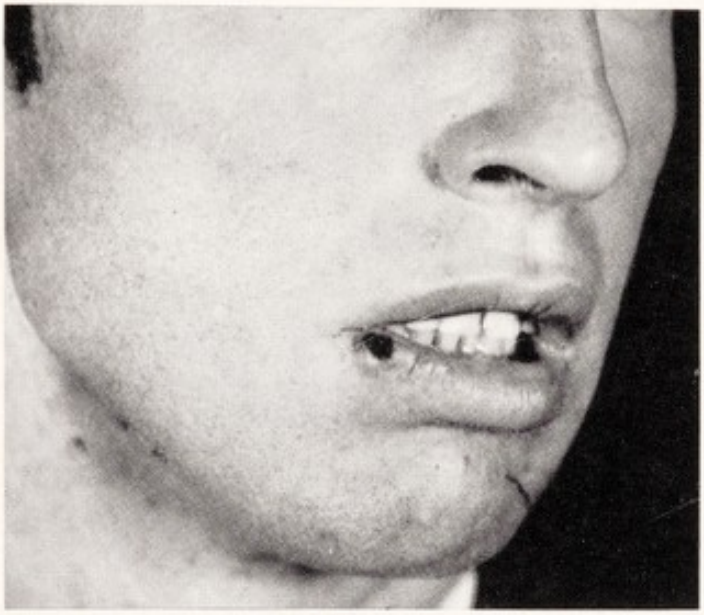

FIG. 20. Granuloma of the lip following a slight trauma. The lesion was soft, slightly elevated and not sensitive. A granular surface became elevated above the adjacent mucosa.

of softness being dependent on their duration. The older ones may be quite firm but it is the firmness of fibrosis and not that of epithelial proliferation. Unless very soft and recent they do not tend to bleed on manipulation. The adjacent area may be slightly hyperemic. The surface may become epithelialized and continue as soft warts for many years.

Histology. The microscopic picture is that of any granuloma, vascular, often with thick walled capillaries between which are round cells and leukocytes. The adjacent epithelium does not take part in the tumor formation. Those of long duration may present the structure of soft warts (Fig. 21) or may have the structure of a keloid.

Tuberculosis of the Lip. Tuberculosis of the vermilion border of the lip without a like lesion somewhere in the mouth, most commonly the

tongue, must be very rare. At least I have seen only a single case. As a corollary to these simple mucosal ulcers are some which are not specific, sometimes seen in undernourished young girls.

Pathogenesis. My single example was the size of a grain of corn when first seen (*A*, Fig. 22). It proceeded to slow healing but the patient died of a pulmonary tuberculosis some ten years later, though there was no evidence of a lung lesion at the time of observation.

Pathology. The lesion was a punched-out one with soft undermined

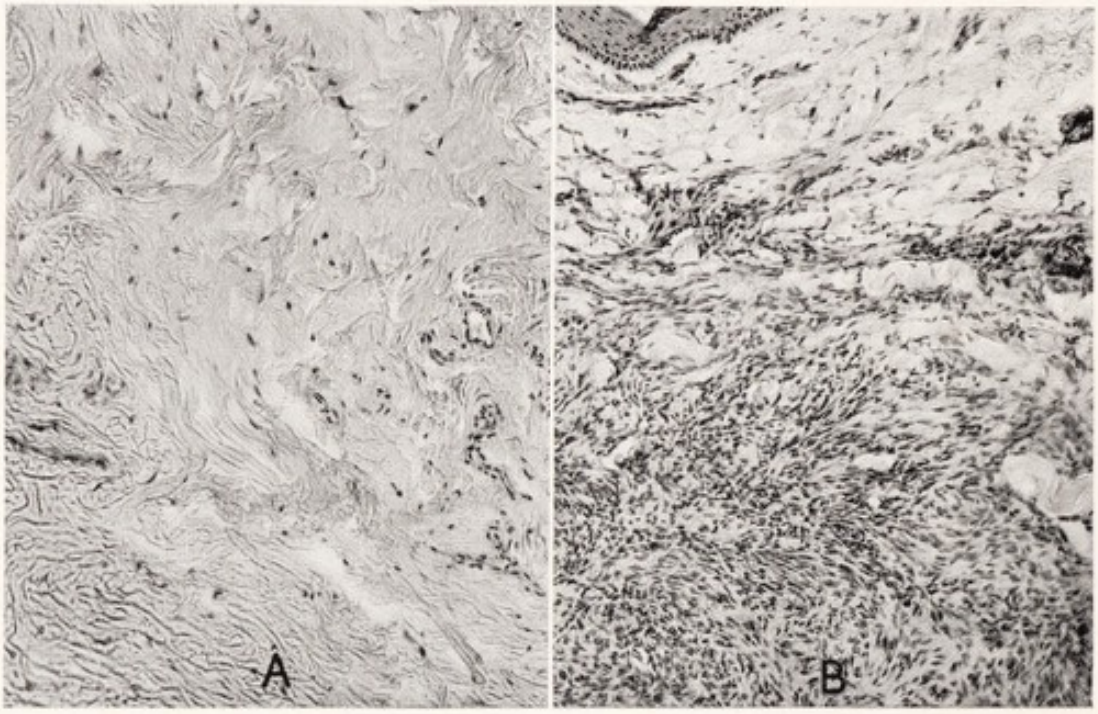

FIG. 21. Terminal stage of a long existing granuloma. The surface epithelium differs but little from the normal. The bulk of the tumor was made up of: *A*, definite keloid bundles and *B*, spindleform connective tissue.

border identical with like lesions of the tongue. Even so the nature of the lesion was not suspected in the clinic.

Histology. The slide showed tubercles.

Chancres of the Lip. Primary syphilitic lesions of the lip are not rare in cities but practically unknown in the short grass country. Even so I have received three in the laboratory in a city which have been removed as malignant lesions and I have stopped operations on a like number which I have seen in consultation. No doubt other surgeons have seen them so that their study is not without importance.

Pathogenesis. These lesions may appear on either lip but all but one of mine were located on the upper lip (*B*, Fig. 22) and in men, a circum-

stance in itself which should cast doubt on the presence of malignancy. Malignancy of the upper lip in males and of the lower lip in women are exceedingly rare. My single case in a woman was located on the lower lip, likewise a topographic assurance against malignancy. Another factor is that they develop in the course of a few weeks, too rapidly for either a benign or malignant tumor. The accompanying adenopathy is acute and lacks wholly the shot-like feel of a malignant metastasis. In fact the nature of the lesion is so obvious that it will not be overlooked if one just thinks of its possibility, even in Kansas.

Pathology. The indurated feel to the examining finger characteristic of lesions occurring in more legitimate fields can be recognized here. The

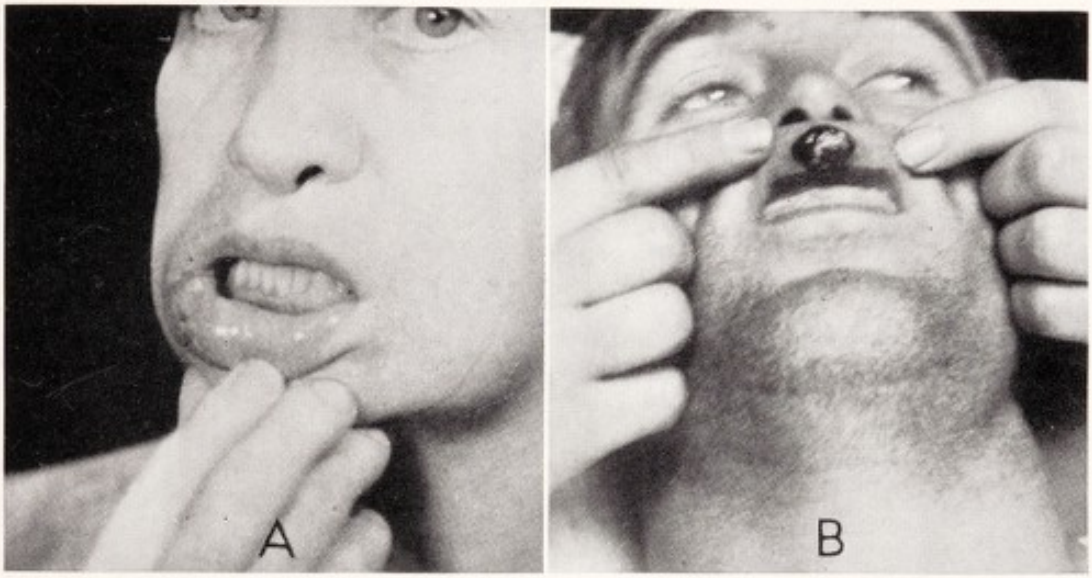

FIG. 22. Granulomas of the lip: *A*, tuberculous ulcer at the outer angle of the lower lip: *B*, chancre of the upper lip.

elevated sharply circumscribed lesions are most characteristic but the small half-submerged lesion has a peculiar disk-like feel that every dispensary student learns.

Histology. Nowadays the *spirochaetes* should be demonstrated. The slide is that of the lesion elsewhere. At least it is easy to see at first glance that the lesion is not malignant.

BENIGN TUMORS OF THE LIPS

Benign tumors of a variety of forms are found on the lip. The most common are the cysts formed in the glands of the mucosa. They form bluish white domed little tumors. Adenomas of the sweat glands are found near the vermilion border and are by no means rare. These are

pure skin lesions and only when they encroach on the vermilion border do they involve the lip. Angiomas both of the lymph vessels and blood vessels, mostly congenital, are not uncommonly found on the lip. They do not differ from like lesions elsewhere.

Cysts of the Lip. Cysts in the mouth are common but occasionally one raises up the vermilion border and is presented as a lesion of the lip.

Pathogenesis. What brings these little cysts about is not known but likely it is safe to say that it is due to some obstruction to the duct.

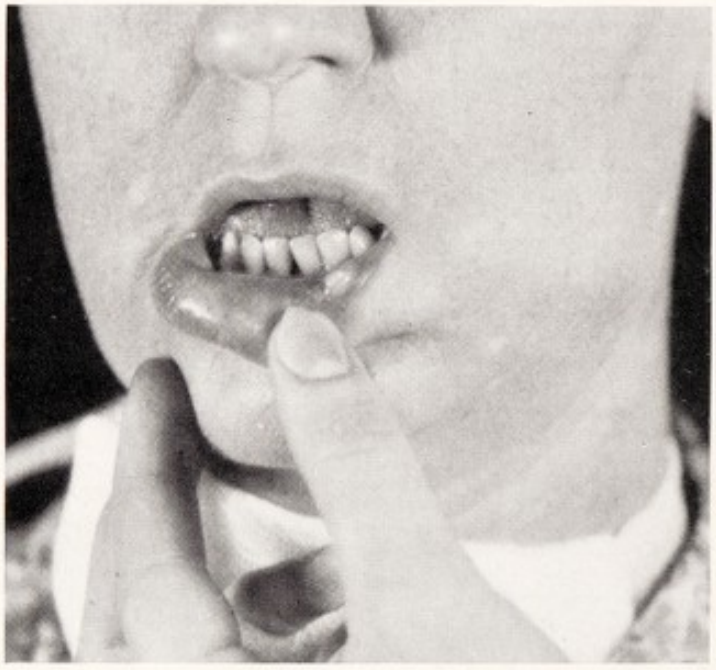

FIG. 23. Cyst of the lower lip the size of a pea.

Pathology. The dome of the cyst is uniform in outline and of a bluish, somewhat translucent, color (Fig. 23). On palpation it seems much larger than appears to inspection. Its spherical outline gives a hint as to its nature. Others located within the mouth further elucidate the problem.

During the course of removal usually the cyst is inadvertently punctured and a thick glary mucus escapes. The section of the capsule shows a thin pearly membrane.

Histology. The slide shows a fibrous cyst wall lined with an indifferent epithelium, cuboidal or more or less flat.

Adenomas of the Lip. These not uncommon lesions are usually found on the upper lip arising in the skin but crowding the vermilion border.

Pathogenesis. They usually develop slowly and exist for many a year without change. Only one in my experience became malignant and this one recurred after I thought I had completely removed it.

Pathology. They form an ovoid tumor low on the upper lip, sharply outlined against the adjacent skin. The skin covering it usually has undergone some change so that it is irregular but not devoid of epithelium.

The tumor is, as noted above, well defined against the adjacent skin. The covering skin is thinned, reddish in color and irregular. The cross-section shows an epithelial mass extending above the general muscular layers of the lip.

Histology. The slide shows a conglomeration of glands.

Lymphangiomas of the Lip. These lesions are usually congenital in origin but may first be noted after the child is a year or more old and are

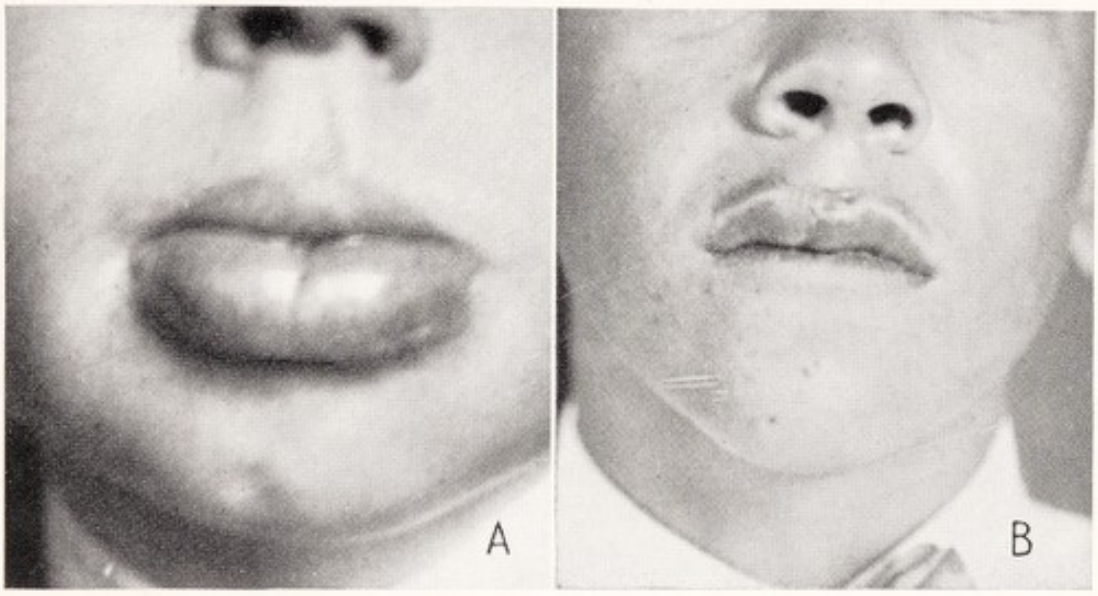

FIG. 24. Lymphangioma of the lips. *A*, M., age 15. Lower lip has been enlarged since infancy. No complaints. Wedgeshaped excision. Cured now 20 years. *B*, Dilated lymphatics of the upper lip following injury.

then likely to be ascribed to some injury. Some indistinct forms do follow injury because definite scars remain to mark the site of the injury.

Pathogenesis. The congenital type occupy the lip uniformly (*A*, Fig. 24), are soft, boggy to the touch, but are not reduced in size by pressure. The acquired type is usually small and there may be a firm area due to the scar of the injury, particularly if this has been recent (Fig. 24). Sometimes the scar forms the greater part of the lesion.

Pathology. The boggy compressible feel without reducing the size of the growth is characteristic. If cut into, the confined lymph escapes. The acquired variety may be made up largely of fibrous tissue after it has been hardened.

Histology. The slide shows the usually large cavities lined with a very flat epithelium. It is worthy of note that some of the smaller vessels from which the lymph cannot escape form cavities lined with a higher epithelium (due to contraction of the cyst wall) and filled with a homogeneous acidophilic material. This forms a structure suggesting a thyroid acinus but strangely enough these have not yet been reported as aberrant thyroids.

Hemangiomas. These lesions are always congenital and always deep red in color.

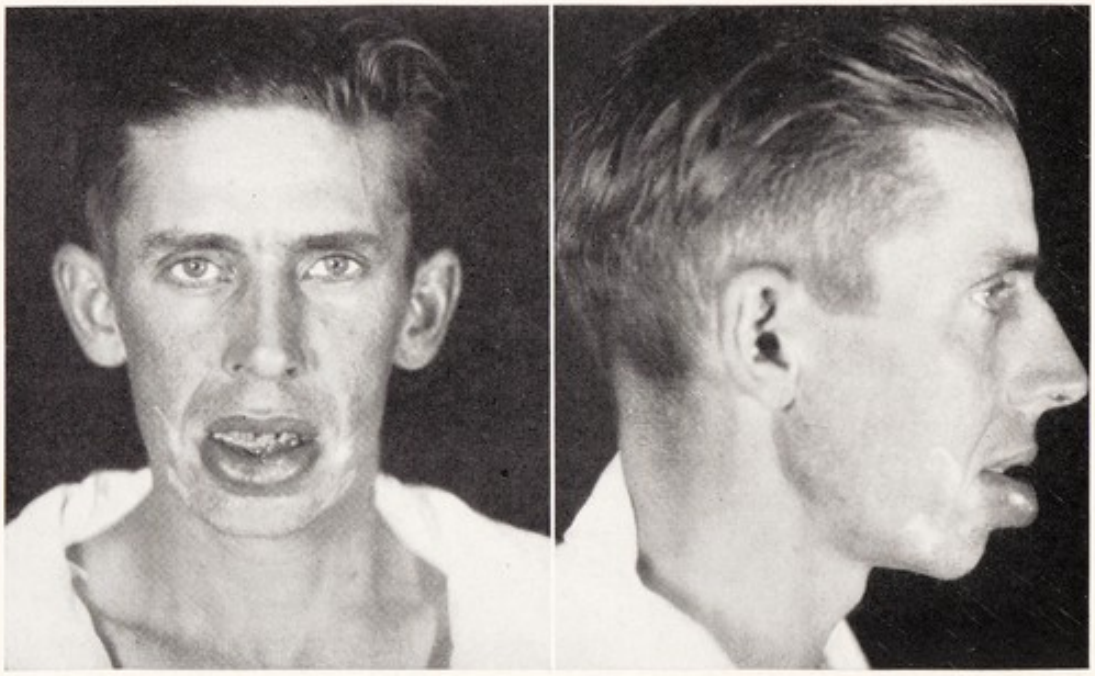

FIG. 25. Hemangioma of the face and lip. M., age 26. Capillary angioma of the face, venous angioma of the lip.

Pathogenesis. Obviously they owe their origin to some disturbance in the vascular apparatus. They are very soft and compressible, and, unlike the lymphatic counterparts, are reduced in size by pressure (Fig. 25). In children crying distends them.

Pathology. When cut into during the course of the operation, hemorrhage is diffuse and after the tissue has shrunk in a fixative there is not much to show.

Histology. Many vessels with flat epithelium. In the contraction these vessels undergo during the course of hardening they may become higher so that a section across a number of them may suggest a gland if the source of the tissue is unknown, and the examiner is in the throes of a mental lapse.

INFLAMMATIONS OF THE LIP

These lesions furnish very little of interest, unless, alas, the patient dies of sinus thrombosis. In some cases the entire lip is inflamed, in others the infection begins at a certain point while the localized lesion remains more or less circumscribed.

The Diffuse Variety. In the diffuse variety the entire lip swells simultaneously.

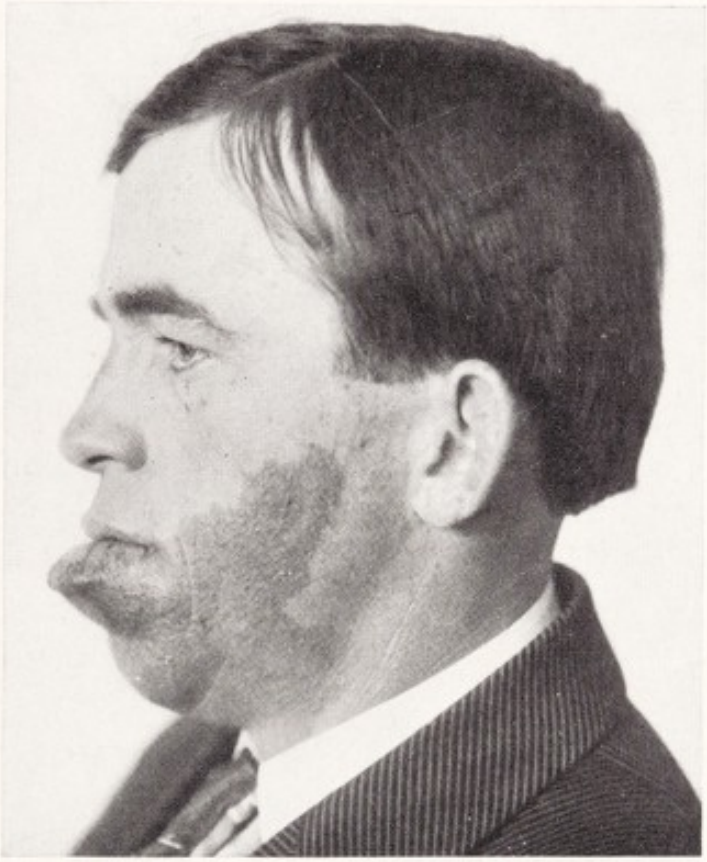

FIG. 26. Diffuse swelling following a superficial trauma. Spontaneous resolution.

Pathogenesis. An injury or a superficial infection is the starting point. A general edema of the lip causing it to protrude (Fig. 26) is evident. The infection does not concentrate at a local point and usually the process ends in resolution.

Pathology. The lip is thick and sensitive to palpation. Usually the adjacent lymph glands are enlarged and tender.

Histology. Slides of this lesion have not come to my notice but of course one could write an accurate description of the findings.

Localized Infection of the Lip. This type is usually found on the upper lip but may occur on the lower.

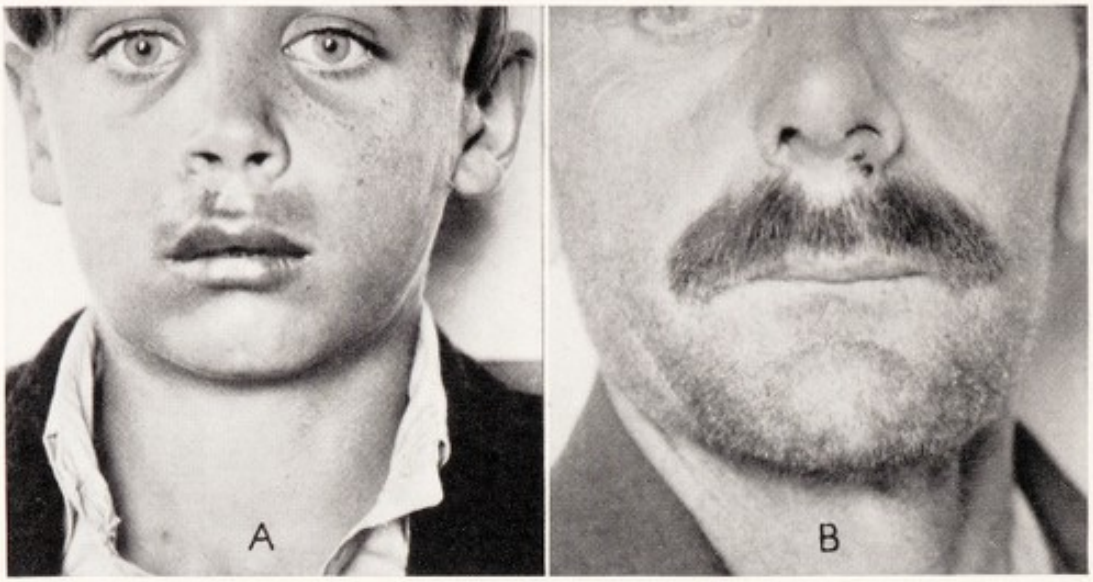

FIG. 27. Acute inflammation of the lips. *A*, Diffuse infection of the upper lip. The tiny lesion is visible at the upper border of the swelling. *B*, Boil at angle of nasal opening.

Pathogenesis. It begins commonly spontaneously as a small pimple, often too small for the camera to see, and remains such if not disturbed. Sometimes the well intentioned ministrations of a tonsorial artist may start the lesion on a wild development. The pimple which marks the site of infection despite its diminutive size may annoy the patient dispro-

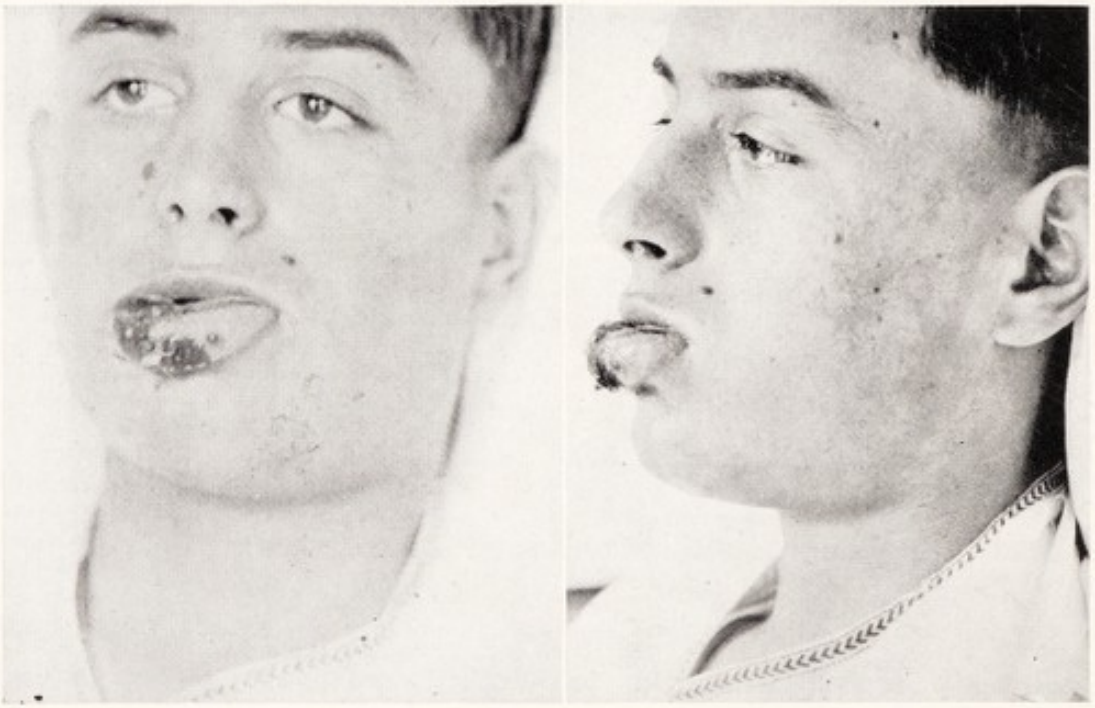

FIG. 28. Lower lip infection. Started as a small lesion. Treatment with salicylic acid and plaster has eroded the adjacent area.

portionately. This is fortunate, for the patient therefore is apt to apply for medical aid early. It seems such a small matter to incise it and thus relieve the patient. This unfortunate meddling may start the infection to ascend, ultimately involving the cerebral sinus, and lead to rapid death. The lower lip is much less commonly affected and is rarely followed by serious extension but a diffuse cellulitis may accompany it.

Pathology. A small infection with a yellow summit is all there is to see. This may be obscured by the swelling about it (Fig. 27). If the sinus becomes involved, induration along the lateral vein is demonstrable and constitutional symptoms rapidly develop usually with a fatal termination. The infections of the lower lip produce extensive swelling (Fig. 28) but usually result in resolution.

Histology. Usually the staphylococcus in pure culture is found in the little lesion—and in the sinus clot.

Literature

The bibliography of the lip is both multifarious and multitudinous and withal offers but little variety so well standardized is the general knowledge. Only a few papers need be cited, chiefly for the bibliography they contain.

Precancerous Lesions. Of interest is Babler's report (*Ann. Surg.*, 1908, 37:331) on a trichinous infection of cancer of the lip. I reported a case of trichina (Clinical Surgery by Case Histories, vol. I, p. 117, Mosby Co., St. Louis, 1922) which was attended by marked epithelial proliferation which might well have been called cancer.

Granuloma of the Lip. Goodman (*Bull. Johns Hopkins Hosp.*, Nov., 1932, 51:263–77) discussed the pathology of perleche. Karcher (*New England J. Med.*, Feb. 21, 1929 200:391) reported a case of combined chancre of the penis and lower lip.

Benign Tumors of the Lip. Boykin (*S. Clin. N. America*, Oct., 1929, 9:1229–30), described lymphangioma of the upper lip. Pilcher, R. (*Brit. M. J.*, May 8, 1937, 1:967–8) reported a "mixed tumor" of the lip.

Inflammations of the Lips. Shelmire, Diseases of the vermilion border of the lip, *South. M. J.*, March, 1928, 21:169–178; Curry, Infections of the lip, *J. Michigan M. S.*, June, 1928, 27:340–1; Coller and Yglesias, *Surg., Gynec. & Obst.*, Feb., 1935, 60:277–90; Brown, Grove and Pittman, Acute infections about the lips, *Internat. J. Orthodontia*, Nov., 1932, 18:1212–17; Pratt, Furuncle of the upper lip, *Am. J. Surg.*, April, 1937, 36: 118–21.

CHAPTER III

Malignant Tumors of the Lips

IN THE present commendable campaign against cancer, stress is laid on the importance of early diagnosis. The theoretical importance of this cannot be gainsaid but in our wage for vigilance we unconsciously imply that if the diagnosis were made early enough cure would be regularly achieved. This also is theoretically correct. We urge frequent pelvic examinations during the cancer age so that malignant lesions may be discovered early, which they rarely are, and mammary glands are becoming as much the object of attention at the end of mature life as they are in the beginning. All this is for the purpose of recognizing malignancy in its beginning.

The irony of all this vigilance lies in the fact that in lip cancers involving a region constantly under inspection, where, if anywhere, early diagnosis should be possible in its earliest stage, permitting treatment of the disease in its incipience, a favorable prognosis should be possible. Despite the favorable location, diagnosis is often not made early and even so the results are lamentably bad. Two factors enter here, the inherent negligence of the patient and the inherent viciousness of cancer in this situation. How bad the results really are in cancer of the lip is obscured by the ease of cure of the local lesion, too often accepted as evidence of the eradication of the disease. For this reason we have no accurate knowledge of the percentage of cures of five years or longer in cancer of the lip. It would seem, therefore, that the campaign should be directed first of all to the early diagnosis of cancer of the lip. It can be done modestly, and since lip cancers usually affect only men, it can also be done safely.

Too often surgeons seem to think a cancer is a cancer without giving heed to the location. That cancers vary widely in the degree of malignancy is often overlooked. Surgeons too often, in case they actually look at the pathologist's report, look only to the last line and if it says "cancer," they feel the diagnosis has been made. Nowhere is this failing more emphatically emphasized than in cancer of the lip. Likely, because of the great vascularity of the lip, and the tendency of the growth to be primarily invasive, the lesions here are among the most vicious of the entire body and the size of the lesion has very little to do with the actual stage of its progress.

Happily, much progress has been made in the early diagnosis and treat-

ment of cancer of the lip. This credit may be shared by patient and doctor alike. Thirty or more years ago, almost the only disease of the lips which reached the surgeon was carcinoma. Minor lesions were disregarded either on the patient's own evaluation, or lack of it, or on advice of the doctor. The result was that many of these had already reached the stage in which the entire lower lip was destroyed and extensive metastases were already present before they reached the surgeon, and then it made little ultimate difference if they ever sought consultation. So greatly has this situation changed that I must go back to old files of pictures to see the local ravages which the lesion can produce and did produce. Naturally, in that day, cancers of the lip were almost always fatal no matter how extensive the operation.

Some observations were made in that early day that have now been well nigh forgotten. We were enabled to study the natural life history of the disease uninfluenced by any treatment. We learned, first of all, that the size of the tumor made little difference in the prognosis and that the rapidity of growth and the spread of destruction depended on a variety of factors, notably age and constitution. We learned also that many of the frightful fungating local lesions existed even many years without gland metastases. In contrast with the huge fungated mass a tiny lesion requiring painstaking examination for its discovery may already be the parent of a numerous gland progeny. Those which are preceded by benign lesions are often of lesser degree of malignancy than those which begin as typical prickle cell carcinoma, particularly if the local ulcerations develop rapidly. These differences should find expression in any statistics relative to the efficiency of any method of treatment in cases of cancer of the lip.

Some especially skilled pathologists estimate the degree of malignancy of a given tumor by its cellular structure. This same degree of accuracy in prognosis can be achieved by a clinical study of the tumor and the patient. The factors noted above have an important bearing on the prognosis and should be included with the pathologic report in any estimate of prognosis. The form and mode of growth are usually not available to pathologic histologists, and they are compelled to depend on cell form in estimating the relative malignancy of the lesion. The ideal is reached when pathologist and surgeon combine their efforts.

In contrast with the days gone by, when the patient displayed his diagnosis, nowadays one's problem is to differentiate early malignant lesions of the lip from those that are not malignant or not yet malignant. As complained of in the chapter on nonmalignant disease of the lips, the problem is much confused by the nonsurgical treatments applied to all

lesions without evidence as to whether they are malignant or not. It cannot be too strongly emphasized that when one cures something, or claims that he has cured something, he should be able by means of the slide to show the skeptic just what he has cured. No report of a cure of malignancy of the lip should be accepted which is not accompanied by microscopic evidence that the lesion was actually malignant. Even with the aid of the slide it may be necessary to observe the patient for a period of years to see what becomes of him. He may enter a minority report and spoil the whole fine diagnosis of the surgeon by dying of a recurrence after diagnosis of a benign lesion.

In the previous chapter I took occasion to deplore and condemn the present tendency to treat all lesions of the lip by the same agent. I believe everyone who essays to treat diseases of the lip should make a pact with Fate indicating just what treatment he shall elect when he himself becomes the victim of a lesion of the lip, whatever its alleged nature. Would he want to remain in doubt or would he wish to know just what ailed him? Such knowledge can be achieved only by a microscopic study of the lesion. If malignant, would he want to take a chance of a permanent cure by its removal, or would he be satisfied with the disappearance of the lesion with the possibility of covering up the local lesion by palliative treatment?

Local recurrences are so rare and gland manifestations may be so late that he is indeed fortunate who has not at some time mistaken the gland metastasis for the primary lesion. Some patients give a misleading history for the purpose of concealing the fact that they have submitted to a method of treatment which they feel reflects on their intelligence in the selection of their previous adviser, as was noted in the preceding chapter. Some patients, too, have been so eloquently assured that their lip lesion has been permanently cured that they actually believe the neck lesion is an independent disease. Some of the measures now employed to cure the local lesion leave no local scar and if the history is deliberately misstated, the surgeon must be able to determine that the neck lesion is an epithelial metastasis by the exercise of his wits. The density of the metastasizing glands is such that a very small exercise of judgment will detect their nature. In this connection I once sat in on a consultation which furnished some amusement. The patient was a large person who sported an astonishingly abundant growth of whiskers. He had a mass beneath his lower jaw as large as a baseball. The first examiner, a surgeon of national reputation, diagnosed a sarcoma. This was in large part dependent on the fact that the patient declared he had no previous lesion of the lip. The second examiner palpated the neck mass,

noting its nature, and began at once to explore the landscape covered by the hirsute. This examiner, pointing to a scar these researches revealed, asked when the patient had had the cancer removed. The patient replied that he had a little growth removed two years previous by a paste, but that it healed promptly so that he knew it was of no importance, hence his negative reply when asked about a previous lesion.

Too many cases even yet are encountered which have reached the stage of not only undoubted malignancy but bearing evidence of gross neglect because of wholly inadequate treatment dependent on an incorrect diagnosis. The term "border-line lesion" is too often employed. It has a different meaning to each observer. Therefore, it cannot be too strongly urged that every tumor of the lip be positively identified, whatever may be required to secure this end. In case of doubt, the growth should be totally removed by a small cautery or excised in such a manner that the whole of it is available for microscopic study. It cannot be too strongly urged that the whole of it should be removed. If this is not done, no one should blame the pathologist if a wrong diagnosis is recorded. Destroying a lesion without identifying it is to be condemned, no matter how innocent the lesion may appear to be.

It may facilitate the understanding of the life history of cancer of the lip to study the various types of beginning. One may accept as typical those that begin as circumscribed local lesions without a previous affection. These may be called primary. In contrast to these are those cancers which are preceded by one of the several nonmalignant lesions discussed in the preceding chapter. These, then, are secondary lesions, the cancer being implanted on a previous nonmalignant state; a keratosis, a chronic benign ulcer, a horny wart. These, on the whole, are less malignant than those that begin as frank carcinomas. In addition, there are other interesting variations from the usual type and course, such as recurrent lesions, cancers in the young, the corpulent, and cancer of the upper lip.

These may be discussed according to the following outline:

I. Primary Cancer of the Lip
II. Secondary Cancer of the Lip
    Secondary to Keratosis
    Secondary to Chronic Ulcer
    Secondary to Papillomas and Horns
III. Unusual Conditions
    Cancer of the Upper Lip
    Cancer of the Lip in the Young
    Recurrent Carcinoma of the Lip

PRIMARY CANCER OF THE LIP

The primary cancer, as above noted, is one which develops without a previously existing benign lesion. It is a localized lesion which in its earliest manifestations bears all the evidence of malignancy and nothing else. That is to say, the patient noticed no preceding lesion which bars us from any information as to the first stages. The important point is that an exceedingly small lesion of the lip is already malignant.

As noted above, there is no region of the body better suited for the study of the genesis of malignancies than the lips. It would seem that in a region so exposed all lesions would be discovered so early that all would be subject to removal before they get out of bounds. It cannot be too

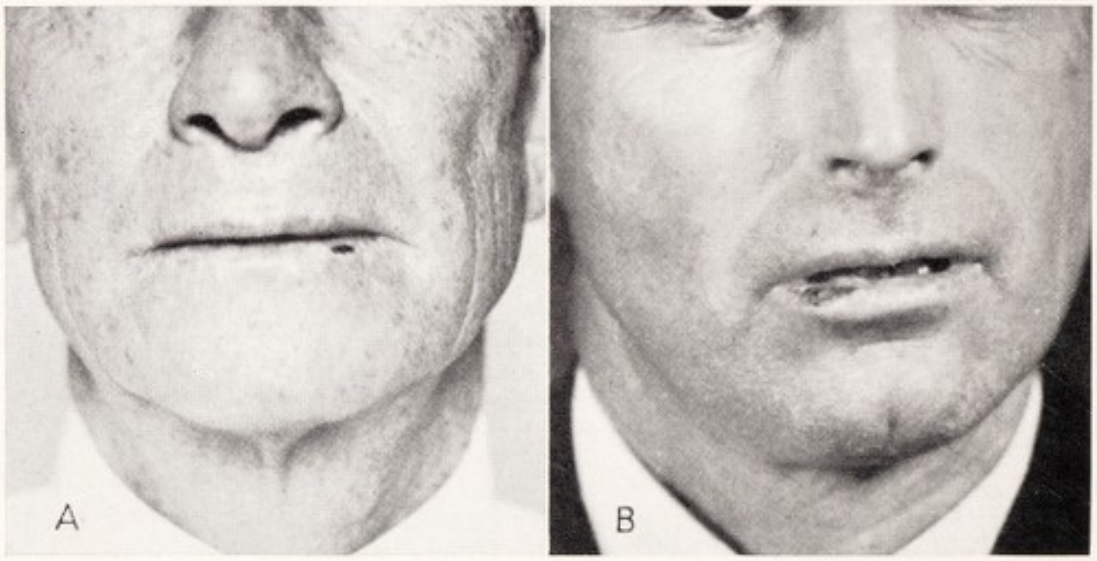

FIG. 29. Early circumscribed ulcerating cancers of the lip. *A*, Early stage when the invasion is less than a millimeter in depth. *B*, Slightly more advanced lesion, still superficial.

often urged that we are still a long way from achieving this end. It is cheering to note that now many sufferers from lip cancers are coming to the surgeon as soon as a lesion is discovered. Perhaps office girls should be appealed to if we hope to secure the patients at an earlier stage of the disease. In fact, the only patients one sees now with advanced lesions are adherents to some cult who expect to be cured by magic or hokum, or the patient is very stupid, which is but restating the same obvious fact. If the surgeon will meet the patient half way the prognosis will be vastly improved.

Pathogenesis. Of course, the cause is unknown, but since cancer of the lip is so much more common in men than in women it goes without saying that it is due to some moral dereliction.

In many cases invasion occurs before the superficial epithelium is destroyed. For this reason, a lesion may have reached some degree of extension before the most observant person becomes aware of its existence. The earliest lesion I have ever seen appeared as a small ulcer reported to have been of only a week's duration (*A*, Fig. 29). Even so, the base was hard and pressure by a slide made it possible to see small cell nests about the periphery. Even so, a confirmation by a slide was a great comfort. Usually the lesion has attained a greater size before a surgeon is consulted so that a mere feel with the fingers establishes the diagnosis (*B*, Fig. 29). Yet even this small lesion had the hardness of unmistakable

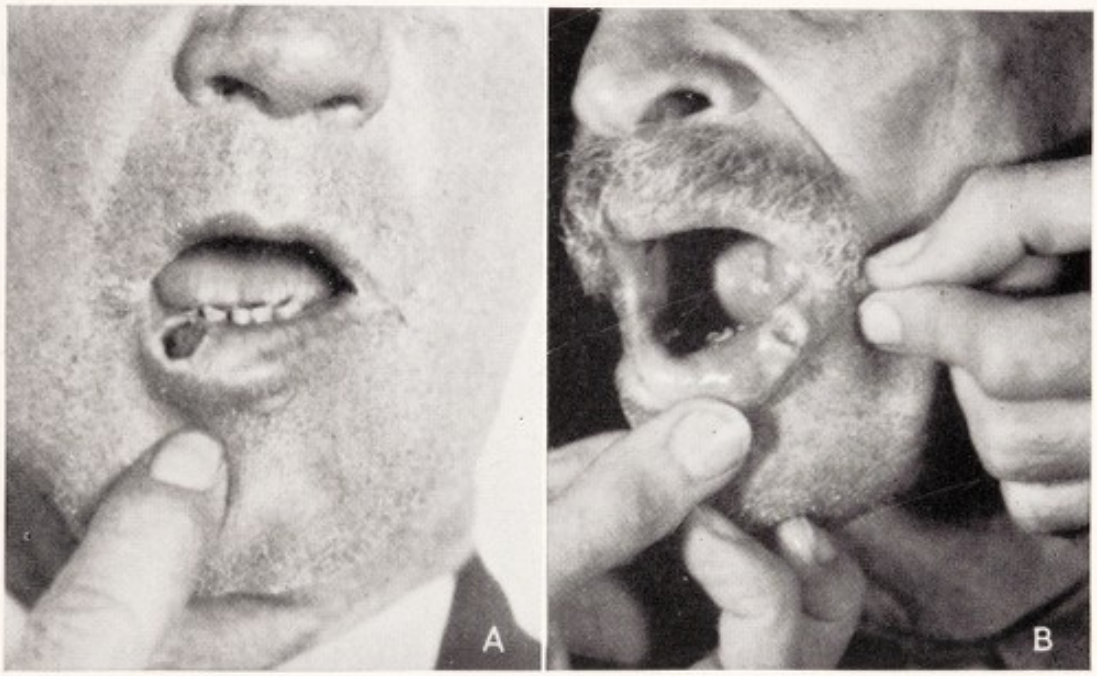

FIG. 30. Well developed cancer of the lip. *A*, The lesion is a centimeter in diameter; the border is elevated and hard. *B*, The lesion is less in extent but deeper. Each lesion on palpation felt as large as a hazelnut.

malignancy. The average case when it comes to the surgeon is larger than those above depicted. The lesion is a centimeter or more in diameter, is slightly elevated about the border, depressed in the center and shows on the surface the fine white pin points of epithelial nests (*A*, Fig. 30). In some cases the lesion is less in diameter but correspondingly deeper (*B*, Fig. 30). To the touch the tumor is larger than it looks and is cancer-hard. These early lesions appear as hard discs in the surface of the lip. Nowhere is the adjacent tissue indurated. These are truly early lesions in most cases.

The lip is an excellent site for the education of the touch for the perception of the peculiar density known as cancer hardness. For the stu-

dent there is no other region of the body where this peculiar hardness can be so well studied as in early cancer of the lip because here very small lesions present themselves for study. It has been likened to the firmness of the heel of a rubber boot. It has the added advantage, since both these means of education should be augmented by feeling and looking at the lesion in the laboratory before, of course, the slide is examined. Here sight can, in a measure, aid his education of the sense of touch.

Not infrequently lesions are much larger when first presented for examination (*A*, Fig. 31). The cause for delay in these cases can usually be ascribed to the fact that some mild measure has been suggested as

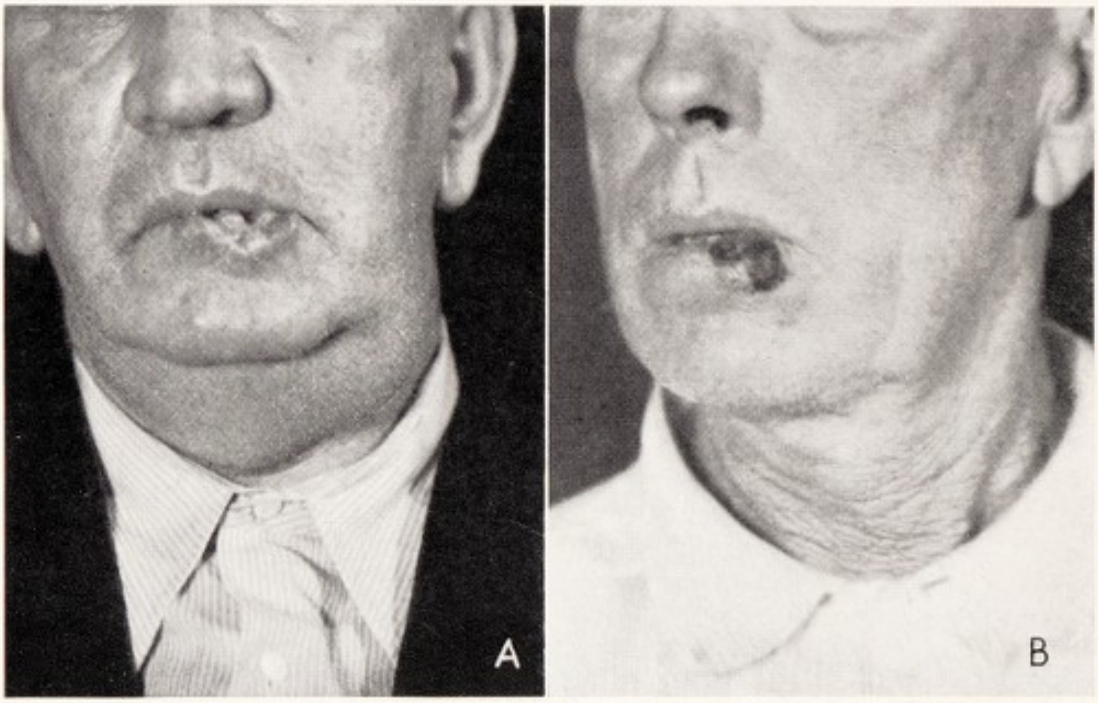

FIG. 31. Well developed carcinomas of the lip. *A*, A distinctive ulcerative process has destroyed half of the edge of the lip. *B*, A nodular protruding mass forms the tumor.

treatment or someone has regarded the lesion as nonmalignant and advised delay. Usually, in such instances, the ulceration is much extended, or the lesion may become elevated above the surrounding surface (*B*, Fig. 31).

Unusual sites of development may cause some hesitancy in the diagnosis because there may be a history of a recent illness. This is observed most often at the angle of the mouth (*A*, Fig. 32). Small lesions may develop beneath the epithelium and still be covered by a layer of epithelium (*B*, Fig. 32). These lesions appear as hard, deeply seated lesions. Their density is characteristic and there is usually sufficient change in the epithelium to show that the surface is being approached. The history

may suggest that the process is an acute one, due to an injury or to some attendant disease. The patient is unaware of the presence of a lesion until some event directs attention to it. Of course, his history dates from the time of discovery.

Cancers beginning without a preceding lesion can nearly always be diagnosed as primarily malignant by the senses of sight and touch. These uncertain states should never find expression as precancerous. It is, from the beginning, the one or the other. In order to achieve a diagnosis in the early cases the surgeon's esthetic sense must be depressed to such a degree that he does not disdain to touch the diseased lip with the bare ungloved finger. The young surgeon should not delude himself. The finger cannot be educated to the diagnosis of cancer through a layer of rubber. Bare finger or frozen section, there is no alternative.

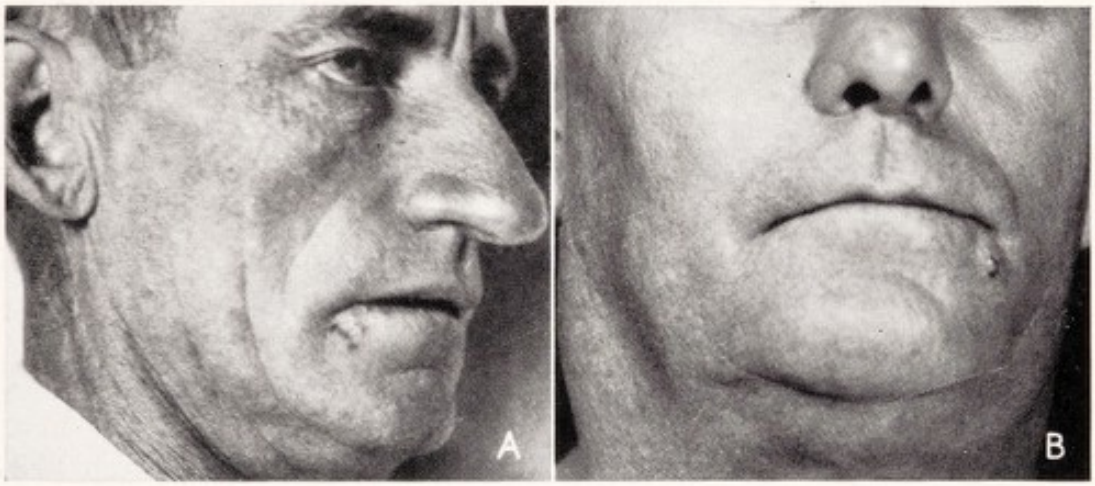

FIG. 32. Early cancers developing at the angle of the mouth. *A*, Carcinoma beginning as a fissure. *B*, Small deep-seated malignant lesion showing chiefly below the vermilion border.

In most cases microscopic examination is not needed. If in doubt excision of the entire growth should be practiced and the tumor examined at leisure. If it is clinically indistinct, a frozen section may but confuse the picture by making the slide from a region that is not expressive of the most offensive part of the growth. Excision may be accomplished either with the knife or cautery. If the cautery is employed care must be exercised lest the characteristic area of the lesion be destroyed, leaving nothing for adequate histologic examination. It is only by the constant comparison between tactile and microscopic evidence that both the touch and eye are educated to the detection of early malignancy.

Even such joint examinations, finger and slide, may go wrong. I have seen lesions which did not appear malignant either to touch or micro-

scopic examination yet showed gland metastases, sometimes not until years later.

Having traced the early life history it remains to take a look at the subsequent course. Fortunately, many are localized when malignancy is first discovered. These local lesions destruction or excision cures. Examination of the local lesion does not tell us whether metastasis has taken place or not. No matter how small the primary lesion may be, we must search the region of the lymph drainage. If none are palpable, we can only say none are palpable now. The future must answer for itself.

The subsequent course after local excision, in rare cases, may be local recurrence. In most instances the local lesion is permanently eliminated. It is the recurrence that announces the failure to cure. Recurrence is manifested in gland metastases, usually in the adjacent lymph glands.

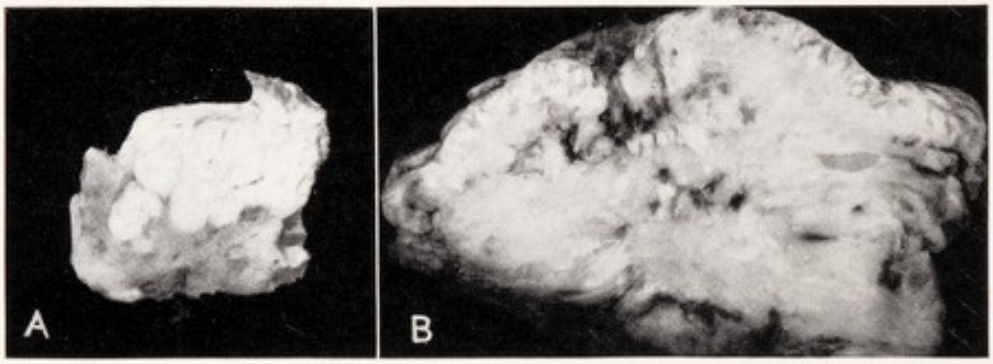

FIG. 33. Cross-sections of a carcinoma of the lip. *A*, The chief tumor mass seems well circumscribed but tiny outlying nodules equally circumscribed are in evidence. *B*, There is but little invasion about the deep ulcerous lesion but the large indurated mass shows many fine points of invasion.

The glands enlarge, adjacent tissues are invaded, ulceration of the skin may result, usually attended by mixed infection. Deglutition is interfered with by infiltration of the loose tissues of the neck and the attendant edema. Emaciation grows apace and happy is he when deglutition-pneumonia brings him relief. Late in the disease massive hemorrhage may close the scene.

Pathology. In order to trace the life history of the disease it has been necessary to discuss simultaneously the pathology to a certain extent. As a matter of fact, the clinic reveals about all there is to know about pathology. In block dissections of the neck glands not palpable in the clinic may be revealed. Even if no glands are palpable during the course of block dissection, it is not evidence that none are present. If there are metastases present the disease is beyond cure, in my experience.

The cross-section of the excised cancer shows a tumor more circum-

scribed. Usually there are small rounded nodules associated with the primary lesion (*A*, Fig. 33). When the clinical examination suggests a larger tumor than the growth seems, it is due to the round cell infiltration about the lesion which is transmitted to the finger but not to the eyes.

On the other hand, what seems to be a large tumor in the clinic shows but little involvement in the cross-section (*B*, Fig. 33). The sense of size imparted to the touch is due to the induration of the adjacent tissue and as such gives evidence of the excessive malignancy of that particular lesion.

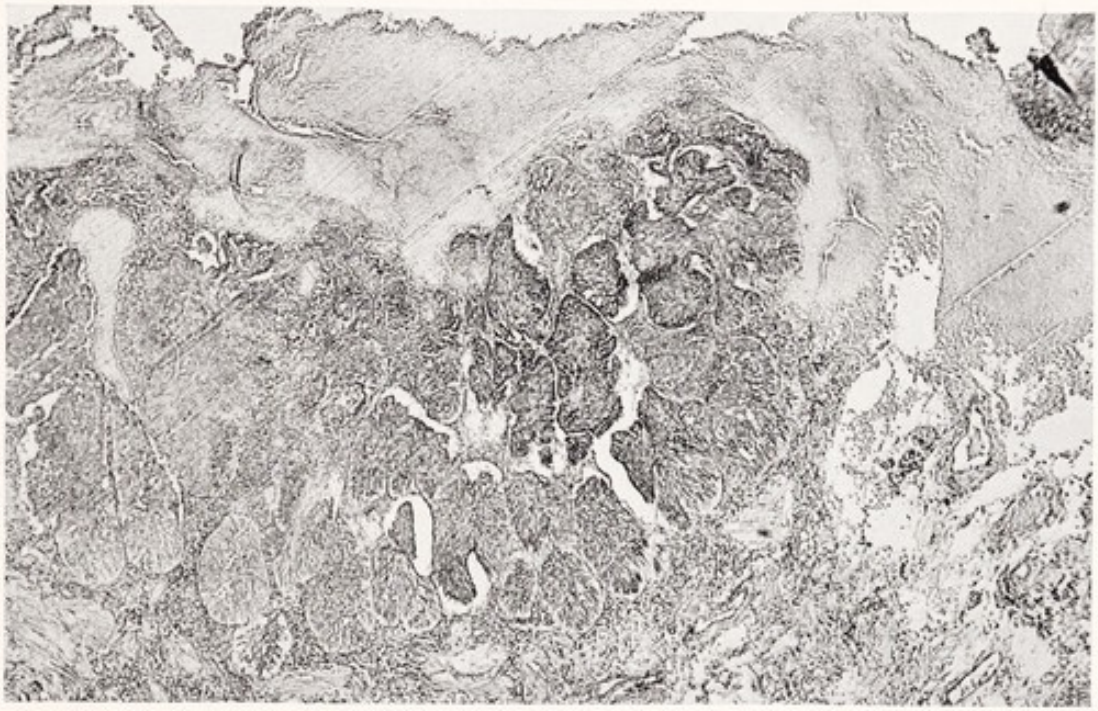

FIG. 34. Early epithelioma of the lip. The small superficial ulceration is breaking down at its summit. The superficial epithelium has been destroyed. The invasion is by mass formation of epithelium surrounded by round cell infiltration without evident epithelial invasion but with definite nest formation.

Histology. Early cancer usually shows a diffuse proliferation of the epithelium, presenting toward the underlying connective tissue a blunt protrusion. This blunt protrusion is surrounded by a diffuse infiltration of small round cells. No cells may be found invading this area but the change in cell type of the epithelium, accompanied by round cell infiltration and changes in the tinctorial reaction of the connective tissue is evidence sufficient that they are on mischief bent (Fig. 34). Other cases show a diffuse pearl formation even about the smallest lesion (Fig. 35), but may be found at astonishing distances from what seems to be a small lesion, even as far as metastases in lymph glands. Cancer nests are usu-

ally found near the initial lesion; with succeeding generations of tumors these are usually lost and a more or less diffuse infiltration of epithelial cells may signalize rapid invasion.

SECONDARY CANCER OF THE LIP

The term "secondary" is used here in the sense that the malignant lesion is implanted on a known benign one. This distinction is justified because it emphasizes the need of scrutinizing every lesion of the lip, no matter how simple it may appear to be, in order to determine its possible

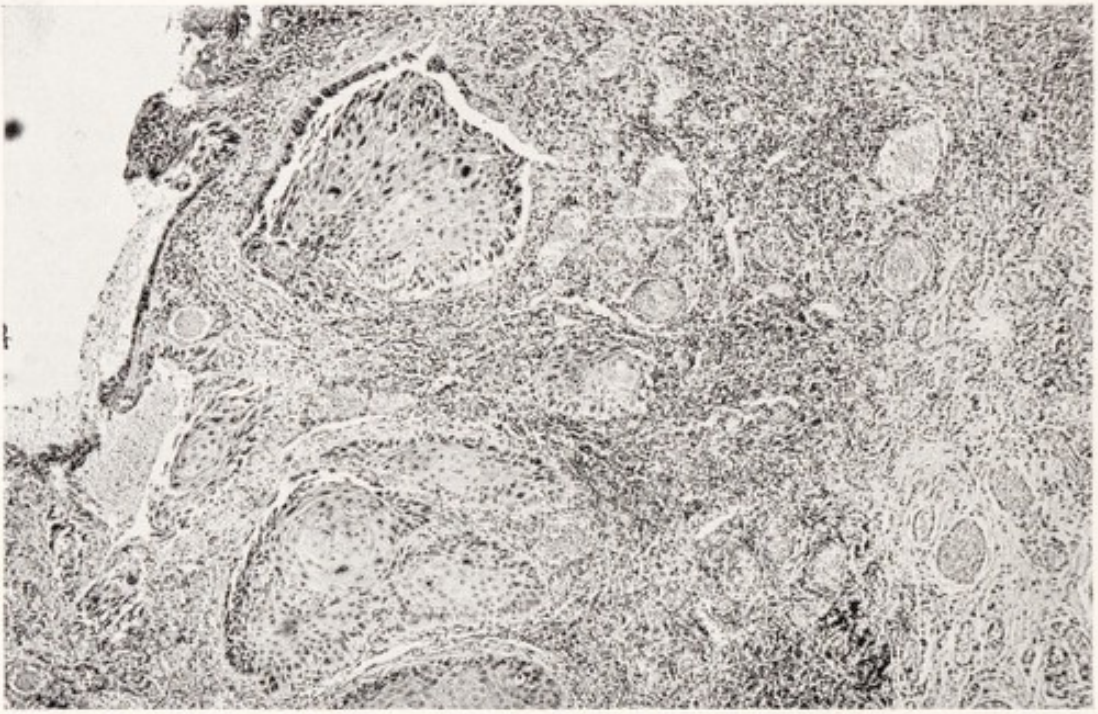

FIG. 35. Early epithelioma of the lip. The surface epithelium is being cast off but there is complete loss of covering epithelium. Beneath are many pearls and diffuse epithelial invasion surrounded by round cell infiltration. The connective tissue has lost its acidophilic reaction.

approach toward malignancy. In fact, every lesion of the epithelial surface of the lip should be looked on, not as innocent, but at best as not yet malignant. Hence none are so simple but that identification and annihilation are in order.

Then, too, there is a difference in that the cancers which have developed on a benign lesion in many cases give a better prognosis than those which begin at once on their malignant course.

In discussing these secondary lesions we may begin where we left them in a previous chapter when discussing their nonmalignant stages.

Malignancies Implanted on Keratoses. Keratoses commonly exist relatively unchanged for a period of years. Those that extend across the greater part of the lip are less likely to become malignant early than more circumscribed lesions. When they do pass into malignancy it is at one point usually, but the entire lip is destroyed simultaneously in rare cases. When keratoses manifest malignancy they do so, naturally, by the downgrowth of the epithelium, usually after several years of palliative treatment. They are always potentially malignant and should be regarded as such.

Pathogenesis. When malignancy begins in the tissue beneath the keratotic cell layer the lesion becomes harder to the touch. The next

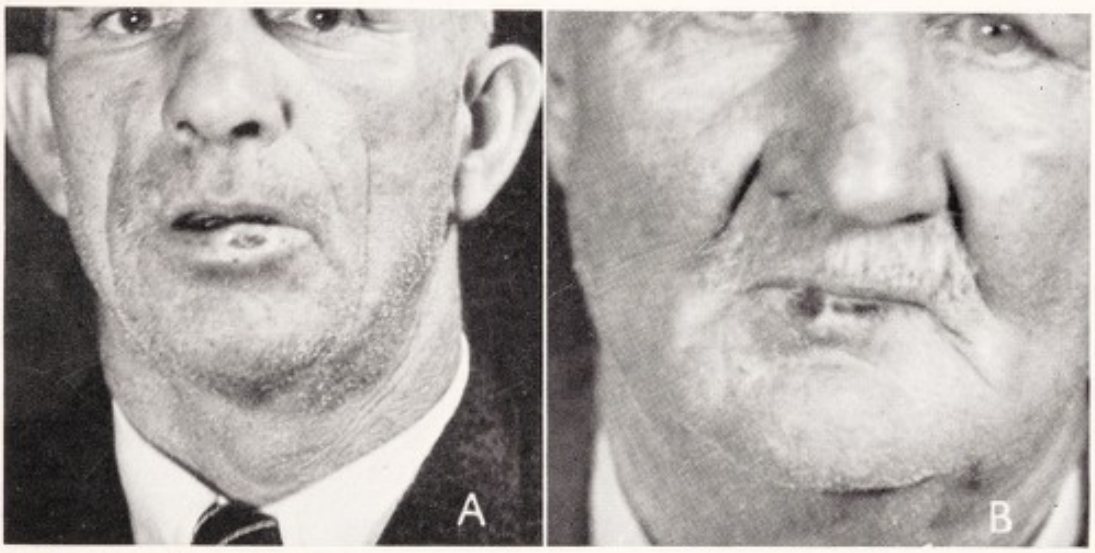

FIG. 36. Early malignancies implanted on keratotic lesions: *A*, localized area which has become malignant; *B*, the entire lip was transformed into a hard indurated mass.

step is for the epithelium to be cast off and a granulating surface is exposed (*A*, Fig. 36). This process may deepen and the localized indurated mass increases until a definite tumor-like nodule has formed (*B*, Fig. 36). The whole keratotic area may become simultaneously or successively involved until the entire lip is converted into a hard surface from which the keratotic appearance is lost (*A*, Fig. 37). The induration may be such that the tumor is much more impressive to touch than to sight (*B*, Fig. 37).

Cancers developing on keratoses may exist for a long time and produce large angry lesions before metastases are formed. On the other hand, sometimes metastases form even before there is positive evidence that the local lesion has become malignant. Because of the superficial nature of the lesion, this type has been a variable, or at least a favorite

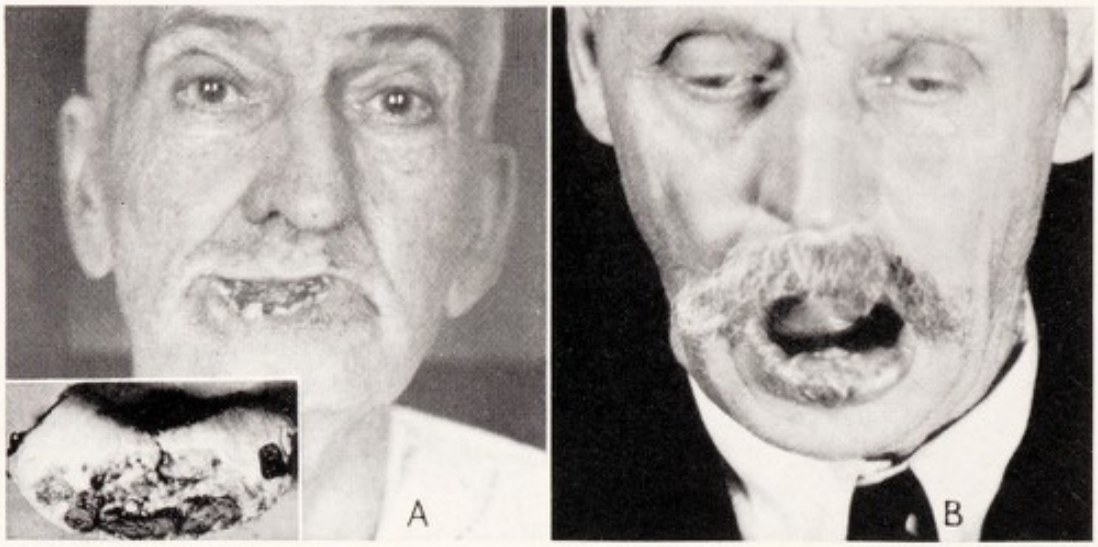

FIG. 37. Carcinomas developing in keratoses of the lip. *A*, Local transformation into malignancy. *Insert*, The growth seen on its surface. *B*, The entire lip was transformed into a hard indurated mass.

object for treatment by irradiation. No doubt many are cured by this means, but many others produce but a superficial cure, and the growth extends beneath.

Pathology. The hardness in the thickened or ulcerated area is typical once the lesion has extended to a malignant degree. However, a keratotic

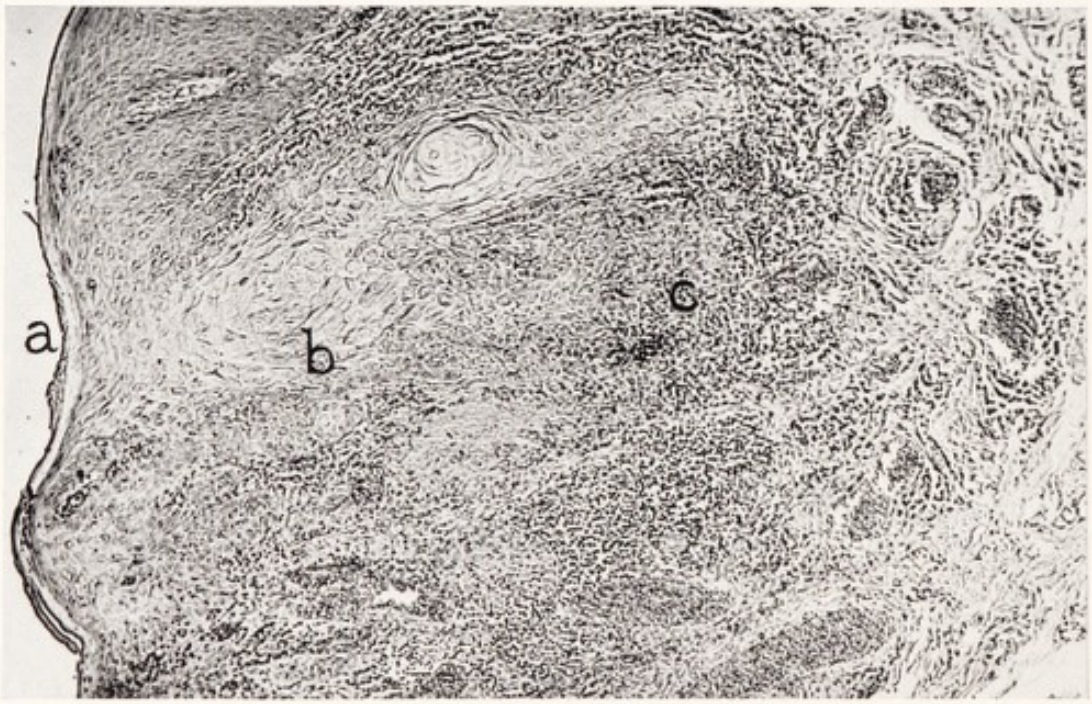

FIG. 38. Early malignancy in an early keratosis: *a*, a thin layer of intact epithelium; *b*, active epithelial proliferation; *c*, extensive round cell invasion of the superficial tissues. The lymphatics at the base of the lesion are distended with round cells.

area is as malignant as its most extensive invasion. Careless palpation of the entire lesion may fail to detect one small area that has reached a malignant stage, yet the slide may show such to be present. The cross-section may show only a thin sheet of malignant change.

Histology. From the foregoing it is evident that the cellular invasion may occupy only a small area of the total lip involvement. Hence repeated sections may be needed in order to gain a comprehensive picture. This means that the entire suspicious area must be available for microscopic study. The cell invasion may not be great. The surface

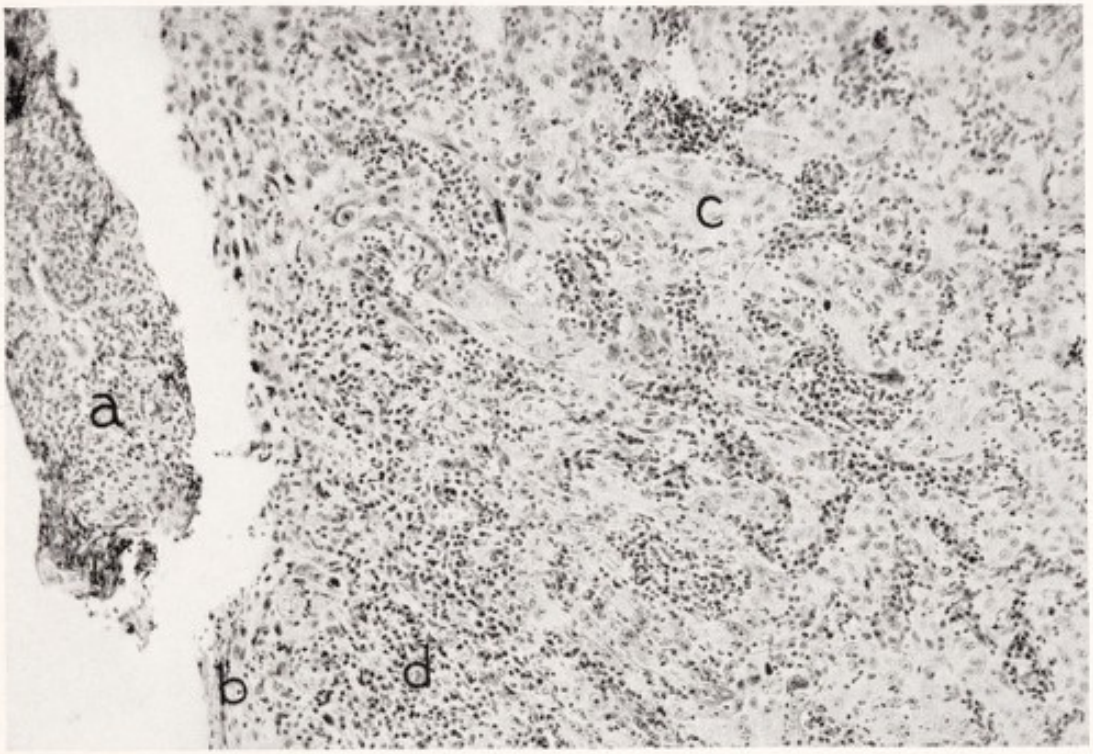

FIG. 39. Malignant keratotic lesion: *a*, a mass of keratotic cells has been dislocated in the course of sectioning; *b*, surface cells at the border of the lesion still in position; *c*, epithelial cell invasion distributed more or less diffusely; *d*, round cell infiltration.

epithelium may still be intact but from it there is a slight downgrowth of cells, adjacent to which is an extensive round cell infiltration (Fig. 38). All this makes up a mass scarcely palpable in the clinic. Once the lesion is definitely malignant to palpation, more extensive epithelial cell invasion is usually in evidence, malignant without a doubt. The cells may be distributed about a wide area. This type is likely to form early metastases (Fig. 39).

Cancer implanted on a Chronic Ulcer. The primary cancers of the lip begin as localized lesions but there is no history of a long existing ulcer. Cancer developing on a previously existing benign ulcer presents a history of a preexisting lesion.

Pathogenesis. As noted in the preceding chapter, chronic ulcers should always be regarded as malignant until proven innocent. In other words, all ulcers should be made available for microscopic study. To destroy them without such study leaves the surgeon in doubt as to what he has cured, if this happy result ensues. Usually when a chronic ulcer becomes malignant the transition is manifested by definite hardness which is not a part of the picture of a benign chronic ulcer. However, clinical diagnosis is not sufficiently accurate for statistical purposes; therefore,

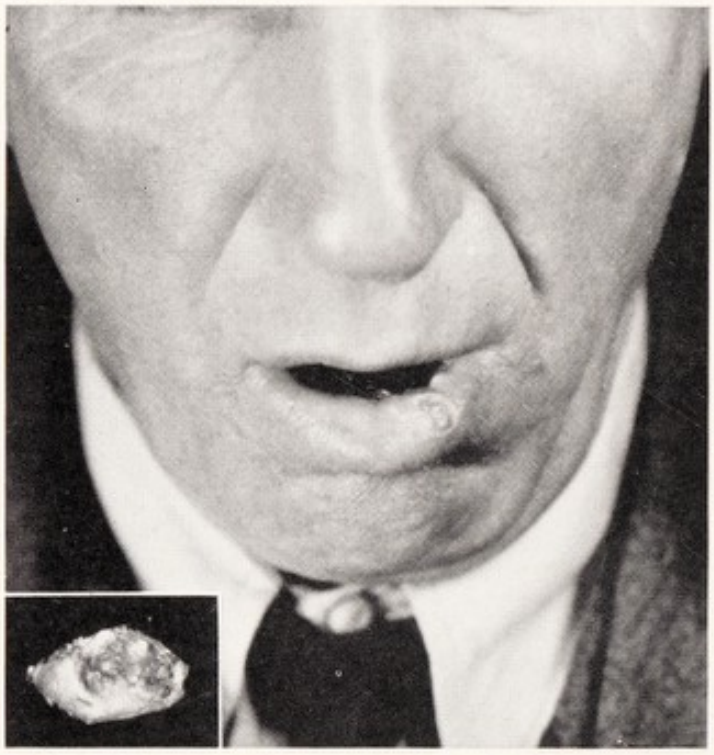

FIG. 40. Early malignant stage in a chronic ulcer. The insert shows cellular activity in the center of the lesion. This mass was definitely of cancer hardness.

though its clinical status is sufficiently accurate for practical purposes, its pathologic status for statistical purposes must be established in the laboratory. The duration of the innocent stage varies greatly. I have known them to exist many years before definite malignant change was noted. One patient presented a lesion which he stated had existed for 18 years, but had advanced to the present stage in the year just past (Fig. 40). It was then painfully definitely malignant.

Pathology. In the earlier stages, the lesion presents a superficial ulcer which forms the thinnest possible disc of hardened tissue (Fig. 41). These, without the history of a pre-existing lesion, could not be distinguished from primary malignancy. The next stage shows the lesion deeper, presenting just more of the same, but then may be diagnosed by

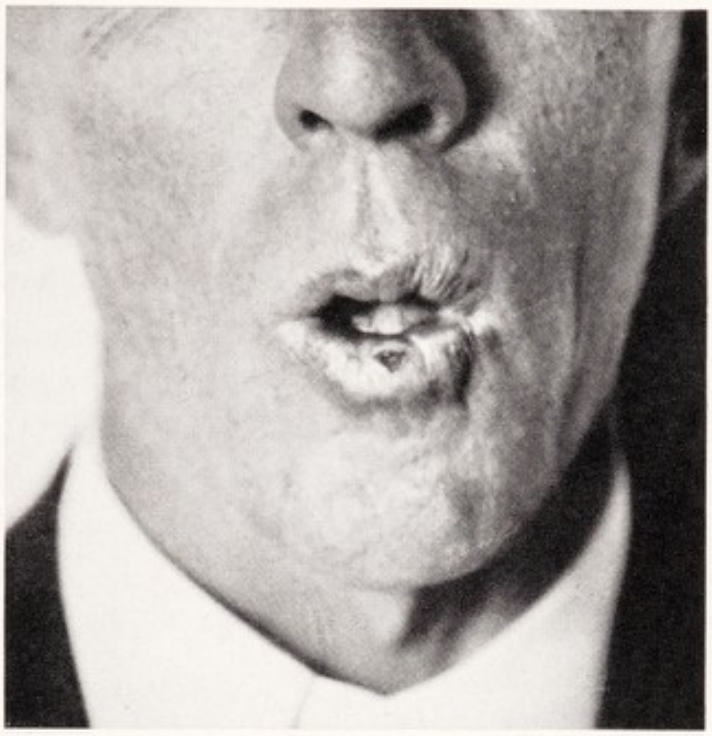

FIG. 41. Cancer secondary to ulcer. The lesion is superficial.

inspection and palpation (*A*, Fig. 42). This type is more prone to form early metastases than those implanted on keratoses. One seldom sees them so early. Usually, there is not only a history of a preexisting ulcer but also that the ulcer has extended more rapidly in recent months (*B*, Fig. 42).

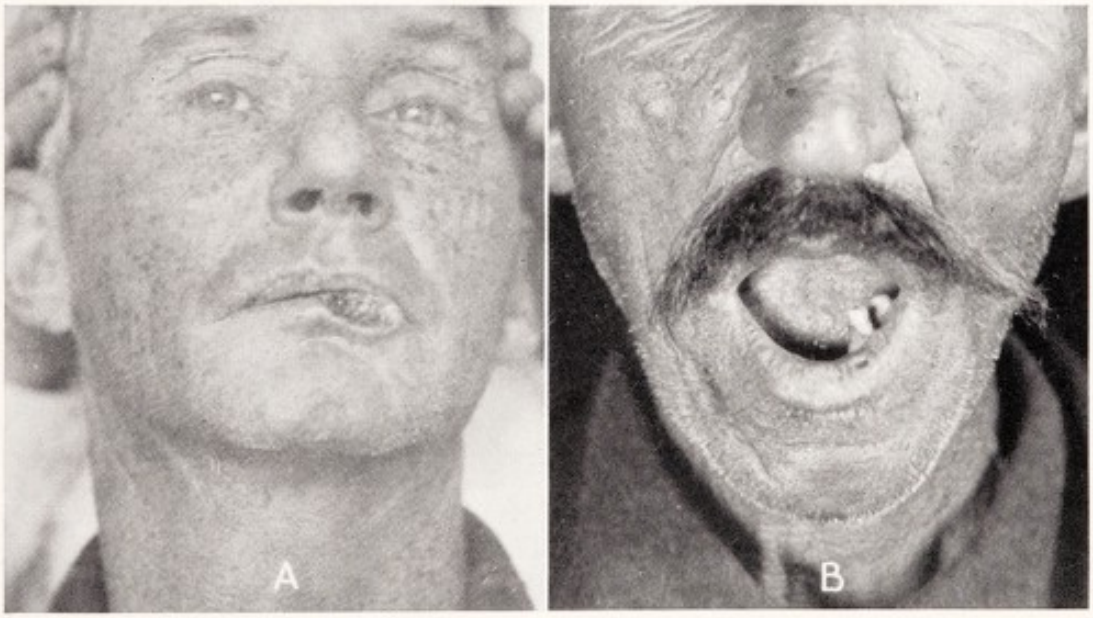

FIG. 42. Carcinomas of the lip developed on ulcer: *A*, the ulcer was caused by a tooth; *B*, the ulcer existed a number of years but had extended in recent months.

Early metastases occur most commonly in small fissure ulcers (*A*, Fig. 43). Perhaps it is the movements such lesions are subject to which form early malignant transition. These, not uncommonly, are associated with metastases when the patient first presents himself for examination. It may be the neck tumor that excites the curiosity of the patient sufficiently to cause him to seek medical advice. This advice may be ignored until the local lesion has begun invasion on its own account (*B*, Fig. 43).

Histology. The earliest changes are found in the thickening of the epithelium about the border of the ulcer which comes to involve the entire base. These may exist as slowly growing ulcers and the reason for

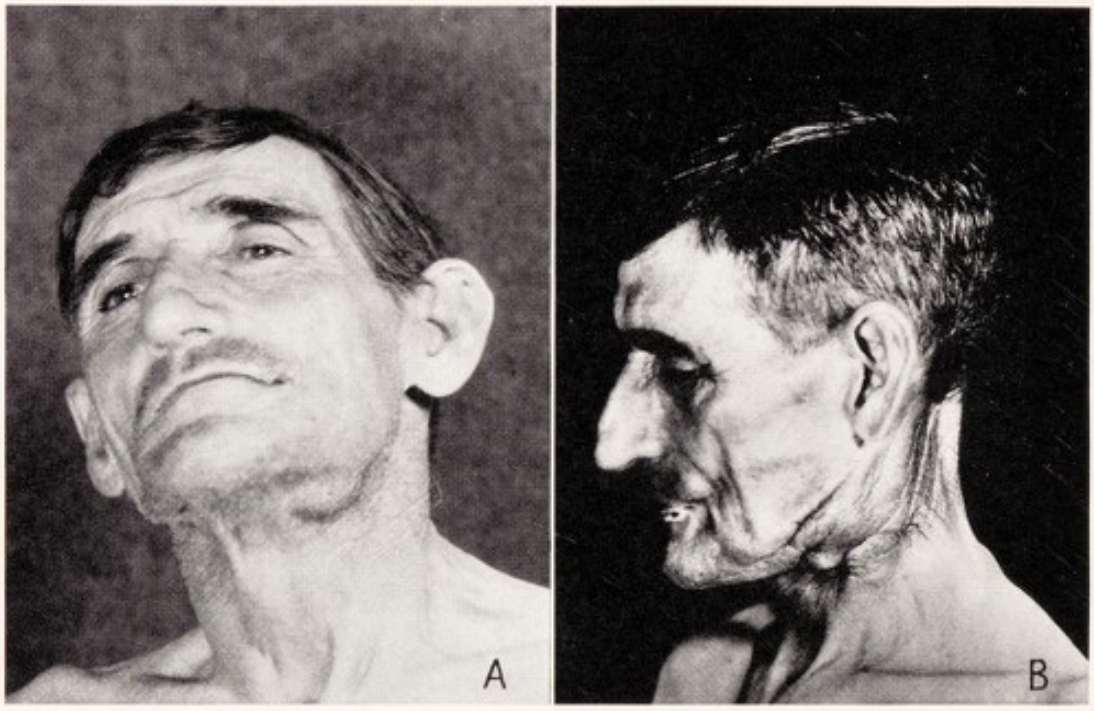

FIG. 43. *A*, Malignant fissure which has already produced an extensive metastasis. *B*, More extensive neck invasion from a chronic ulcer developed in a leukoplakic patch. There is a keratotic area on the lip in addition to the very small ulcer at the angle of the mouth.

this is seen in the close resemblance of the cell nests of basal-celled ulcers of the face (Fig. 44).

Malignant Papillomas. Under this head may be included all elevated lesions which have a benign stage and later become malignant.

Pathogenesis. These tumors may begin as simple warts or as is less common the early lesion begins as an ulcer and later forms a protruding epithelial mass at some point of its border. Malignancies may form about the base of cutaneous horns. The harder the horn the less likely it is to become malignant. The smaller ones showing a lesser degree of hardening are more apt to become malignant.

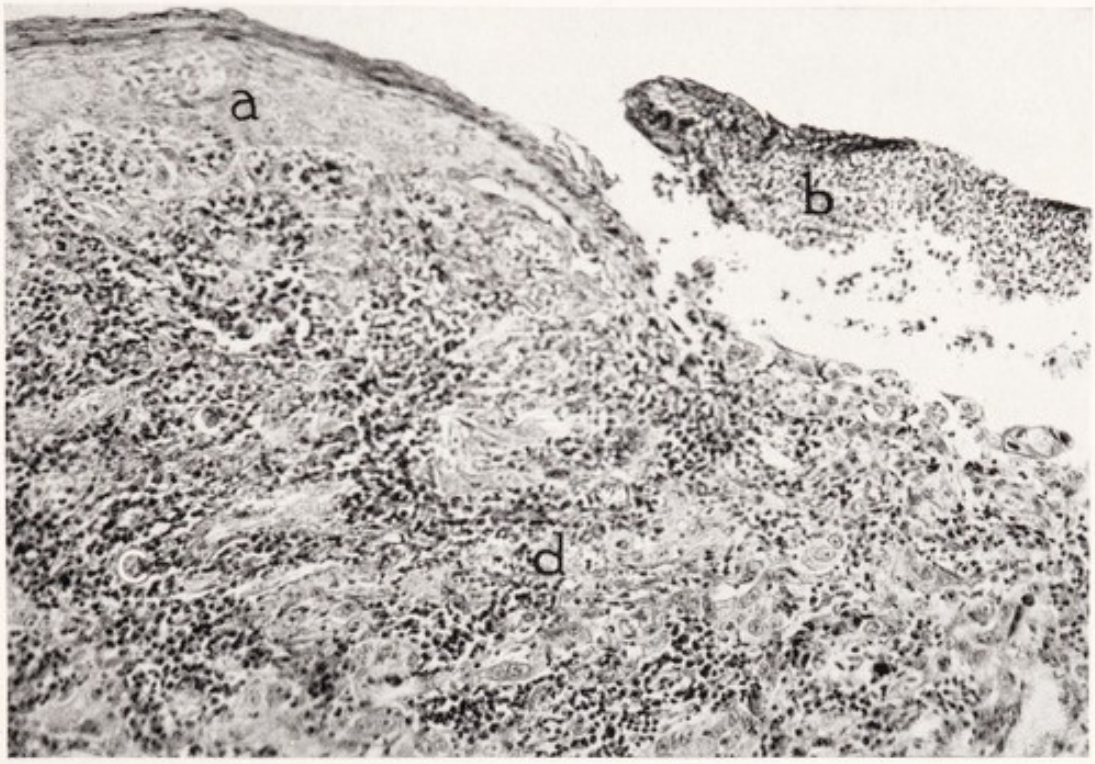

FIG. 44. Ulcer of the lip unchanged for six years: *a*, epithelium of the lip showing some proliferation; *b*, ulcer covered with a scab; *c*, round cell infiltration; *d*, scattered epithelial cells in the granulation tissue. (There were metastases in the cervical lymph glands.)

These in the beginning may be but slightly elevated, beginning in an old ulcer (*A*, Fig. 45). In later stages, the elevation extends distinctly above the surrounding border. These are the result of attempts at cornification of the epithelium, hence a less pronounced degree of deviation. This might indicate that this type is less malignant than the preceding but this does not seem to be the case. As a corollary to these is

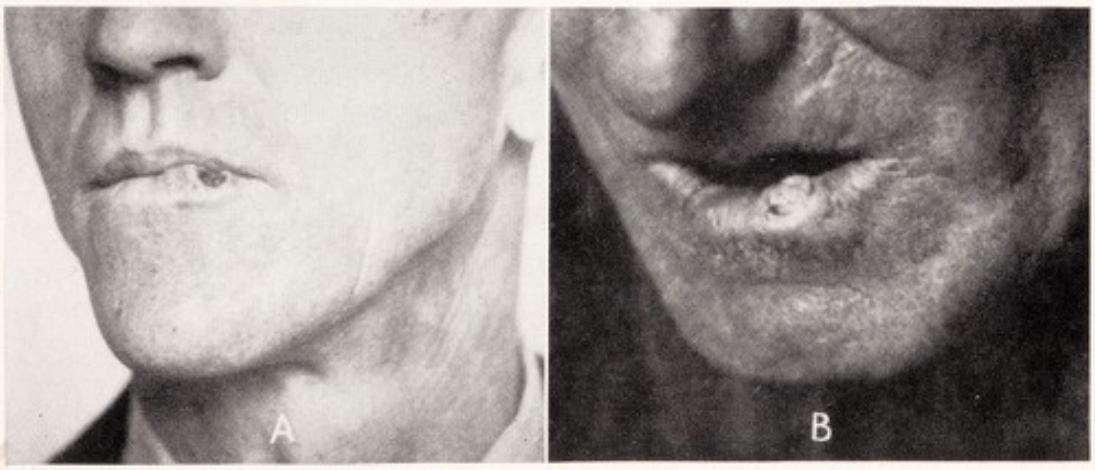

FIG. 45. Papillomatous carcinomas of the lip. *A*, Very early elevation of the base of the ulcer. *B*, Small horn which has undergone malignant change.

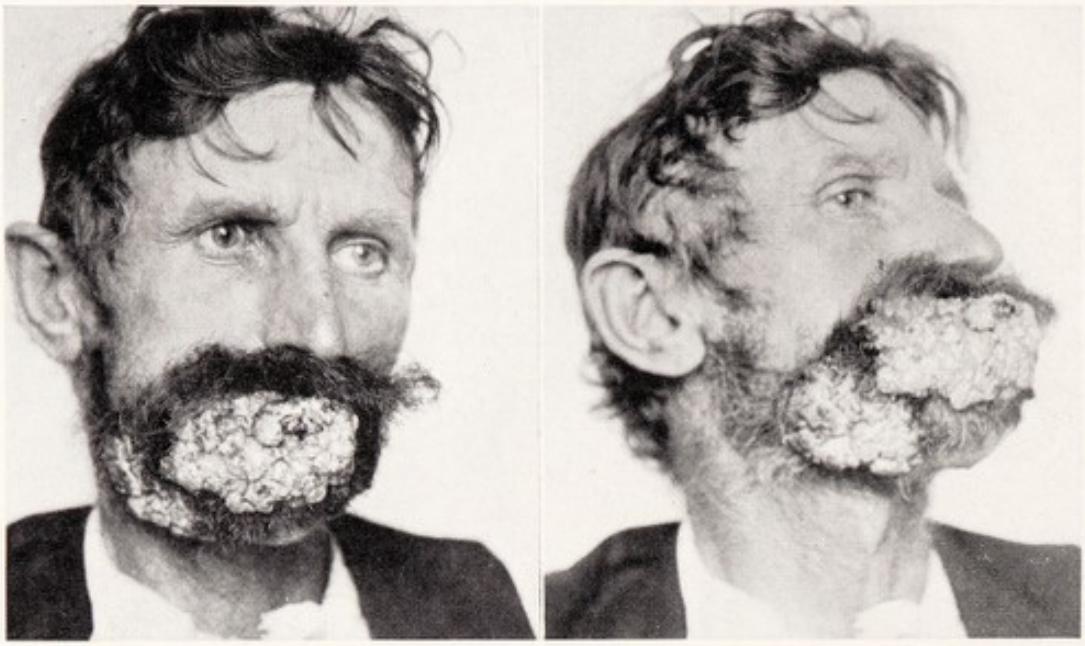

FIG. 46. Papillary carcinoma of the lip. A wart preceded the more rapid growth by a number of years. The metastasis is seen to be fungating under the angle of the jaw.

the cutaneous horn that becomes malignant. The difference is that when a horn becomes malignant, it is the epithelium about the horn that extends and not the cells of the base of the horn itself. This cellular proliferation produces a rim about the base of the lesion which is perceptible on palpation (*B*, Fig. 45).

In the old days, it may be mentioned as of historical interest, we saw really fungating masses which permitted diagnosis by inspection (Fig. 46). This patient did not have adequate medical care, obviously, but for the simple reason that he did not desire it because the lesion did not cause pain.

Pathology. The beginning of malignancy is manifested by hardening of the tumor itself without macroscopic extension about the border (Fig. 47), but this increase in firmness is characteristic. In the case of the horn

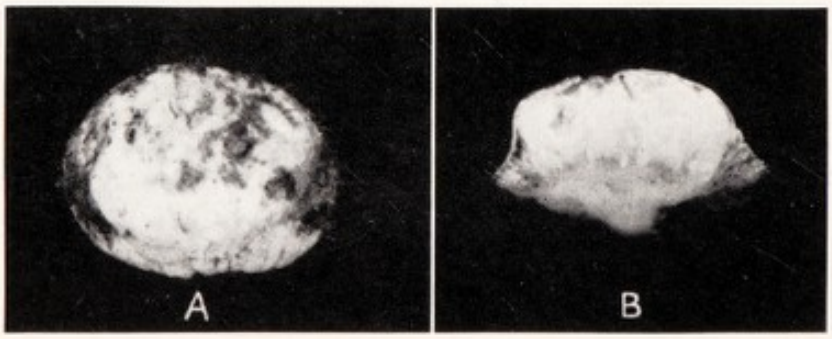

FIG. 47. Protruding carcinoma of the lip. *A*, The base is sharply circumscribed. *B*, The cross-section shows the slight tendency to invasion.

there may be no definite induration palpable but it becomes less movable and may be painful on manipulation.

A less common form of carcinoma of the lip is somewhat protuberant but there is no history of an antecedent lesion and there may be but slight if any ulceration. They appear as deeply seated masses covered by reddish epithelium or beginning ulceration. These are prone to be very malignant.

Histology. The characteristic feature in the early transition to malignancy is the mass development of the epithelial cells without the formation of cell nests but which are bordered with round cell infiltration.

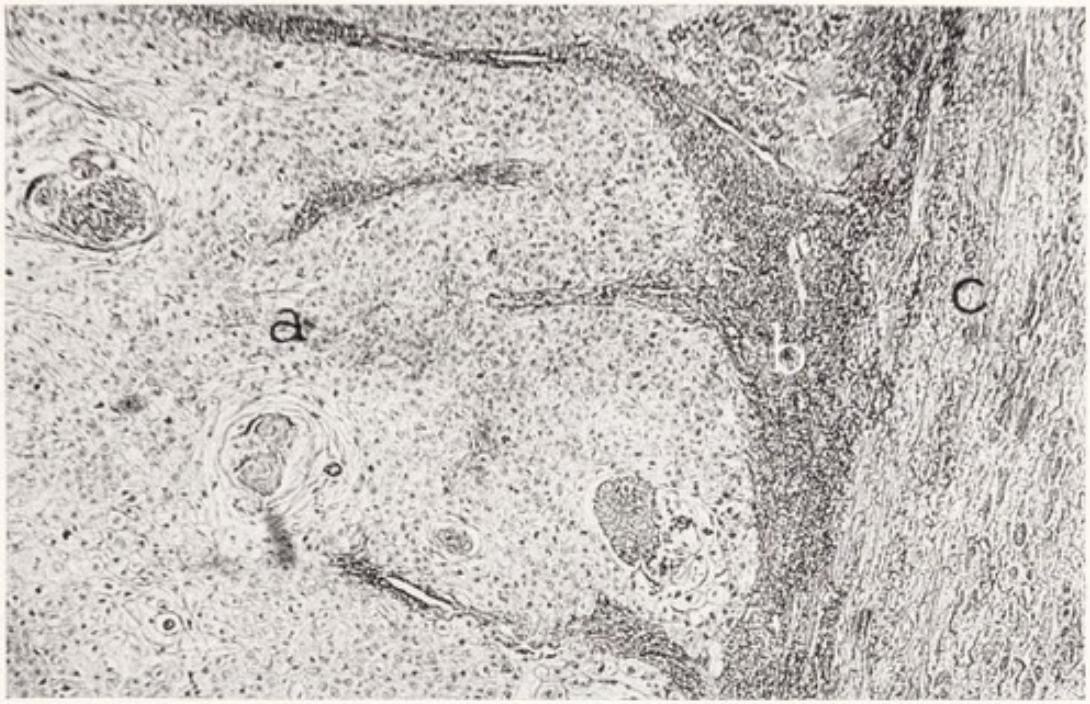

FIG. 48. Malignant papilloma of the lip: *a*, diffuse proliferation of epithelium with but little change in cell type; *b*, extensive round cell infiltration; *c*, fibrous tissue base which has ceased to be acidophilic.

In the presence of such a round cell infiltration, one is not warranted in excluding malignancy no matter how slight the epithelial changes. Of course, when the cell proliferation extends more deeply, forming nests, possibly with pearl formation, the diagnosis is easy but is then no longer an early lesion.

Round cell infiltration alone is suspicious of malignancy. A slight invasion is sufficient to warrant a diagnosis of malignancy (Fig. 48).

UNUSUAL CARCINOMAS OF THE LIP

There are several types of cancer of the lip of especial interest which will bear separate consideration because of their clinical course. These

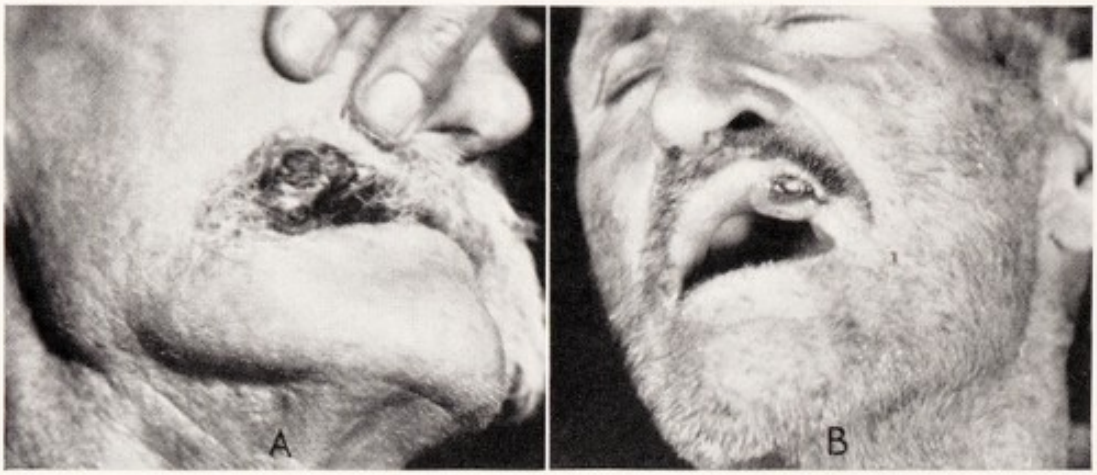

FIG. 49. Cancers of the upper lip. *A*, Adenoma which had become malignant. *B*, Ulcerating carcinoma. There was already metastasis in the neck and mediastinum.

are carcinomas of the upper lip, carcinoma of the young and recurrent carcinomas.

Carcinoma of the Upper Lip. The upper lip is not commonly the site of cancer in men, but in women it is more common in this situation than the lower lip. I have seen but one cancer of the lower lip in a woman.

Pathogenesis. They may begin on the upper lip near the vermilion border in an adenoma and extend downward invading the lip secondarily (*A*, Fig. 49). Some begin in the upper lip resembling like lesions in the lower lip (*B*, Fig. 49). Some of them begin as adenomas of the skin and reach the vermilion border late without destroying it (Fig. 50). The

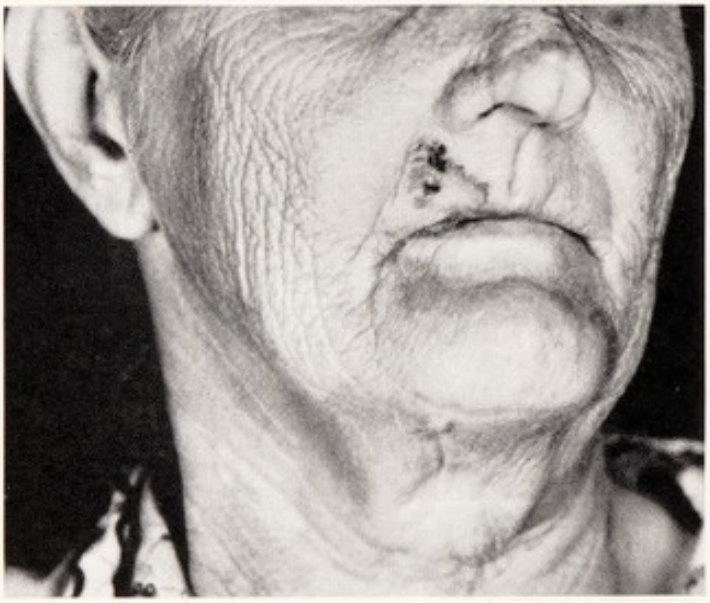

FIG. 50. Adenoma of the lip. The growth first affected the skin and remained so located several years. The vermilion border became gradually involved.

latter type is less malignant. As a matter of fact, they are really skin lesions. Nevertheless, unless radically removed, they tend to recur.

Pathology. The cancers of the upper lip do not differ materially in appearance from those of the lower lip. They form rounded hard tumors usually only in part ulcerated. If seen early, they may be confined to the skin, not yet having invaded the mucous surfaces. The ulcerated type does not differ in physical character from like lesions of the lower lip and has the same tendency to early metastasis.

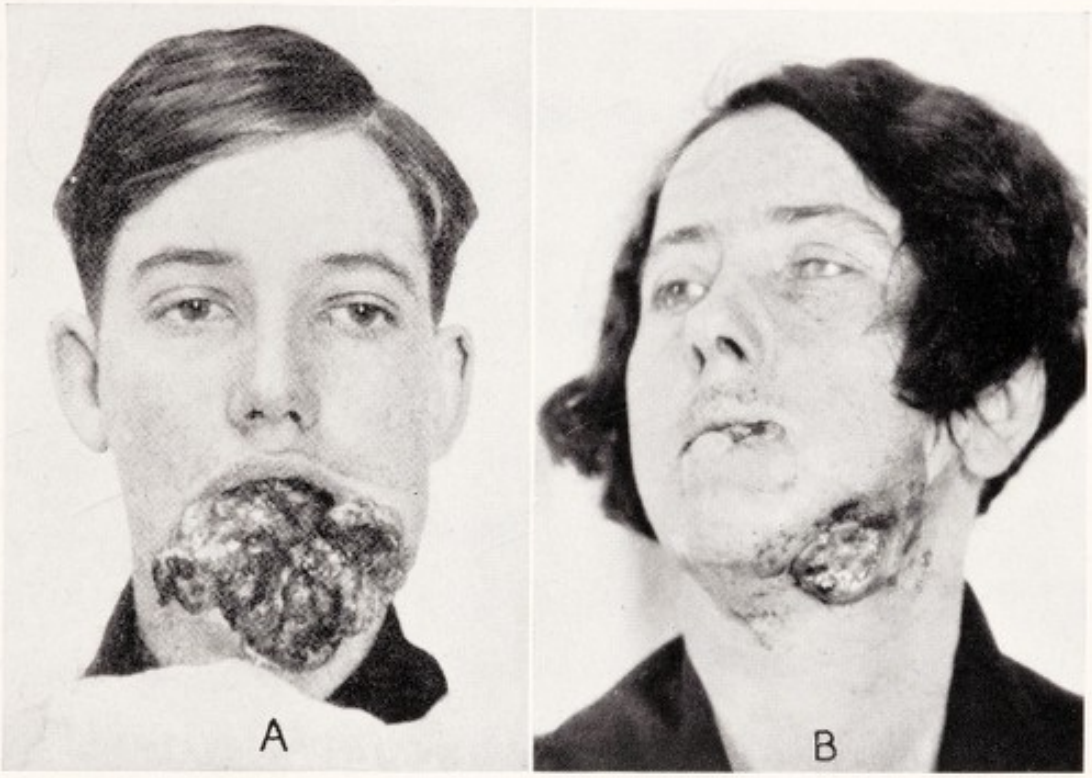

FIG. 51. Cancer of the lip in early life. *A*, Huge ulcer in a boy of sixteen. *B*, Ulcerating metastasis in a woman of thirty-five. The lip lesion was treated as a chancre.

Histology. Those beginning on the vermilion border have the same structure as those of the lower lip. The adenomatous type may retain their gland structure for a time but ultimately lose it entirely. The few metastases that I have examined bore no evidence of gland derivation.

Carcinoma of the Lip in the Young. Like all epitheliomas of the young, those of the lip make a very rapid growth. Fortunately, they are rare.

Pathogenesis. The growth is usually so rapid that the lesions are not observed in the early stages. Likely they are primary without any precedent lesion. They tend, of course, to rapid destruction.

Pathology. This may find expression in extensive local ulceration, the entire lip being destroyed within a few months (*A*, Fig. 51). These ulcerations may attain huge proportions before there is gland involvement. These are apt to be mistaken for inflammatory lesions in the early stages. On the contrary, the local lesion may remain small and the lymph glands become involved early and associated with induration and consequent sensitiveness to pressure. These are sometimes mistaken for primary syphilitic lesions because they occur in young persons, are of rapid development and are accompanied by extensive glandular invasion (*B*, Fig. 51). They are really deceptive lesions. Such mistakes are possible only when the physical characteristics of the local lesion are disre-

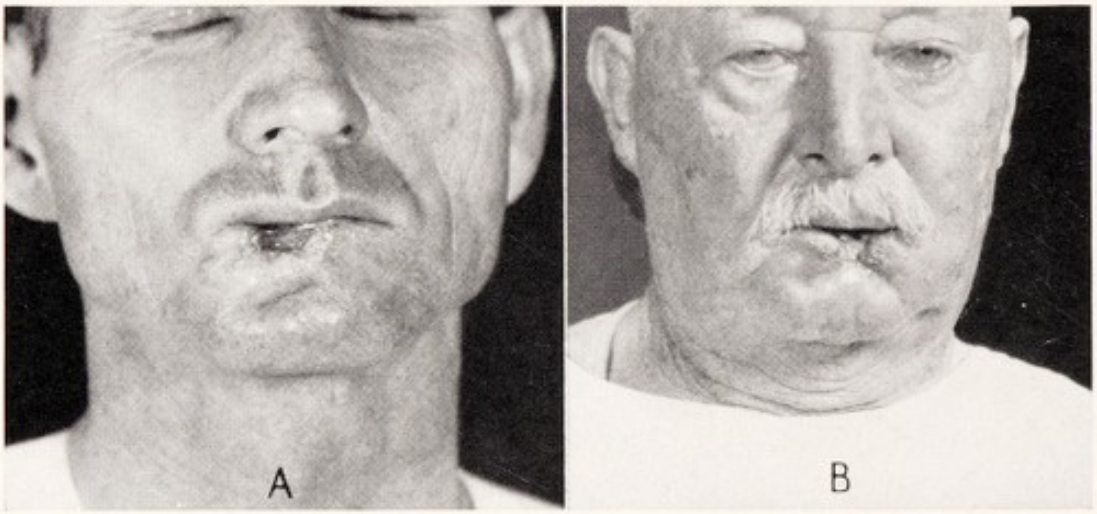

FIG. 52. Recurrent cancer of the lip after paste treatment. *A*, The entire skin covering the lip is a red, firm, indurated mass. *B*, Cured three times. The tip of the chin presents diffuse malignant invasion.

garded. A lip lesion in a young person suggests chancre and a positive Wassermann stops all further investigation. The cancer hardness is as evident here as in the more slowly growing forms affecting older persons. It is the destructive ulceration that misleads.

Histology. The cell form bespeaks the rapid growth in the excessive cellularity and the stainability and in the number of mitoses.

Recurrent Carcinomas. By this term is meant the reappearance of a cancer at the former site of the lesion after it has been caused to disappear by the deliberate act of somebody. Invasion of lymph glands after the removal of the primary lesion is not properly called a recurrence. The metastasis was present before the primary lesion was removed.

Pathogenesis. When a growth reappears at the former site following some form of treatment, the first evidence is usually a hard thickening

beneath the epithelium about the scar. The development of a single nodule is unusual. The result is the patient is apt to regard the gradual hardening with curiosity rather than with alarm because it is not attended by ulceration. In the old paste days, the recurrence appeared before there was complete resolution from the caustic (*A*, Fig. 52). Ulceration may take place after healing has taken place. If the cures were repeated, a puckered yet indurated mass resulted (*B*, Fig. 52). In some cases, after the use of paste, large masses of granulation tissue formed within a few weeks after the use of the paste. One should not be frightened at these. The quack may have destroyed a benign lesion

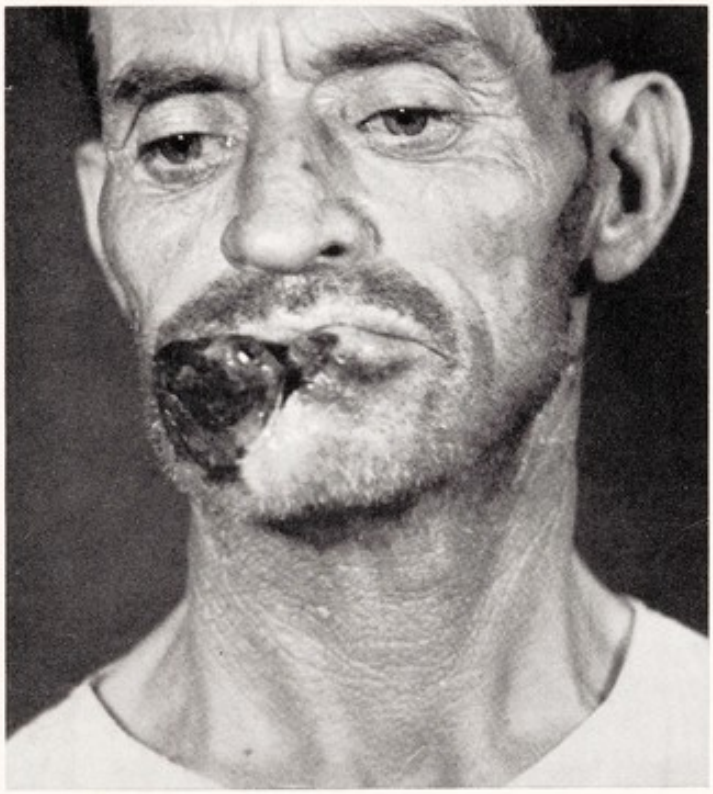

FIG. 53. Carcinoma of the lip, recurrence after cure from zinc paste. There was recurrence about the border but the bulk of the tumor was made up of exuberant granulation tissue. There were no metastases.

(Fig. 53). In either event, local destruction soon followed, resulting in the entire skin about the lesion being destroyed. Like in recurrences in the skin after operation for cancer of the breast, local recurrence is indicative of inadequate primary treatment. The surgeon is not accountable for metastases, but he is for local recurrences.

Recurrences after irradiation have annoyed the surgeons in the past and there are still grounds for grumbling. Radiologists were prone to present for our delectation, in six or nine months after lesions disappeared,

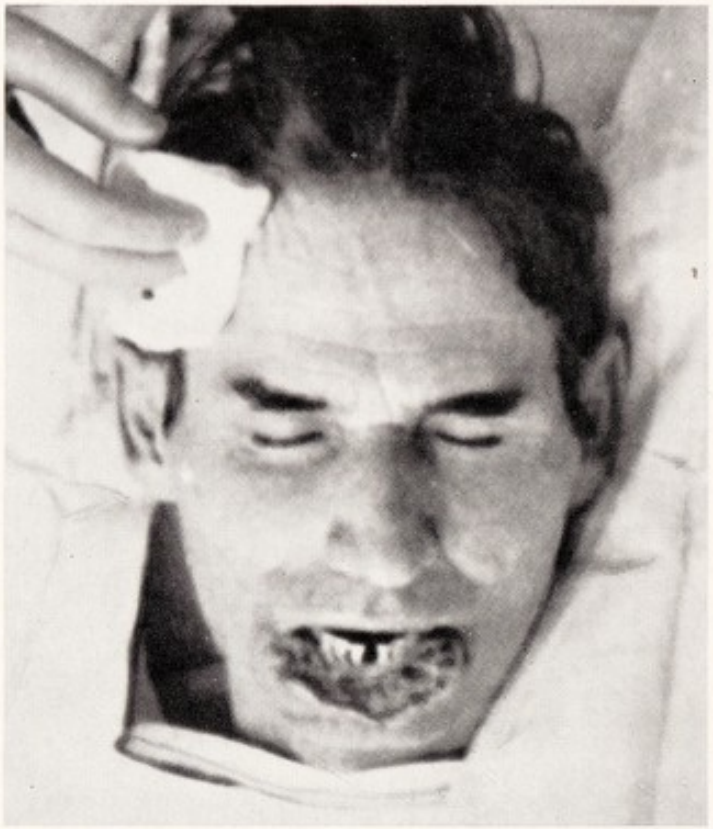

FIG. 54. Extensive local recurrence after cure of a leukoplakic area.

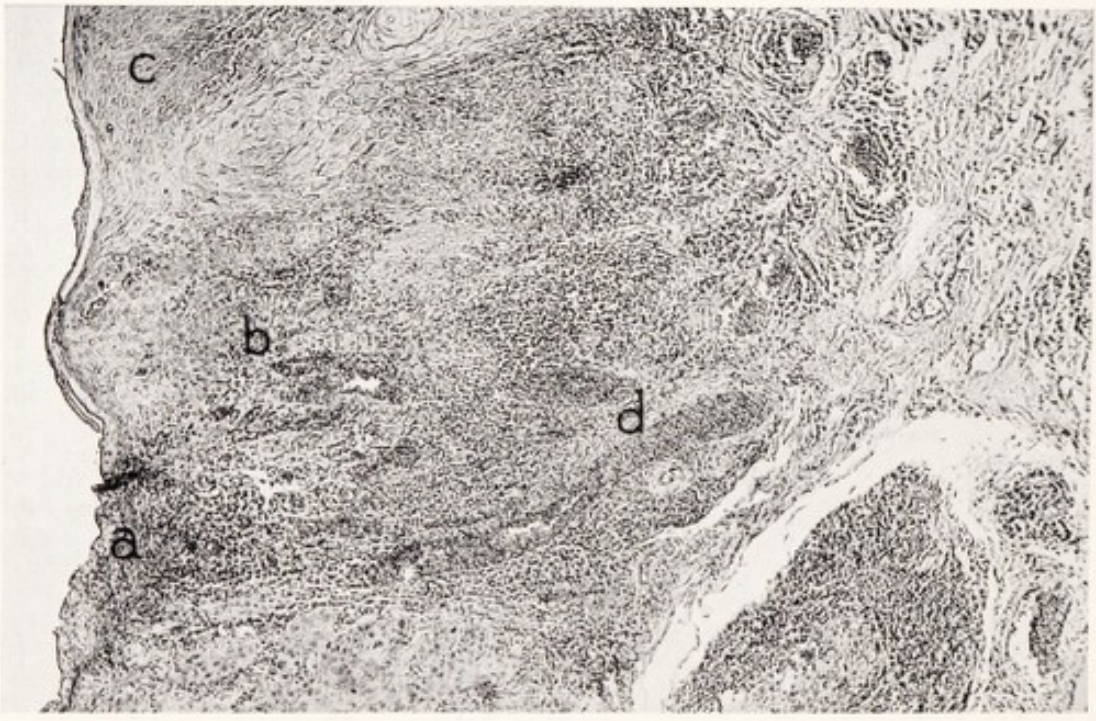

FIG. 55. Recurrence of cancer of the lip after a cure of a year: *a*, small area in which the epithelium is destroyed; *b*, extension of the epithelium downward; *c*, relatively normal epithelium; *d*, round cell infiltration, extending deeply into the subjacent areas.

areas nicely covered with skin. Smooth surface following irradiation sometimes was followed by local recurrences and metastases. Surgeons have a right to demand of radiologists the same rules they apply to their own efforts, a slide to show what was treated and the result after five years. We should lend ear to nothing else.

Pathology. The developing hardness at the site of the old treatment is usually sufficient to declare the nature of the trouble. However, the recurrence may be deeply seated below an intact epithelial surface.

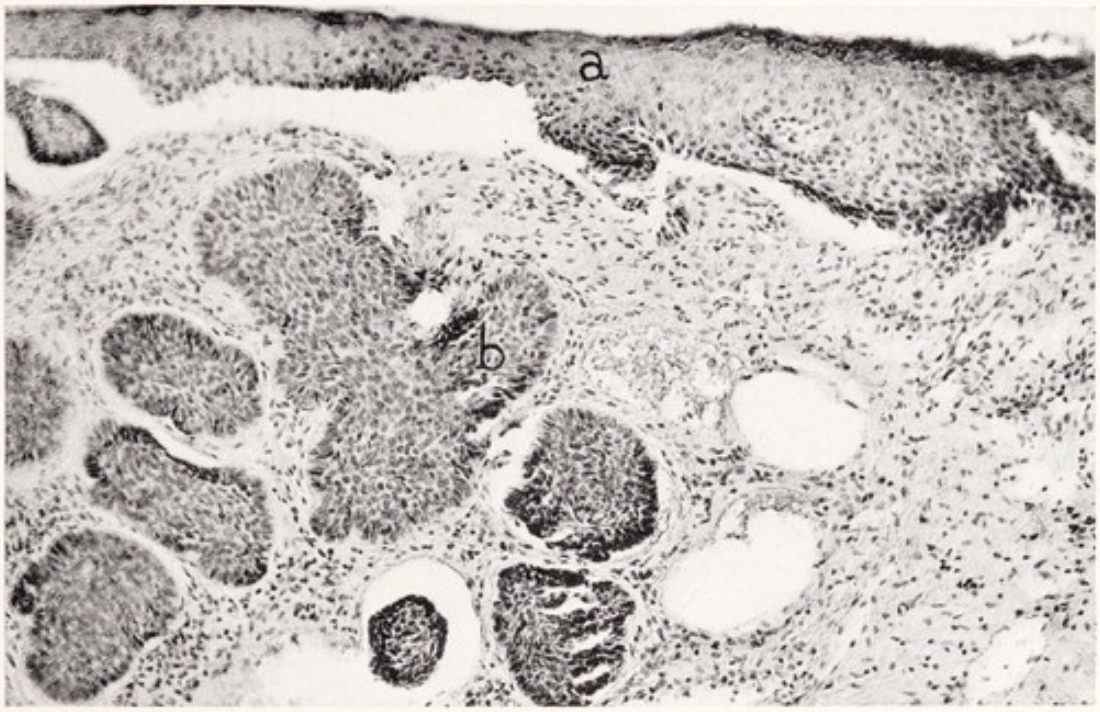

FIG. 56. Recurrent cancer of the lip: *a*, surface epithelium intact; *b*, malignant cell nests which evidently never were cured because they are surrounded by areas of connective tissue not extensively infiltrated by round cells. Also the cell type in the cancer nest differs abruptly from that of the covering epithelium.

After ulceration has occurred the matter is simple enough (Fig. 54). The ulceration may occur in the skin away from what was once the vermilion border. The extensive thickening is due to edema and round cell infiltration. The actual malignant growth may make up but a small part of the mass.

Histology. The cell structure of recurrences varies. It may extend from the covering epithelium (Fig. 55) or may show deeper invasion independent of the covering epithelium (Fig. 56). The superficial epithelium was restored but active cell nests continued in the depth. Such slides explain why to inspection, especially in the lantern slide, the lesion

seems healed, but in a very short time—months, even weeks—may show a wide ulcerating area (compare Fig. 26).

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Malignant Diseases of the Lip. Nearly all malignant diseases of the lip are of epithelial origin. A few sarcomas of the lip have been reported, none too convincing.

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Sarcoma. Cholnoky, *Am. J. Cancer*, Nov., 1934, 22:548–54; Falisi, *J. A. M. A.*, June 23, 1928, 90:2015–17; and Markley, *ibid.*, August 2, 1913, 61:334–5.

CHAPTER IV

Benign Lesions of the Mouth and Tongue

THERE is a host of benign lesions which affect the tongue and adjacent soft parts. These, for the most part, are of little clinical importance for they remain just what they are in the beginning, yet they require a diagnosis and as such are of pathologic interest. Though usually easily recognized and the pathologic sequence predictable, every now and then the slide pulls us back on our haunches, much to our chagrin and delight. No one but a pathologist can picture the association of those two emotions.

Like most benign lesions their importance lies in knowing what they are not. Chief of these are the true ulcers. Knowing benign ulcers is an important part in recognizing the malignant ones. Some benign tumors have an importance quite their own. Though not malignant they possess an annoying possibility if neglected. For instance, the lymphangiomas of the tongue if neglected, by undergoing repeated reactions, reach such a huge size that the mouth is no longer able to harbor them. Many of the lesions which are found in this region may be classified as annoyances, annoyances indeed in which the surgeon as well as the patient may share. Foremost are the cysts.

Protean as these benign lesions of the mouth and tongue are, it is possible in a measure to group them in a general way, though some violence is required in order to accomplish this end. The object here is to emphasize clinical rather than a pathologic unity. Unfortunately the difficulty of securing satisfactory photographs of lesions in this region greatly embarrasses a satisfactory presentation. The following will serve the purpose with the greatest possible brevity:

I. Ulcers of the Mouth and Tongue
   - Simple Ulcers
   - Chronic (Decubitus) Ulcers
   - Tuberculosis
   - Syphilis

II. Angiomas of the Mouth and Tongue
   - Lymphangiomas
   - Hemangiomas

III. Congenital Anomalies of the Tongue and Cheek
- Macroglossia
- Macrocheilia
- Macromelia

IV. Benign Tumors of the Mouth and Tongue
- Cysts
- Connective Tissue Tumors
- Papillomas

V. Inflammatory Lesions of the Mouth and Tongue
- Glossitis
- Diffuse Inflammation of the Floor of the Mouth
- Inflammations Due to Sialoliths
- Vincent's Infection
- Leukoplakia

This classification omits the various minor lesions of little consequence to the surgeon, notably those due to constitutional disturbance.

ULCERS

A variety of ulcers may be found in the oral cavity. The common ones are due to injury from a broken tooth or a defective dental apparatus but may develop through unknown causes. The surgeon is compelled to give heed to these, ill prepared as he may be to comprehend all the constitutional relations. Specific ulcers should be considered as a group of their own but since they must be compared to the nonspecific it is convenient to consider them together. Tuberculosis is not at all uncommon but syphilis is rare in this region. However, at that, one must be on the lookout particularly when confronted by visitors from the cities.

Though many of these lesions do not threaten the life of the patient they cause great annoyance and the patients are grateful for relief, for they are prone to gage the ability of the surgeon by the relief they obtain rather than by the skill displayed.

Simple Nonspecific Ulcers. Simple ulcers are usually more or less diffuse and their cause is unknown. Many high sounding and ornate names have been applied to them but it has cleared up nothing. They are usually found on the dorsum of the tongue, less common on the edges, in which respect they differ from the decubitus ulcers. They differ from the decubital ulcers chiefly in that there is no object which would account for their origin.

Pathogenesis. Since any chronic lesion may end in malignancy the responsibility of detecting the border-line is great. Because of the

induration about them they have a firmness that may be distressingly like an early malignancy and is often mistaken for such. The lesion may shift its location, healing at one point only to break down at another. They seem not prone to form malignancy but may persist for years without notable change. One suspects a pernicious anemia or some disturbance of the digestive apparatus.

Even when the history states that they have existed for a period so long that it would seem to exclude malignancy, a cancer may have become established in a recent period. In some cases a diagnostic section is

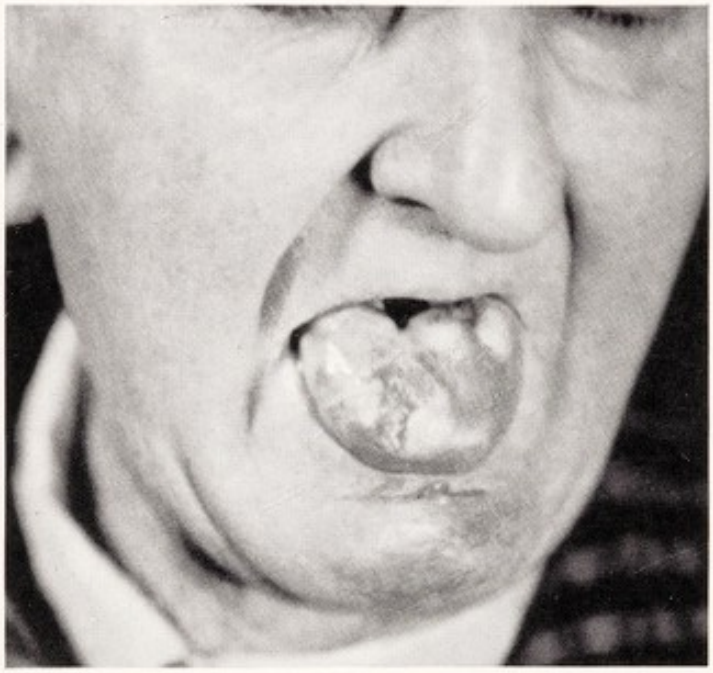

FIG. 57. Chronic ulcer of the tongue. There is a deep ulcer at the tip, more superficial ones on the dorsum.

indispensable. In such case the ulcer should be excised totally if possible or at least a sufficient area so that a really efficient microscopic study can be made. The most difficult cases to interpret are those in which the patient exhibits a local point of distress though the lesion covers a much wider area. This causes one to suspect that at that point the ulcer may be advancing. If such a point is complained of it is the area which should be excised for microscopic examination.

In the most of these cases there is a constitutional disease in the background and the entire patient requires careful examination. The entire tongue may show nutritional changes, the epithelium being shiny and thinned and may be exfoliating. The patient may complain of a sense of dryness and pain from irritation or coming spontaneously.

Even if a localized lesion is found, care must be exercised in accepting it as the whole trouble. Generally speaking, if a localized lesion is found without a local cause there is some constitutional disorder in the background.

Pathology. The mucosa about the lesion is smooth, pale and shiny but it may be red particularly near the ulcer or at the border of the ulcer. The ulcer may be so superficial as to appear to be but an excoriation or covered with a leukoplakic layer (Fig. 57). At some points the mucosa has obviously been destroyed (Fig. 58). The edge may be irregular,

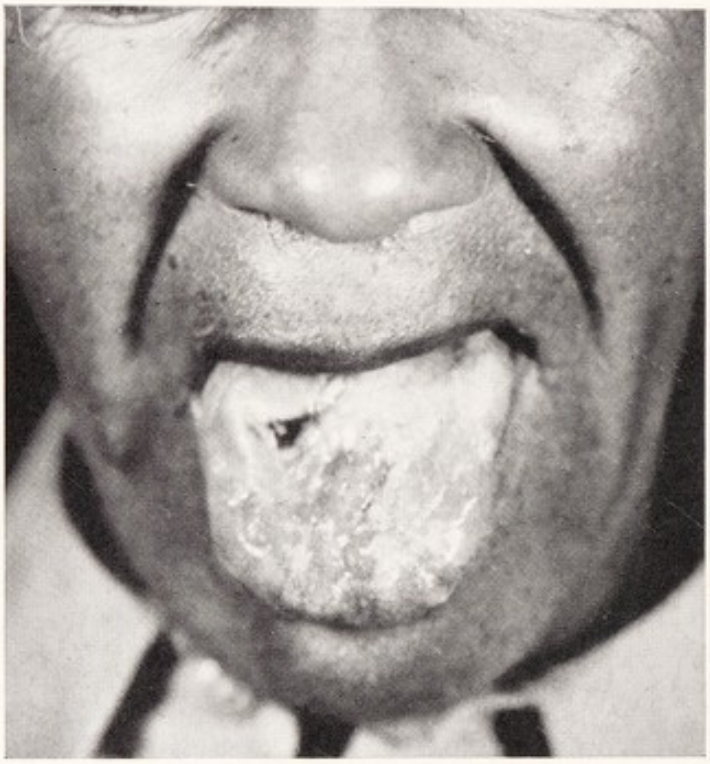

FIG. 58. Chronic ulcer of the tongue. Irregular ulcer involving much of the dorsum of the tongue. Duration ten years. Complicated with leukoplakia.

slightly elevated, not undermined and but little or not inflamed. In long standing ulcers the border may feel hard to the examining finger. The bed is usually made up of dirty gray granulation tissue. Sometimes the ulcer is more acute being sharp walled with overhanging edges and the base reddish. The border is soft to the touch. The border may be irregular but sometimes there are several ulcers grouped together. Because of this softness, malignancy can be certainly ruled out.

Histology. The picture is that of a reactionless lesion, a few round cells and but feeble attempts at regeneration of the epithelium. In the chronic ones which most closely resemble malignancy in the clinic, the

edge is made up of palely staining muscle with very little round cell infiltration, therefore wholly different from malignancy. The failure of the tissue to stain with any dye suggests that the change may be within the muscle and the loss of epithelium is due to this (Fig. 59). In some areas the lesion is superficial, not fully devoid of epithelium, while in others there is a true ulcer. One area may heal while there is extension to other not previously involved areas.

Chronic Decubitus Ulcers. In this group may be placed those ulcers which present an obvious cause. Otherwise they are like the simple

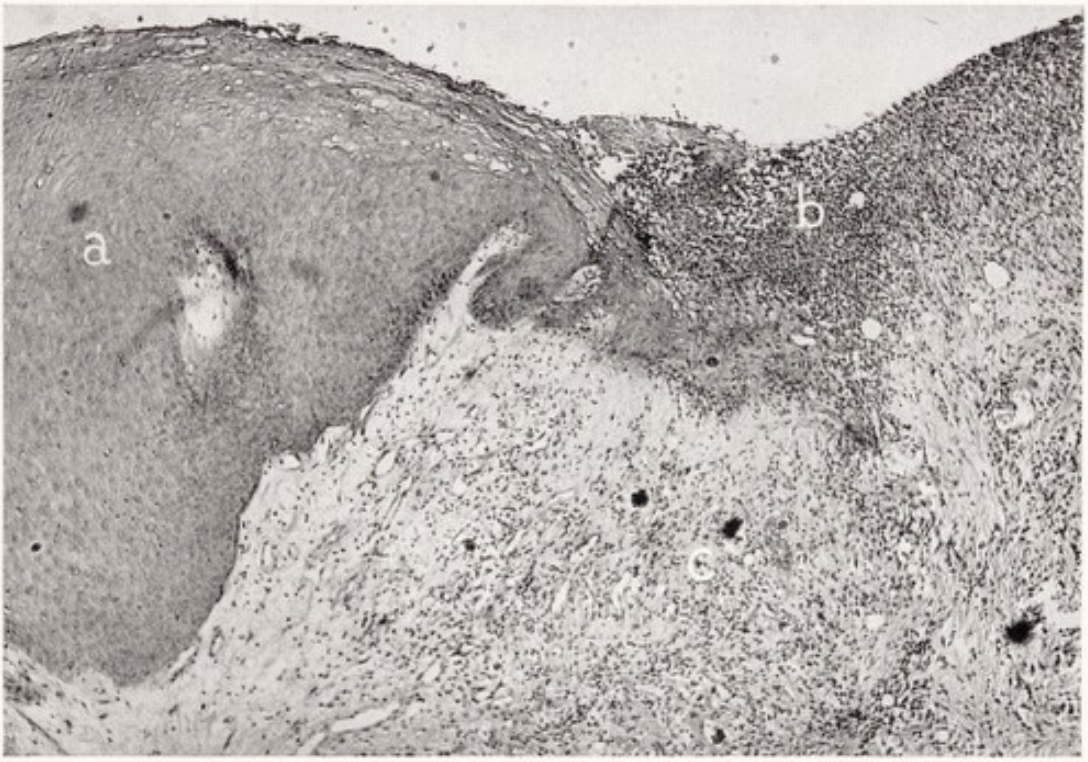

FIG. 59. Slide from a chronic ulcer of the tongue: *a*, the epithelium is considerably thickened but without change in cell type; *b*, the epithelium is lost and the surface is covered with more or less degenerated cells; *c*, granulation tissue-like area showing many vessels and some round cell infiltration.

variety. Decubitus ulcers are located adjacent to some irritating object such as a broken tooth or a defective dental apparatus. It is common knowledge that they may be forerunners of malignancies. A considerable number of them are already malignant when the patient first seeks advice. Because of this tendency these are of the most importance. Even though there may be an obvious cause for the existence of the ulcer one must not forget the possibility of a constitutional disease in the offing. Perhaps the denture would not have caused an ulcer except for the constitutional disease.

Pathogenesis. Constant irritation by a broken tooth or a faulty denture produces an ulcer which of course becomes infected as soon as the epithelium is broken down leaving an indolent superficial ulcer. They commonly continue as such indefinitely unless the offending cause is removed. If the offending source of irritation is removed and the ulcer does not heal, malignancy must be suspected or expected for if not already malignant it is likely to become so. If malignancy develops on the base of the ulcer, they present the usual course of a cancer in this region though usually less malignant than those developing without an evident exciting cause.

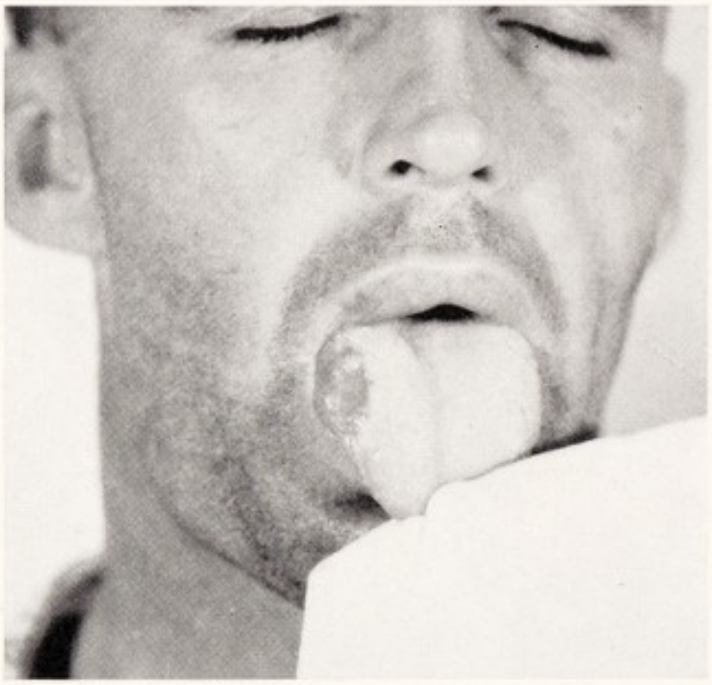

FIG. 60. Chronic ulcer of the tongue. Had severe pain in lower jaw. Had ganglionectomy three years later. The ulcer continued.

Pathology. A superficial soft bordered defect greets the examiner. Neglected teeth are the common cause. The base is grayish or reddish and the border but little changed (Fig. 60). In some cases when the irritating object is localized the border is clear cut, indurated and reddened. Such conditions may seem to present a border-line condition when they really do not. Beginning malignancy is manifest by a border which is becoming hard. Touch is a more reliable guide than is sight. Usually they are definitely the one or the other. When there is doubt the entire lesion should be excised. This is good treatment in either event. Early in the course of the ulcer it may be seen with difficulty. Therefore if a patient complains of pain at a definite site an ulcer should be assumed to exist.

Histology. The structure is that of a chronic ulcer. Sometimes the epithelium piles up and if but a small bit of tissue is at hand for examination it may be mistaken for beginning malignancy, therefore the need for securing adequate tissue for examination. If the whole lesion is available this error is not likely to occur. The slide usually shows much the same picture as the simple variety except that the round cell infiltration is usually greater due likely to the constant irritation.

Tuberculosis Ulcers. Tuberculosis of the tongue is fairly common particularly in patients of reduced health, especially in those who already show evidence of lung tuberculosis. In fact if a patient has a tuberculosis ulcer of the tongue one may assume that there is a lung involvement even if it cannot be demonstrated at the time. Conversely if an ulcer is found in a patient with pulmonary tuberculosis one had best assume that the local lesion is of the same nature even though there appears to be a local cause for it. The ulcer is most commonly situated on the tip of the tongue, always a suggestive incident, but may occur anywhere on the tongue, floor of the mouth or cheek.

Pathogenesis. A superficial round ulcer is the typical beginning lesion. This may remain unchanged for many months, even years. Sometimes they heal spontaneously or as the result of treatment, perhaps to recur at some other point. The base of the ulcer may become invaded by deep cell infiltration producing a thickened mass beneath it or the deep lesion may appear first, the superficial lesion not appearing until months afterwards. If the lesion is extensive a mass of considerable size may be produced so that a cancer may be imitated. The ulcer may be very small in comparison with the tumor mass within the tongue. I once removed half a tongue under this misconception, not discovering the error until slides had been prepared for my class, much to my chagrin since the class had witnessed the operation for "carcinoma." Recurrence and healing may continue for years producing a much deformed organ. In most cases the patient ultimately dies of pulmonary tuberculosis. Some of the spreading ulcers one reads of in the literature have not been proved histologically and quite possibly were of the simple variety above discussed. I have never seen a proved tuberculous ulcer imitate a simple chronic one.

Pathology. The ulcer is soft, superficial with a dirty gray base (Fig. 61). The edge of the ulcer is usually slightly overhanging and decidedly soft to the touch. That is, usually it is superficial but as noted above may produce a considerable mass.

Histology. An adequate slide presents a simple picture of tubercu-

losis but if a timid biopsy is depended upon nothing but inflammatory tissue may be disclosed or the attempts at healing may show epithelial proliferation that may resemble carcinoma to the credulous microscopist.

Syphilitic Ulcers. When the fates invented syphilis they gave the medical profession a break by giving the tertiary lesion a kidney shape no matter where located. In no region is this providential forethought of greater value than in specific lesions within the mouth. Most ulcers are located in the fauces and on the tongue. In the latter location the pathognomonic shape of the lesion is commonly encountered. Unfortunately in the oral cavity this peculiar form of the ulcer cannot always be made out especially when located on the fauces. The Wassermann reaction may be useful but often one must resort to the therapeutic test. The therapeutic test should be tried before a biopsy is attempted because if it is syphilis the lesion is likely to be much larger after operative removal of an adequate diagnostic piece.

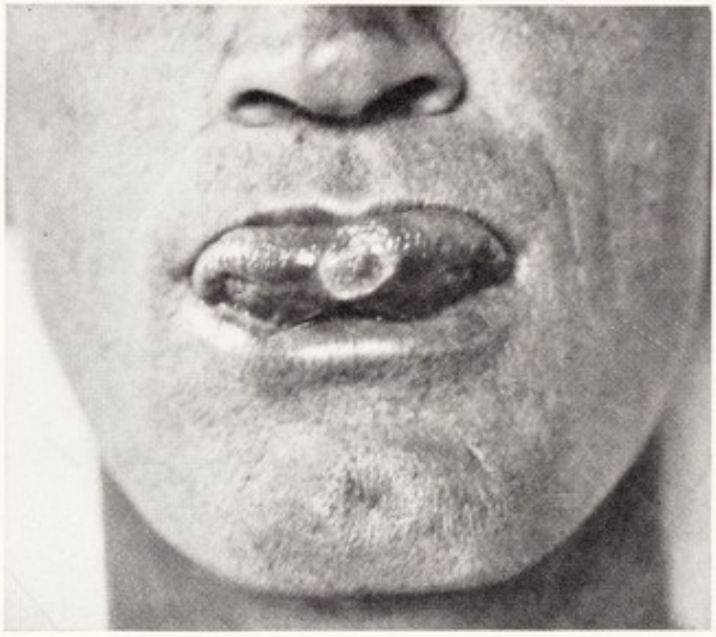

FIG. 61. Tuberculous ulcer of the tip of the tongue. It is circular with a finely granular base and overhanging border.

Pathogenesis. Late lesions of the tongue are preceded by a gumma the breaking down of which produces the ulcer. This remains as a deep punched out ulcer situated on one-half of the tongue parallel with the long axis of the organ. This remains indefinitely until treatment is instituted. Long neglected lesions may heal to a greater or less extent and break down again producing confusing pictures.

Pathology. When the lesion is observed in the gumma stages, it presents an ovoid enlargement, firm or elastic or fluctuating depending on how near it has approached to perforating the covering mucosa. Once the ulcer forms it is ovoid with a dirty base and distinctly overhanging edges. When situated on the pillars or palate the ulcers are more superficial and may be multiple. Once when I was very, very young I excluded syphilis of the pillar because the patient was such a nice lady. Here, especially if long neglected, healing may take place in some regions while breaking down occurs in others, producing irregular lesions. The results may be a much scarred surface. When successive ulcers have formed and healed a rough corrugated surface may result, so it is said. Possibly this form belongs with the simple chronic ulcers. Syphilis is too often named rather than proved.

Histology. If the breaking down edge of the ulcer is secured in the biopsy the caseated area with retained nuclei characteristic of this disease may be found. Otherwise breaking down granulation tissue with the thickened vessels is all that can be demonstrated.

ANGIOMAS OF THE MOUTH AND TONGUE

Angiomas involving either the lymphatic or blood vessels are of course congenital lesions, though they may not declare their presence for a number of years after birth. Though innocent enough in the beginning, both may cause serious consequences in the course of the years, the lymphangiomas by causing tumorous enlargement of the tongue, the hemangiomas by causing hemorrhage.

Lymphangiomas. Lymphangiomas of the tongue are usually overlooked until the child is several years old since they do not cause obvious enlargement of the organ until reaction has taken place.

Pathogenesis. Usually in the beginning these tumors appear as small apparently insignificant lesions, giving little evidence of the serious conditions which may follow unless the proper treatment is instituted. Occurring in children parents naturally are reluctant to permit any operation involving the removal of a part of the tongue, the lesion giving so little evidence of its potentialities.

When first observed, lymphangiomas form small elevated areas on one side or at the tip of the tongue. On inspection the tongue does not seem to be greatly enlarged (Fig. 62) but when grasped between thumb and finger an incompressible nodule is found. Inspection may reveal but little and the untrained observer is prone to belittle the apprehension of the surgeon. Inspection shows an irregular surface which may show

small blister-like nodules but little elevated above the surface (*Insert*, Fig. 62).

This condition may remain stationary for years but sooner or later the organ becomes inflamed from causes unknown. Each recurring inflammation leaves the lesion somewhat larger than before. Repeated recurring inflammatory attacks ensue until ultimately there is a considerable increase in the size of the mass. This increase in size is maintained after the reaction subsides. The recurrence of these attacks results in producing a huge tongue which taxes the capacity of the oral cavity to contain it, making deglutition difficult. Removed early the small excision ends the difficulty; neglected the whole tongue must ultimately be excised. I once observed a case in which the inflammation

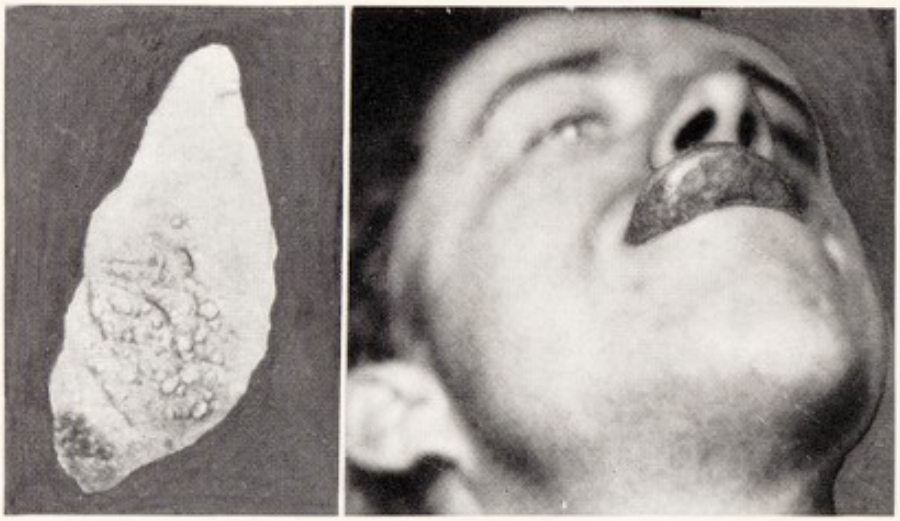

FIG. 62. Lymphangioma of tongue. M., age 14. Tongue has been inflamed at intervals of six months for several years. Mass occupied right half of tongue for two inches. Small clear cysts cover surface of tongue (*Insert*). Excision. Cured 16 years.

recurred about once a year for twelve successive years but despite emphatic warnings operation was refused until the entire tongue became involved producing a very distressing condition, not the least part of which was the distress of the mother when she realized that the repeated warnings given her had a real basis in fact. She berated me for not making the end-results emphatic enough.

Pathology. The small submerged blister-like lesion characterizes the early stages. Only careful palpation reveals the real extent of the lesion. Allowing the mother herself to make these palpatory investigations aids materially in convincing her that there is really a lesion present. This strategy gives the responsible party a better idea of the extent of the lesion and aids in securing consent to the needed early operation. After

repeated reactive exacerbations, cysts may no longer be visible and a huge incompressible organ may be all that greets the examiner.

Histology. Early, small cysts are characteristic. Late, large amounts of fibrous tissue, lymph spaces of varying size with many leukocytes and large mononuclear cells may form the whole picture (Fig. 63).

Hemangiomas. Hemangiomas may vary from a small area of dilated vessels located on one side of the tongue to huge masses involving cheek, floor of the mouth as well as the tongue. These are sometimes associated with hemangiomas of the neck even extending to the thorax and axilla.

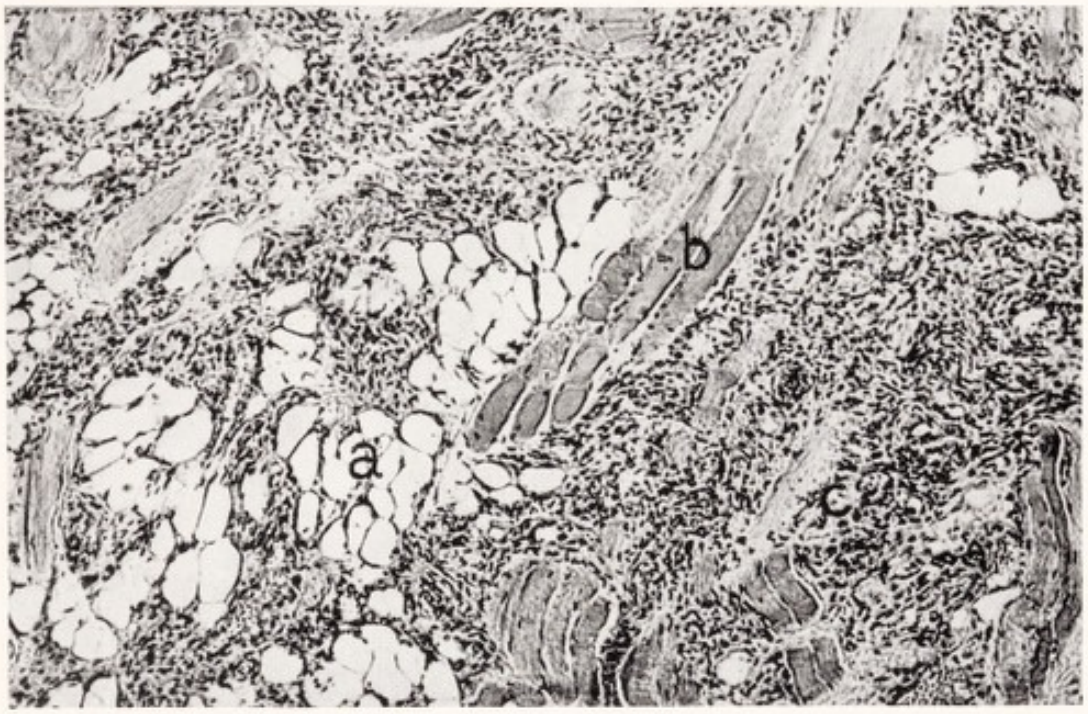

FIG. 63. Lymphangioma of the tongue which has recently undergone reaction: *a*, lymph spaces; *b*, muscle fibers of the tongue; *c*, round cell infiltration and newly formed connective tissue.

Pathogenesis. These lesions though congenital often focus the attention of the parent after the child has attained the age of several years. Because of their color and tendency to become distended they usually come to the attention of the mother much earlier than the lymph cysts. In many cases, however, they do not appear until years later, even not until adult life is reached. The first patient I observed was 48 years of age at the time of operation.

Hemangiomas tend to remain stationary for a number of years not being affected by periods of exacerbations as in the case of the lymphangiomas but in time increase in size is sure to result, and in rare cases they

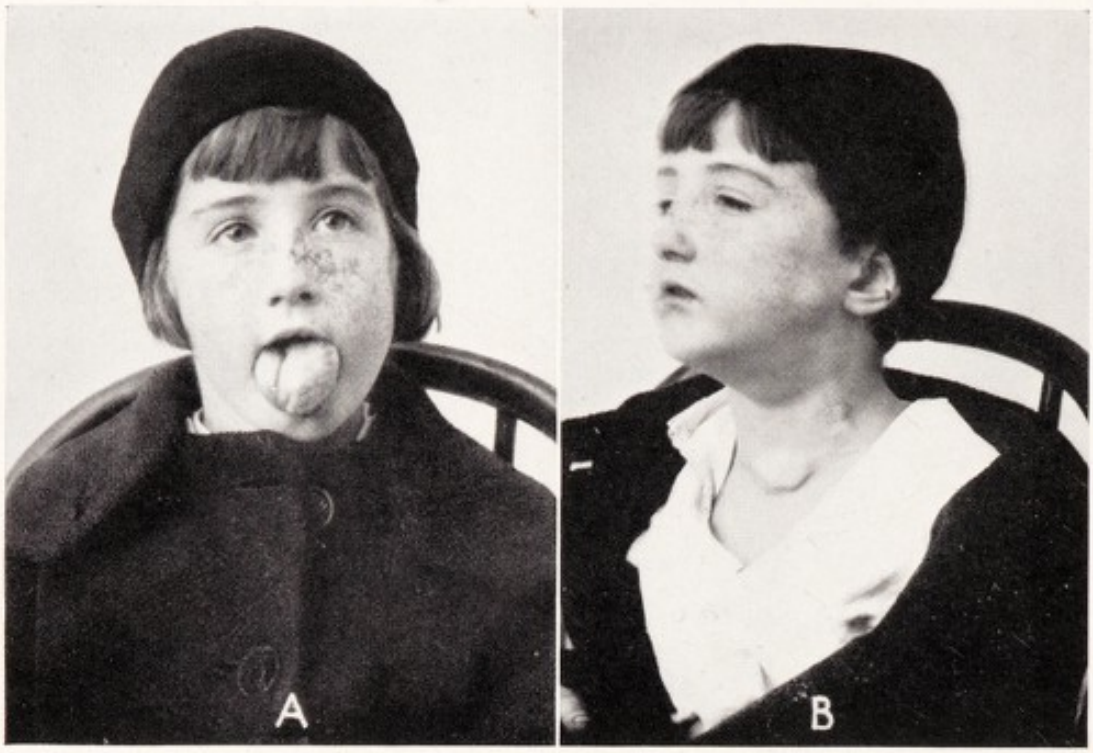

FIG. 64. Cavernous angioma of the tongue and neck. F., age 10. *A*, The left half of the tongue is much thickened; *B*, the parotid region, the neck and the suprasternal notch show dilated vessels.

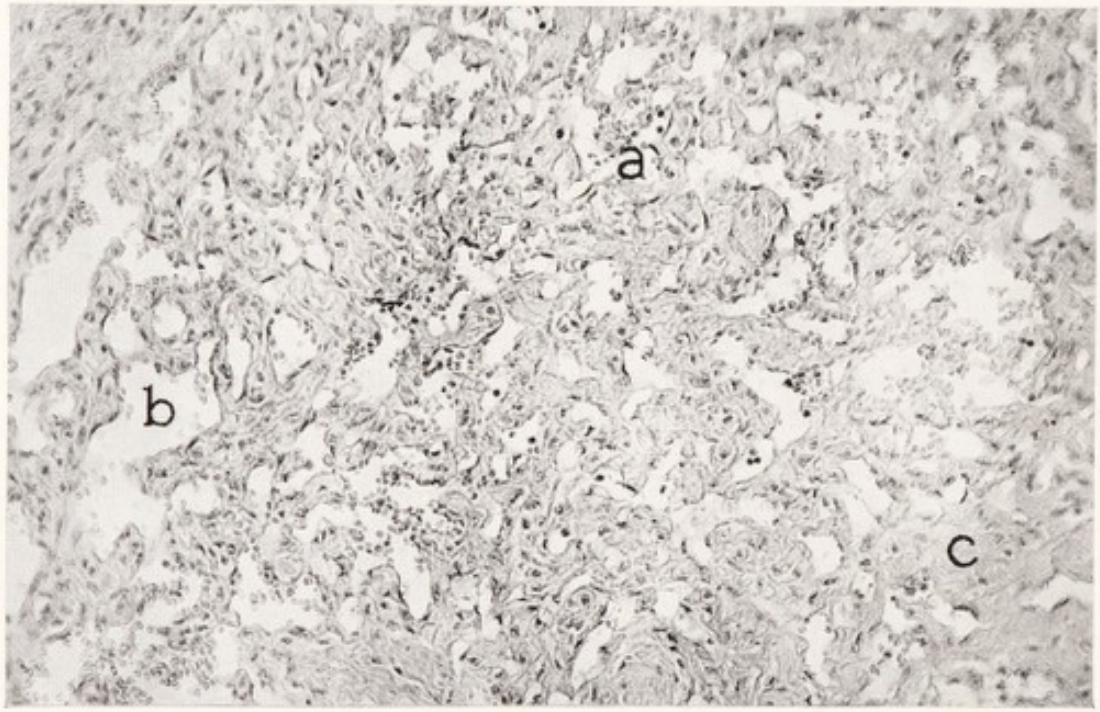

FIG. 65. Hemangioma of the tongue: *a*, the vascular spaces are small for the most part but separated from each other by a considerable connective tissue; *b*, larger spaces near the periphery; *c*, connective tissue capsule.

become thrombosed which may produce fatal complications or ulcerate and cause alarming hemorrhages.

Pathology. The color declares their nature and this is confirmed by their compressibility in which characteristic they differ from the lymphangiomas. In children in early cases the lesion may be made evident by causing the child to cry. Usually one look at the average surgeon by the child is an adequate excitant. The size varies greatly.

In most cases the dilated vessels are confined to one-half of the tongue (*A*, Fig. 64). The tongue lesions may be associated with dilated vessels

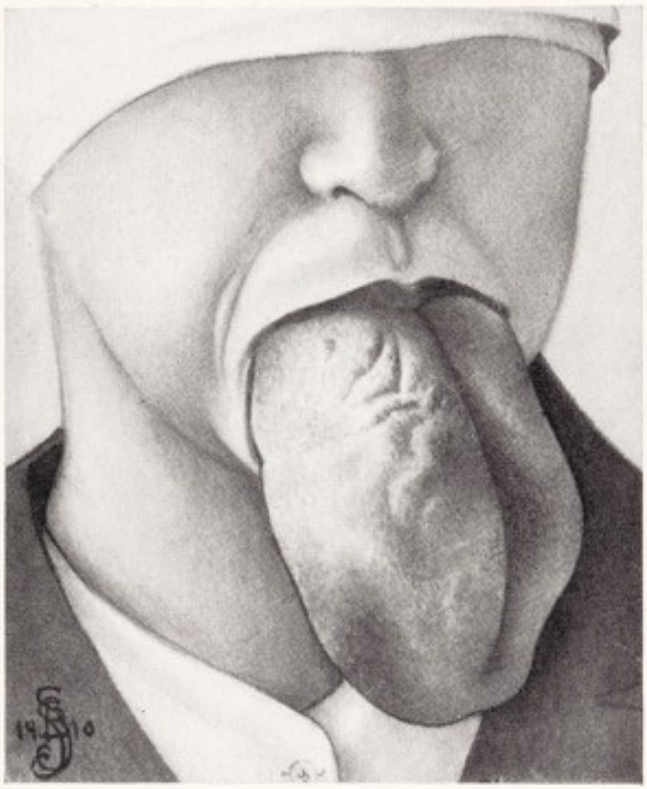

FIG. 66. Macroglossia. The huge organ has been protruded for years.

of the neck (*B*, Fig. 64). I once saw a patient in whom a large angioma occupied the mediastinum. It is well therefore to examine the entire patient before attacking the tongue.

Histology. The veins are thin-walled and the intervenous tissue may be much or little. There may be associated dilated channels usually small. Those of the tongue are usually small (Fig. 65) even when the lesions of the neck and elsewhere are cavernous.

CONGENITAL ANOMALIES

The lesions here discussed, though they may be associated with enlarged vessels, are attended by the presence of hyperplasia of other

tissue which gives them their peculiar characteristics. They involve usually one organ only, namely the tongue, lips, or cheek. The location in special regions has given rise to separate high sounding names, to wit: macroglossia, macrocheilia and macromelia. Each of these lesions are made up of increase of all of the tissue of the organ, vascular spaces, connective tissue proliferation and usually an increase of muscle fibers.

Macroglossia. Giant tongue is a rare lesion, fortunately. The beginnings date from infancy but in some cases the history is not clear. In a sense the lymphangiomas and hemangiomas produce a big tongue but

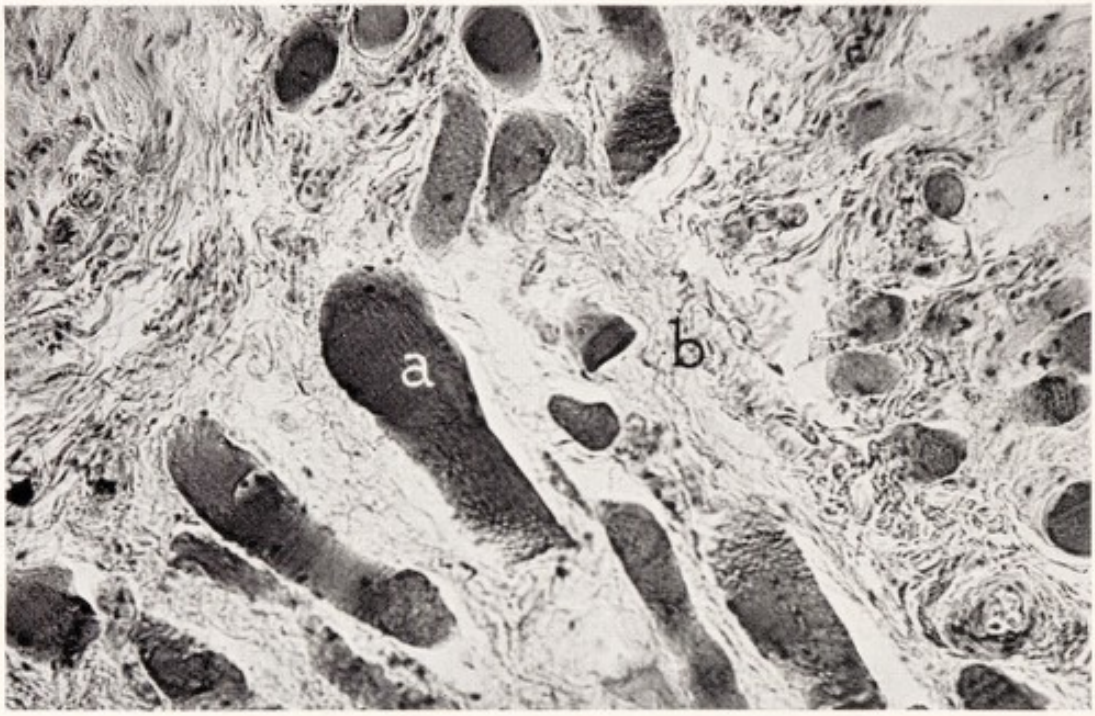

FIG. 67. Macroglossia: *a*, the muscle fibers are irregularly formed and very much enlarged. They vary in stainability; *b*, connective tissue is much increased, containing few ovoid and spindle cells. The fibers are separated by a stainless material.

they are not attended by an increase of all the specific elements of the organ as is the case in macroglossia.

Pathogenesis. The history of the beginnings is usually obscure but usually an abnormally enlarged tongue is noted soon after birth. The organ is large, sometimes huge, before it comes under observation. Their long duration is usually attested to by the deformed jaws, the teeth usually being more or less parallel with the long axis of the tongue. When the tongue becomes too large to be harboured in the oral cavity the patient does the obvious thing—sticks it out (Fig. 66). Apparently these lesions do just one thing, grow gradually larger, gradually and not by acute exacerbations as is the case as noted in the lymphangiomas.

Pathology. The entire tongue is involved but retains more or less its original shape though certain regions show greater density than others. The feel is firmer than a normal tongue and it lacks almost entirely the capacity to move. Pressure on the teeth may cause local ulcer.

Histology. In the enlarged tongues apparently the muscle fiber changes are dominant (Fig. 67). The lymphoid and blood spaces are not marked by increased size or numbers. A considerable part of the bulk is made up of stainless material such as one notes in elephantiasis.

Macrocheilia. The term implies merely a thickened lip. Usually the

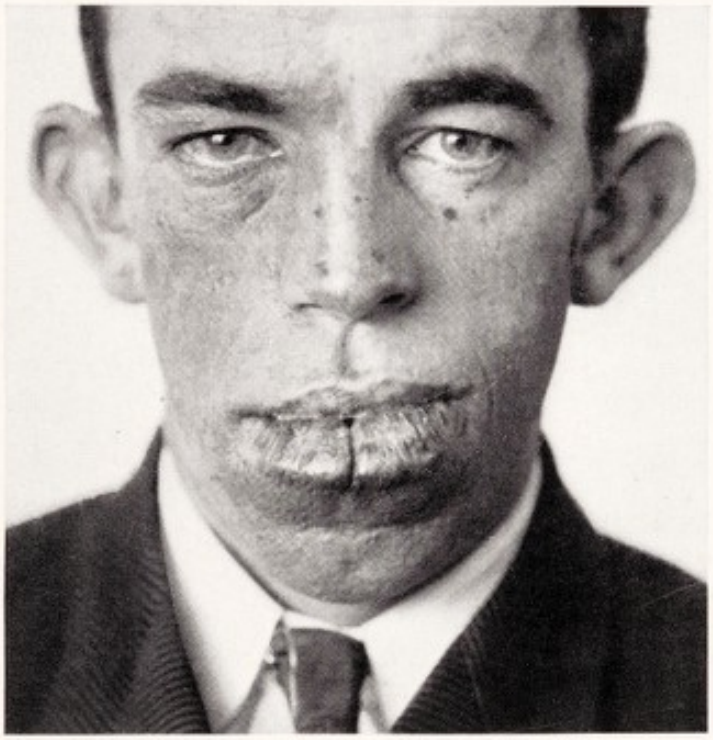

FIG. 68. Macrocheilia. The thick incompressible lip is associated with a capillary nevus.

increase in size is due to an increase of the muscle and connective tissue; usually the lymph and blood vessels add more than their normal portion to the increase in size of the organ.

Pathogenesis. The lip seems less prone than the tongue to gradually increase in size. Usually when they have attained a size several times the normal they remain stationary (Fig. 68). The increase in size may be so little in evidence that the disorder is neglected until the child reaches a self-conscious age.

Pathology. In most cases the surface epithelium is unchanged. Nothing except increase in size of the organ is in evidence. Usually only the lower lip is involved which makes the increase in size more conspicuous.

The surface capillaries may be dilated and the appearance be that of a hemangioma. This idea is dispelled when the organ is grasped between thumb and finger and is found to be but little compressible. The feel of the lip may be that of the normal organ, the only difference being that there is more of it but in some cases the whole enlarged area is definitely firmer than normal.

Histology. The bulk of the tissue is made up of an increase of the muscle and connective tissue (Fig. 69) though the blood vessels and

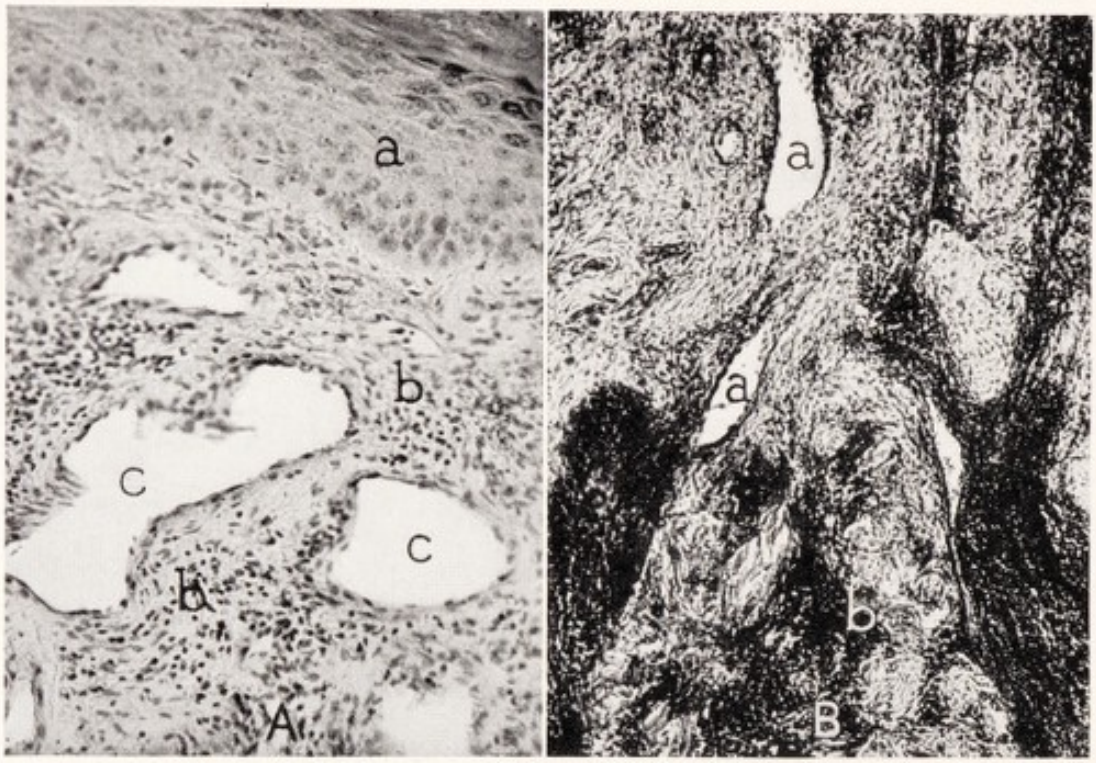

FIG. 69. Macrocheilia: *A*, *a*, covering epithelium of the lip irregularly thickened; *b*,*b*, connective tissue varying much in cellularity; *c*,*c*, dilated blood vessels; *B*, deeper slide, chiefly connective tissue, possibly neurogenic; *a*,*a*, lymph channels; *b*, connective tissue.

lymph channels may be more prominent, the proportion varying greatly in different cases.

Macromelia. This deformity involving the cheek is the same as that involving the lip, the difference being only that of topography. It is usually apparent at birth but is commonly overlooked for some months or even longer. The state is usually first discovered by some well wishing neighbor who inspects the new arrival in the hope of finding something wrong.

Pathogenesis. Thickened cheek may be made up largely of connective tissue, likely of neurogenic origin, and dilated lymph channels but

sometimes the muscle fibers and even the fat of the sucking pad may take part. Naturally the thickened cheek protrudes into the cavity of the mouth as much as possible. When the child develops teeth the protrusion may be impinged upon.

When the enlargement of the cheek is discovered soon after birth, the thickening may be but slight so that the doctor comes into the case as a referee to settle a family or communal controversy. When the doctor deftly uses a bimanual touch to aid his estimate usually the audience is

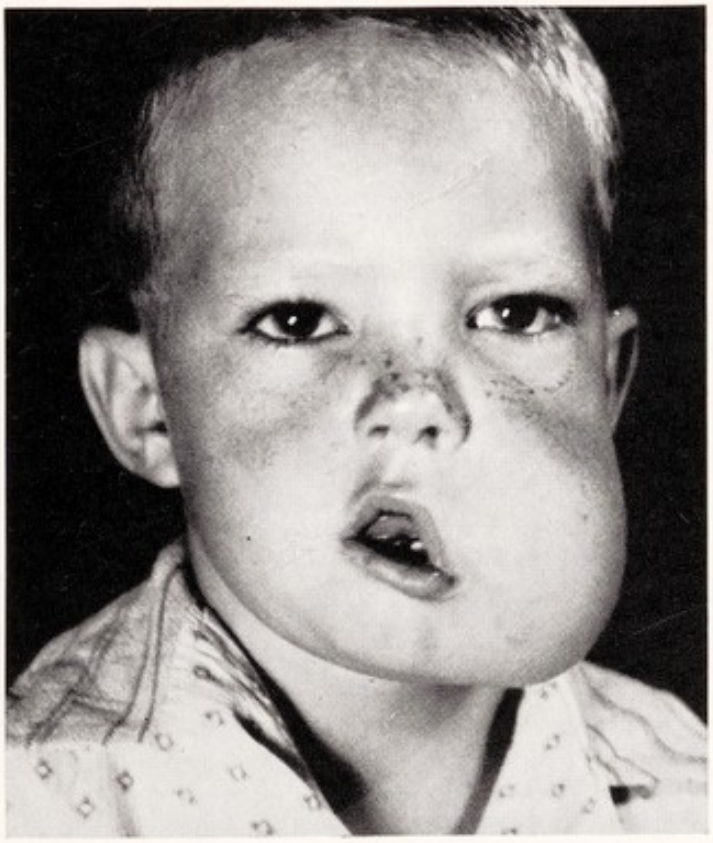

FIG. 70. Macromelia. The enlarged parts are firm, incompressible.

chagrined by the failure to institute this aid. Usually the size gradually becomes more pronounced but having attained a certain size, the condition is prone to remain stationary.

Pathology. The deformity is usually evident on inspection (Fig. 70). The enlarged parts are firm instead of flabby as in cavernous lymphangioma which they resemble on inspection. The weight is manifest by the obvious sagging of the cheek when it is lifted up in the hand.

Histology. The slide, like the analogous thickening of the lips, shows a varying increase in connective tissue, muscle fibers, blood and particularly lymph vessels (Fig. 71).

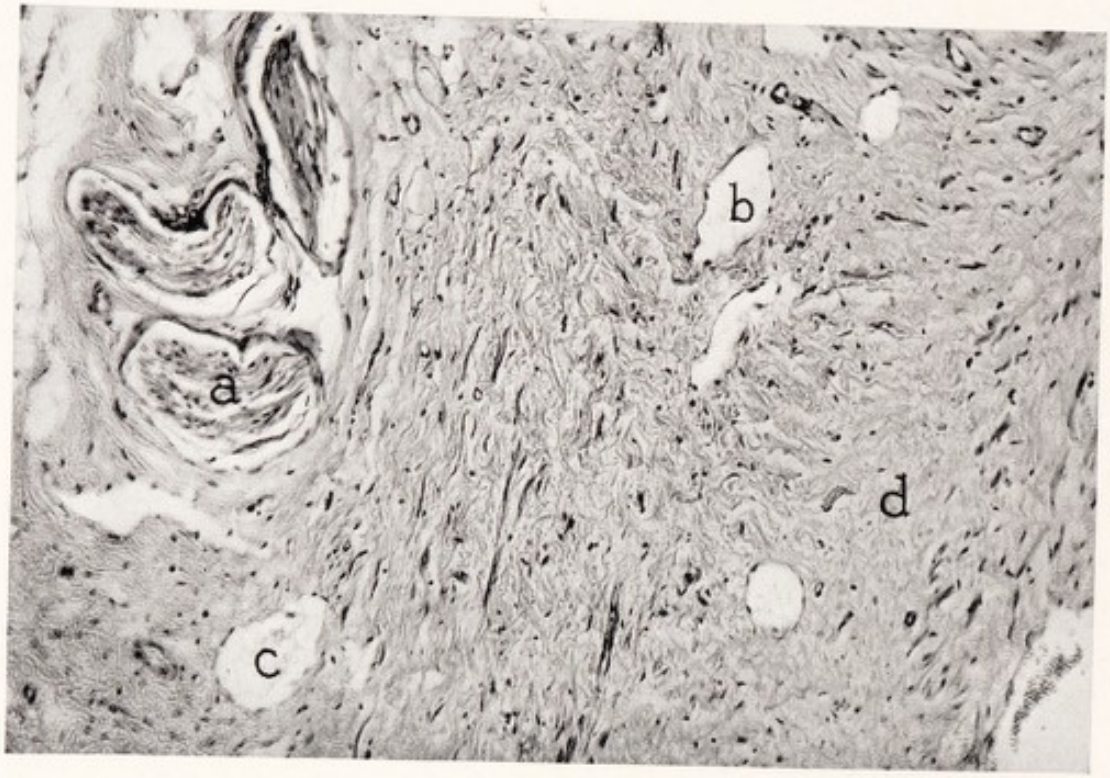

FIG. 71. Macromelia. The slide is made up chiefly of fibrous tissue: *a*, nerve fibers; *b*, lymph spaces; *c*, blood vessels; *d*, wavy connective tissue containing a few small deeply staining spindle cells.

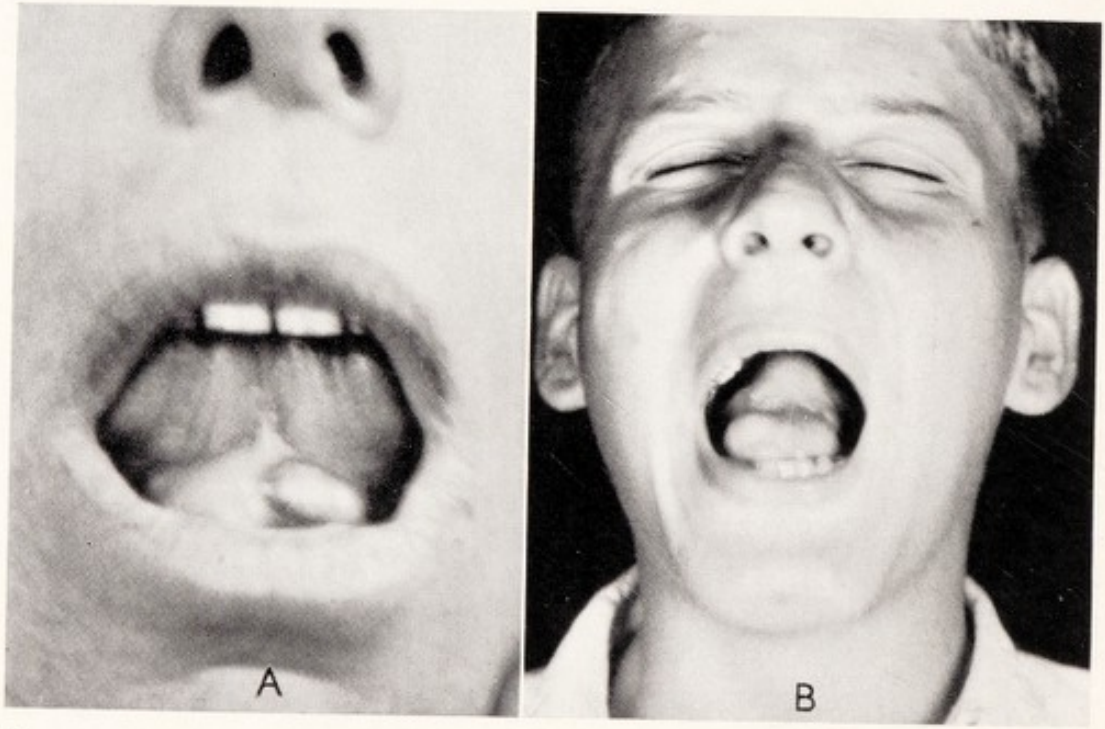

FIG. 72. Ranulas. *A*, An ovoid tumor occupies the space under the tongue and to the left of the frenulum. *B*, Bilateral.

BENIGN TUMORS OF THE MOUTH AND TONGUE

A number of benign tumors are found in the oral cavity, none of great importance. Some are congenital, most of them acquired. Cysts, vessel tumors, connective tissue tumors, and papillomas of a number of types are found here.

Cysts. A variety of small cysts are found about the cheeks, lips and tongue due to closing of the ducts of glands, and developmental disturbances. Ranulas and cysts on the bottom of the tip of the tongue or

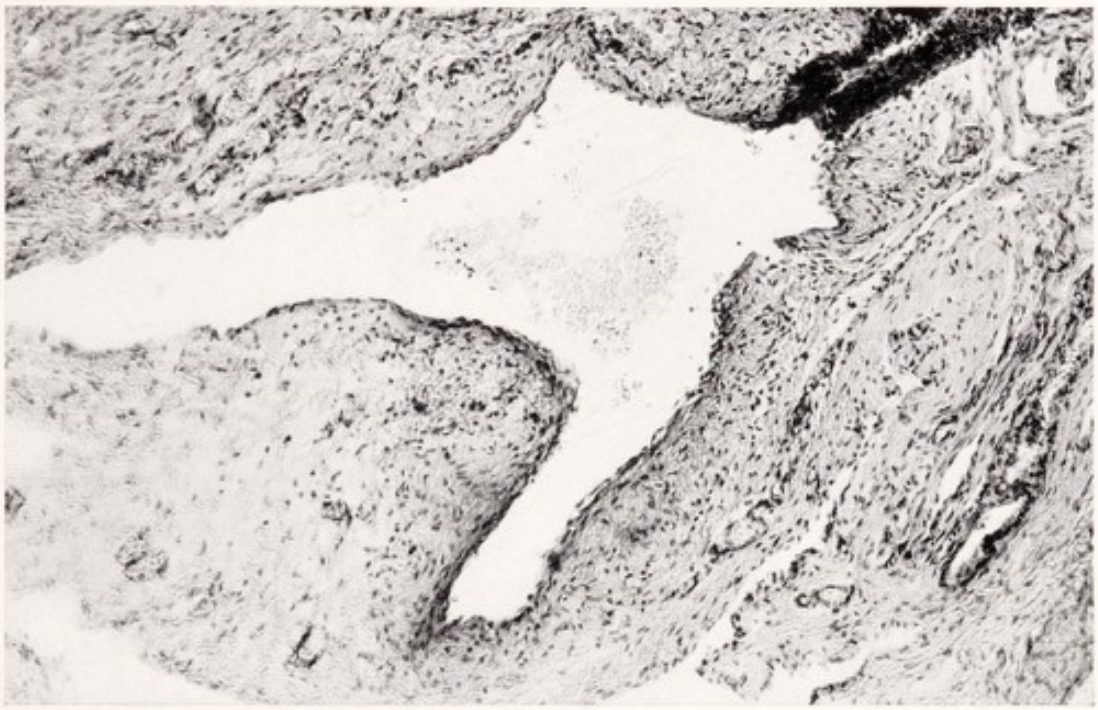

FIG. 73. Section of a collapsed ranula. The cyst is lined with a stratified squamous epithelium partly destroyed. The wall is made up of loose palely staining connective tissue with a few mostly spindleform nuclei. Immediately below the epithelium the connective tissue cells are large, oat-shaped and deeply staining.

cheeks, cysts the result of maldevelopment of the thyroglossal duct and others of obscure nature are encountered.

Ranulas. These are bluish cysts situated beneath the tongue. They are supposed to be due to obstruction of the sublingual ducts but if so they cause no disturbance of the gland.

Pathogenesis. Ranulas may be congenital or may occur at any period of life. They are not more common in women than in men. They form bluish tumors situated beneath the tongue either on one or both sides of the frenulum (Fig. 72). They may become so large that they interfere with the action of the tongue and produce protruding tumors in the neck. Usually they are larger than the end of a finger. Sometimes cysts

are encountered which seem to be attached to the undersurface of the tongue rather than to the floor of the mouth as is true of the ranulas.

Pathology. Their blue color, soft to the touch, situated just lateral to the frenulum constitutes the findings. When cut into, a more or less tenacious fluid escapes.

Histology. The cyst wall is made up of a fibrous capsule lined with a stratified epithelium (Fig. 73). The cysts attached to the bottom of the tongue are lined with columnar epithelium (Fig. 74).

Thyroglossal Cysts of the Tongue. These are situated in the midline

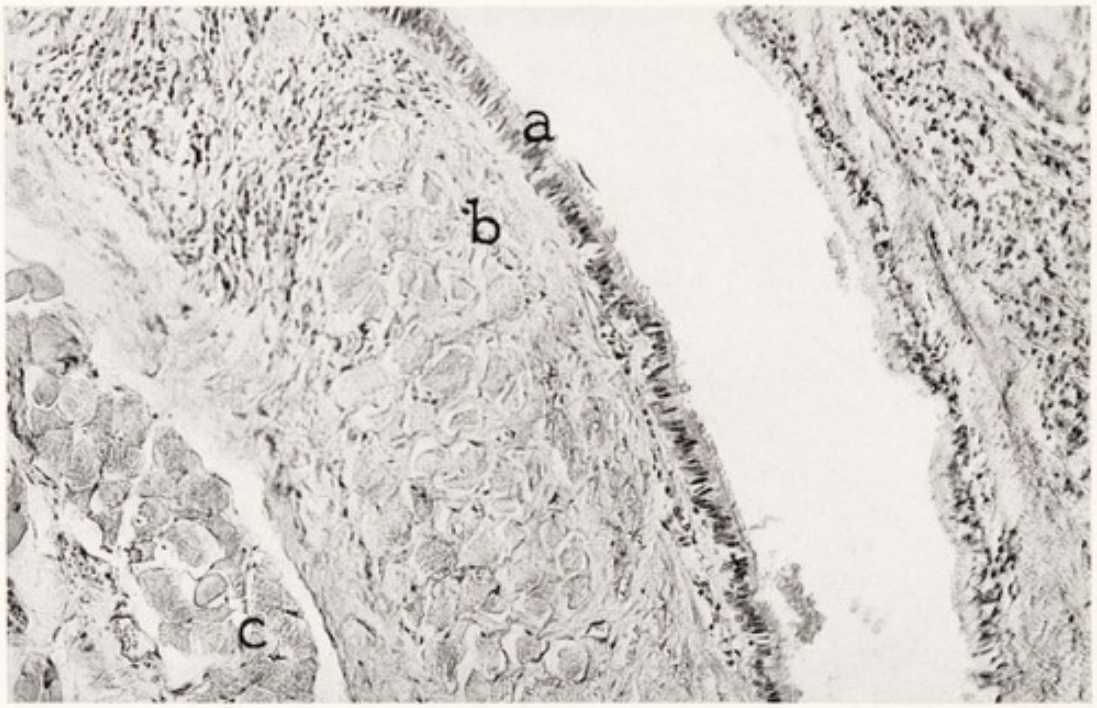

FIG. 74. Slide from a sublingual cyst: *a*, the cyst wall is lined with a distinct columnar epithelium; *b*, beneath this on the tongue side is a muscle layer; *c*, the protruding side of the cyst is made up of connective tissue containing lymphoid cells.

just in front of the epiglottis and, as the name suggests, are due to some disturbance in the obliteration of the thyroglossal ducts.

Pathogenesis. They usually appear in midlife as rounded objects at the base of the tongue. They may not be visible until the tongue is protruded far out. There may be considerable complaint of disturbance of deglutition. The cause of the complaint is not apparent unless the tumor is examined bimanually. A tumor that seems small to inspection may be recognized as a deeply seated tumor the size of a walnut.

Pathology. The tumor is smooth, spheroidal and elastic and situated exactly in the midline appearing as a bluish dome (Fig. 75).

Histology. The fibrous wall is lined with a flat or cuboidal epithelium as is true of the same much more common structure in the neck.

Cholesteotomas (Fibromas, Mucoid Cysts). As a corollary to the above may be mentioned a number of innocent lesions which are found in the midline of the tongue anterior to the site of the thyroglossal cysts. Some contain cholesterin crystals, some a thick mucus, some are wholly fibrous.

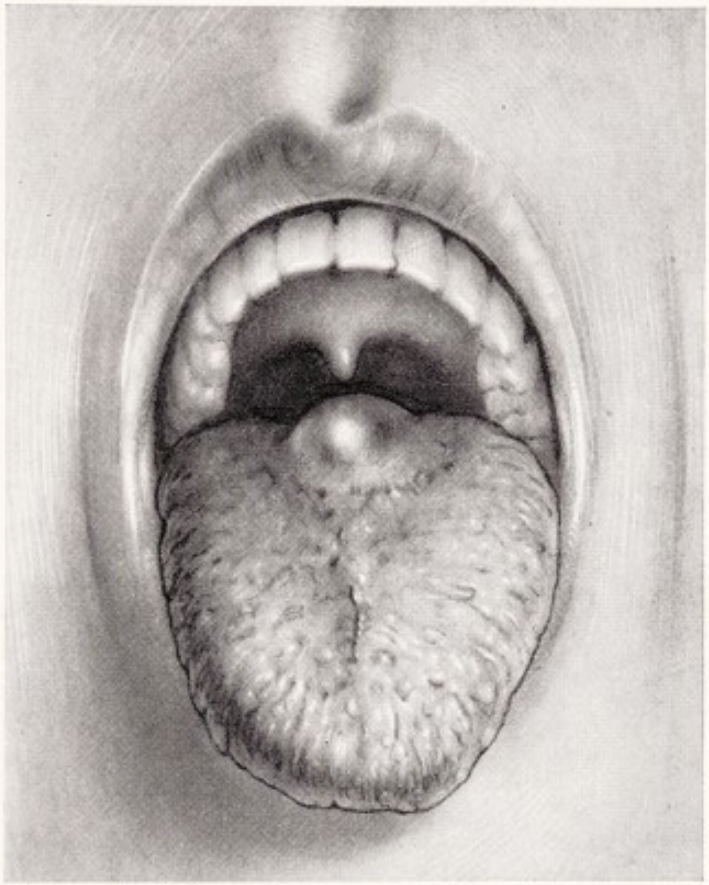

FIG. 75. Thyroglossal cyst of the base of the tongue.

Pathogenesis. These tumors are usually the size of a pea when presented for inspection. They cause no trouble, just curiosity, and seem not to develop further. Because of these facts it requires considerable persuasion on the part of the surgeon to induce these patients to sacrifice their tumor on the altar of science. Sometimes the cysts lose their tops and an ulcer results.

Pathology. All are smooth elevations, situated in the midline. The solid ones are whitish (*A*, Fig. 76), the mucus-containing ones may be bluish (*B*, Fig. 76). The ulcerated ones present an ovoid punched out defect with sharp edges and a granular base (Fig. 77).

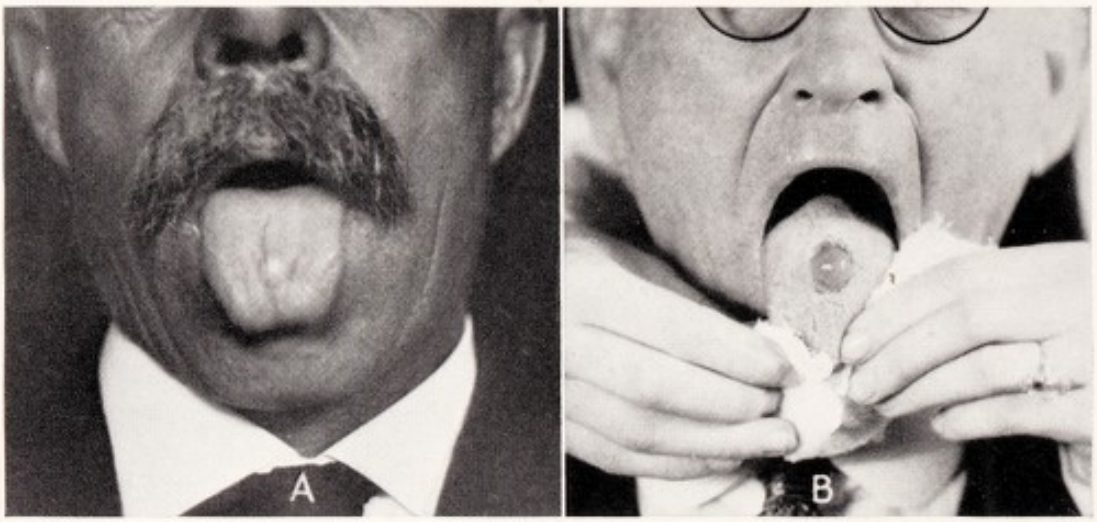

FIG. 76. Connective tissue tumors of the tongue: *A*, Cholesteatoma; the tumor was filled with cholesterin crystals; *B*, a cyst of the tongue contained a thick mucus.

Histology. The structure of the cholesteatomas is a whitish structureless material quite like similar lesions in the mastoid region. The cysts are lined with cuboid epithelium surrounded by a connective tissue wall. The ulcers devoid of epithelium contain a granular wall.

Mucous Cysts. These may develop wherever mucous glands are found. They occur at any adult age.

Pathogenesis. Usually they attain the size of a pea but may become as large as a hickory nut. They are usually situated on the cheeks produc-

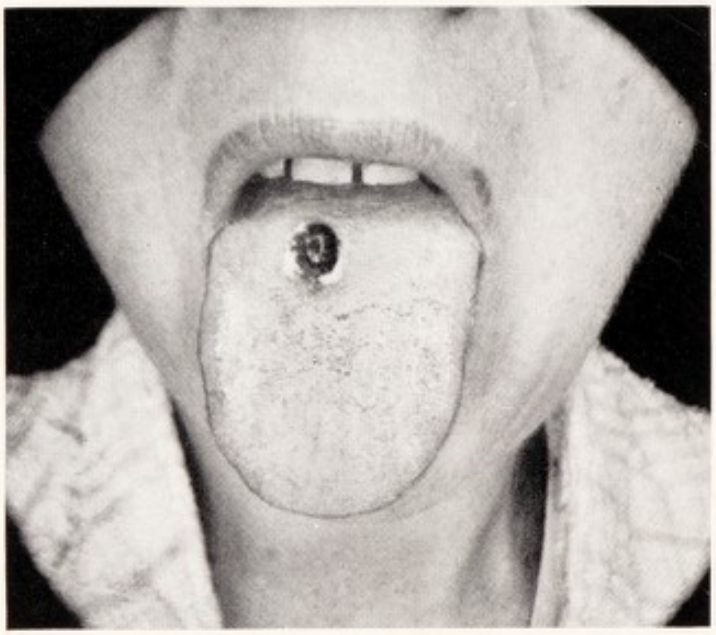

FIG. 77. Ulcer of the midline of the tongue. It started in a "big pimple." Painless but sensitive to pressure.

ing small shiny tumors which protrude above the surface (*A*, Fig. 78). Sometimes they achieve a size that is disturbing, especially when they occur in the tongue (*B*, Fig. 78).

Pathology. They are smooth, whitish in color and may move with the mucosa.

Histology. The cyst wall is fibrous, lined with a flat epithelium.

Connective Tissue Tumors. Fibromas, lipomas, and myxomas, even chondromas, or any combination of these, may be found in the cheeks,

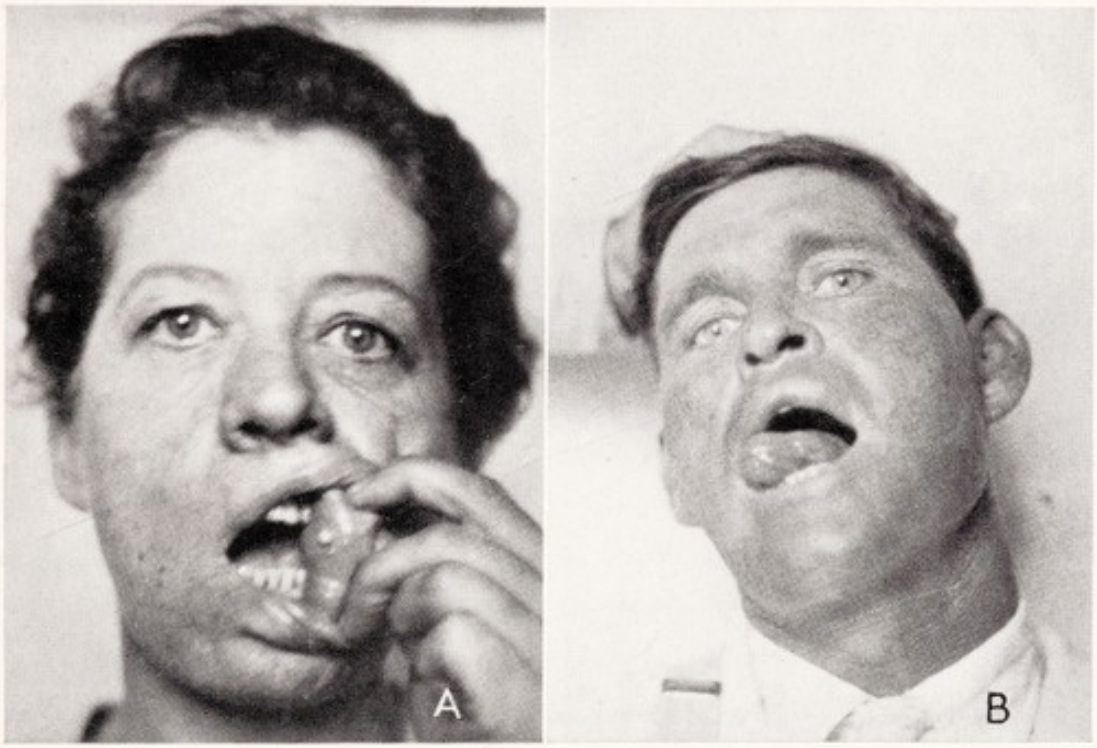

FIG. 78. Mucous cysts: *A*, pea-sized cyst protruding from the left cheek; *B*, cyst of the tongue the size of a hickory nut.

less commonly on the tongue. Small fibromas in the cheek lying just beneath the mucosa are the most common.

Pathogenesis. Fibromas may be soft connective tissue tumors or harder, made up of dense fibrous tissue, both counterparts of warts of the skin. When first discovered they are the size of a millet seed and attain the size of a pea or larger and then remain stationary. They may be pedunculated (*A*, Fig. 79) or they may have a broad base (*B*, Fig. 79). They may cause disturbance in a mechanical way if they become involved in the masticatory act. Lipomas are most common in the sucking pad. They extend in the cheek, producing unsightly tumors. Myxomas are sometimes found here and these may become sarcomatous. I have seen two such.

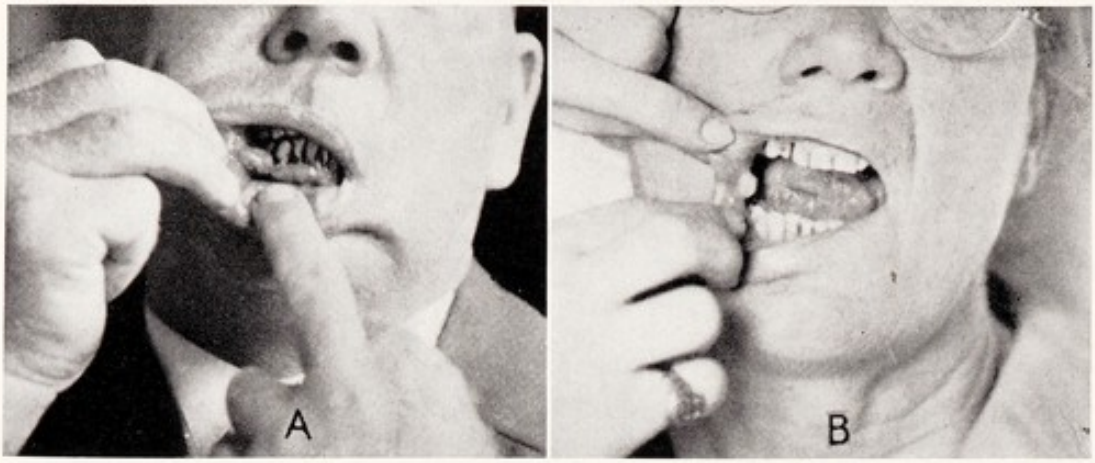

FIG. 79. Papilloma of the cheek: *A*, pedunculated fibrous papilloma; *B*, broad based papilloma.

Pathology. The appearance of these various types is characteristic. They remain stationary after they attain a certain size, except rarely the myxomas.

Histology. The microscopic structure is as simple as the gross appearance. The fibromas are covered with a stratified epithelium but the bulk of the tumor is made up of wavy connective tissue (Fig. 80).

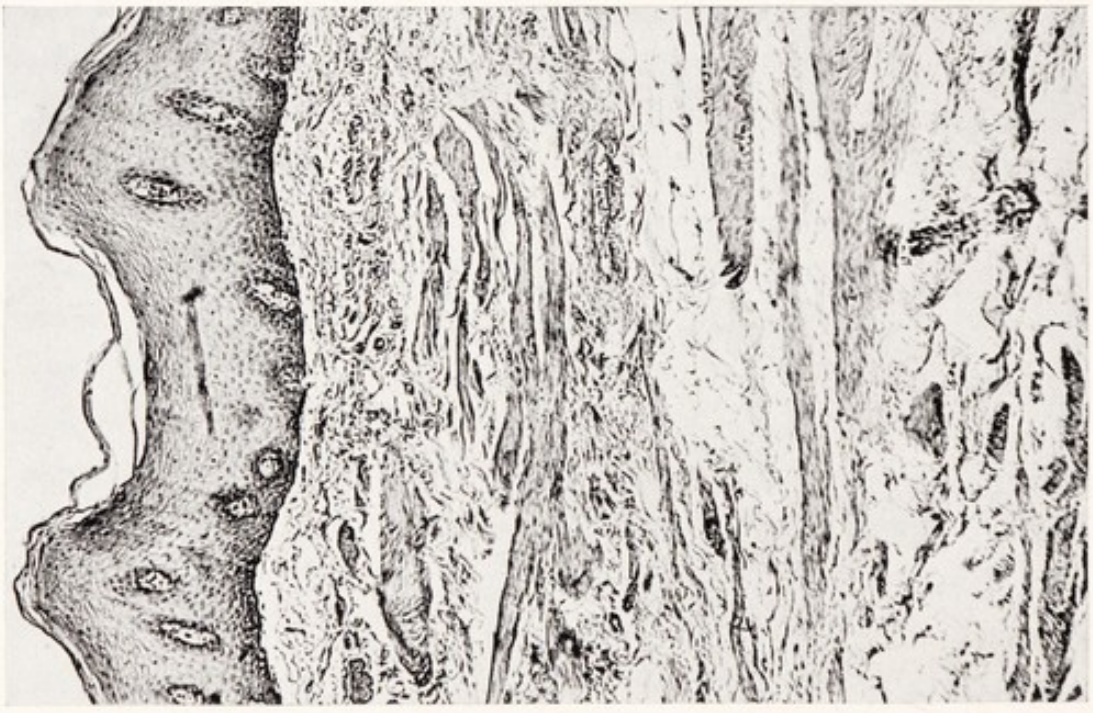

FIG. 80. Slide from a fibrous papilloma of the cheek. A layer of stratified epithelium forms the surface. Beneath is a loose wavy connective tissue containing many lymph spaces. Interspersed are round cells. The entire tissue bespeaks a soft loosely constructed tissue with few cells.

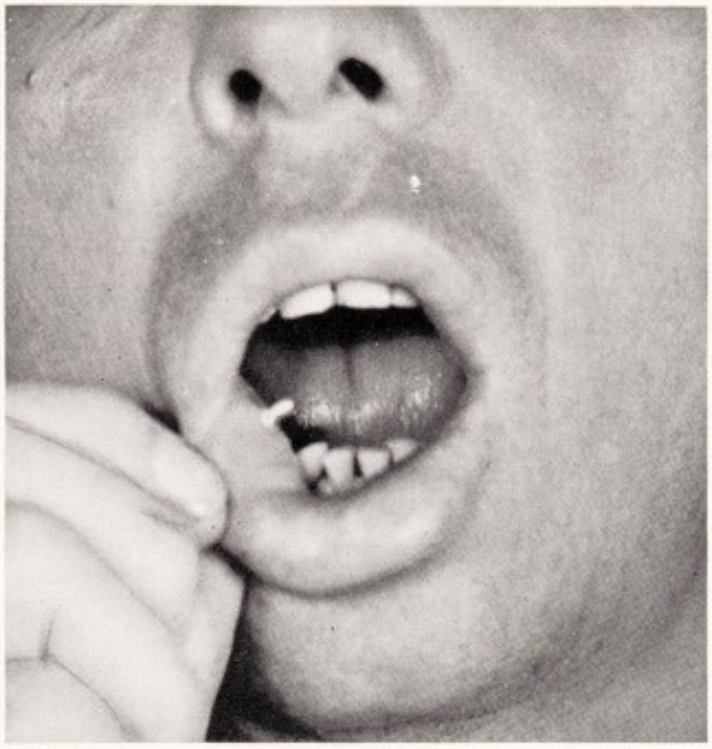

FIG. 81. Papillary horn of the cheek nearly half an inch long.

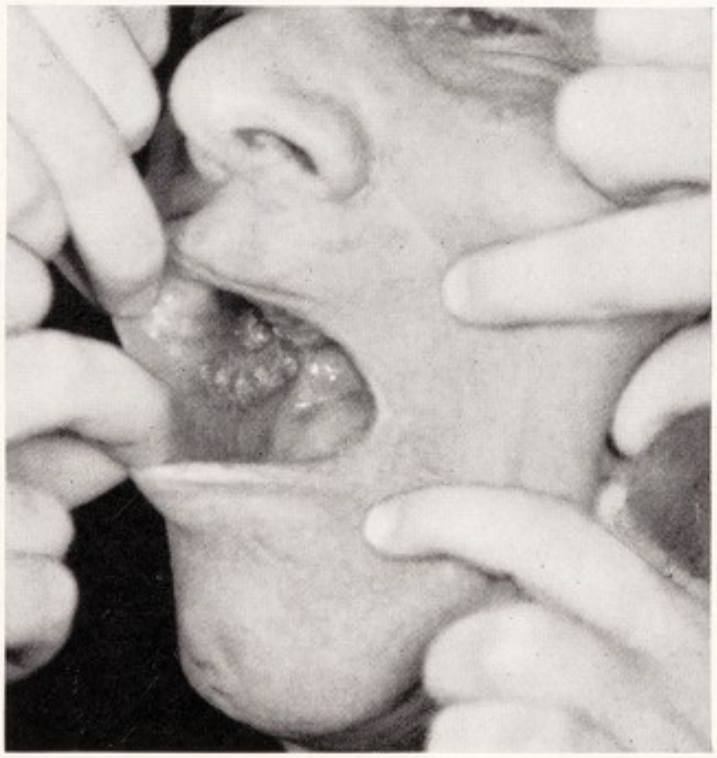

FIG. 82. Fungiform papilloma of the cheek covered with whitish cells.

Epithelial Papillomas. Horny warts are rare on the cheeks, less rare at the base of the tongue and about the pillars of the fauces. They do not seem to tend to become malignant.

Pathogenesis. Usually the tumors are not observed until, because of mechanical interference, they annoy the patient. Their rate of growth is slow. Patients endure these structures for astonishing periods of time. They cause annoyance by interfering with the act of chewing. The fungoid type sometimes invades the base. These in fact should be regarded as semimalignant.

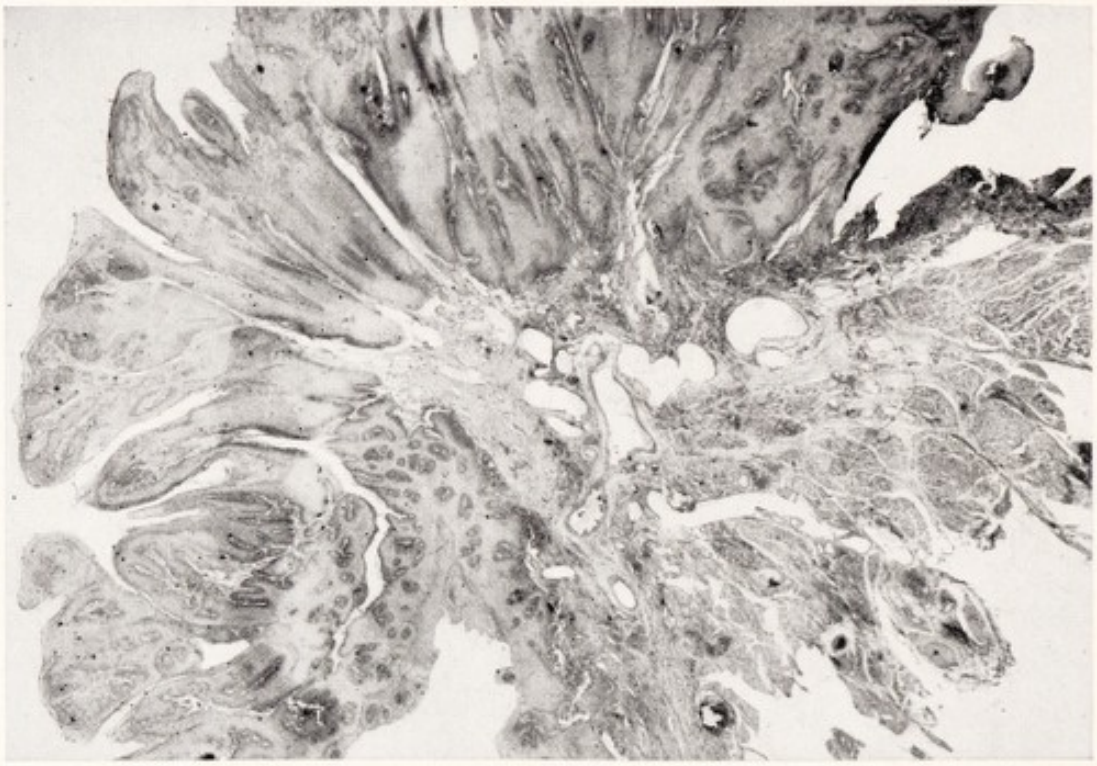

FIG. 83. Papilloma of the cheek. The mass of the tumor is made up of hornified epithelium, the nuclei of which stain poorly. The base is made up of connective tissue without cellular infiltration. The vessels are large and numerous.

Pathology. As the name indicates these little tumors are hard to the touch. In the region of the pillars of the fauces they may occur in clusters. When solitary they may attain a large size (Fig. 81). The villous type may occupy a considerable area (Fig. 82). The surface may be covered by whitish scales suggesting a kinship with the leukoplakias. Like them these sometimes become malignant.

Histology. They have the form of villous papillomas elsewhere, long finger-like processes made up of epithelium (Fig. 83). Their entire growth energy is directed upward, therefore they are not malignant.

INFLAMMATIONS OF THE MOUTH AND TONGUE

This group of lesions is of interest because the inflammation is apt to be diffuse and the site of the beginning may not be easy to determine. Usually there has been a local trauma and the problem is to locate its site. Unfortunately the problem is much broader. Specific infections, constitutional states and conditions wholly unknown must be included.

Glossitis. Inflammation of the tongue is not common but acute inflammations, even to the formation of abscess, is sometimes observed.

Pathogenesis. Pain, burning, and swelling, symptoms of any infection, dominate the picture. It may remain such for days, gradually subsiding without leaving a trace, and leaving one in the dark as to cause, or an abscess may form which when drained is followed by rapid recovery.

Pathology. Inspection reveals redness and thickening, the latter becoming more impressive on palpation. The increased heat makes the complaint of burning comprehensible. A fluctuant area may develop indicating an abscess.

Histology. The microscopic picture is that of inflammation but care is needed lest some underlying lesion be overlooked. One can secure a specimen for microscopic examination when it is necessary to drain the abscess, it may be remarked.

Diffuse Inflammation of the Floor of the Mouth. A variety of conditions may cause infection in the floor of the mouth. The commonest cause in the wheat belt in the old days of hand binding was a wheat beard. In these days of combines and harvesting by remote control, the affection is rare.

Pathogenesis. Usually there is a lack of history and a diffuse cellular infection may be the only clue. Edema under the tongue, even manifest under the chin, may be the dominant physical finding. The tongue may become involved in the inflammation. Suppuration with abscess may follow. Rarely a general edema threatens respiration.

Pathology. Marked edema is the prominent finding. The offending foreign body may be hidden in a fold of the edematous mucosa. Because of the edema, a deep fluctuation may be overlooked.

Histology. If of long duration large mononuclear cells may dominate the field, suggesting a sarcoma as is found in woody phlegmon.

Sialoliths. Stones in the duct of the submaxillary or sublingual glands may produce a diffuse swelling in the floor of the mouth differing from the above in that it is unilateral, usually with a somewhat localized area at the site of the sublingual gland.

Pathogenesis. The duct being occluded by a stone, the secretions are penned in by the resulting infection. As ball valve stones in the gall-bladder, spontaneous drainage may occur providing relief which is likely to be followed by a renewed attack after months or years. The gland may become the site of abscess formation.

Pathology. A unilateral swelling in the floor of the mouth is the usual picture. The induration may be most marked at the site of the outlet duct. The localized thickening is apt to harbor the stone where it may be demonstrated by needling. The gross findings are those of acute infection. The x-ray may show a stone (Fig. 84).

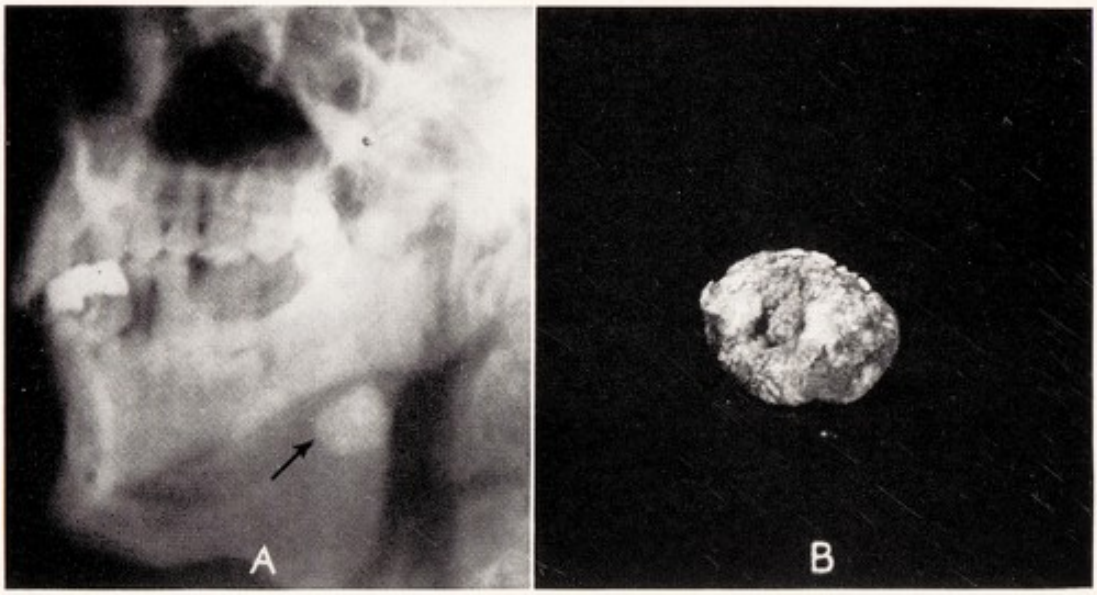

FIG. 84. Sialolith. *A*, X-ray of a stone lying below the edge of the jawbone. *B*, Stone after removal.

Histology. The microscopic structure is that of inflammation.

Vincent's Infection. This troublesome disease is due to a specific organism. It produces an intense reaction about the teeth and adjacent structures.

Pathogenesis. As to how or why the infection comes about is not clear. A pronounced inflammation rapidly develops, usually accompanied by constitutional reaction and sometimes blood changes of a serious nature. Unless correctly treated the disease may continue indefinitely.

Pathology. Acute reaction forms the picture not distinguishable from other inflammations by mere inspection.

Histology. The discovery of the spirillum makes the diagnosis. It is safest to hide behind the pathologist in making these examinations.

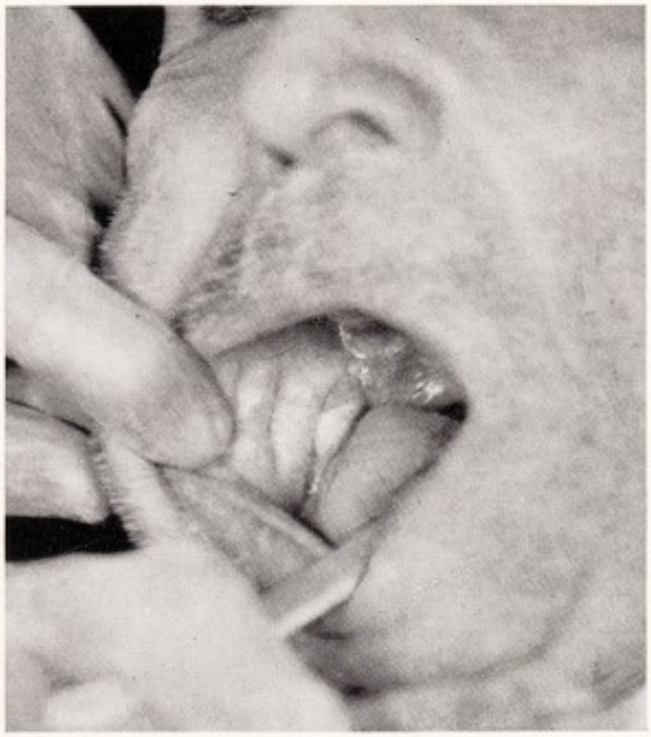

FIG. 85. Leukoplakia of the cheek and palate of mild degree which remained stationary many years.

Leukoplakia. This troublesome affection may as well be classed under the inflammations as anywhere else since we do not know what it is or what causes it. Since it occurs chiefly in men, it must be due to some

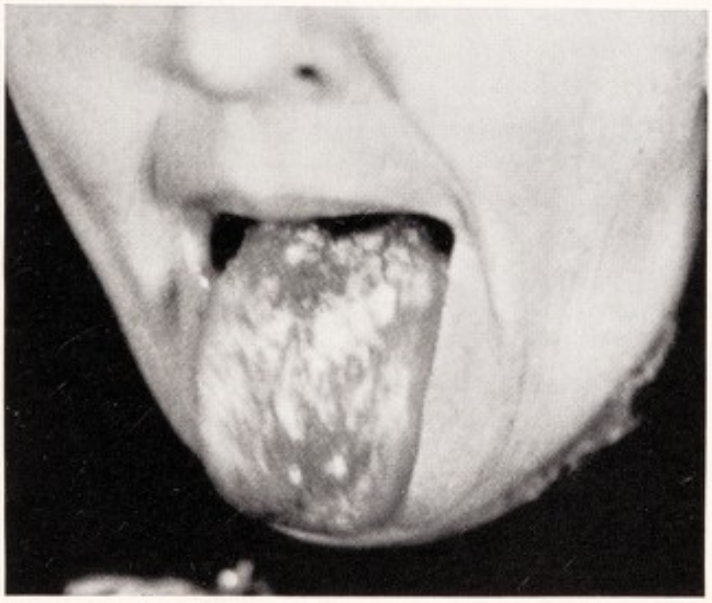

FIG. 86. Leukoplakia of the tongue and cheek. Tumor-like masses were formed (the cheek lesions were even higher).

moral dereliction but unfortunately for this theory it does not spare the nice men. Certainly it is not due to syphilis nor to smoking.

Pathogenesis. The usual initiatory lesion is a hyperemia following which a whitish granular surface develops. The process may remain stationary for years or may gradually invade new surfaces until a large part of the cheeks or tongue become involved. The whitish covering may remain there and be limited over many years (Fig. 85). On the contrary the white areas may become so piled up that veritable warty structures are formed (Fig. 86). The general tendency is toward extension in area and in depth and some become malignant. Sometimes the appearance of metastatic nodules occurs in the neck before it is realized that the local lesion has become malignant. One may well use caution. I once had a patient with an old leukoplakia who developed a tumor in his neck. This was supposed to be a metastasis. When the neck tumor was removed it proved to be a typical lympho-epithelioma, just an independent lesion. This shows that curiosity has its rewards.

Pathology. The whitish color of a more or less diffuse lesion is characteristic. When early and limited, the lesion is soft to the touch. When an approach toward malignancy begins, the lesion becomes firmer and when the border-line is definitely past, cancer hardness may be detected.

Histology. The whitish degenerated epithelium makes a characteristic picture though good sections are not easy to obtain. A surface of structureless material overlies a cellular layer in which the outline of the cell is lost. Beneath this is a layer of cells relatively normal. When a malignant stage is approached the basal layer proliferates and invades the underlying structure in a typically cancerous manner.

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CHAPTER V

Malignant Lesions of the Mouth and Tongue

THE epithelial malignancies within the oral cavity represent a variety of lesions so that the term "cancer" means but little. They differ so much in prognosis that it is necessary to consider the various regions involved as entities. Some lesions for instance are amenable to vigorous attack surgically, while in others operative procedures are useless to say the least. The stage of the disease is of the utmost importance. Because of the moist soft state of the mucosa of the oral structures, very tiny lesions are perceptible to the ungloved finger which are wholly unimpressive to sight alone. Lesions less than the size of a millet seed can be felt. The patient also can detect them early, particularly when situated on the top or sides of the tongue. When a patient "feels something," his complaint should receive attention even though casual inspection reveals nothing. Palpation may perceive a small induration only, though such lesions are usually easily recognized by the touch as already malignant. Such areas should be secured for histologic examination. This can be done by excision with a small cautery or removal with scalpel and the resulting cavity cauterized. To destroy the whole lesion is to deprive the surgeon of the only means he has of obtaining an education. The plea for material for study is particularly needed now, when most surgeons lean toward treatment by cautery or irradiation.

While lesions on the top or sides of the tongue are usually discovered by the patient those situated on the floor of the mouth present a wholly different story. Here the lesion may attain astonishing dimensions before the patient discovers it. Enlarged glands in the neck not uncommonly bring the patient to the doctor. Even visceral metastases may produce symptoms before the mouth lesion is discovered.

Leukoplakia, considered with the nonmalignant diseases of the mouth and in the chapter on the lips, in its early stages, is a very common benign lesion in the mouth. It must also be considered with the malignant diseases for it has been my experience that it will ultimately become malignant. So certain is this to happen, that the lesion should be destroyed as soon as it is recognized. Otherwise a lesion evidently benign at one visit may be definitely malignant when the patient again presents him-

self, perhaps after several years. The extent of the lesion may be so great at the first examination that its removal is uninviting, yet it will never be less and one might as well begin. If extensive, a patch at a time may be destroyed. If such a procedure is followed and careful microscopic examination of all tissues made, one will be surprised to find many areas already showing invasion.

The alleged relation of leukoplakia to syphilis has led to much frittering delay. That syphilis may produce lesions which somewhat resemble leukoplakia is of course true, but the differentiation can be made. Also it is to be remembered that a four plus Wassermann does not make of a leukoplakia a syphilitic lesion. Also a diagnosis of syphilis will not prevent a true leukoplakia from becoming malignant.

Epithelial papillomas likewise are often long neglected; though usually benign when first presented, they are worthy of careful study in the laboratory. These lesions present surprises not only in the laboratory but also in the after course.

Nowhere does the surgeon have a better chance to show his acumen than in dealing with the lesions of the mouth. Early diagnosis is possible only by careful inspection and palpation. The patient with lip cancer exhibits his lesion, the one with malignant lesion of the mouth only presents a suspicion and it devolves on the surgeon to find it and then to diagnose it after he has located it. Usually when the lesion has reached the obvious stage the prognosis is bad.

The several types cannot be adequately presented in abstract terms of cytology. Topography and type are more important, at least to the surgeon, than histologic classification in determining the likely clinical behavior of a given case. Larger, though technically operable, lesions in the floor of the mouth or at the base of the tongue are best sent at once to the radiologist. In these the diagnosis is placarded and excision adds nothing to the diagnosis.

Location also plays a role in prognosis. The lesions on the tip of the tongue differ in malignancy from those of the base, and the fungating form, no matter where situated, give a better prognosis than the ulcerating. Cancers situated on the floor of the mouth and on the cheek, if discovered at all early, give the best prognosis because of the lesser blood supply.

Least of all in malignancy is a leukoplakic patch which has just crossed the border. Some of these respond promptly to irradiation but my experience has been that they return and show excessive malignancy. Therefore the surgeon should regard leukoplakic spots as malignant, or potentially malignant, and destroy them at sight as belonging to his own

special province, after of course having secured material for histologic examination. On the contrary an ulcerating carcinoma with metastasis may be delayed in its progress by the radiologist but is likely to be speeded up by the surgeon. There is no place in tumor treatment where team work between radiologist and surgeon is more important.

All epithelial lesions of the oral cavity are treacherous. Metastases may have formed from what, on local examination, appears to be a borderline lesion. Furthermore distant metastases may form before the local lymph glands have become involved and even before the local lesions have become sufficiently impressive to send the patient to the doctor. Thus a liver tumor or dyspnea may be the patient's chief complaint. Therefore in every malignancy of the mouth one should harken to any other complaints the patient may offer us. Conversely, in obscure general conditions a thorough inspection of the oral cavity, particularly the floor of the mouth, may save the surgeon from making a foolish diagnosis. This is not an abstract observation.

The various types of the malignancies of the oral cavity may be presented in the following order. To these may be added a few rare lesions.

I. Malignant Leukoplakia
II. Carcinoma of the Tongue
III. Carcinoma of the Floor of the Mouth
IV. Carcinoma of the Cheek
V. Rare Tumors in the Oral Cavity

Of necessity the discussion of the differences in these various groups becomes more a clinical study than a pathologic one and the basis of this presentation is clinical experience rather than laboratory study.

MALIGNANT LEUKOPLAKIA

In this disease there is a peculiar keratosis involving the mucous membrane of the oral cavity. The peculiar whitish gray color is very striking and may cover considerable areas of the mucosa. The nonmalignant stages were discussed in the preceding chapter where our ignorance of the nature of the beginnings of the process was duly set forth. It needs therefore to be added here that we are equally ignorant as to why it advances from a benign to a malignant stage. Because of the slowness of this transition, a careful apprehensive watching of this disease well repays the effort. Indeed I believe that the presence of leukoplakia should not be tolerated. It should be destroyed on sight. Because of the wide distribution, this may seem to be a formidable task. But so much is certain, it will never be smaller, nor farther removed from malignancy.

The cheeks are more commonly and more extensively involved than the tongue or the floor of the mouth as a rule, but apparently when diffuse the lesion is less apt to become malignant than when there is only a lesser or more circumscribed patch. This seems particularly to be true when located on the tongue. Therefore, localized patches should always be regarded with particular suspicion.

Pathogenesis. When first noticed, a fine thin membrane covers so lightly the mucosa that it looks like one could wipe it off. This becomes thicker until a heavy white membrane forms a definite covering of the mucosa completely hiding it, and finally a replacement of the whole mucosal area. The mucosa, together with its leukoplakic covering, glides freely over the underlying tissues in a perfectly normal way in the premalignant stage. The loss of mobility signalizes a beginning malignancy. On the tongue this mobility is of course lacking and a uniform membrane is seldom seen. Usually it is patchy from thicker and thinner areas being interspersed.

In many cases, usually after some years, at some area the white patch becomes elevated above the surrounding area. The area is thicker than the normal mucosa. Small papillary projections form here and there. When the lesion is more advanced, the white plaque becomes exfoliated and a reddish area is exposed. Such areas should be regarded as likely malignant. Tobacco produces hyperplasia of epithelium on a rabbit's ear. Naturally the question arises, what, if any, effect has the use of tobacco in stimulating the developing of leukoplakia from the benign to a malignant stage. Nothing definite is known but it is plausible, quite apart from the question as to its capacity of initiating the process. To say the least, tobacco exerts no curative effect.

A warty elevation may form somewhere on the surface and, particularly when located on the tongue or cheek, may be subject to trauma in the process of mastication. That this may tend to aggravate the condition is plausible, certainly it does cause distress to the patient and one may say, as a general rule, if a leukoplakic area causes the patient pain, it should be regarded as malignant until proven innocent.

One of the commonest forms involves the anterior extremity of the tongue. The lesion is prone to pile up like the frosting on a cocoanut cake. Despite their formidable appearance these are not necessarily the most dangerous types. In fact, when a more advanced stage is reached, the thickness and flakiness of the cell excess lessens and definite wart-like nodulations, devoid of a leukoplakic covering, appear. In other words when the lesion appears to be better it is worse, a thing that should be remembered while local treatment is being used. With this advance of

the lesion, the mobility of the tongue lessens because of the exudate in the submucosal tissue. Sometimes one can make a presumptive diagnosis of malignancy by noting the manner in which the patient protrudes his tongue.

Even when the above stage has been reached, though the epithelium is invading the subjacent structures, the degree of malignancy is perhaps not great and complete removal ends the trouble. This is true because sometimes the invasion is by round cells with but relatively little invasion of the epithelium. If neglected, however, the process goes on and metastasis forms. This is particularly likely to occur early in isolated patches at the border of the tongue which escape the notice of the patient, or the growth of the lesion may go unheeded by the patient because someone sometime has advised the patient that leukoplakia is an innocent lesion and advises him to treat it with neglect. The only safe procedure is to get rid of leukoplakic areas wherever and whenever discovered. This of course may seem a somewhat idealistic view-point to the patient because sometimes the lesion is so extensive that the destruction of all the lesion seems like a formidable procedure. At least one can observe the patient from time to time and cauterize such lesions as become elevated or dense about the area in question. By so doing the doctor will have the satisfaction of knowing relatively closely the exact time when the lesion got out of bounds. This experience will reflect to the benefit of the next patient.

Sometimes ulcerous lesions are observed apparently superficial in character, not the ex-site of a leukoplakic area but there are such areas in other regions of the mouth. One must assume here that the ulcer represents the site of a former leukoplakic lesion. Therefore, while apparently in the beginning stage, they are actually at the border-line of malignancy or beyond. In fact watchful waiting usually results in regret and is useful only for statistical purposes. This reminds me of a like incident of a different nature. A patient was brought to the hospital bleeding from an abortion. A young resident was called but the bleeding continued so Dr. Chesky was summoned. "What are you doing?" was naturally the first question. The resident replied that he was watching. This brought forth the agitated query, "What will that avail you but to fix the exact time of her death?" That is about all vigilance will do in watching a leukoplakia become malignant.

Pathology. From the superficial benign lesion, which appears as though the mucous membrane had been painted with silver nitrate, to the thickened lesion of malignancy is a gradual process. When located on the cheek the buccal mucous membrane is movable with the lesion. When

located on the tongue, of course, it is at no time movable as above noted. When a normally mobile mucosa becomes fixed beneath a leukoplakia patch, the border-line of malignancy has been reached. In regions where the mucosa is fixed there is a less elastic feel in the region involved. If the white covering is removed and the red surface palpated, the unmistakable hardness of cancer can be detected by the ungloved finger at a very early stage. This is true of lesions no larger than a few millimeters in diameter. It is of the utmost importance to remember that malignancy

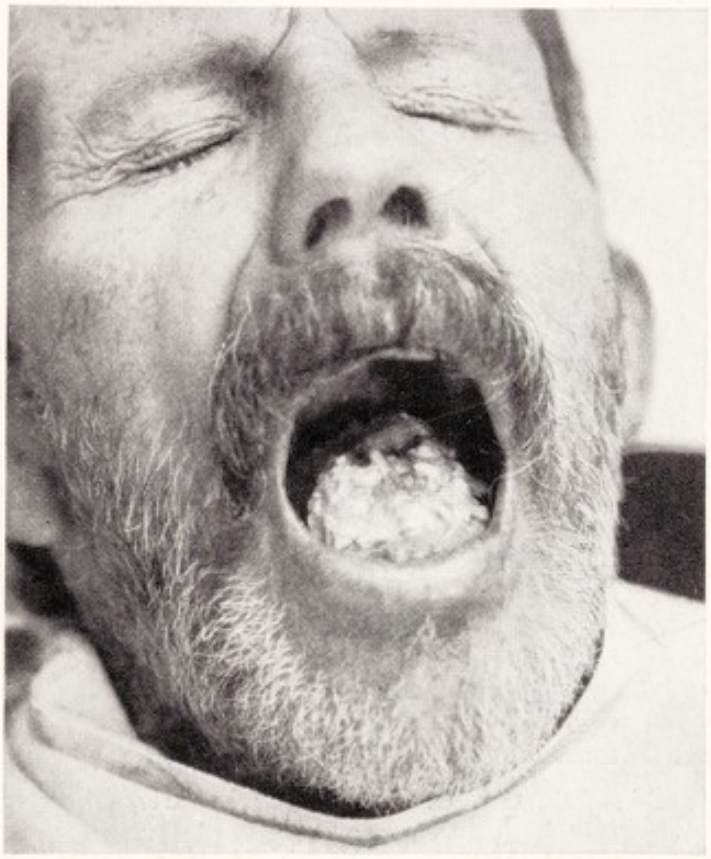

FIG. 87. Leukoplakia of the tongue. The area presents a thick pellicle which sticks more tightly than appears.

usually begins at one small point. The appearance of the lesion, as set forth, gives a clue to the approach of the danger before the physical evidence of malignancy is positive. Thickness of the covering is no evidence of the degree of progress toward malignancy. This may be evidenced by the lack of mobility. The patient may not be able to protrude his tongue (Fig. 87) while one with a thicker lesion may be able to do so (Fig. 88). Comparatively thin lesions may already show invasion.

As the disease progresses a thickened firmer area of the tongue beneath the leukoplakic covering is evident to palpation and a tumor mass

may have formed which may attain the volume of a hazelnut before notable ulceration takes place (Fig. 89). Therefore both sight and touch may be used with deliberation and care in the study of these lesions. This thickening may occur in small lesions. The casting off of the surface, leaving a small ulcerating lesion, warns that the lesion has reached a dangerous stage (Fig. 90), in fact likely is already malignant. However be it remembered that malignancy is compatible with the absence of apparent thickening of the underlying tissue. Metastases may develop from what appears to be an early and a small lesion. A hard gland in the neck may

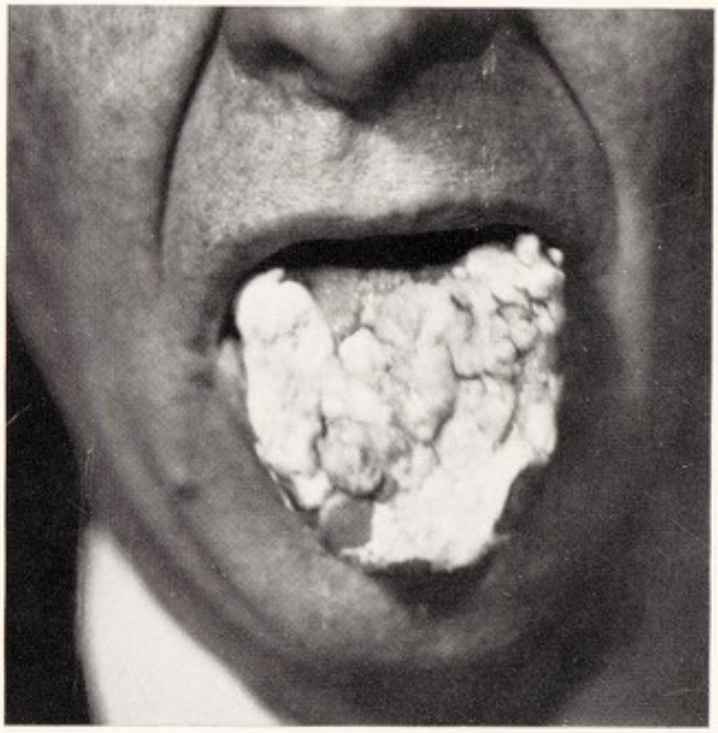

FIG. 88. Leukoplakia of the tongue. The exudate is wart-like and has lost some of its silvery appearance.

be the first evidence that impresses one with the seriousness of the intra-oral lesion. This happily is a rare occurrence but it is frequent enough to caution one to feel the neck in all cases.

Early lesions when cut across are flat, like a basal celled cancer of the face, the thin border can barely be palpated as a hard ring. When the mass is large, the regular features of cancer are in evidence. The gland metastases are characteristically hard and shot-like.

Histology. The leukoplakia patch in the innocent stage is made up of flat palely staining cells covering the normal epithelium. In the first stage toward malignancy the epithelial cells are more deeply staining than normal but they are sharply defined from the underlying connective

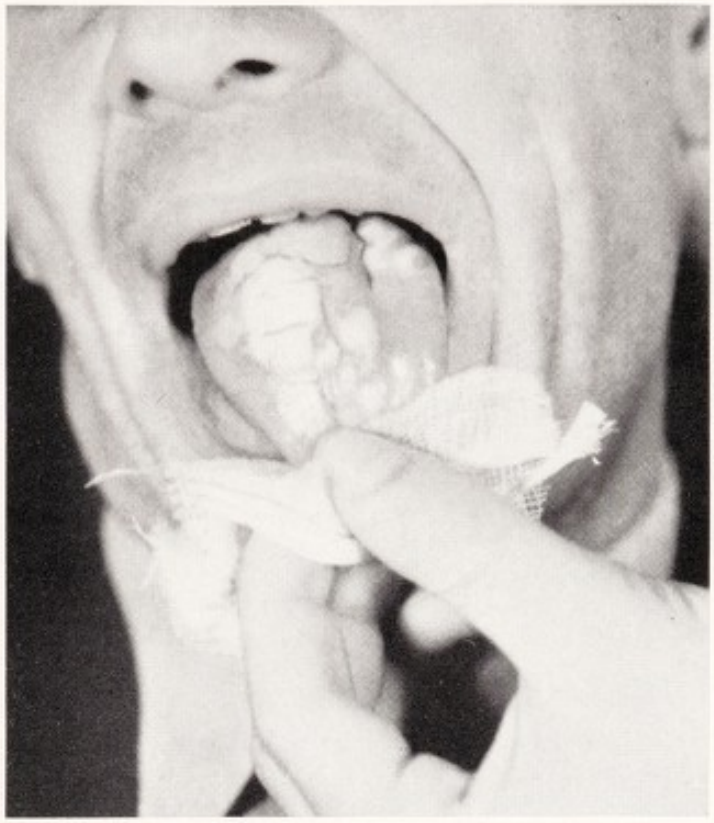

FIG. 89. Leukoplakia of the dorsum of the tongue. The lesion is elevated above the surrounding mucosa. It is thicker at several small points than over the larger area. These advanced points are on the medial edge of the lesion and show beginning invasion.

FIG. 90. Leukoplakic patch on the side of the tongue. At the front part of the patch the white portion has been exfoliated and a small red area is present. This area is definitely malignant.

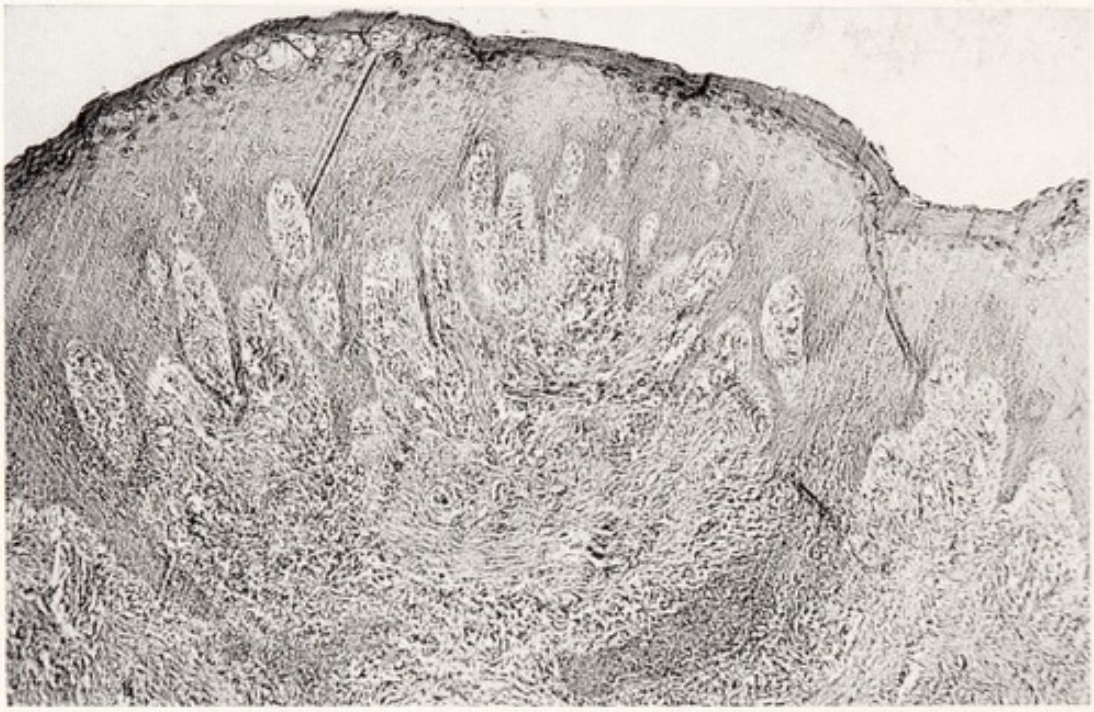

FIG. 91. Leukoplakia of the tongue. The epithelium shows hypertrophy but no definite change in cell type. There is abundant round cell infiltration but some thickening of the vessel walls. The superficial muscle bundles are partly replaced by fibrous tissue.

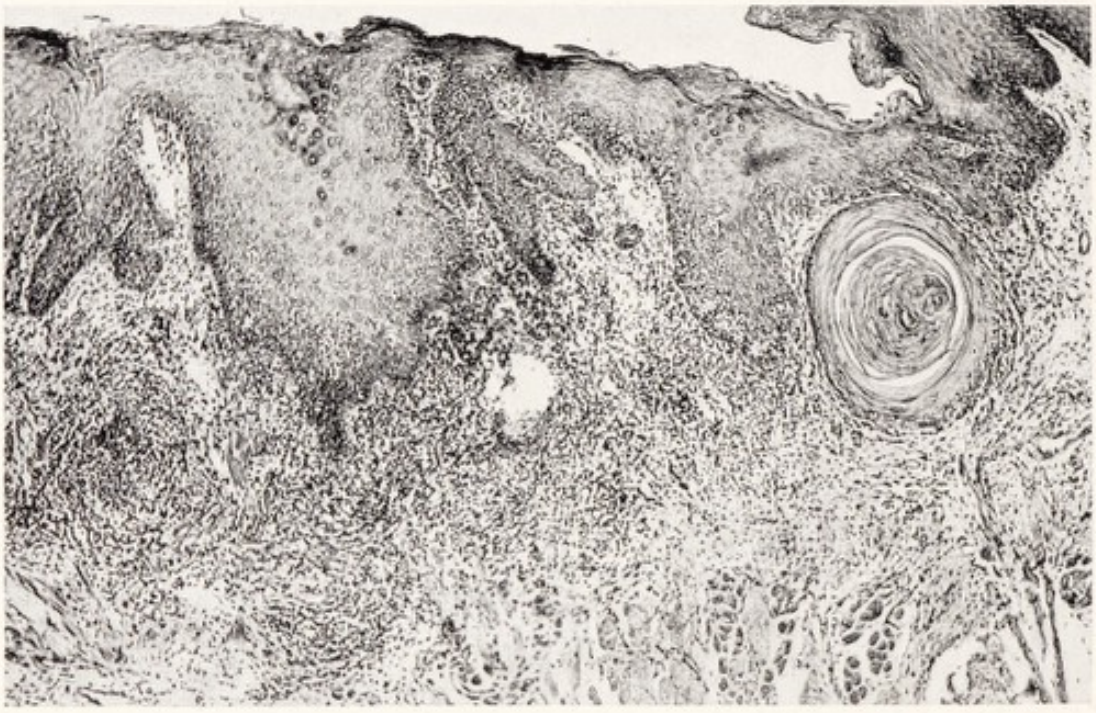

FIG. 92. Early malignant stage in a leukoplakic area. The superficial white scales have been lost in the process of sectioning, the remaining epithelium extends downward in irregular columns with marked change in cell type and is preceded at the advancing border by abundant round cell infiltration.

tissue; however there is beginning round cell infiltration adjacent to the epithelium (Fig. 91). As the stage of malignancy is approached, the deeply lying epithelial cells, in addition to being more deeply staining, begin to change in cell form. There is round celled infiltration beneath the epithelium (Fig. 92). This is the border-line stage to say the least and had best be regarded as already malignant.

In the next step the epithelial cells more definitely invade the underlying connective tissue, producing typical nests (Fig. 93). One reason for the excessive malignancy when located in the tongue is the great

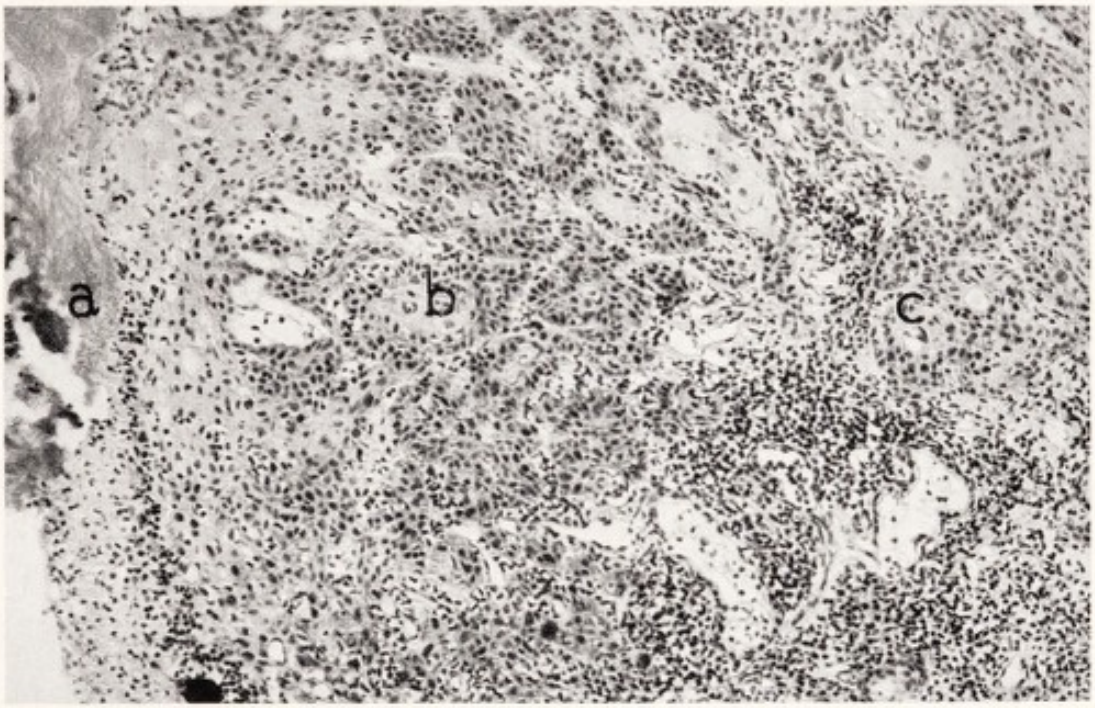

FIG. 93. Malignant leukoplakia of the tongue: *a*, but a small part of degenerated epithelium remains, the rest having been extruded; *b*, abundant cell nest formation; *c*, extensive round cell infiltration surrounds the advancing epithelium. There are many dilated vessels.

normal vascularity of this organ. This is usually apparent at the advancing border of the malignant lesion. These nests may be marked by extreme keratinization.

When ulceration appears, the entire superficial epithelium is lost and the borders undermined by the growth (Fig. 94). This type is characterized by extensive invasion of the deeper structures and is likely to be followed by early metastasis. Even in the absence of such early gland invasion, the stage of curability is past. When leukoplakia is cured by irradiation, the area may become covered by a relatively normal-appearing epithelium, while lying deeply in the tissue may be definite nests of

epithelial cells. Evidence of cure is not the disappearance of the white covering but the absence of cell nests beneath, provable only by histologic examination or by the lapse of time, never by edict alone no matter how erudite.

CARCINOMA OF THE TONGUE

Cancer of the tongue to the old surgeon brings to mind's eye one of the most dramatic, if not the most formidable, operations in surgery. The incision was made below the border of the jaw and the tongue pulled

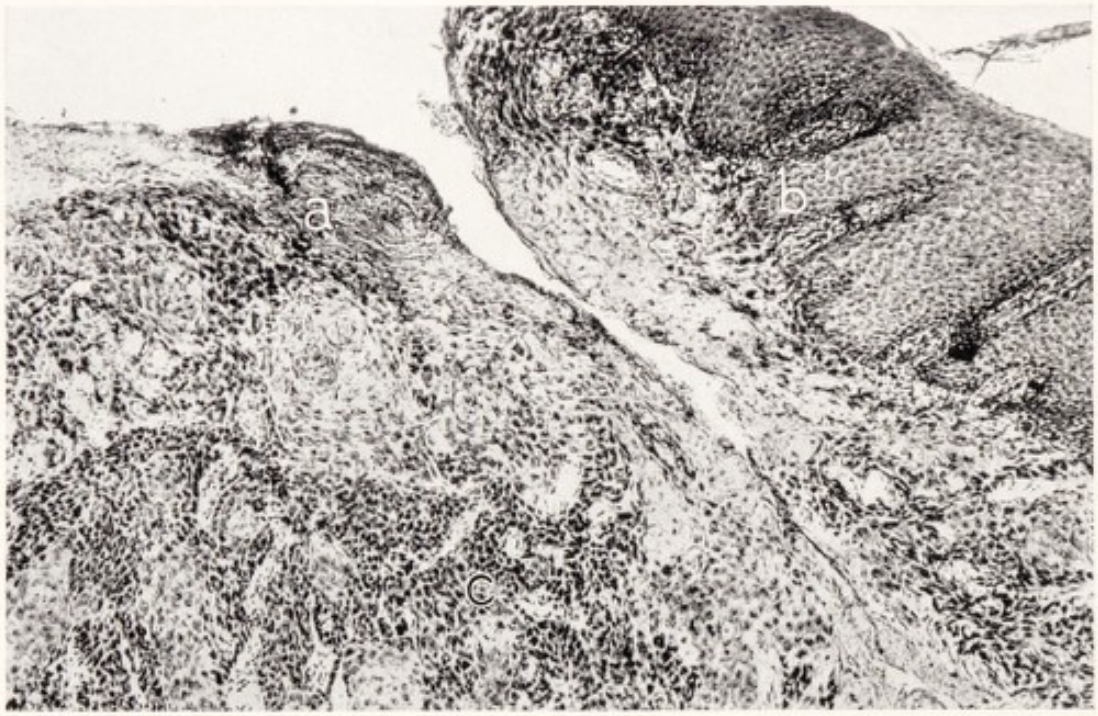

FIG. 94. Malignant leukoplakia of the tongue: *a*, the malignant mass is without epithelial covering, it having been lost in the progress of the disease; *b*, the near normal epithelium is undermined by the advancing growth; *c*, the cell nests are abundant and show a varying stainability with some tendency to keratinization.

over the neck. After the old technic it was a bloody operation, largely because of the old manner of giving anesthesia, and everybody became covered with blood. The happiest after-thoughts are two: most of the patients died of pneumonia but it gave the young surgeon material for the accurate topographic study of cancer of the tongue, and the autopsy permitted a detailed study of the glands of the neck. Under local anesthesia the operation became bloodless but the patient's efforts at swallowing made the surgeon regret his operative skill. Yet there is no more terrible death than that from cancer of the tongue. I am glad to turn the page.

Carcinomas of the tongue are fortunately not common as compared

with those of the lip or of the alveolar border. They are of excessive malignancy particularly in the vigorous in middle age. Fortunately the patient usually seeks aid as soon as a lesion is discovered. The reason is that the lesions of the tongue cause discomfort early. There is no neglect as is so common in cancer of the lip. This shows that patients are more responsive to physical discomfort than changes in the landscape of their features.

Unfortunately they are often lesions of vigorous early life when this vigilance avails them little. But they also occur in the earlier midperiod, less commonly in the aged. It is distinctly more common in men than in women. This certainly does not seem right.

No diagnosis carries less meaning than the diagnosis "cancer of the tongue." In the old emaciated anemic person with fungiform or a small ulcerous lesion with elevated border and but little extension into the depth of the tongue, there is a fair prospect of a cure. At the other end of the scale is the young florid individual with a malignant ulcer with invasion —the outlook is utterly hopeless. Early diagnosis is the other determining factor but of far less prognostic significance. It seems strange that the mouth, our most used organ, can harbor these lesser malignancies for months without attracting attention, or at least exciting the apprehension of the patient. This neglect is not at all uncommon in the aged and ignorant, especially when they are both.

The professional attitude is not always commendable. Too often a small ulcer of uncertain nature is allowed to continue untreated. This is particularly true when there is an apparent cause, such as a broken tooth. An ulcer of the tongue of uncertain nature must be excised completely, and with a margin, on sight. To treat, or better said, to tease an ulcer of unknown nature with caustic or even with irradiation without a definite diagnosis is to do something that should not be done. A biopsy, a removal of a part for the purpose of diagnosis, is something to make the judicious grieve, stupid timidity. If the lesion is small the whole thing should be removed, if too large to remove completely the diagnosis certainly can be made at the clinical examination, making a biopsy unnecessary for diagnosis but a slide must be obtained for record. This can be obtained without detriment to the patient at the moment treatment is begun. To cut out a piece "for microscopic confirmation" of a lesion which is veritably screaming its identity smacks of pseudo-science, or something else.

Pathogenesis. Though some carcinomas of the tongue begin as leukoplakic spots, as above noted, and a few begin as benign papillomas, and a few begin in ulcers caused by broken teeth or defective dental ap-

paratus, the majority just begin. A "sore place" appears, a tiny ulcer, slightly painful, nothing more. Usually there is just a small ulcer a few millimeters across and less deep but some are more deep than broad. So small are they that they can scarcely be seen but may be felt distinctly. The patient rarely brings them in at this stage. Too often they are a centimeter across and half as deep, with edges that fairly cry out their incurability. Even when the whole lesion is superficial, scarcely below the mucosa, yet it is hard to the touch, to the ungloved finger.

Sometimes, on the other hand, the superficial lesion is small but there is a considerable mass within the body of the tongue, not apparent on inspection but strikingly plain when the tongue is grasped between thumb and finger. In such cases, quite commonly the mucous membrane covering the deep lesion rapidly breaks down once it starts producing a large ulcerating mass within a few weeks. In this type the patient is apt to declare that the lesion is of a few weeks' duration which brings the suspicion of gumma, until one palpates the lesion.

Associated with such lesions sooner or later the lymph glands become involved, usually first those in the posterior digastric triangle. However metastases may develop when the tongue lesion is small. I have on several occasions had to tell the patient that he had a primary lesion of the tongue when he came, because of a tumor he had discovered in his neck.

Once metastases have occurred, the progress toward a fatal termination is rapid. The surgeon becomes just a recorder of events and a sorry tale it is. The radiologist may be able to slow the process. The ulcerous lesion extends, the glands enlarge. Pain harasses the patient and the nutrition fails. He starves to death, dies of a deglutition pneumonia; only the favored of the gods die of hemorrhage before the final scene has had time to develop.

That carcinomas located at the base of the tongue give a poorer prognosis than those nearer the tip is generally recognized. Whether the difference is due to the fact that the diagnosis is made later is difficult to say. I have never seen an early cancer at the base of the tongue such as one sees in the protrudable part.

The usual rule that epithelial malignancies develop more rapidly in the young man than in those of advanced years holds with emphasis in case of the tongue; nothing stays them and operative efforts seem only to speed them. In the aged, sparse and anemic, on the contrary, the progress may be slow and offer a fair prognosis.

Pathology. Like many cancers on exposed surfaces one sometimes has the opportunity to study the pathology when the lesion is very

small, so small indeed that the low power of a microscope can literally encompass the entire region of invasion. So small may they be that they are scarcely perceptible to sight as the patient indicates the site of his annoyance. The ulcer, even when that small, has the hard border that cannot be mistaken, the tumor bulk being made in part of associated round cell infiltration. Most difficult to judge is the small superficial ulceration, little more than an excoriation, but the excoriation has substance. Such conditions may be confusing when there are present other lesions of a benign nature (Fig. 95). I have never yet seen a cancer of the tongue that did not carry its own label at the first visit of the patient. The normal tongue is soft, the lesion is hard, if only a little hard, to the ungloved finger. Nevertheless, anyone with a particle of curiosity, confronted with such a small lesion, will instinctively remove it, body and

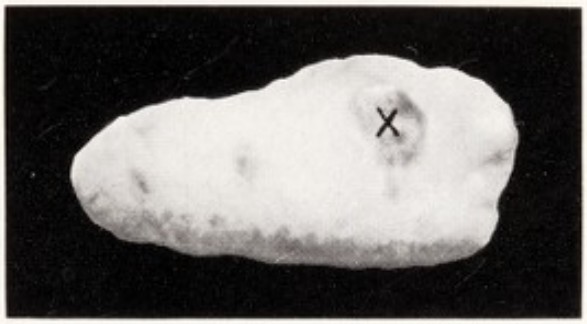

FIG. 95. Early carcinoma of the tongue. M., age 36. A lesion a few millimeters across first noticed three weeks before. A "diagnostic section" of the size shown in the figure and a block dissection of the neck did not check the growth.

soul, preferably with a small cautery. A confirmatory microscopic slide is a great comfort.

When the lesion is larger, particularly if deeper, the recognition is easy. One can see the diagnosis, but the opinion should be confirmed by the touch, with a finger in its native state.

At some time in the course of events the regional lymph glands become involved, palpable as deeply seated nodules. They begin so early that their presence should be assumed though they are not palpable; the younger the patient the more likely is this true.

Less often, instead of ulceration, deep infiltration may be the first sign of malignancy. There is just a hard place beneath the point of irritation. Early a fungous formation in which the growth extends above the surrounding mucosa is rare in cancer of the tongue. Such lesions are slowly growing and their complete removal sometimes results in cure. Small fungiform tumors, superficial in nature, are sometimes seen in younger individuals, forming superficial ulcers with elevated borders

FIG. 96. Fungus carcinoma of the tip of the tongue. The growth protrudes through the mucosa.

FIG. 97. Large carcinoma situated in the base of the tongue. Fibromuscle bundles separate the various tumor nodules.

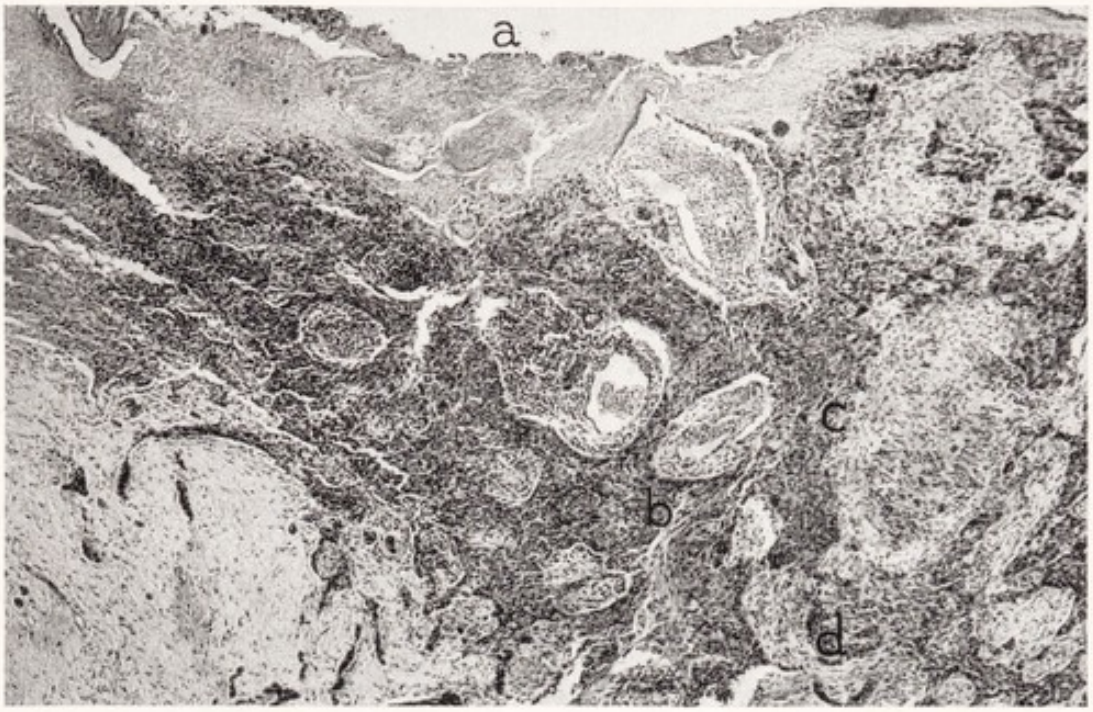

FIG. 98. Very early cancer of the tongue: *a*, superficial epithelium is absent; *b*, extensive epithelial infiltration extending between the muscle bundles of the tongue; *c*, round cell infiltration; *d*, muscle bundles separated by round cell infiltration.

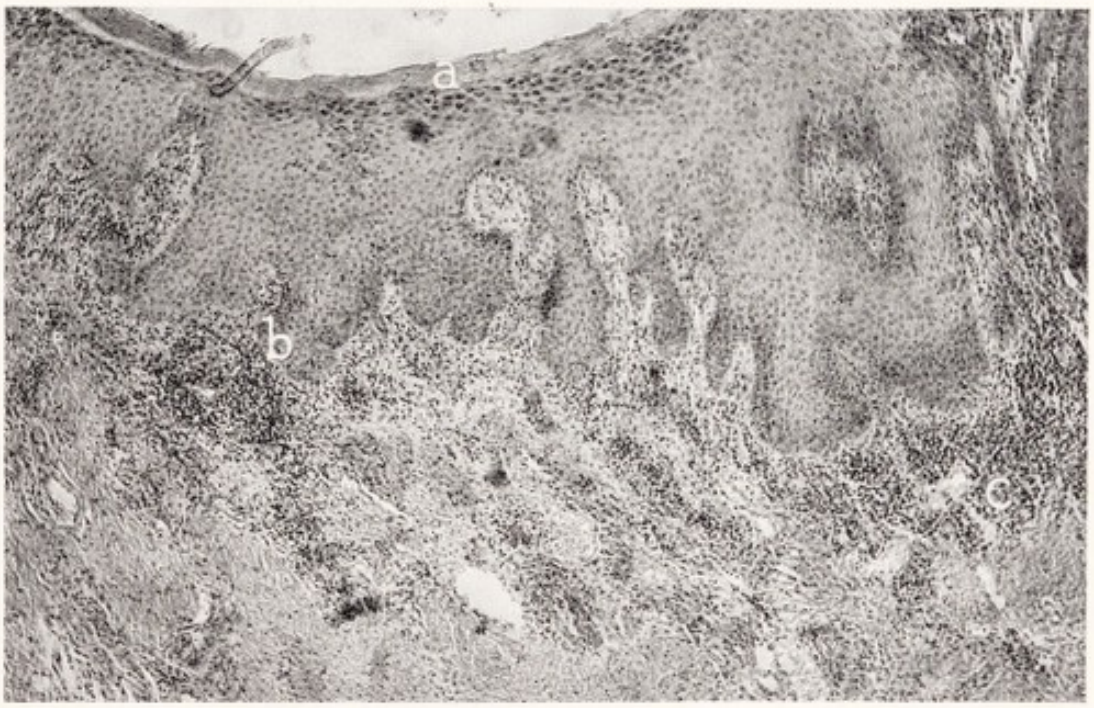

FIG. 99. Very early carcinoma of the tongue: *a*, the surface epithelium is retained; *b*, there is pronounced extension of the epithelium downward with pronounced change in cell type and stainability; *c*, extensive invasion of the muscle by round cells. There are many vessels.

(Fig. 96). These give a poor prognosis. Large bosselated masses may form within the tongue, particularly if slowly growing as one sees them, sometimes in the aged, or when located near the base of the tongue (Fig. 97).

Histology. Cancers of the tongue are easily recognized by the microscope because they start out in such a business-like way. Growths so small that they may be encompassed in a low power field leave no doubt as to the nature of the lesion (Fig. 98). Lesions so early that the superficial epithelium is still intact are sometimes encountered (Fig. 99). Cancers of the tongue if at all advanced show pearl formation. Even early, at

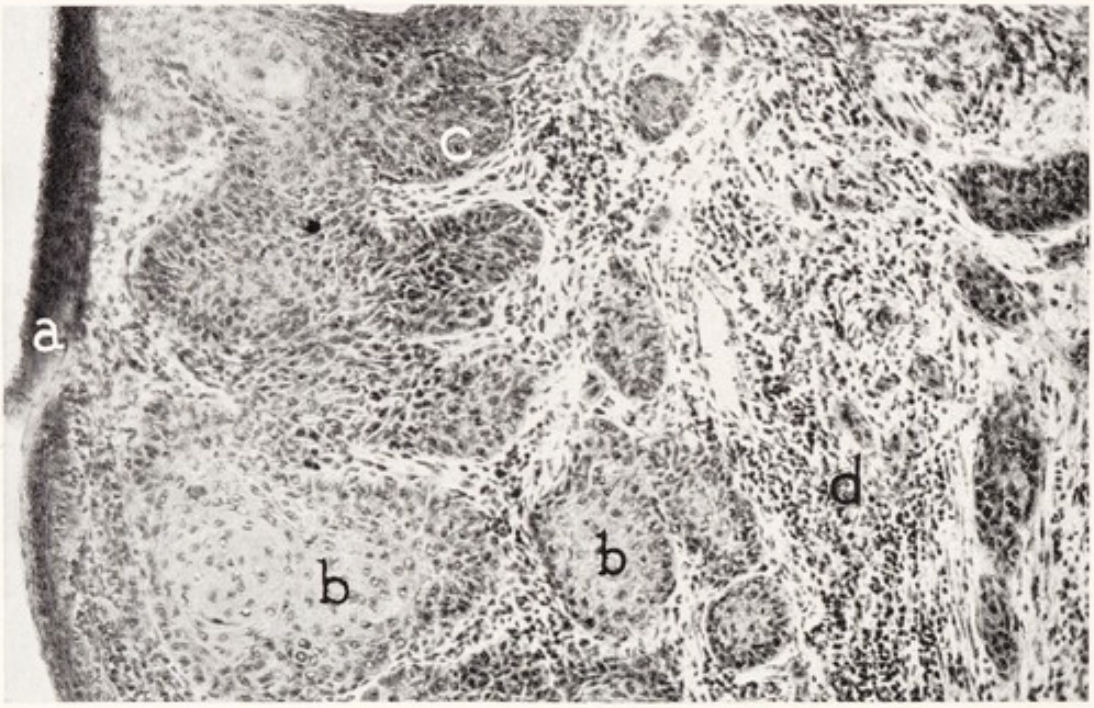

FIG. 100. Early cancer of the tongue: *a*, partly destroyed superficial epithelium; *b,b*, beginning pearl formation; *c*, advancing cell nests showing marked change in cell type; *d*, extensive round cell infiltration about the advancing cell columns and invading the muscle bundles of the tongue.

least small, lesions usually show beginning keratinization (Fig. 100). Metastases, even to the third and fourth generation, may be formed chiefly of pearls. On the contrary, very small lesions may escape detection. Even earlier lesions may be encountered in which excision is done on suspicion (Fig. 98).

CANCER OF THE FLOOR OF THE MOUTH

Carcinomas of the floor of the mouth cause many surprises because they are so commonly overlooked until local or distant metastases have formed. Despite this fact these tumors give a better aggregate prognosis

than cancer of the tongue. The reason for this, likely, is to be found in the vastly less abundant blood supply in the tissues of the floor of the mouth.

Pathogenesis. These not uncommonly are developed on a previously benign lesion. Such cases may be but mildly malignant. Unfortunately, these are usually neglected until extensive lesions have developed so that it is impossible to say whether or not they were preceded by a lesser lesion. Instead of a superficial lesion a solid tumor may occupy the floor of the mouth. This is particularly likely to be true if they have been cured a time or two by conservative means.

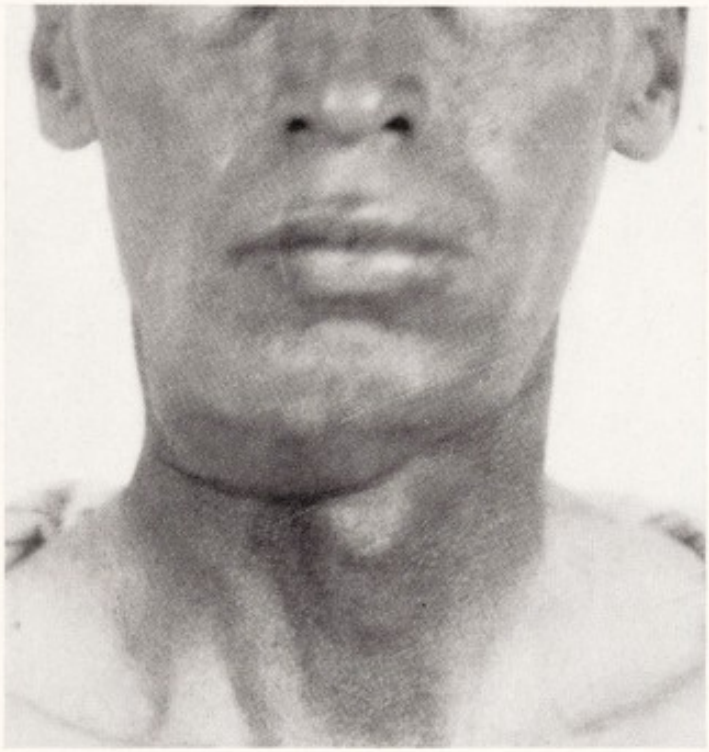

FIG. 101. Carcinoma of the floor of the mouth causing a bulging beneath the chin.

It is astonishing how long lesions in this location may be overlooked. I once had as a patient, an exceptionally capable physician 75 years of age, who came because of a series of tumors of his neck the size of hickory nuts, having wholly overlooked an ulcer the size of a Kansas sales tax token in the floor of his mouth. He was incredible when his digit, at my direction, explored the local lesion. Not rarely, too, patients with cancers in the floor of their mouths have distant metastases, notably in the liver, when they first present themselves for examination. I once did an autopsy on a man who showed multiple metastatic cancer of the liver. Prolonged search for the primary tumor finally led to the discovery of a sizable cancer beneath his tongue.

In most instances there is a superficial ulcer in the floor of the mouth with a firm hard border. I have, however, seen a lesion so small that it could be encompassed in a single field of a low power lens. The degree of induration varies. Usually, if not irritated by being cured a few times, they remain as ulcers for many months, even years, as one sometimes sees in obstreperous patients of advanced years. On the contrary, sometimes a bulging tumor under the chin may be the complaint that sends the patient to the doctor (Fig. 101). These are due to continuous or discontinuous growth rather than to metastasis. More commonly the tumorous mass protrudes upward, producing a tumor under the tongue.

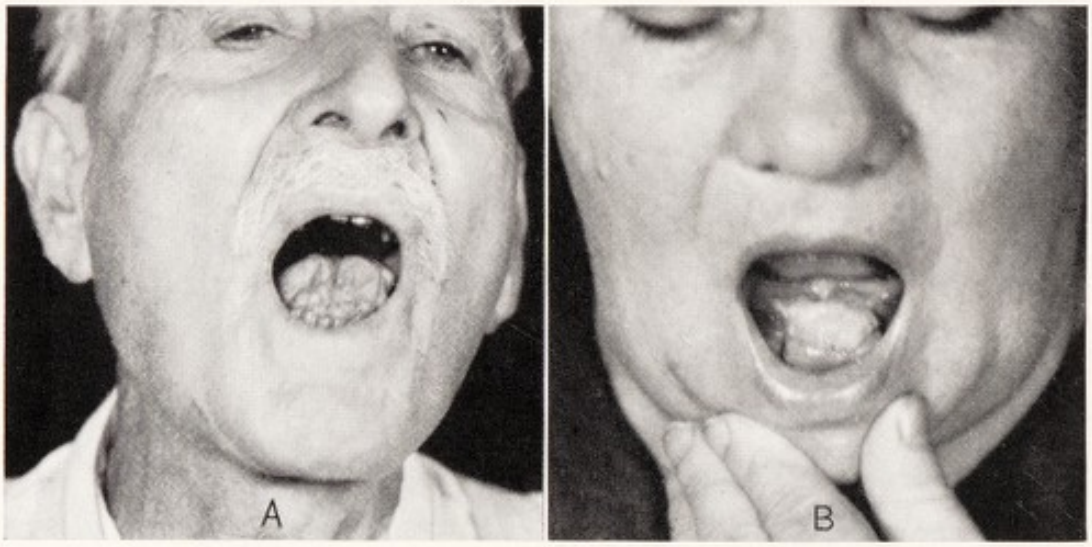

FIG. 102. Carcinomas in the floor of the mouth: *A*, Recurrence after operation. *B*, Primary tumor complained of because it interfered with the use of the dental plate. The alveolar border was not involved.

These may attain a considerable size before the patient complains (Fig. 102).

The type of lesion most commonly overlooked by intelligent people is a superficial ulcer with but little extension into the depth (Fig. 103). Rarely one finds a lesion so early that it is barely perceptible, the excision of this lesion resulting in a cure of eight years to date.

Death is most commonly caused by obstruction of the esophagus and trachea since the tumorous mass in the neck due to metastases plays a greater role than the ulcerous mass in the floor of the mouth.

Pathology. The typical cancer of the floor of the mouth presents an ulcer with hard borders. Large tumors may form filling the whole floor of the mouth which bulges into the neck, particularly in young and phlethoric patients. Sometimes one is fortunate to discover these lesions as a very early stage presenting a small hard papilloma with but a

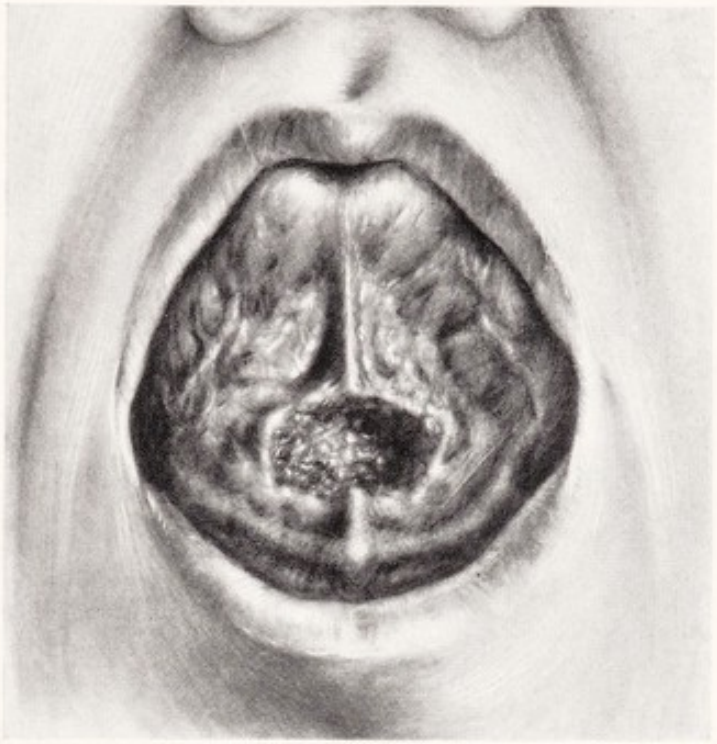

FIG. 103. Carcinoma of the floor of the mouth. The granular base is surrounded by a firm hard ridge. This patient came because of a tumor of the abdomen. He had a metastatic tumor of the liver. He had not noticed the ulcer beneath his tongue.

beginning invasion. One such tumor which I removed presented a small soft fungoid tumor which showed malignant change only in the depth of the fissure (*Insert*, Fig. 104).

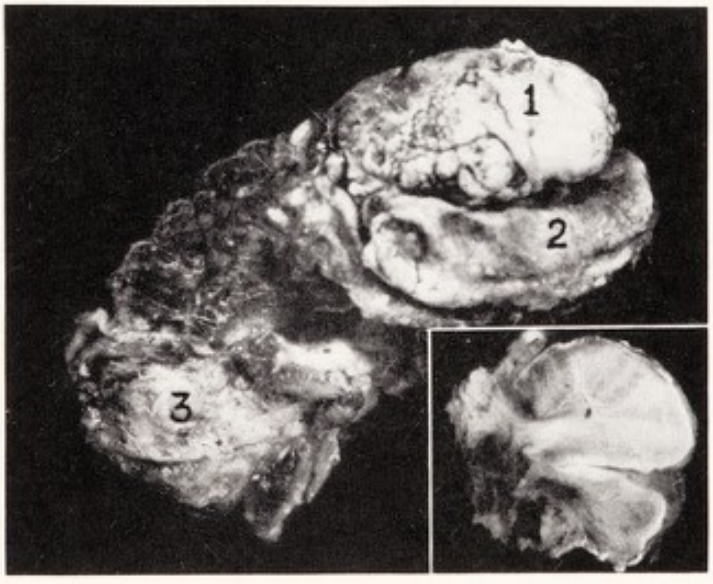

FIG. 104. Carcinoma of the floor of the mouth: *1*, the primary tumor appears as an ovoid nodule at the top; *2*, mucous membrane of the floor of the mouth; *3*, below is a malignant mass separable from the primary tumor. *Insert*, Cross-section of the tumor showing an ovoid protruding nodule.

Some tumors present a rounded mass the surface of which still retains a covering epithelium expending their growth into the loose tissues of the neck. The tumor after removal may show, besides the cancerous masses, a series of discontinuous growths (Fig. 104), and sometimes a series of secondary abscesses.

Histology. Likely because of the nature of the epithelium from which they spring cancers of the floor of the mouth tend to form undifferentiated cells, which accounts likely for their tendency to form distant metastases. They may even lose all evidence of nest formation, particu-

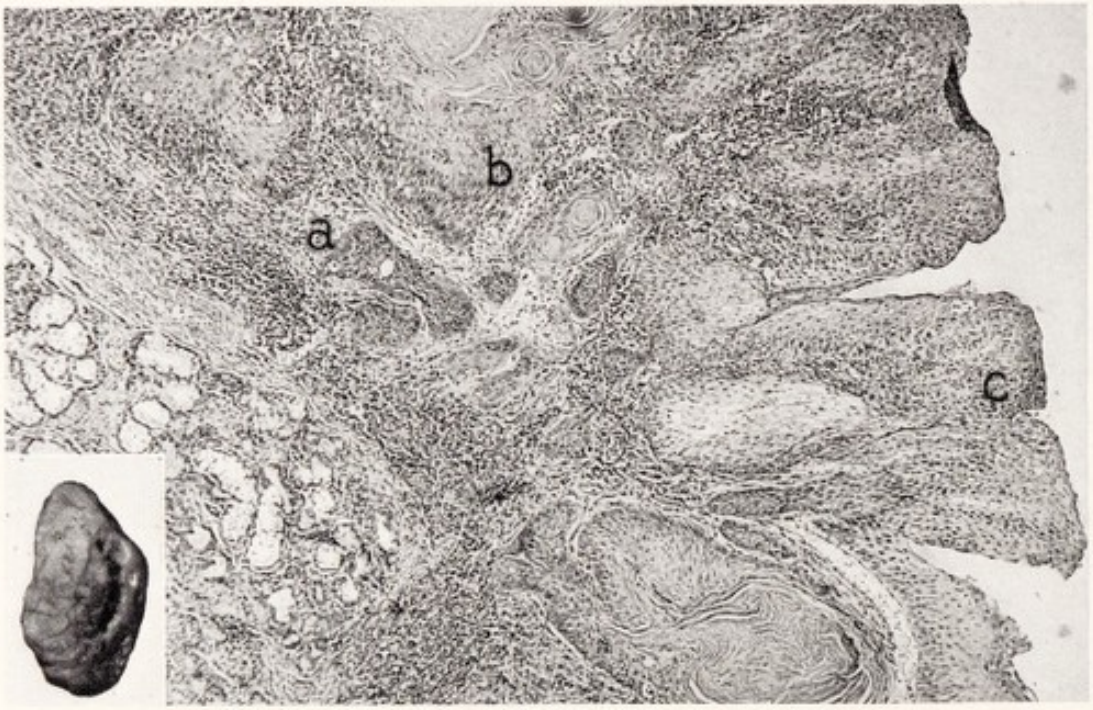

FIG. 105. Early cancer in the floor of the mouth: *a*, extensive round cell infiltration surrounding a mucous gland; *b*, pearl formation with adjacent changes in cell type; *c*, the surface epithelium is papillary in type. *Insert*, the tumor natural size. The lesion is confined to the depth of the small crevice.

larly in the metastases. On the contrary, as above noted, one may discover the tumor when it is yet so small that ulceration has not yet begun (Fig. 105). Even very early lesions in which the cell changes seem to be just beginning may run a rapid course. These are characterized by extensive round cell infiltration and early change in cell types.

CARCINOMAS OF THE MUCOUS MEMBRANE OF THE CHEEK

Epithelial malignancies develop in the cheek quite as commonly as in the tongue and, as is the case in that organ, they are quite often situated at the site of a broken tooth or a defective dental apparatus. These

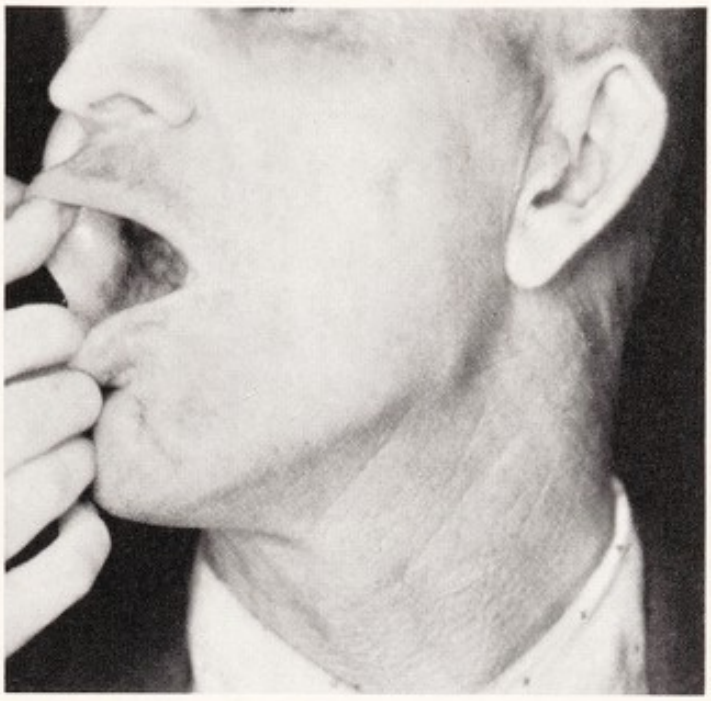

FIG. 106. Papillary carcinoma of the cheek just lateral to the gums.

cheek tumors give a worse prognosis than lesions of the alveolar border, likely because of the abundance of blood and lymph vessels in this region.

FIG. 107. Early malignancy in a papilloma of the cheek. The tumor presented a hard cornified mass: *a*, the epithelium of the cheek is retained; *b*, the point at which the growth has destroyed it; *c*, dense cornified tissue; *d*, cell proliferation with round cell infiltration.

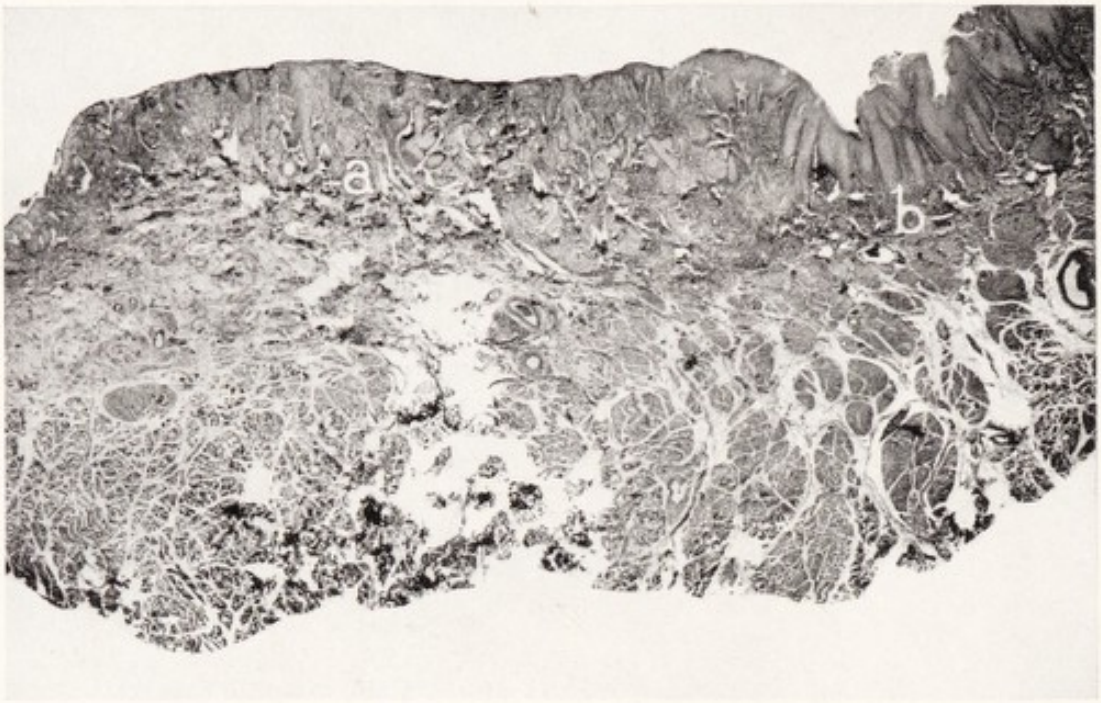

FIG. 108. Flat papilloma of the cheek: *a*, papillary thickening of the epithelium without invasion; *b*, epithelium staining more deeply and beginning to invade the subjacent tissue. The round cell infiltration is more impressive than the invasion of the epithelium or the change in the cell type.

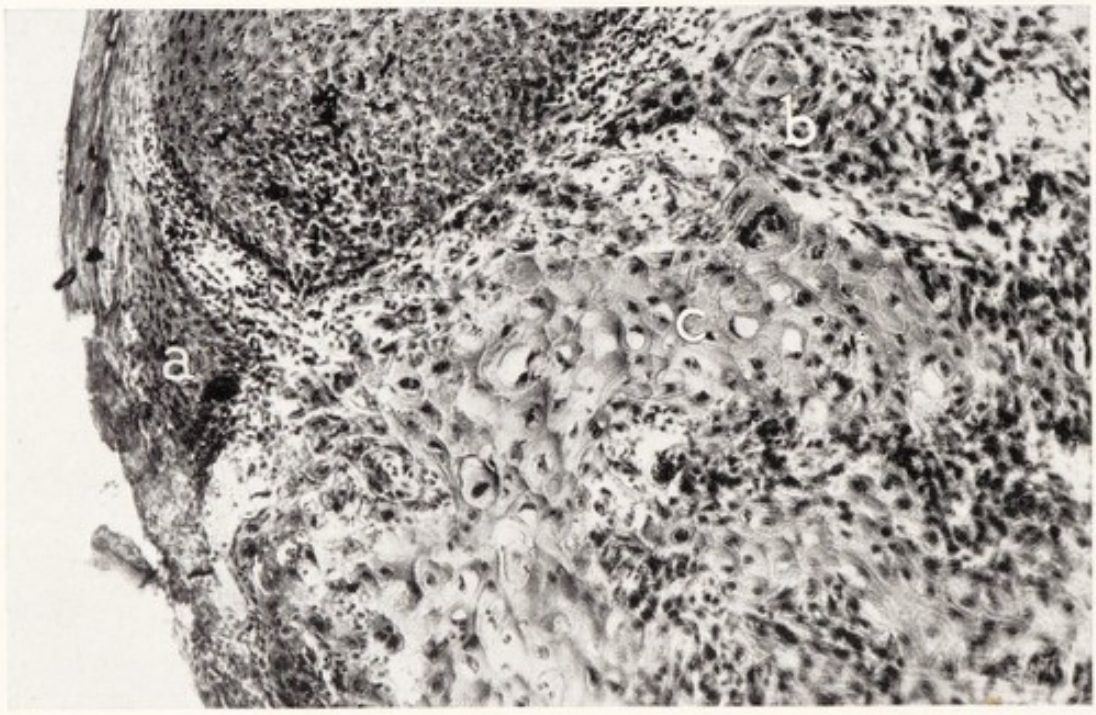

FIG. 109. Very early carcinoma of the cheek: *a*, epithelium of cheek partly destroyed; *b*, epithelial hyperplasia extending into the cheek; *c*, epithelial cells undergoing keratinization without definite pearl formation and staining but palely. The round cell infiltration is but moderate.

Pathogenesis. An indurated ulcer or a papillary tumor marks the beginning. This may represent a distinct defect in the tissue but quite as often there are excrescences both at the bordering mucosa and in the floor of the ulcer. Sometimes a veritable cauliflower growth is produced. Some of these are less violently malignant and seem quite amenable, at least for a time, to local cauterization. The tumors may remain localized

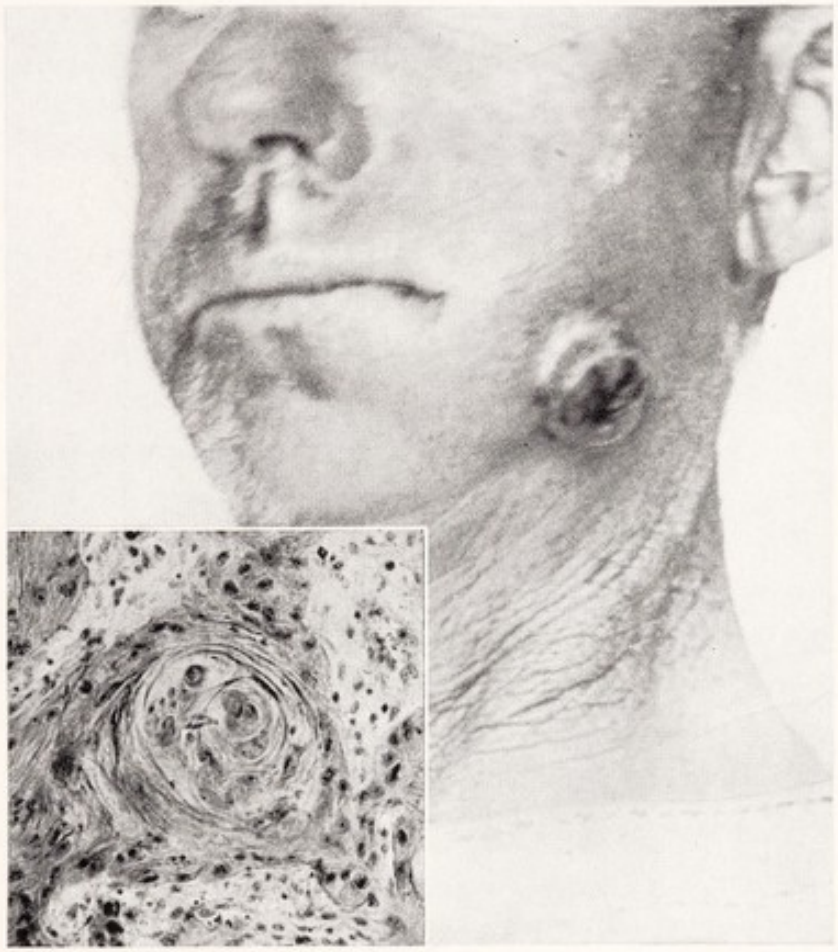

FIG. 110. "Collar button" cancer of the cheek. M., age 60. Following wart on cheek a tumor with open top developed. A continuous growth developed in the cheek. *Insert*, Slide shows squamous cell epithelioma.

but may come to involve the entire mucous membrane of the cheek and the pillars of the fauces. Metastasis is usually early, even before the local growth has been discovered by the patient. The end stages do not differ from those of the tongue. They are more prone to bleed, and if the facial artery is eroded a fatal hemorrhage is the result.

Pathology. Cancers of mucous membranes of the cheek may begin as ulcers but may also begin as papillomas (Fig. 106). The papilloma may be destroyed and an ulcer result secondarily. These papillomas may be

little impressive on inspection or on cross-section (Fig. 107), yet destroy the patient. Instead of being either papillary or fungiform they may present a hard wide plaque (Fig. 108).

Histology. Like cancers of the tongue, epithelial pearls develop early and may be retained for a long time even in the face of early metastasis. On the contrary, the epithelium may undergo early degeneration, suggesting origin from a leukoplakic area even when none is apparent (Fig. 109).

Clinical experience leads one to diagnose malignancy in the slide on very little positive evidence. Experience has shown that change in cell type with moderate round cell infiltration may be followed by recurrence.

FIG. 111. Oral end of a collar button cancer of the cheek. It presents a large ulcerated mass.

RARE MALIGNANT TUMORS OF THE CHEEK

As a corollary to the above is a type of cancer which involves simultaneously the mucous membrane of the cheek and the skin of the face (Fig. 110). These are very appropriately called *collar button cancers of the cheek.* The invasion of the mucous membrane of the cheek may exceed the surface growth (Fig. 111) even though the surface lesion only was noted. They are excessively malignant; though seemingly circumscribed they are but stimulated by any operative procedure no matter how radically the operation is done. They may remain stationary locally if not disturbed while metastases extend rapidly, producing huge lesions of the neck.

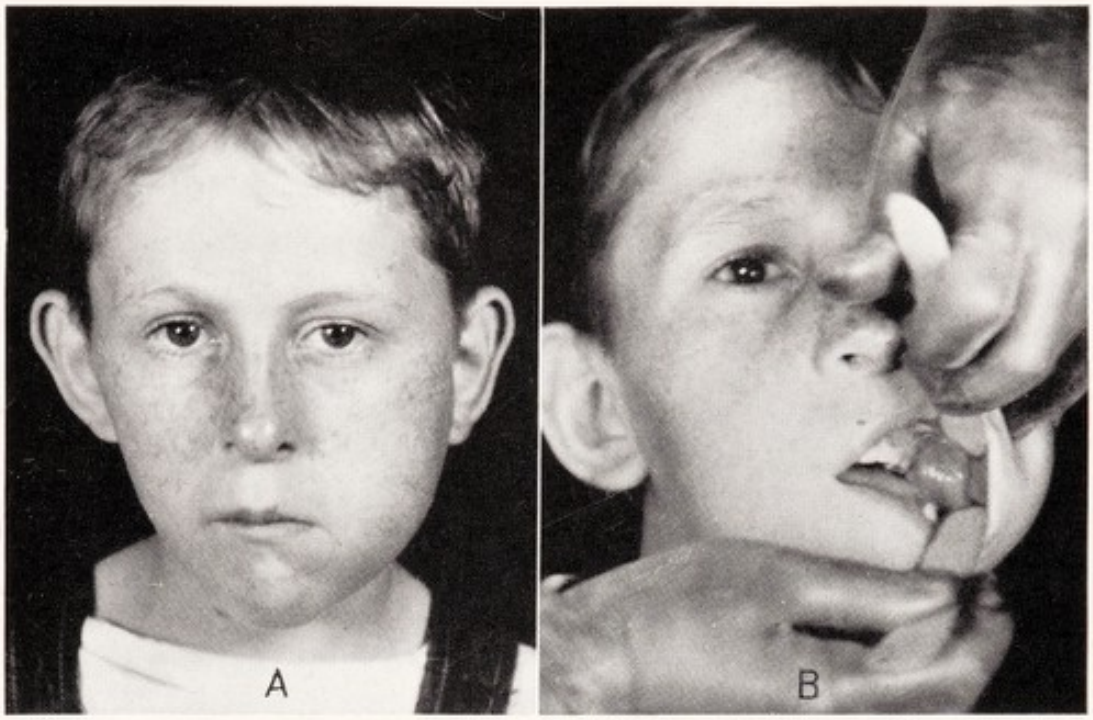

FIG. 112. Sarcoma of the cheek: *A*, bulging of the cheek; *B*, bulging into the mouth.

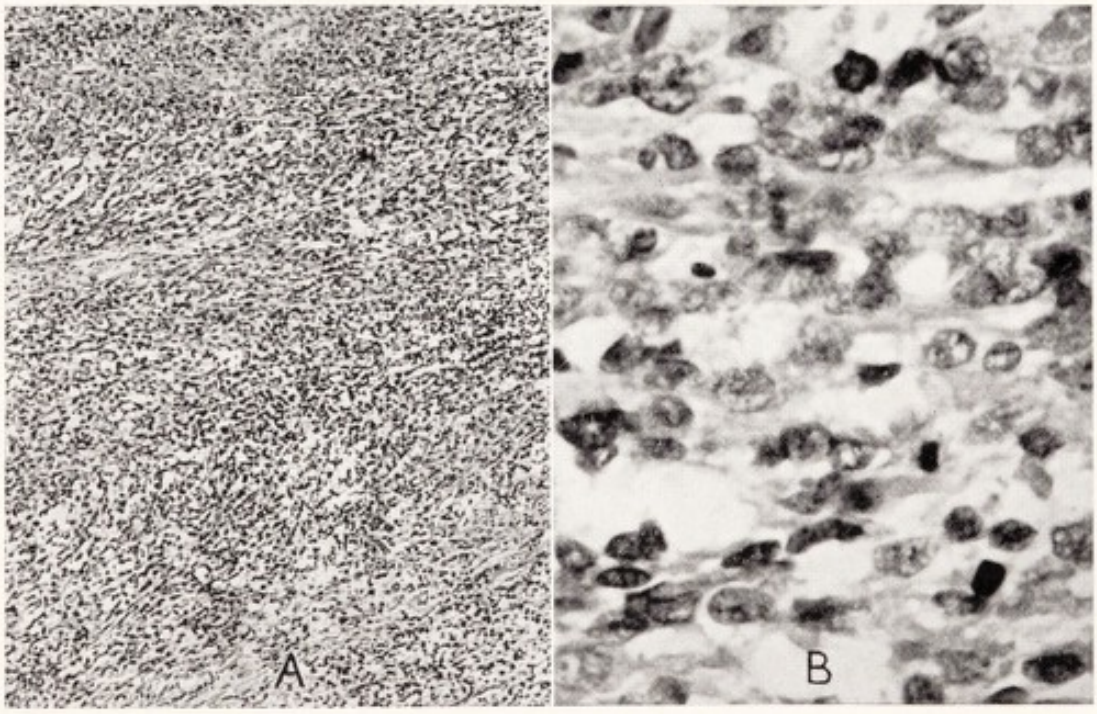

FIG. 113. Sarcoma of the cheek: *A*, very cellular with sparse connective tissue; *B*, the cells varied much in size and stainability, many undergoing degeneration. There is little connective tissue, the intercellular tissue being for the most part granular and refusing stain.

Myxomas of the cheek are sometimes located in the region of the sucking pad. The lipomas which develop in this region have been discussed in the chapter on benign tumors. Myxomas in this region should be regarded as forerunners of sarcomas and treated accordingly, to wit: a careful dissection of the tumor together with its capsule.

Very malignant *sarcomas* are sometimes observed in this region. I have seen two, both rapidly fatal. They begin as pseudo-encapsulated tumors in the site of the sucking pad. They produce a uniform bulging of the cheek with corresponding bulging into the oral cavity which, on pal-

FIG. 114. Melanoma of the cheek. Natural size. It was encapsulated and was in part cystic. It seemed to be primary.

pation, shows a spheroidal tumor (Fig. 112). After removal the lobulations of the tumor are apparent which, on section, shows an expansile growth. Both were round celled in type, and the rate of growth was quite in harmony with the type (Fig. 113). I have seen a single *melanoma* in this region. If there was a primary melanoma on the skin surface of the cheek it was quite overlooked (Fig. 114). It produced a circumscribed tumor the size of a hickory nut when first observed. When cut across it had the appearance of a melanoma of a lymph gland, at least it was encapsulated. In the slide the cell type gave a preview of the extreme malignancy the clinical course exhibited. Early metastasis developed in the liver.

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CHAPTER VI

Granulomatous Tumors of the Gums

FIBROUS tissue lesions of the gums have one thing in common, to wit: they are tumors of the gums. They require separate classification in order to distinguish them from each other as an intellectual achievement and for the purpose of separating them from more serious lesions. The surgeon's concern is in distinguishing those which permit of local removal and those which require the removal of more or less of the bony parts.

There is no term applicable to all members of the group. Some of them are typical granulomas. These may terminate in typical fibrous tumors. The epulides can be entered here only by the exercise of strong arm tactics if one considers them strictly on their merits. But they may resemble very closely the end stage of granulomas. Furthermore, an epulis may be covered with a layer of connective tissue. Therefore, even on microscopic study, the fibrous tissue may fall in the section. Therefore, though it may be poor pathology to include these in one group it is convenient for practical surgery.

As so often is the case in a group of diseases one considers together some members which are simple, others more complex. In such a case one is apt to fix on the more simple and overlook the more serious lesions. Thus the simple tumors of the gums, the granulomas, resemble the epulides and these shade into the more complicated and more serious tumors of dentigerous origin. The problem is confused because the gingerly snipping off of a piece for microscopic examination may give the wrong lead, a fibrous bit or a small section showing giant cells may fail utterly to disclose the malignant part lying more deeply. I have seen so many disastrous results of inadequate examination when the lesion is located on the upper jaw, that I have come to regard all tumors in this location as serious. Discovering that a tumor is an epulis does not help us much. We must know its location and topography. The only way one can avoid such errors is to lay the entire tumor, no matter what its name, on a board so that one can examine its several parts. The surgeon may not be able to say in detail during the course of the operation just the nature of the tumor with which he is dealing but he can know if he is dealing with tumor tissue requiring serious consideration and when the lesion is, so to say, only an inconvenience.

The first groups have for their ground plan simple granulation tissue which develops about roots of teeth. These tumors may be devoid of a covering epithelium, being simply the proud flesh of the older authors. Some of these acquire an epithelium. Others are fibrous epithelial-covered tumors when first observed. The genetic relation cannot be certainly known. They are often designated fibrous epulis, a wholly misleading term. They have nothing in common with the epulides. This is incorrect because the epithelium is directly united with the tumor beneath. Connecting them with fibromas and very cellular fibromas, sometimes called fibrosarcoma, is possible only when working with a high-powered lens and little experience.

The true epulides, the giant-celled tumors, are believed generally to form the great bulk of the tumors of the gums and by some the term is made to include all the tumors of the gums, as the original meaning of the term indicates. Epulides are giant-celled tumors. In their clinical behavior they lie midway between the benign granulomas on the one hand and the deeply lying malignant tumors on the other.

Giant cell tumors are rightly regarded as only locally malignant, but some of the more rapidly growing have an outpost of more malignant nature, the giant cells belying the more serious lesion beneath. Politicians sometimes nominate a respectable citizen for office purely as a blind and when he is elected they mount him on a slide as something benign while the malignant growth continues unhindered.

It was possible for me in my years of country practice to observe early lesions of patients who went to the city surgeons for operation. Many of these, both tumor and patient, returned after months or years when the tumor had taken on malignant changes. Usually such recurrences proved to be dentigerous tumors which had had a giant cell protrusion, assuming there was an attempt at scientific diagnosis—a rather gratuitous compliment.

On the other hand, whether giant cell tumors take on active proliferation of connective tissue with subsidence of the giant cells, as is sometimes assumed, I am unable to say. I shall, rather reluctantly, discuss these as fibromas, pure and simple, in order to retain the name "epulis" unsullied as a benign giant cell tumor. In my experience, when an epulis becomes malignant the diagnosis was wrong. The epulis was an innocent bystander, the more malignant parts having been overlooked.

The granulomas, on the other hand, are fibromas to their end stage; they remain fibrous tumors. It is their early cellularity that may cause confusion to the embryo microscopist. They are not fibrous epulides. The fibrosarcomas I have seen have been simply organizing granulation

tissue which some microscopist tried to diagnose from a slide with a high-power lens without knowing anything of the origin of the tissue. It was the lens which was high powered, not the pathologist.

Real sarcomas of the jaws that I have seen were plain bone sarcomas, without any distinction referable to the location on the jaw or alveolar border. In fact, the alveolar border is usually spared. The sarcomas of the jaw I have seen required no slide examination for diagnosis. They fairly radiated malignancy.

All of the central bone sarcomas I have been privileged to examine proved to be a mistaken diagnosis, being central giant cell tumors or adamantinomas. Periosteal sarcomas are very rare and do not differ from those situated on the long bones.

Certain vascular tumors of the gums have been classed as angiomas. Such a specific term seems unnecessary because many of the granulomas are distinctly vascular and might without a stretch of imagination be classed as angiomas, save for the fact that the vessels take no part in tumor formation. They are as easily cured as are all granulomas. For these several reasons I am including them with the granulomas.

Finally there are the curious gum hypertrophies which come apparently without cause, not tending to become anything but fibrous hypertrophies.

I shall, therefore, divide the fibrous tissue tumors of the gums into granulomas, epulides and fibromas of the gums.

I. Granulomas of the Gums
II. Epulides
III. Hypertrophy of the Gums

These have in common the one factor that they are either inherently benign, or are so if approached by a competent surgeon.

GRANULOMAS OF THE ALVEOLAR BORDERS

As intimated in the foregoing, granulomas as here considered are small tumorous masses, the result of infection about the teeth, which tend ultimately to heal through a process of fibrosis and epithelialization. Innocent enough themselves the importance lies in knowing what they are not.

As the name implies, granulomas are the result of reaction to infection. They form small tumors the size of a wheat grain to that of a grain of corn, seldom as large as a hazelnut, developing about the roots of teeth or on the alveolar border where a tooth has recently been. Included also are the fibrous tumors covered by epithelium no matter what the degree of vascularity. They are but variations in development and the end stage of a granuloma.

Pathogenesis. Granulomas are just that. They develop from a gum the site of irritation and the resultant infection. The tooth may itself be free from disease but usually there are deposits on its surface which irritate the adjacent gum. The tooth may be diseased and its removal may not cure the granuloma and it may continue to exist, even to increase in size after the tooth is removed. If a granuloma develops at the site of a tooth which continues after the tooth has been removed, it should be regarded with suspicion because there is a possibility that there is a

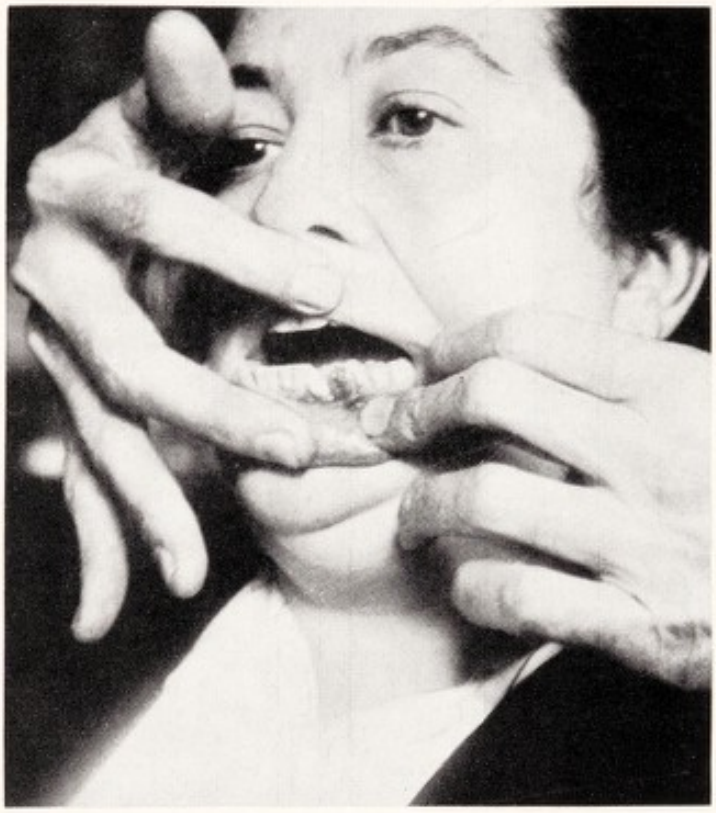

FIG. 115. Granuloma pyogenicum of the alveolar border. The tiny growth is about half the size of the adjacent tooth.

more serious deeply lying lesion which was the factor that destroyed the tooth. It must be regarded as a more serious lesion until proved otherwise. The best means of differentiation is by exploration, curet in hand in order to determine once for all whether there is something of more importance in the depth.

If the curet proves there is nothing in the depths of the jaw it is not a deeply seated tumor protruding to the surface. I once purloined a jaw carrying a typical granuloma. Sarcoma had been diagnosed from a tiny biopsy by a professor of pathology in one of our state universities, not in Kansas, and the jaw was resected by the professor of surgery in the same institution. That was a fine specimen, half a jaw carrying a tiny granu-

loma. Fortunately, the tiny biopsy the surgeon removed did not disturb the outlines of the tumor for exhibition purposes. The moral is such tissues should never be sent to the laboratory. The curet should decide the problem. If the pathologist must shoulder the responsibility of diagnosis, hand him the curet and he will make the diagnosis in the clinic and confirm his diagnosis in the laboratory.

Granulomas may involve a single tooth or several teeth. They are especially likely to cause confusion when they are extensive. The history

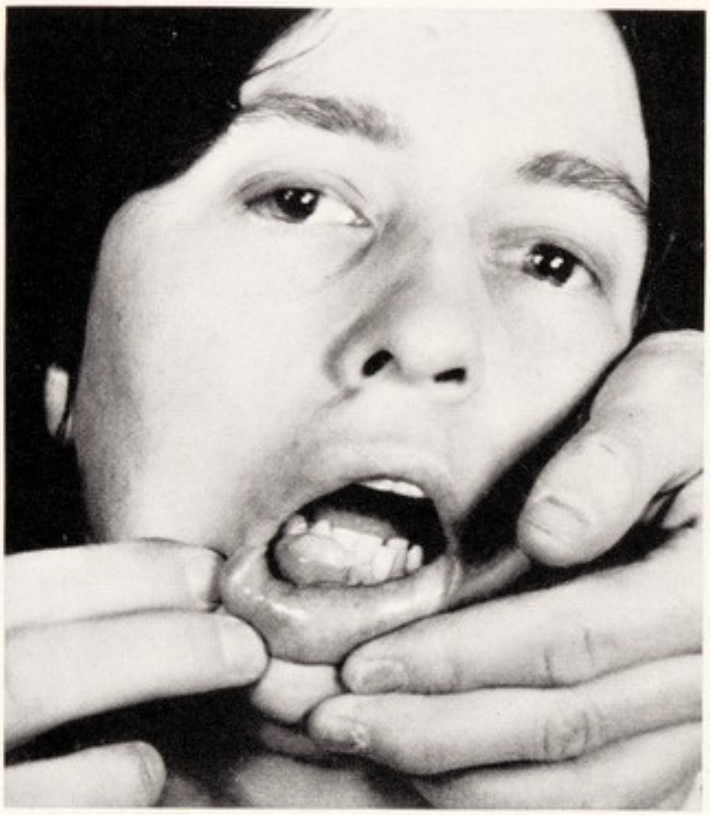

FIG. 116. Fibrous granuloma of the gum. The tumor was smooth, elastic, covered with epithelium but contained no giant cells.

is that they have developed in a month or two. Rarely the complaint is that the toothbrush causes bleeding. Moreover, most of these lesions develop in jaws, strangers to the toothbrush as well as to the dentist, occurring chiefly in neglected teeth not rarely the subject of a pre-existing pyorrhea. In fact if there is no evidence of previous infection about the site of development it is a matter for apprehension. However, they do develop in the mouths of those who practice a careful oral hygiene. This is important to keep in mind lest the diagnosis of granuloma might offend the patient, like discovering lice on the heads of their children.

These lesions begin as soft growths of the gums adjacent to a tooth or several teeth. They usually attain a size larger than a grain of corn and

then cease developing (Fig. 115) but if several teeth are involved a large fungous mass may be produced. They may remain stationary for many months and, if superficially removed, may attain their original form in a few days, the rapid recurrence of which may frighten both the patient and the doctor. As they grow older they become firmer and less red and approach in appearance the true epulides (Fig. 116).

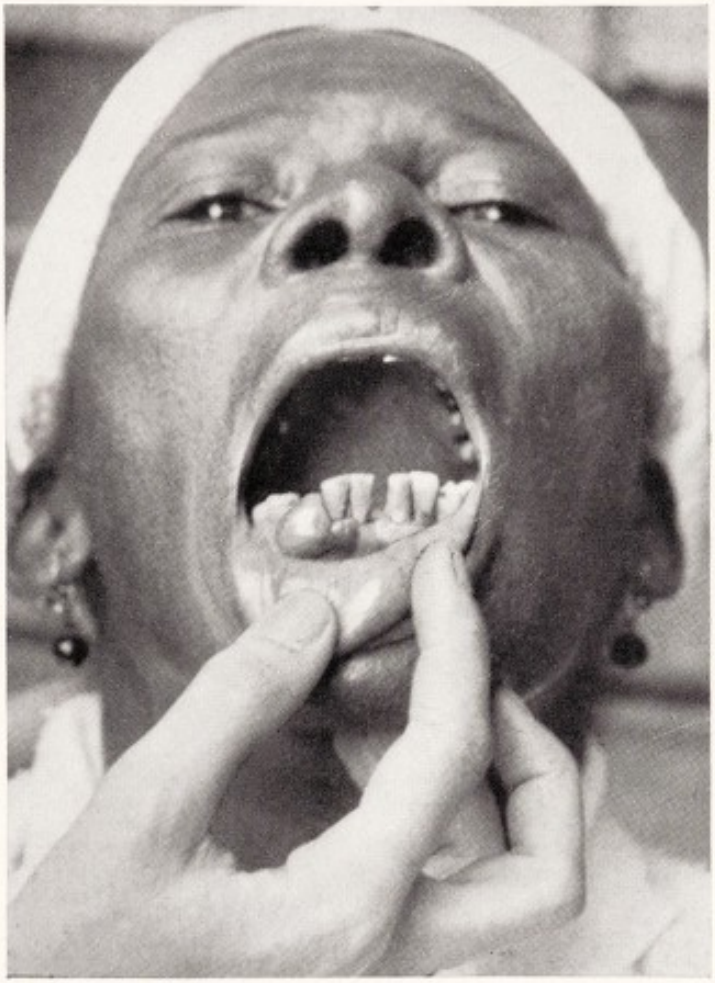

FIG. 117. Epulis and granuloma of the lower jaw. The larger nodule sprang from between two teeth and contained giant cells, the smaller from the alveolar border at the root of the second incisor tooth was a granuloma.

One sometimes encounters growths which are larger than typical granulomas and are covered with epithelium when first observed. I was fortunate once in being able to follow the genesis of such a tumor. An exuberant granuloma was removed only to recur; the primary tumor had no epithelial covering. The recurrence, long neglected, acquired an epithelial covering over a dense keloid-like connective tissue. Complete removal ended the trouble.

Larger bluish, somewhat softer, tumors are sometimes observed.

These features being expressive of vascularity are sometimes called angiomas or angiomatous epulides. I believe there is no need to separate these from the granulomas if there is an absence of giant cells, a thing that may need to be determined in the laboratory. There is no clinical need for a separate classification.

Pathology. As may be surmised from the foregoing these lesions are usually soft to the touch in the beginning and may bleed but the older ones may be covered with epithelium, as above noted, and being firm suggest epulides. They may remain deeply red for a long time after they be-

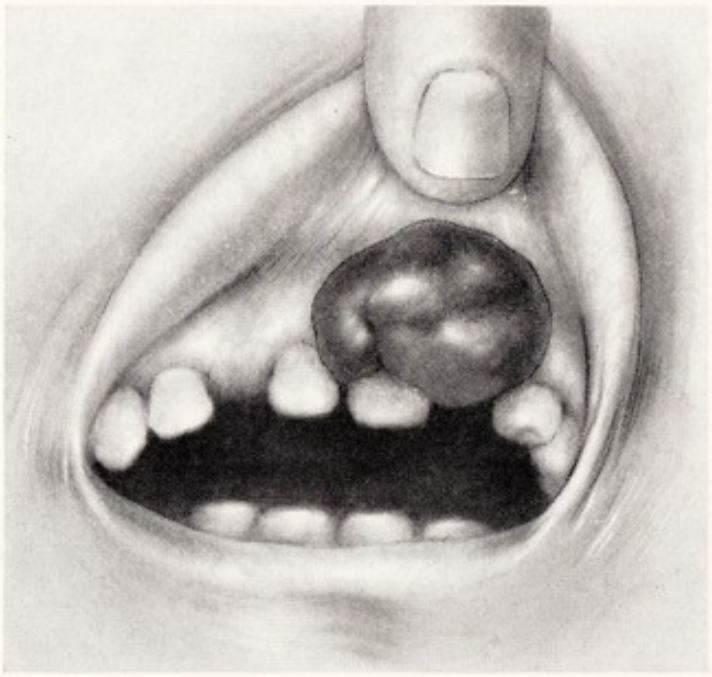

FIG. 118. Localized bosselated epulide of the upper jaw. The tumor was bluish red. The tumor, tooth and alveolar border were removed. There has been no recurrence.

come covered with epithelium (Fig. 117). The most vascular may have a distinctly bluish color (Fig. 118). They appear to extend up out of the tooth socket, as indeed they do, belonging to an individual tooth, or teeth, and do not protrude impartially from between the teeth as epulides do. If a number of teeth are involved, in an exceptionally neglected mouth, the appearance may be somewhat formidable until examined in detail. As an end result, as noted above, these tumors may attain an epithelial covering and then the anatomic distinction from the epulides disappears except for their location and history of development. These but partly epithelialized growths show the relationship particularly well. Sometimes, when the tooth is removed, the granuloma remains adherent to the tooth (Fig. 119).

FIG. 119. Fibrous granuloma of the gum. The little tumor remained adherent to the tooth which was removed under suspicion that the growth was an epulis.

When cut across the color is uniform, usually red in the early cases, as any exuberant granulations subject to the variations above indicated. The very vascular ones may shrink up to a small part of the original size when placed in hardening fluid. The old fibrous ones may have almost a keloid hardness, in fact they are wholly comparable with keloids.

Histology. The histology from the foregoing is obvious; several points are worthy of note. When the granuloma remains adherent to the tooth the section shows the loose attachment to the tooth (Fig. 120). The early

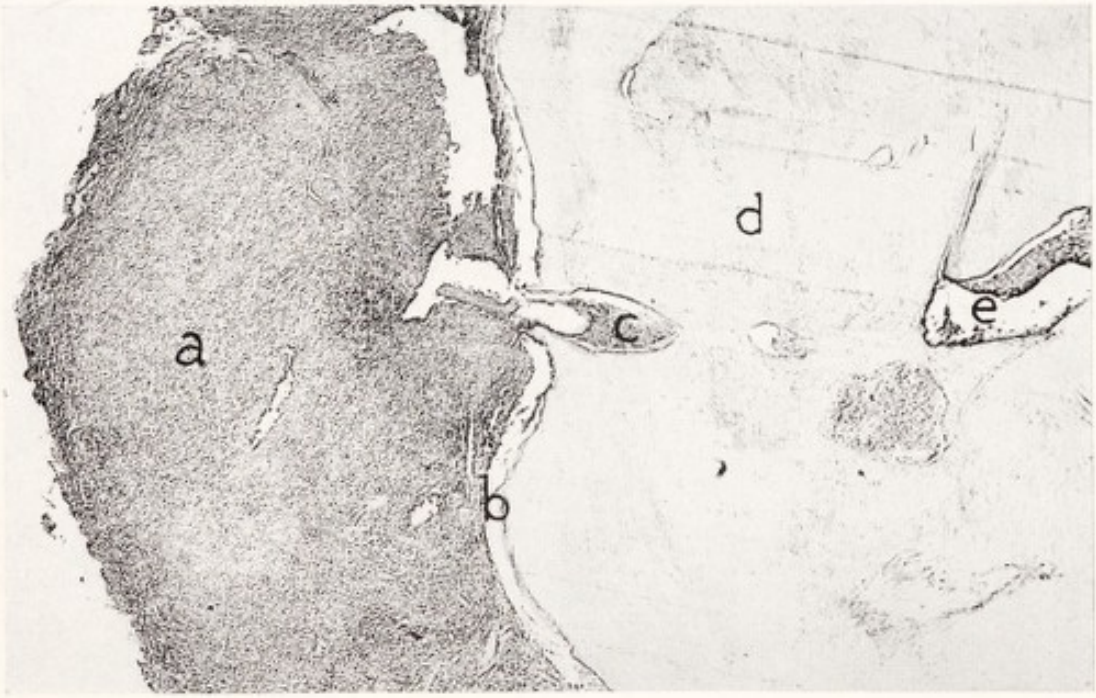

FIG. 120. Section of a granuloma attached to the root of a tooth: *a*, granulation tissue containing areas of necrosis and thin-walled blood vessels; *b*, dividing line between the granuloma and the root of tooth; *c*, apical vessel which supplies blood to the granuloma; *d*, the tooth root; *e*, section of the apical vessel.

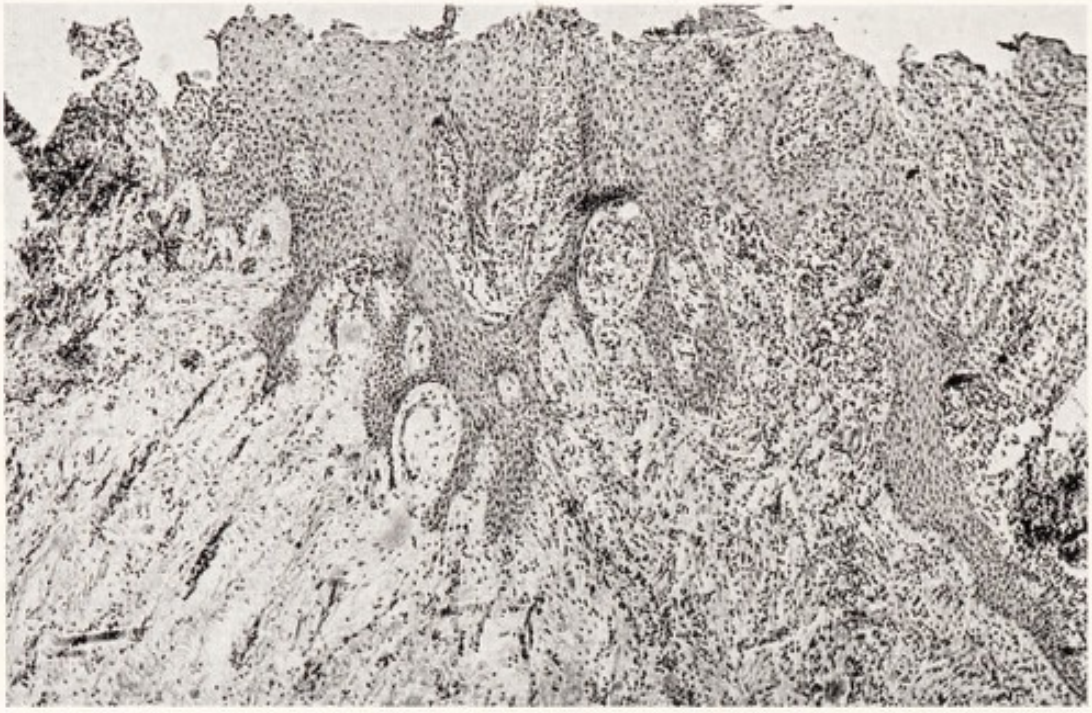

FIG. 121. Mature granuloma of the alveolar border. The surface is covered with an irregular epithelium. Beneath is maturing fibrous tissue with extensive round cell infiltration. The vessels show the development of fibrous tissue characteristic of aging granulation tissue.

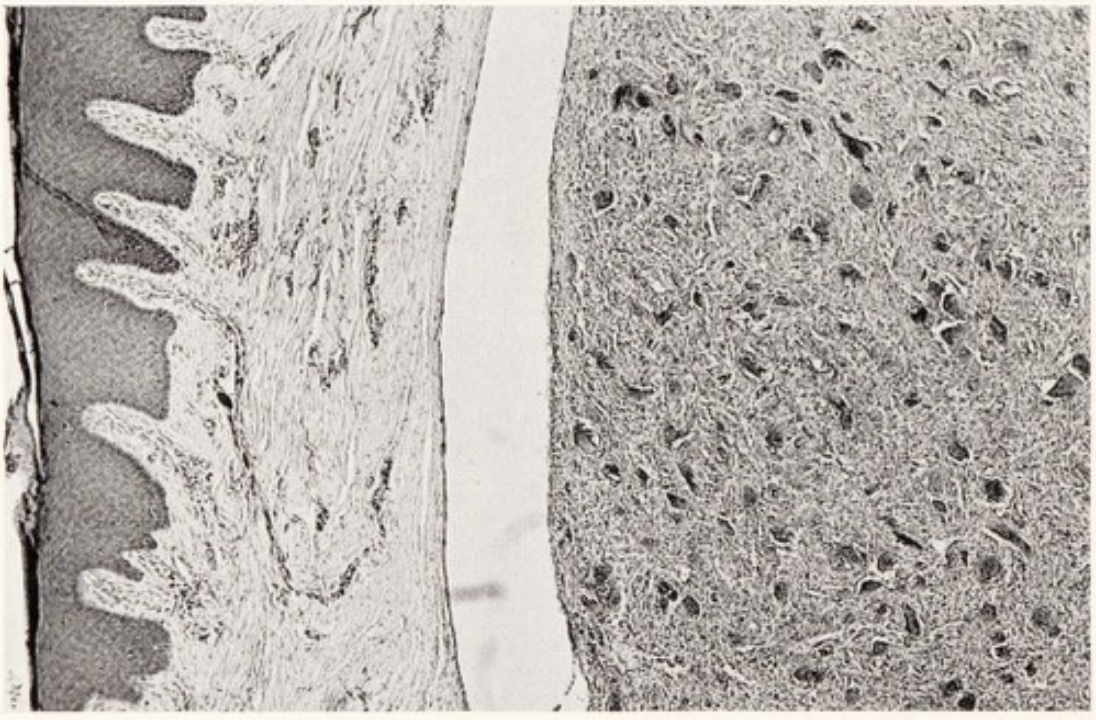

FIG. 122. Slide showing epithelialization of a small pyriform tumor situated beside an incisor tooth. The epithelium is irregular in development showing long columns of epithelium extending into the depth. The round cell infiltration is diffuse, not collected about the cell columns. The fibrous tissue is keloidal, indicating its source.

cases show the vascular and cellular infiltration, the earmarks of granulation tissue. Because of the large numbers of large vessels with adjacent cells it has suggested to some a small round cell sarcoma as above noted. This is true of granulomas of some months' duration. The mistake is possible, of course, only if the microscopist is ignorant of the size of the tumor from which the biopsy was taken. Nobody ever saw a small round cell sarcoma the size of a grain of corn. Even so, furthermore, though round cells or endothelioid cells may be arranged near the vessels there are always polynuclear cells intermingled which should warn the judi-

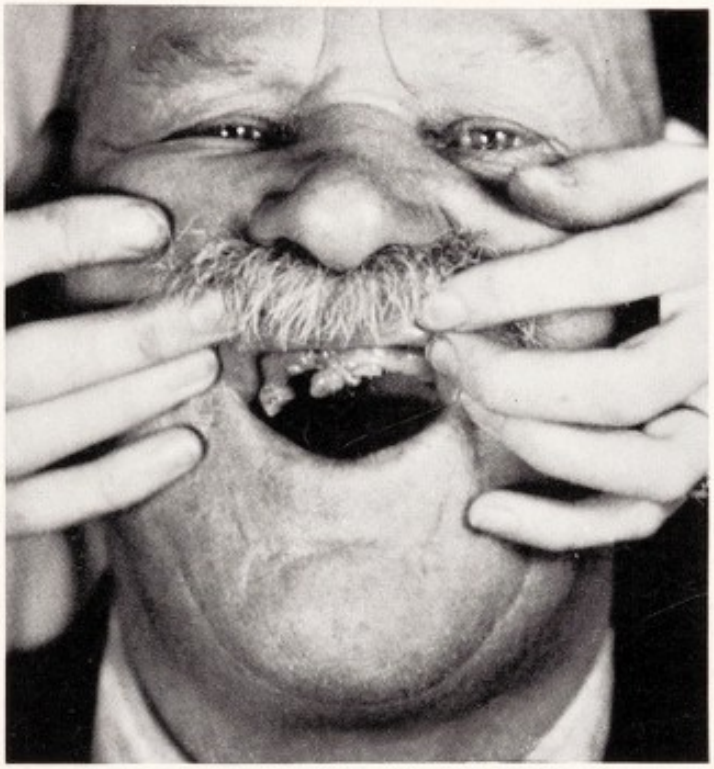

FIG. 123. Combination of a fibrous tumor and an epulis. The covering epithelium is squamous. Below is a fibrous layer containing few cells. A line of separation divides the fibrous area from the typical epulis beneath. The multinuclear giant cells and the abundant fibrous tissue is that of a typical giant cell tumor.

cious (Fig. 121). In the older lesions when these cells are absent there is a thickening of the vessel walls quite foreign to a neoplastic growth. Even more distinctive is the association with the subepithelial connective tissue. This is obvious when epithelialization is not yet complete as well as when the entire tumor has received its epithelial covering. This is in contrast to epulides and other expanding tumors. Therefore, to call these tumors fibrous epulides is untenable.

There is yet another possible source of error. When epithelialization occurs there may be an unequal development of cells with consequent extension into the depth of some of the cell columns (Fig. 122). This

with associated round cell infiltration has frightened some pathologists when confronted with a slide made from a mite of a biopsy. Epithelial malignancy simply does not begin that way, a fact perfectly obvious if the entire growth is at hand for examination.

Sometimes epulides carry on their summit a layer of connective tissue carrying a regular epithelium, the real epulis lying deeper (Fig. 123). In such cases a timid biopsy may remove only epithelium and connective tissue missing wholly the epulis beneath. I have known of this error being made by pathologists who trusted the surgeon to remove the diagnostic tissue.

EPULIS

The term "epulis" was used by the ancients to include all tumors of the gums. Of course the term means just that and nothing more. That was a good term for the ancients because all they had to do was to call it something and their day's work was done, but we, responsible for the cure of the lesion, find it necessary to specialize. We now know that there are a variety of things that can happen to the alveolar process. Now the term is reserved for certain relatively benign giant celled tumors of the alveolar border and is here used in that sense. Relatively benign, indeed, for like giant-celled tumors of long bones they sometimes take on malignant growth under certain conditions and locations. However, it must be remembered, that not all giant-celled tumors are epulides. Very violent periosteal sarcomas sometimes show areas in which giant-cells are found. The term "sarcomatous epulis" formerly applied to them was an attempt to straddle an uncertainty, a need which is sometimes still present if one neglects to consider all the possibilities. When an epulis goes wrong it is something more than an epulis, that is, a tumor of the alveolar border harboring more malignant structures. Most commonly a central giant-celled tumor or an adamantinoma was overlooked. This viewpoint is important because the need of not overlooking anything when dealing with epulides is ever present. When we say that a neglected epulis has "extended" we mean that the part we overlooked has in the course of time thrust itself on our attention. This is to say, the examination of a diagnostic section may be useless because it gives no evidence as to structure of that part of the tumor which was not examined. Much of my experience with epulides has had to do with the second operation after the extra-alveolar part had been removed by some one else. Properly, epulides removed in anything like a reasonable time after their appearance result in permanent cure, even after much neglect and operative abuse.

Pathogenesis. The source of these tumors has been much in dispute,

recent researches apparently indicating some relation to the process of shifting from the temporary to the permanent teeth. I like this theory because it calls attention to a deeper origin than the old views of periosteal or peridental origin.

Assuming such a developmental origin for the epulides aligns them with the dentigerous tumors. Nevertheless, these growths sometimes appear to develop on the alveolar border between the teeth or along the alveolar border across several teeth but when very small seem to be attached to the root of a single tooth in which case they may closely

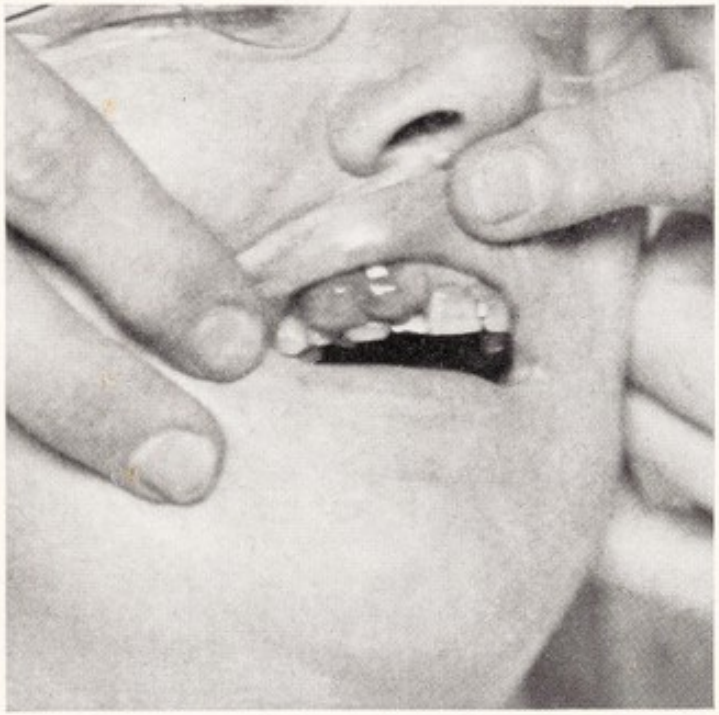

FIG. 124. Epulis arising from a single upper incisor. It recurred after the pulling of the tooth. Removal of the alveolar border stopped the process.

resemble the granulomas. We may say with confidence that the great majority do attach here because when removed with this attachment in mind there is no recurrence. Furthermore, we know from clinical experience in what regions this superficial attachment is most likely. Those about the incisor teeth of the lower jaw are always such; adjacent teeth of the upper jaw hold about the same relation. The molar teeth of the lower jaw are less certain. Associated deep giant cell tumors or dentigerous tumors are occasionally found. It is the region of the molar teeth of the upper jaw where deeper extension is to be expected, if, indeed, it is not the rule. The vast majority of the patients on whom I have been required to do secondary operations have been in this region. Even at the primary operation, I believe the deeper areas should be explored to

the extent that the teeth involved and their alveolar sockets should be removed. In smaller ones removal of the teeth and exploration of the sockets may be sufficient. Certain it is that the entire disease must be removed and it is easier to do this at the first operation, and, incidentally, it is safer for the patient.

Epulides vary in size from a pea (Fig. 124) to that of a hickory nut but, particularly in the upper jaw, may become much larger and yet be benign tumors (Fig. 125). They form rounded tumors protruding be-

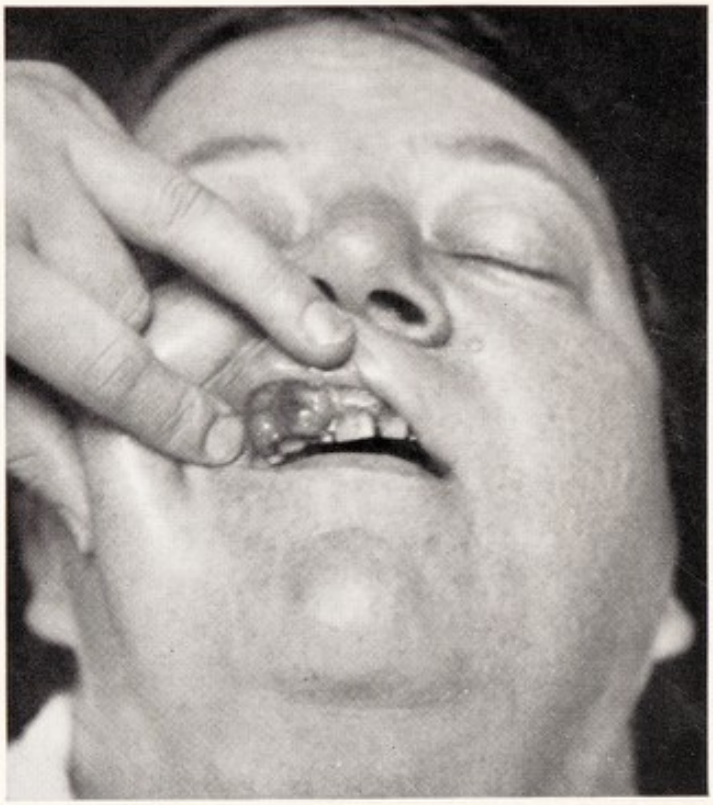

FIG. 125. Epulis arising from a single upper incisor but extending along the alveolar border. The removal of one tooth and the adjacent bone cured the tumor.

tween or about one or several teeth. Those developing in the upper jaw are seldom confined to a single tooth but may be so. They may be localized about a single tooth and extend for some distance along the alveolar border (Fig. 126). They may arise about a single tooth, and by extension may involve the adjacent teeth. After inadequate operation followed by recurrence (Fig. 127), it always requires the removal of a considerable border of the alveolar process. The extension may extend beyond the alveolar process on to the hard palate demanding the sacrifice of the entire upper jaw except the orbital plate (Fig. 128). In rare instances the initial lesion may be rapidly growing and involve the alveolar border

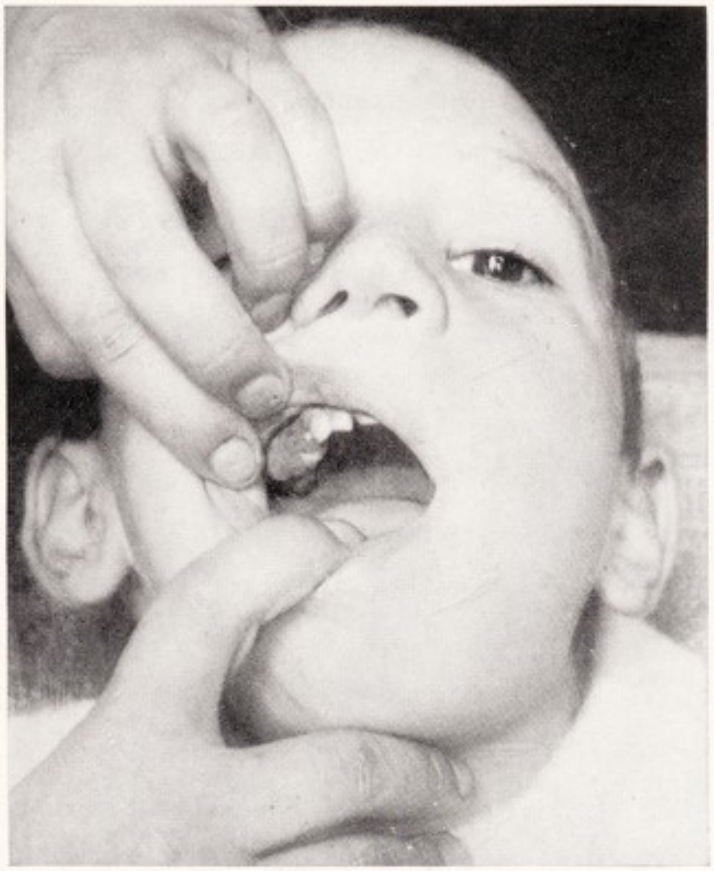

FIG. 126. Epulis arose about one tooth but invaded adjacent teeth requiring the removal of three of them.

and considerable of the hard palate before the patient seeks advice (Fig. 129).

Pathology. The epulides of the lower jaw are uniformly small and of slow growth. The epulides of the upper jaw adjacent to the molar or

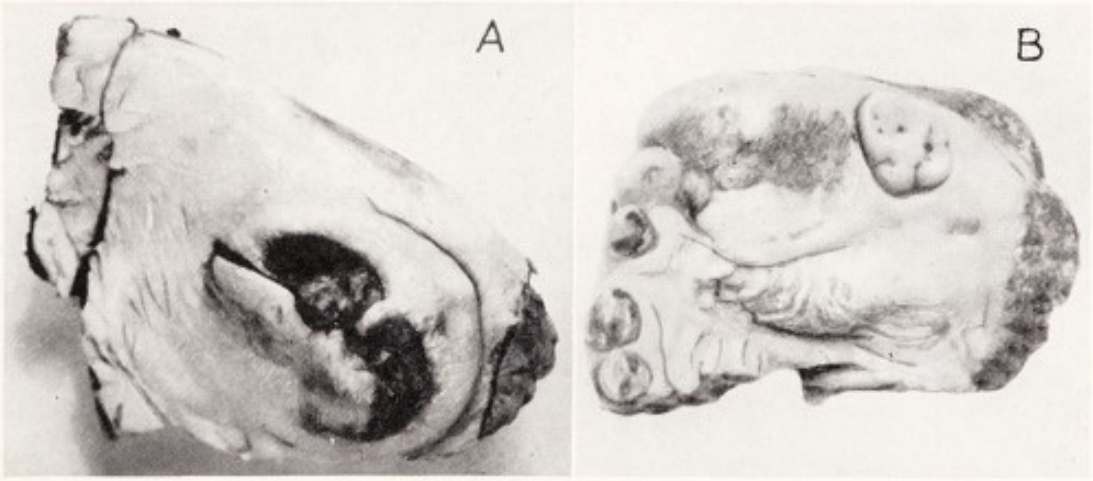

FIG. 127. Recurrent epulis of the upper jaw in a boy aged nine. Permanent cure followed removal of the alveolar border.

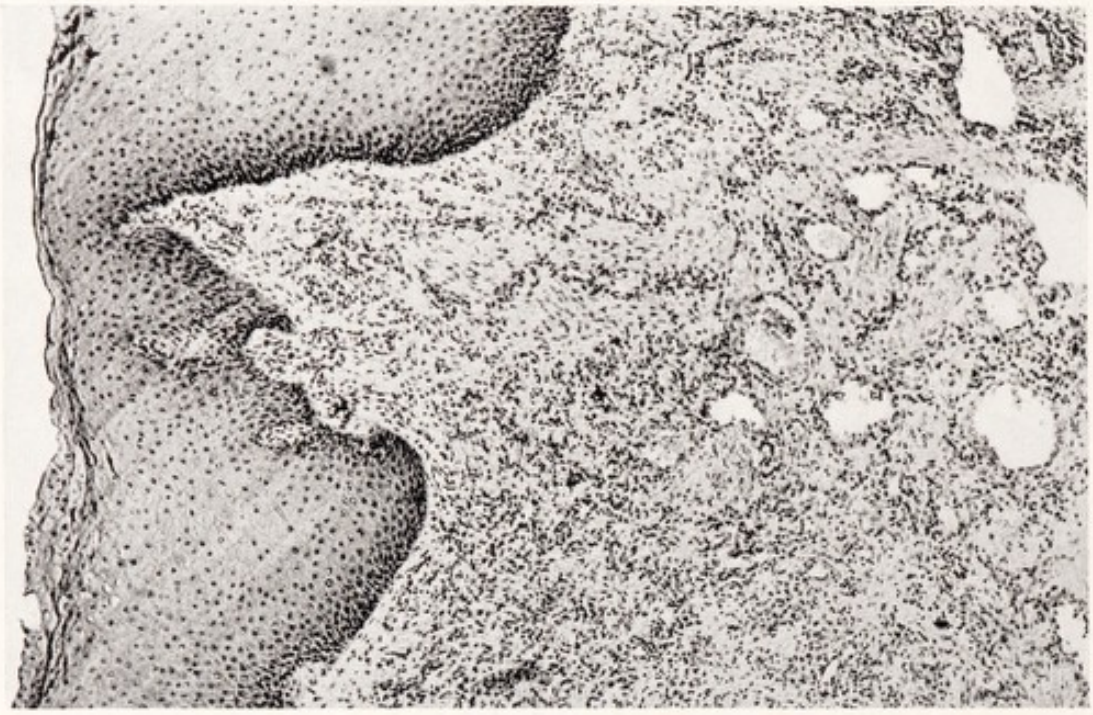

FIG. 128. Recurrent epulides after incomplete removal. *A*, The tumor which formed a mass on the hard palate had been incised as an abscess. *B*, Diffuse recurrence on the alveolar border. Both patients remained free from recurrence.

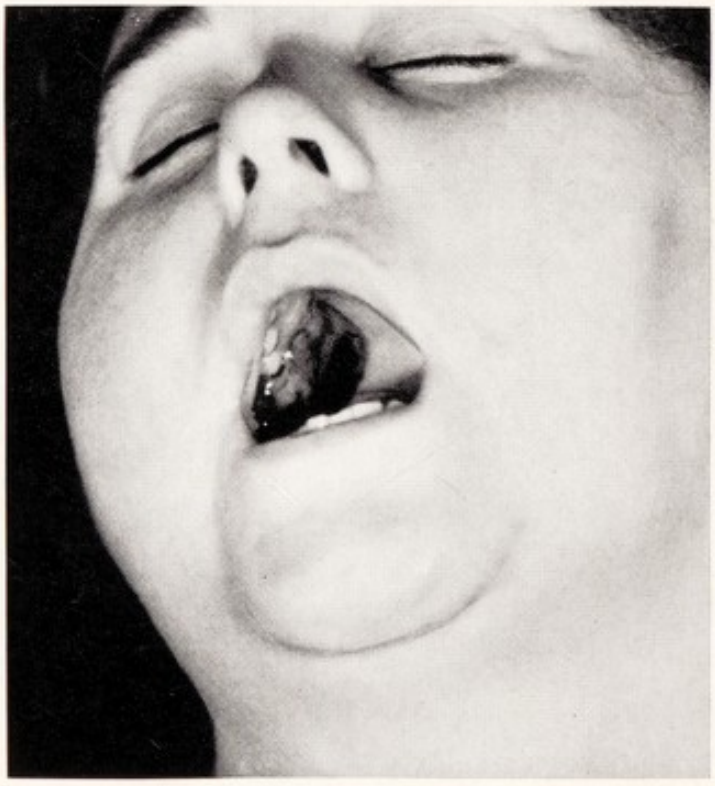

FIG. 129. Large rapidly growing epulis involving a large part of the alveolar process and the hard palate.

bicuspid teeth, on the contrary, are usually large when first observed. The recurrences are always larger than when first observed before the unsuccessful operation. Even recurrences when radically removed usually remain cured. But if removed too often, they may lose patience and really become malignant. If, with the local recurrences of the tumor, there is an expansion of the alveolar border one may expect some additional tumor growth with the antrum, that it was of dentigerous origin and not an epulis.

This is likewise true when the recurrence invades the hard palate. This extension usually is less malignant than the tumors that invade the

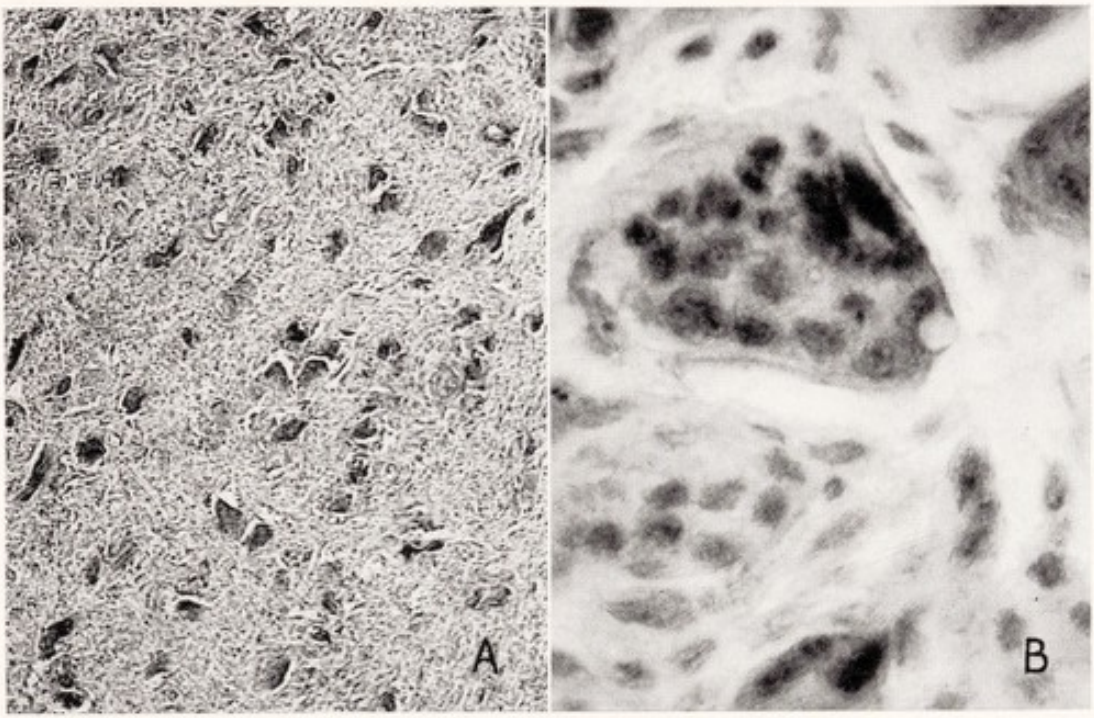

FIG. 130. Slide from a small epulis: *A*, the dominant feature is the large irregular multinucleated giant cell lying in an abundant fibrous tissue containing but few cells; *B*, the nuclei are distributed more or less regularly throughout the tumor. The nuclei are uniform in size and stainability and contain deeply staining nucleoli.

alveolar border and antrum. In harmony with this the palate extensions are pure giant-celled tumors.

The surfaces of the tumors have the color of the mucous membrane adjacent. In fact, the mucosa may be continuous over the surface of the tumor and be independent of it as above noted. They may be bluish red if the vascularity is great. The tumor may be attached by either a narrow or a broad base. Those with a broad base are the more apt to be growing rapidly.

On section the tumor is mottled reddish gray but may be brownish.

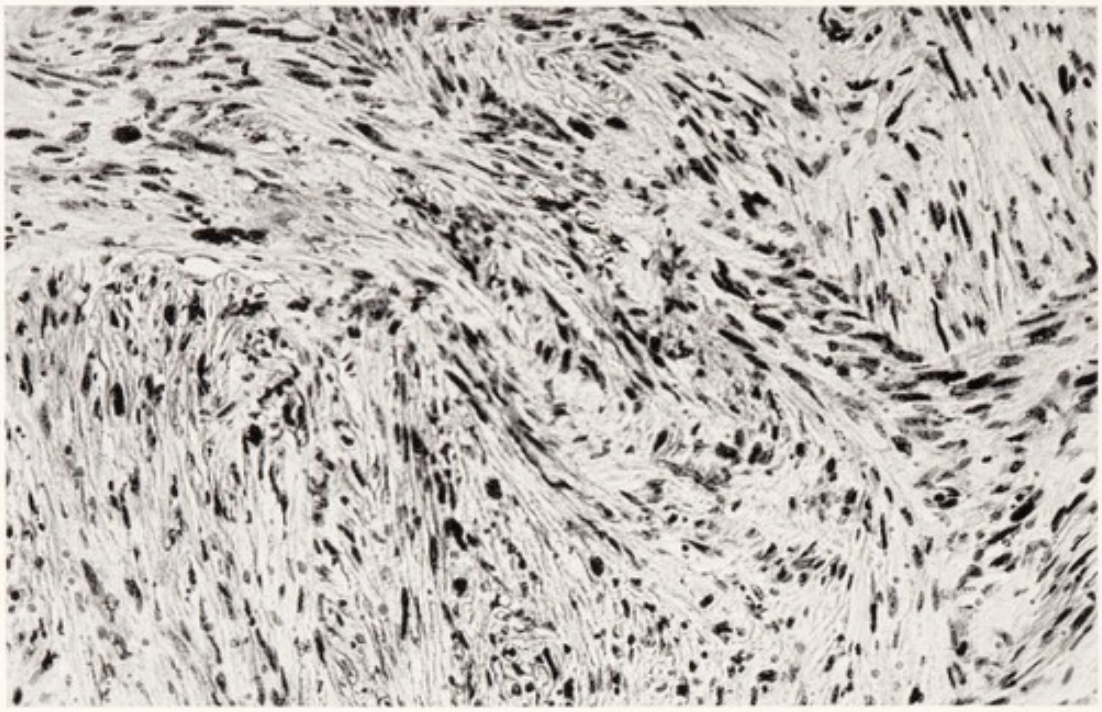

FIG. 131. Recurrent epulis: a large area of spindle celled fibrous tissue without the presence of any giant cells. The spindle cells are prominent throughout the area; they vary in size and stainability. The fibrous bundles dominate the field and are deeply staining. The vessels are few in number and have well defined walls.

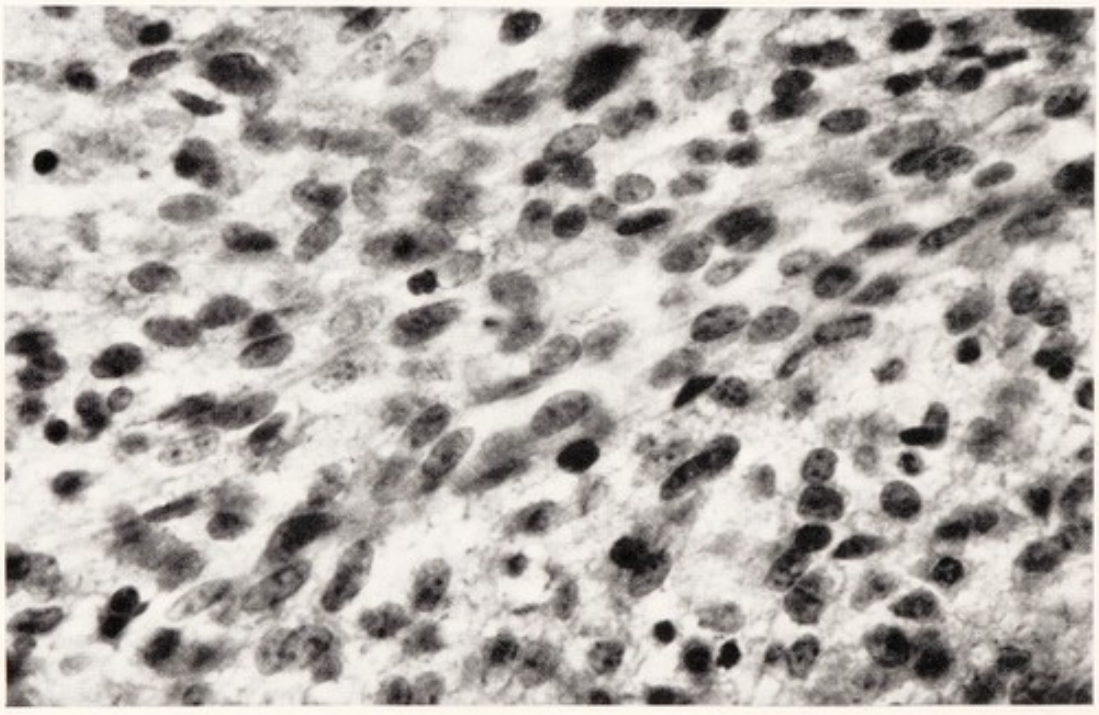

FIG. 132. Recurrent epulis after fourth operation. The entire field is made up of large round and spindle cells ranging from pale to deeply staining. The nucleoli are deeply staining. In some areas is seen a state of degeneration. The intracellular tissue is granular and stains but slightly or not at all.

Histology. A slide of a typical epulis is a thing for the sophomore student, so typical are they. The distinctive feature is the presence of giant cells (Fig. 130) and the freedom of the mucosa from the tumor proper. The intervening connective tissue may contain many round and spindle cells. It is the character of these cells that spells the rate of growth. Recurrences are prone to contain more fibrous tissue containing spindle cells of varying sizes (Fig. 131). As the growth approaches malignancy the cells increase in size and number at the expense of the giant cells. When a distinct stage of malignancy is reached all trace of giant

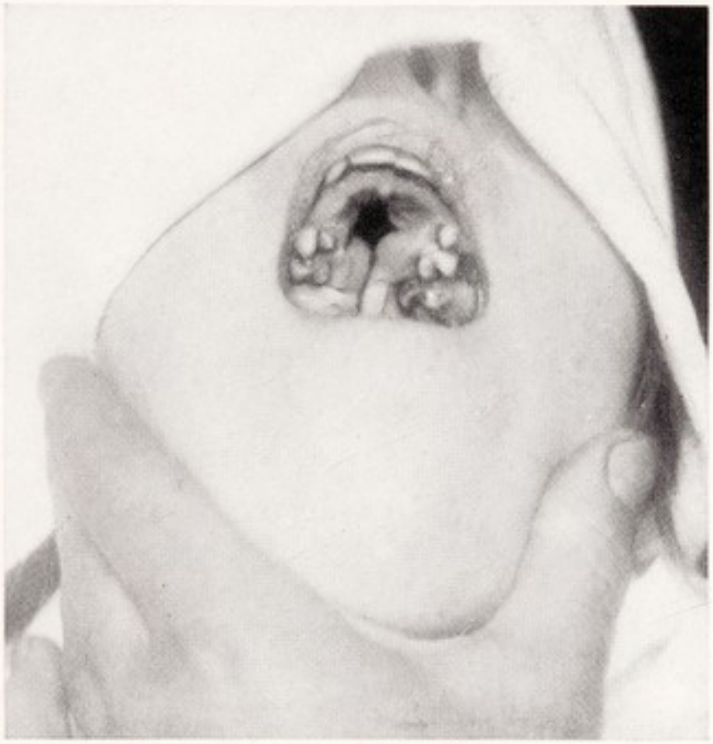

FIG. 133. Hypertrophy of the gums. Boy, age 14. After the hypertrophied tissue was removed there was no recurrence after 3 years.

cells and even of well-formed connective tissue is lost (Fig. 132). Needless to say that when this stage is reached cure is impossible.

HYPERTROPHY OF THE GUMS

This curious lesion may attend some disturbance of development, at least it begins in early life because the alignment of the teeth is much disturbed.

Pathogenesis. The patient notices a gradual thickening of the gums. So insidious is the growth and so devoid of disturbance that usually the deformity has attained a considerable size before medical aid is sought (Fig. 133). The excess tissue can be resected, the denuded surface

heals over and the general condition improved so far as mastication is concerned. Of course the teeth are not realigned. In none of my three cases has it been possible to obtain an after history. The treatment is not pleasant even if one attacks but a small area at a time.

Pathology. The nodulated enlargement and the irregular arrangement of the teeth declare the nature of the trouble at a glance. On palpation they are densely hard. The mucous membrane is not affected.

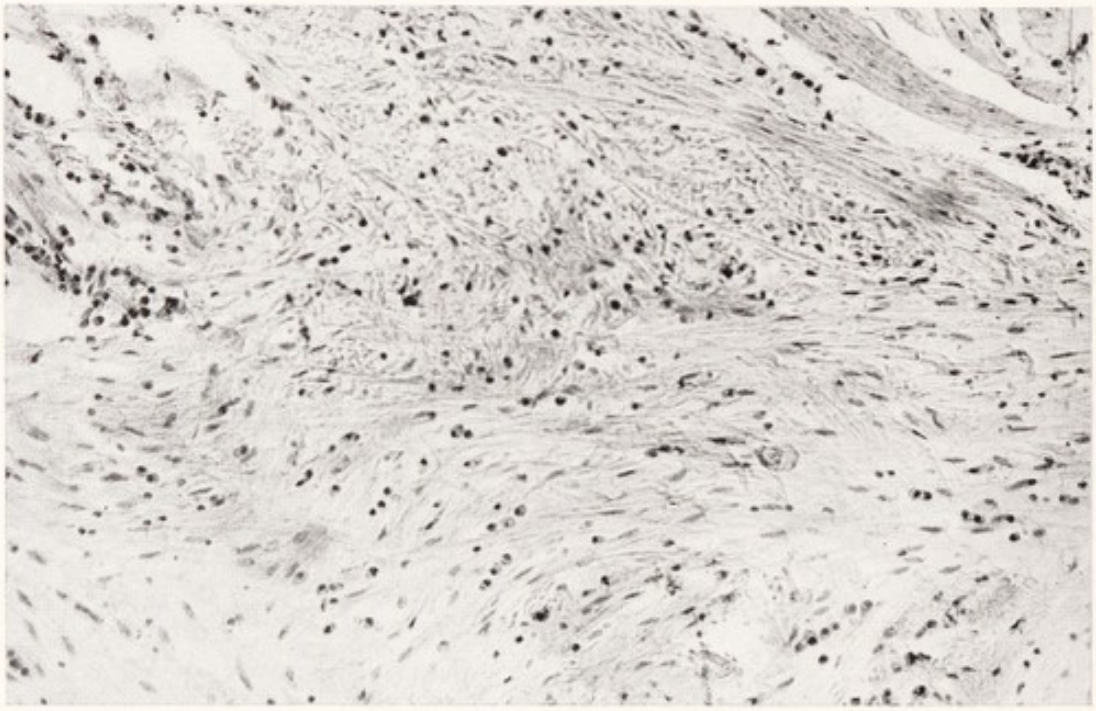

FIG. 134. Slide from hypertrophied gums. The density of the fibrous tissue varies as does the stainability. Many large cells with pale protoplasm and deeply staining nuclei are interposed. Some areas stain palely as though mucinous in nature. Large vascular spaces are present.

Histology. The structure may be densely fibrous, almost keloidal in structure but in part it is made up of fibrous tissue with cells suggesting the structure of elephantiasis (Fig. 134).

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CHAPTER VII

Malignant Tumors of the Jaws

THE various structures surrounding the oral cavity are so interrelated that it is difficult to separate the various regions for the purpose of presentation. In this chapter it is designed to study those lesions which attack the jawbone. The epulides studied in a previous chapter involve the bone to a certain extent, but their involvement never becomes the major lesion. Malignancies of the tongue, floor of the mouth and cheek do not involve, at least not until very late, the bone itself. Likewise, the dentigerous tumors use the jawbone chiefly as a covering to be pushed aside to permit their growth. For this chapter there remain but the malignant epithelial diseases of the alveolar process which in themselves are a distinct group only because by nature of their location they extend down into the tooth sockets, and in doing so in this way the bone destruction becomes the major lesion, at least to the operating surgeon.

The sarcomas are distinct enough clinically but are very rare diseases. They have, in general, the characteristics common to sarcomas of the bone.

Included in this chapter also are a number of benign bone lesions of little importance clinically, and of less pathologic interest.

These varieties may be discussed in the following order:

I. Carcinoma of the Jaws
II. Sarcoma of the Jaws

CARCINOMA OF THE JAWS

By carcinoma of the jaws may be understood epithelial malignancies developing on the alveolar border, and by invasion destroying the bone. Quite like other carcinomas in anatomic structure, though developing less rapidly than other tumors of the mouth, the difference in growth is likely due to the lesser blood and lymph supply of the gums.

It is customary in discussing the carcinomas of the alveolar process to note that they begin adjacent to some ill-fitting dental apparatus or directly about a broken tooth. However, the tumor may begin about a sound tooth or on gums long devoid of any teeth. The disease is most prone to develop in mouths ill kept, but those who practice careful dental hygiene are not exempt.

The most common site is about the molar teeth. Patients tolerate cancers for months or even years before seeking advice. They regard the inconveniences they suffer as a part of the sore mouth with which they have long been afflicted. Here, as usual, those who need advice the most do not seek it and they come to us after irreparable damage has been done.

Pathogenesis. Two general types may be distinguished, the ulcerative and the fungating. The former may continue as a superficial ulcer while the growth is extending downward destroying the bone. This is the type which the patient is most apt to overlook or neglect. The fungating type, as the name implies, produces an elevated lesion which expends its energies by producing an outgrowth long leaving the bone unaffected. Because of the obvious tumors this type produces mastication is interfered with, and sometimes hemorrhage helps to cause the patient to seek relief.

We know that cancer of the jaw must begin as a small lesion but, when the patient first presents himself, in most cases it has attained such dimensions that there is no question as to the nature of the lesion. The local invasion may not be confined to the mucosa for a long time but may invade and rarefy the underlying bone. This invasion is usually confined to the alveolar border, leaving the body of the jawbone intact so that removal of the alveolar border accomplishes as much as the resection of the jaw. Once the body of the bone below the alveolar border is involved, no treatment is of avail. At least in none on whom I have resected the jaw was the disease stayed. In my earlier years I resected a number of jaws with cure, but the resection was unnecessary, I suspect, judging from later experience.

I saw one patient who did not heed the lesion until a severe hemorrhage occurred attending the erosion of the artery. I have seen more hemorrhages occur a week or so after the removal of the alveolar border and cauterization of the base. This experience is so exasperating that now preliminary ligation of the external carotid is a regular precaution, I may add in passing. I have seen several instances in which the patient disregarded the lesion on his gums until a spontaneous fracture excited his curiosity. I have also felt the bone fracture in my hands as I sought to remove the affected part.

The adjacent lymph glands remain free from metastases for a longer time than in the epithelial tumors affecting the soft parts. Sooner or later the lymph glands become affected and, due to secondary infection, may ulcerate, causing diffuse cellulitis of the connective tissue of the neck with general sepsis and edema of the larynx. The oral ulceration

may interfere with nutrition and the patient quickly becomes emaciated and, with this exhaustion, deglutition pneumonia is apt to initiate the terminal stages.

Pathology. The ulcerative type may not be impressive on inspection, and there is little to impress the photographic lens, but the examining

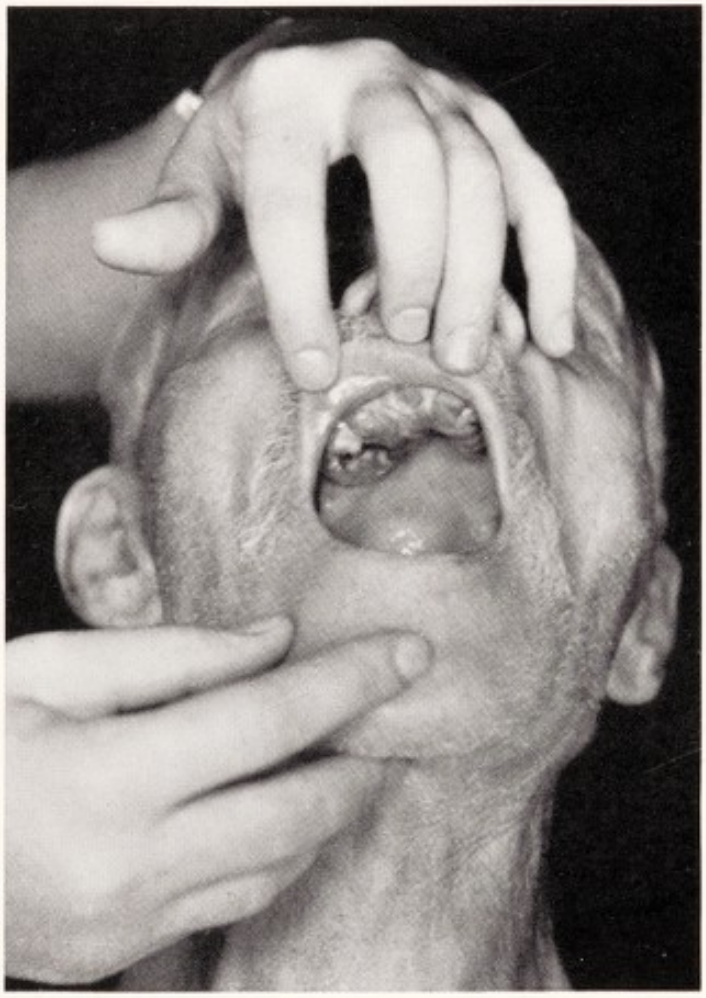

FIG. 135. Carcinoma of the gum. First noticed the lesion three weeks before. A deep craterform ulcer occupies the site of the bicuspid tooth. Stumps of the incisors are still in place.

finger detects the ulcer with a hard rough border which is unmistakable (Fig. 135). Commonly, there is a tumor about the site of one or several teeth. The disturbance is regarded as due to defective teeth or pyorrhea and the offending organs are withdrawn. The lesions do not heal and the advice of the surgeon is sought. By this time the hard ulcerating border is easily recognized (Fig. 136).

The fungiform type may protrude as a solid mass, the surface being fairly smooth and without notable ulceration (Fig. 137). More com-

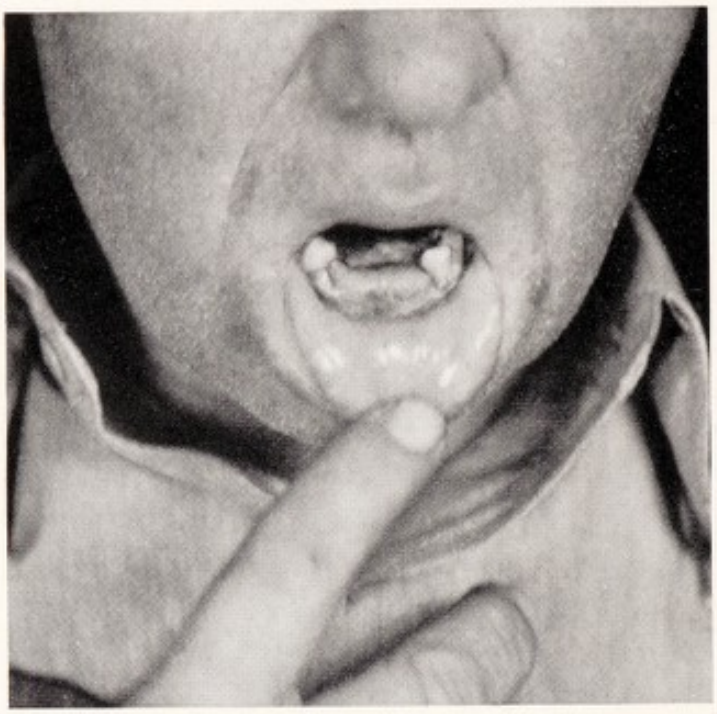

FIG. 136. Carcinoma of the jaw. A solid roll of cancer occupies the alveolar border previously the site of the incisor tooth.

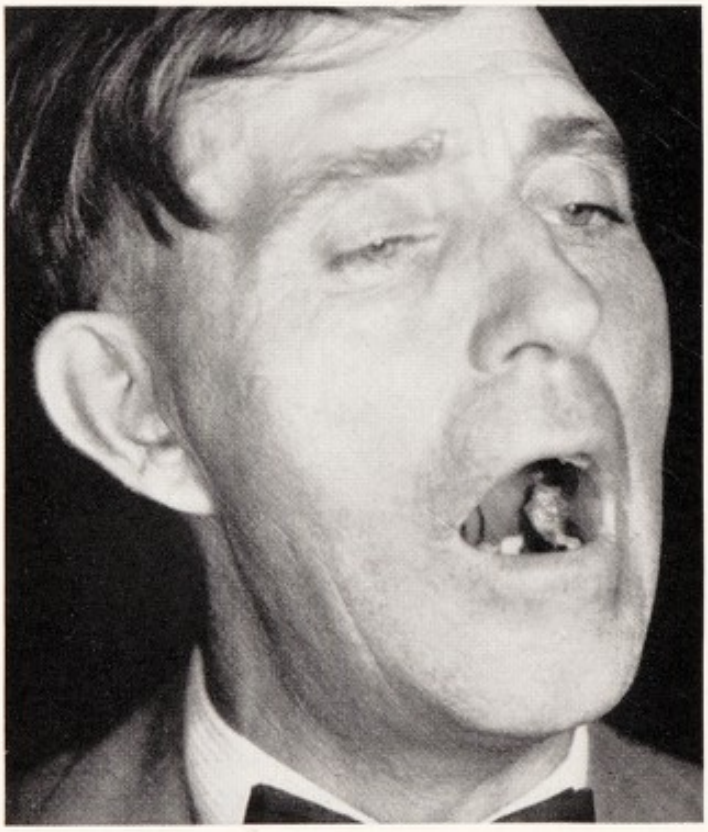

FIG. 137. Carcinoma of the gum. A solid protruding mass occupies the site of the second and third molars.

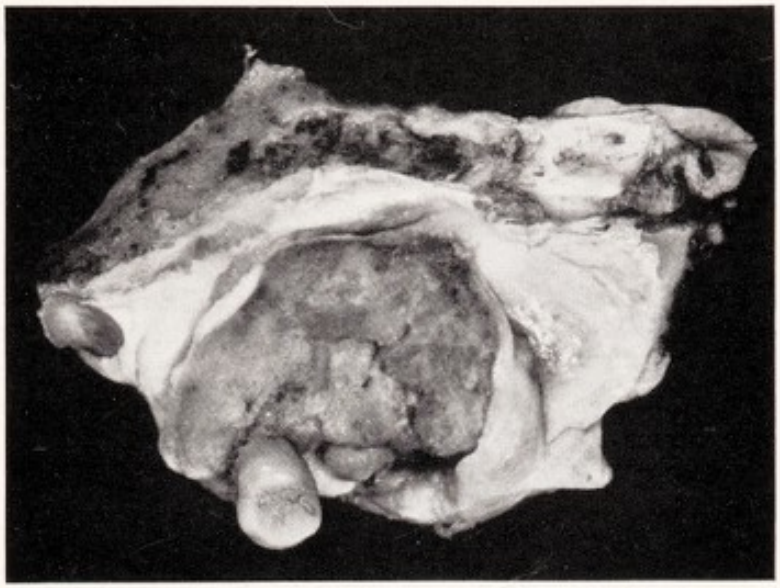

FIG. 138. Carcinoma of the gum. A fungiform mass with a roughened surface extends from the bicuspid back. Free from recurrence fifteen years after cauterization.

monly, the surface is papillary, being covered by small mountain-like elevations, which are in part destroyed, producing a tumor that is primarily papillary but is becoming ulcerous (Fig. 138). This may extend to such a degree that what was once a papillary tumor has become a purely ulcerous process. These tumors are usually observed while the

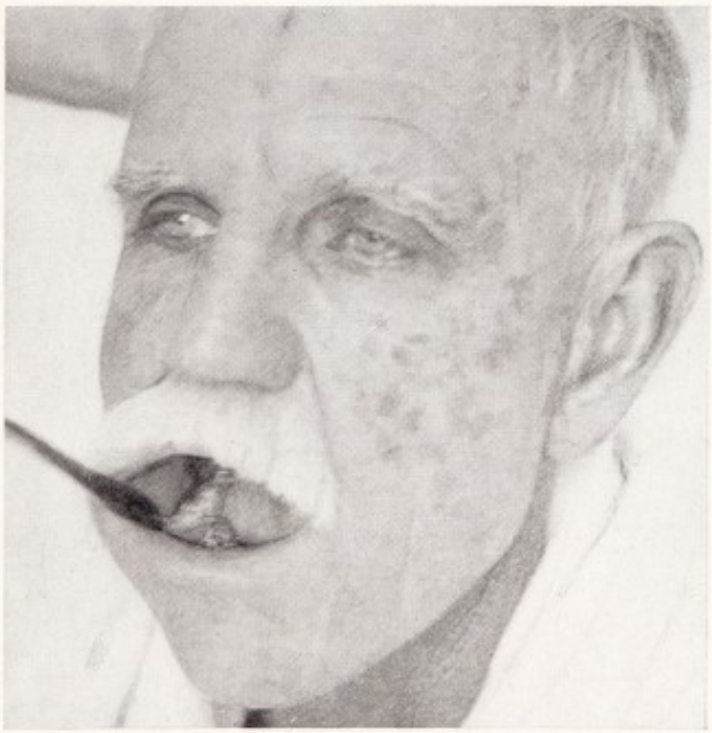

FIG. 139. Epithelioma a part of an adamantine tumor protruding through the alveolar mucosa but without involving it.

bone is still unaffected, as shown by the x-rays. This is due in part to the nature of the growth, but also because of the interference with mastication the patient becomes interested and seeks relief.

In carcinomas of the upper jaw, an added degree of circumspection must be exercised. Adamantine tumors sometimes become malignant in the epithelial elements. These may protrude through the gums, producing an ulcer which is epithelial in structure, and consequently is a car-

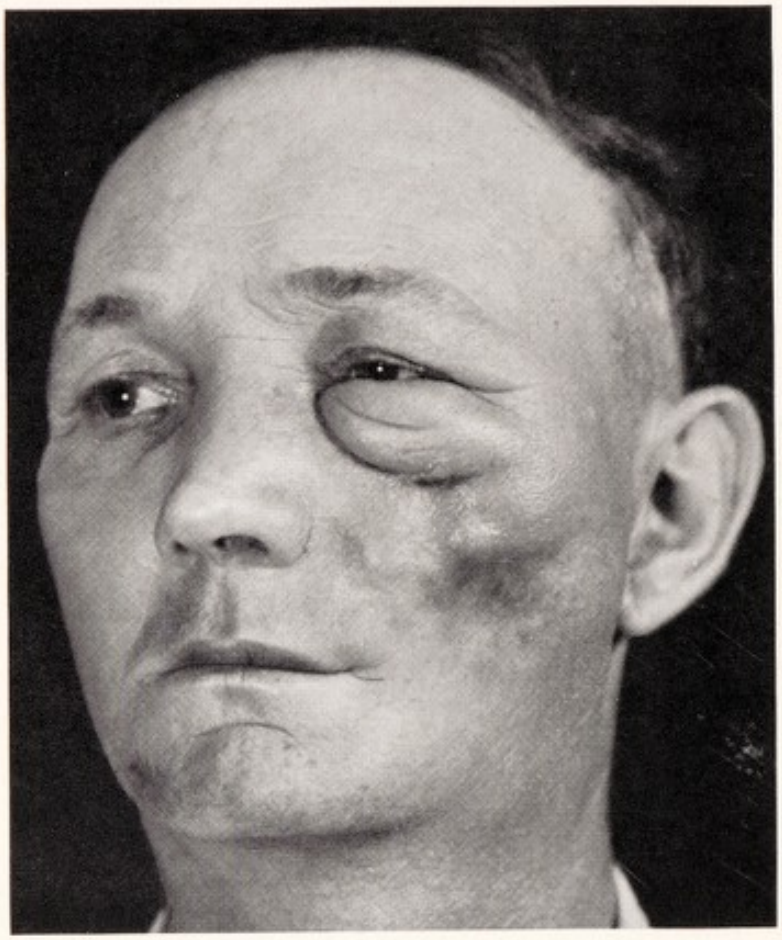

FIG. 140. Carcinoma of the antrum invading the external wall and the plate of the orbit.

cinoma when palpated, or a diagnostic section is examined. Such errors need not occur if the examiner will carefully inspect the growth and refrain from removing a diagnostic section. It will be observed that the epithelium of the gum is not engaged in the process, the growth destroying it by pressure from beneath (Fig. 139). The second point is that other elements of adamantine tumors lie deeper and will be revealed by an x-ray plate. The case noted in the figure had, beside the carcinoma on the top, an adamantine structure beneath and an area of typical giant cell tumor at one border.

Such tumors lead one to the nature of epithelial malignancies of the antra. Typical epithelial tumors occupy the antral cavity without involvement, apparently, of the alveolar border. These may be of squamous type, becoming so because of metaplasia of the epithelial lining of the antra caused by suppuration. The possibility is development from dentigerous rests. The two forms need not be confused. The adamantine epitheliomas produce expanding tumors easily discernible on inspection, while the primary epitheliomas do not notably expand the bone but tend

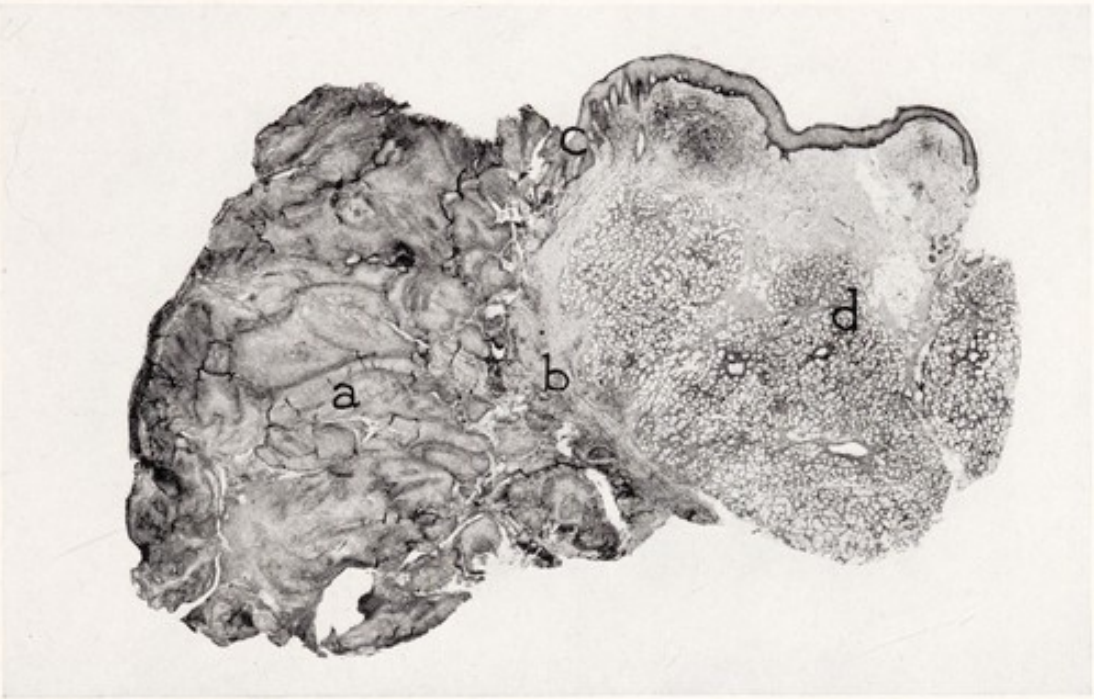

FIG. 141. Papillary projection of a gum adjacent to an area which had become malignant: *a*, hard keratotic nodule protruding from the surface of the gum; *b*, there is slight round cell infiltration but no apparent invasion; *c*, epithelium of the alveolar process terminating at the base of the tumor; *d*, section of the alveolar process.

to invade it (Fig. 140). The bone may be destroyed and the tumor invade the cheek or orbit, and still there is no expansion.

Histology. The study of the slide is not uninteresting. Very early the elevation may be hard, keratotic, resembling the horns which form on the lip (Fig. 141). There may be but little evidence of malignancy. The surface is made up of keratinetic cells, poorly staining and, in part, degenerating (*A*, Fig. 142). The structure of these cells may be lost, others illy staining, there remaining but a deeply-staining nucleus (*B*, Fig. 142). The base of a typical early papillary carcinoma presents an active proliferation of the epithelial cells with the tendency to keratinization (Fig. 143). The surrounding tissue is extensively infiltrated with

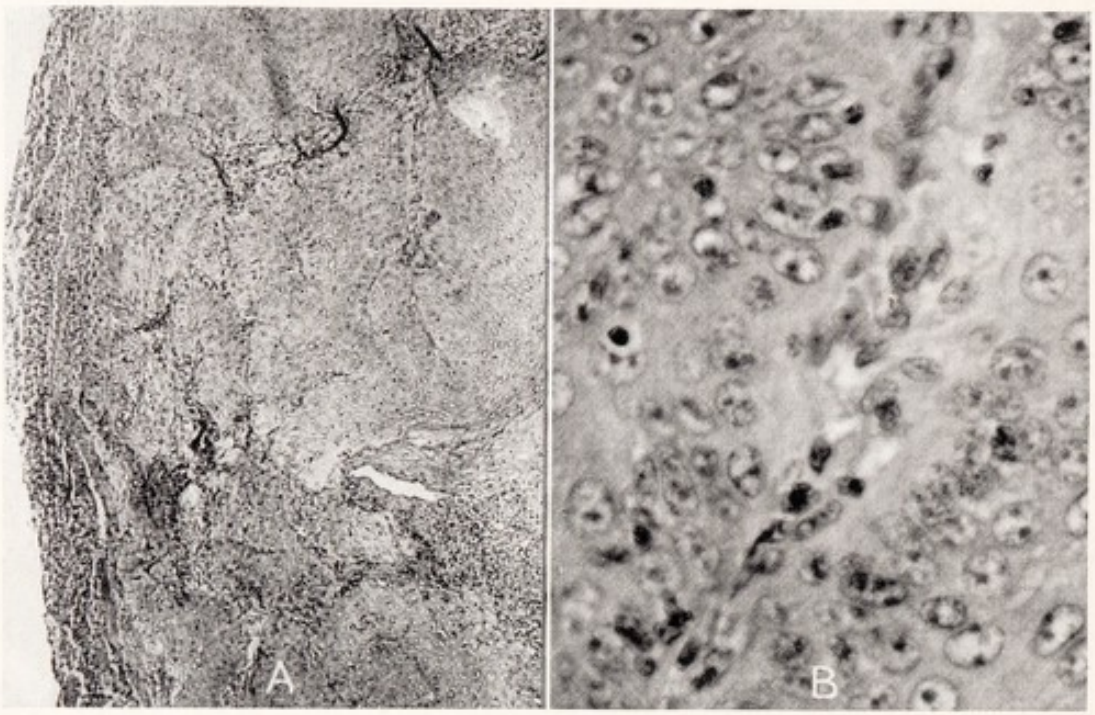

FIG. 142. Papillary carcinoma of the gums: *A*, the surface is covered with debris beneath which is a piling up of ill-formed epithelial cells; *B*, high power of the slide shows the epithelium in a state of degeneration. Only the nucleoli are well preserved.

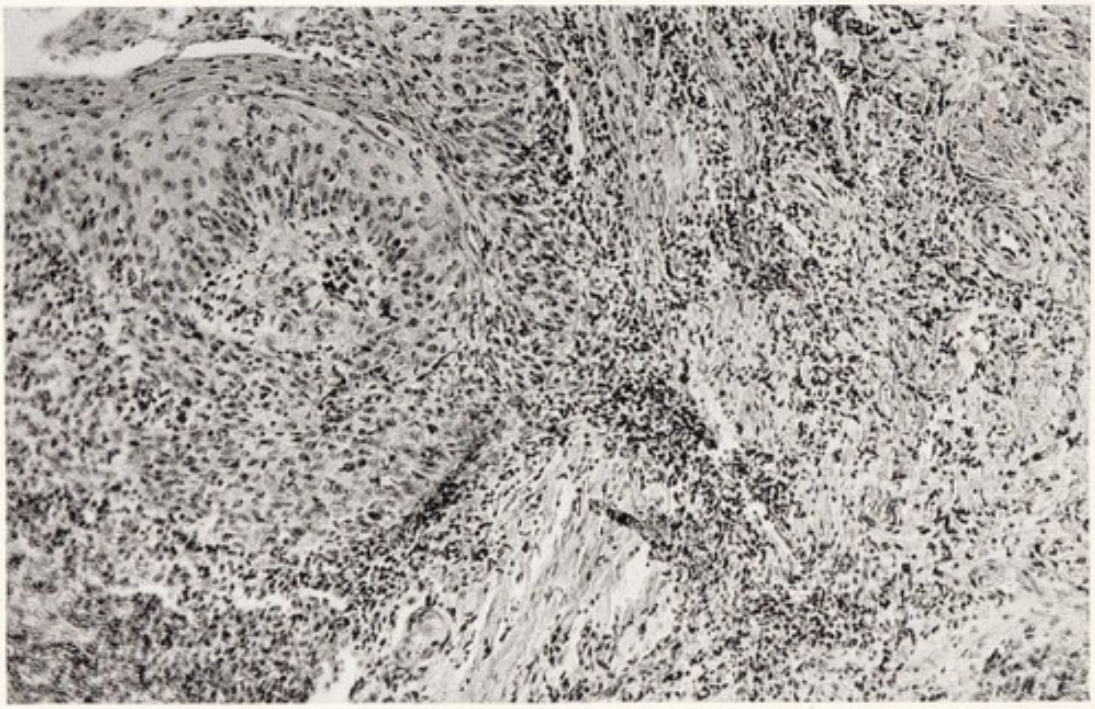

FIG. 143. Papillary carcinoma of the gum. The bases of the papillary projection show a proliferation of cells with a tendency to early keratinization, hence but little nest formation and only moderate changes in cell type. There is abundant round cell infiltration beneath.

round cells. This is the type one cures with the cautery. The metastases, also, are not without interest. The affected glands may show only here and there a pearl (*A*, Fig. 144) without other evidence of epithelial invasion. On the contrary, the changes in the cell type may be so great that all alveolar arrangement may be lost (*B*, Fig. 144).

SARCOMA OF THE JAWS

The various types of sarcomas may be found in the periosteum and in the antra. The various types of connective tissue are represented. I once had apparently a pure myxoma of the antrum and I expressed a

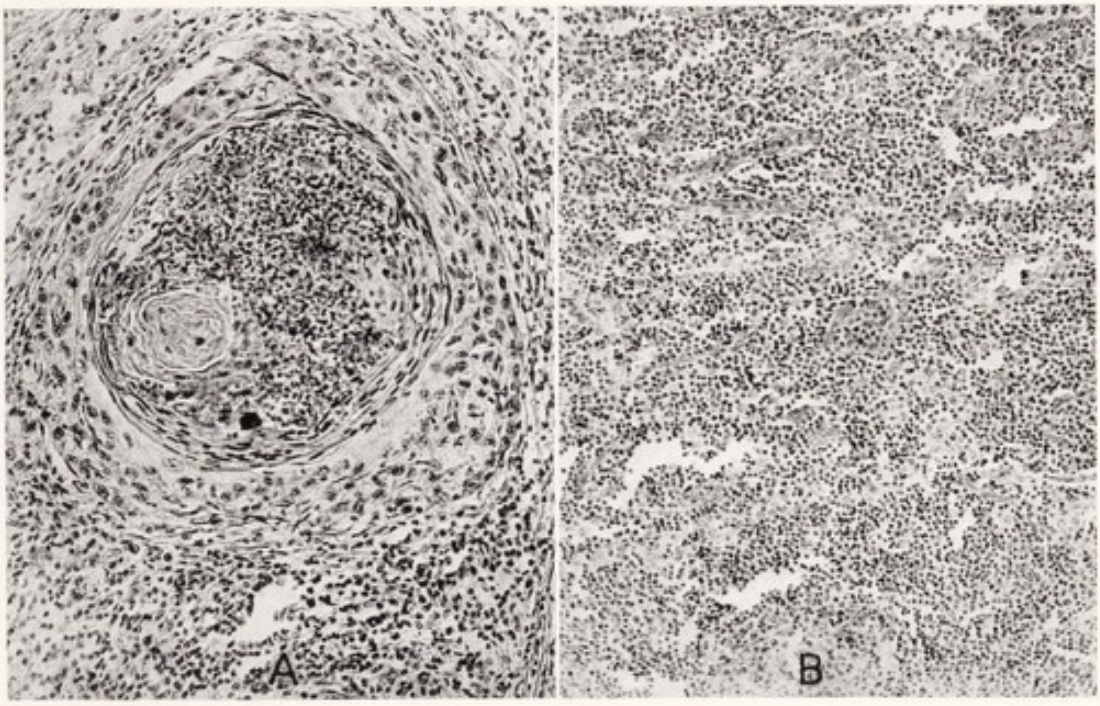

FIG. 144. Lymph gland metastasis in papillary carcinoma of the jaw: *A*, only small pearls are found scattered about without epithelial nests; *B*, diffuse cellular invasion without definite formation of alveoli. The connective tissue is reduced to granulating illy staining bundles.

favorable prognosis. I have learned by experience that an elevation on the gums which has a broad base, or a loose tooth without pyorrhea, one had better regard the lesion as malignant until it is proved otherwise. The prognosis of sarcoma is always bad, no matter how early diagnosed, but it saves the dignity of the surgeon to be able to anticipate the unfavorable result.

Pathogenesis. The early sign of a periosteal sarcoma is a tumorous elevation that is diffuse. The most common form is found on the lower jaw. They sit on the edge of a bone like a mudball on the edge of a board. Usually the spread is rapid. The subfascial type, it is said, grows more

slowly. The sarcomas developing in the antra make themselves manifest by expanding the bone, then perforating it and spreading into the surrounding tissue. Occasionally they grow into a nostril, filling it and announcing their advent by a bloody nasal discharge.

Pathology. As above intimated, a submucous tumor, especially if diffuse, or one periosteal, should be suspected of being a sarcoma. A biopsy is necessary to give it a specific name, but the prognosis can be made without such detail.

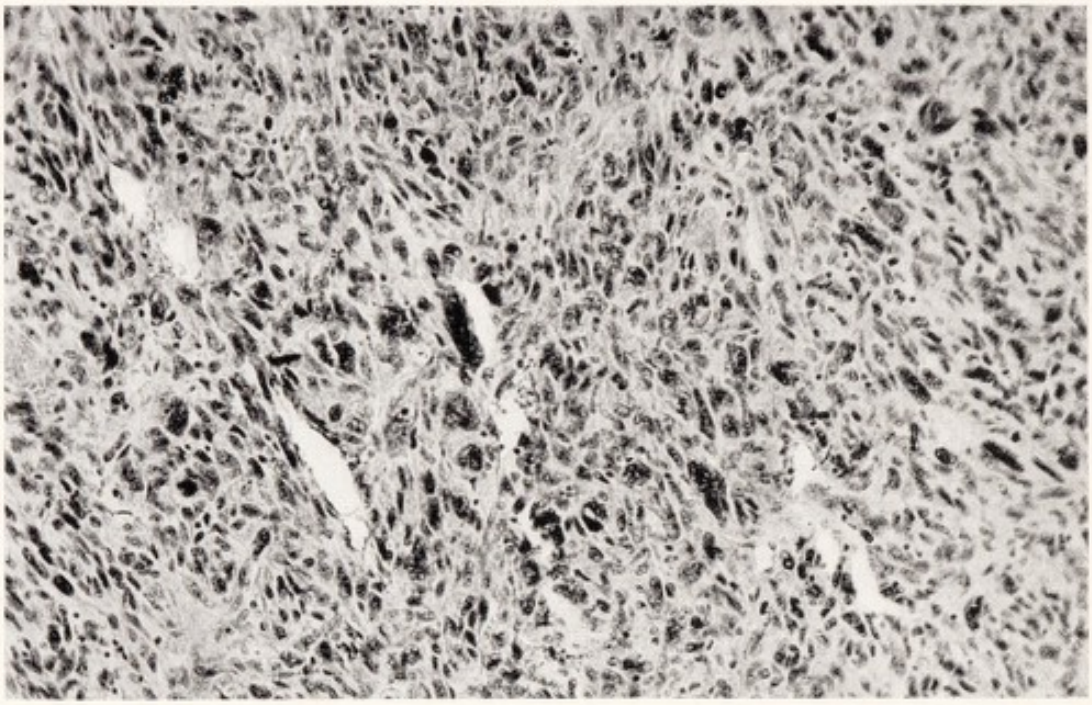

FIG. 145. Periosteal sarcoma of the jaw. The slide is made up of cells of varying sizes and shapes and degrees of stainability. The vessels are large and numerous with very thin walls or none at all. Some cells are in process of degeneration. The connective tissue is near the vanishing point.

Histology. All cell types are represented. Fibrosarcoma and myxosarcoma are commonest in the antral cases. The periosteal sarcomas, in my experience, are very cellular round cell or spindle cell tumors. They fairly breathe malignancy (Fig. 145).

Literature

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CHAPTER VIII

Tumors of Dentigerous Origin (Mixed Tumors)

SURGEONS have suffered much from over-classification of diseases in many situations, but nowhere more than in the developmental tumors of the jaws. Root cysts, dentigerous cysts, adamantine cysts, adamantine carcinomas with cysts, fibromas, myxomas, and sarcomas are but a few of the things the surgeon is asked to keep in his mind's eye. These are but subdivisions of a single process and attempts to designate them by familiar terms more often mislead than guide the surgeon. Too often the attempt at subdivision leads to the focusing of the attention on but one part of the tumor, and it usually results from an inadequate examination of the tumor in the laboratory, or perchance the diagnosis of the whole is based on a biopsy.

Nowhere is the disastrous result of trying to teach surgeons too much pathology more clearly emphasized. Subdivisions as above indicated become but words which cannot be translated into tangible things clinically, and have led to much confusion. If one arrays before his mind's eye the experiences of many years, while the individual tumor forms differ greatly, there is a certain uniformity in the course of the disease; one type of tissue dominating the picture in one case, quite another type in another, but the course is the same, a tumor of low malignancy, if attacked early and adequately, but all capable of being teased to malignancy by timid tinkering. A cyst, a myxoid area, a giant-cell area, and an epithelial area are individual only under the microscope. Collectively speaking, they are a part of a congenital slowly growing process, the removal of which results in permanent cure. One might compare the multiplicity of pictures in mixed tumors with women's clothes; there is endless variety and each has a name, but mere man learns after awhile that they all belong to the same group, and as to the name and when or where worn—the less he knows the better. Detailed knowledge is all right for some folks; detailed knowledge of this group of tumors may be all right for the pathologist, he is responsible only to his science, but the surgeon is responsible to his patient.

The point to be emphasized is that in the clinic and in the operating room these tumors are simple enough. The tumor must be removed

completely. But there is a vast difference between complete and radical operations, measured by the removal of tissue. The removal of diseased tissue fulfills the requirements. A radical operation implies the removal of the environment. This is the reason why the surgeon must know these tissues—in the raw. The tissue once removed, one can take it to the laboratory and separate the various areas into all kinds of funny structures. Even then the attempts to give each area a specific name is likely to lead to a compound term which only causes confusion, and it may be emphasized, leads to a mistaken prognosis.

Obviously this chapter is not intended to glorify my own prowess as a jaw surgeon. My experience has been sprinkled with mistakes and disasters, exemplified by needlessly radical operations in my earlier years, as was the habit of the times. Resection of a jaw was a thing that brought the staff-room loafers clattering to the operating room, music to the young surgeon. But that was all wrong. However, there is this consolation; if I had had more sense I would have fewer specimens, for radical operations did preserve the tumor in its relation to its environment. Nowhere else is it so necessary to plan the operation as one proceeds. If there is tissue that does not belong there, remove it, and when all foreign tissue has been removed the operation is finished. Clinical examination, including x-ray, gives one only a general idea as to the plan and extent of the operation. Details must be worked out as the operation proceeds. The simple operation, I may add, which results favorably for the most patients, produces but a mass of tissue difficult to reconstruct in the laboratory. Such successes are neither interesting nor instructive when viewed in the museum.

The important fact is that, like most tumors of congenital origin, these are nearly all slow growing, usually curable by adequate operation if done in reasonable time. This may be stated as a general axiom. It must be emphasized, however, that many of them are capable of being led to disastrous ends by neglect, especially when aided and abetted by surgical prodding. Time was when all jaw tumors were called sarcomas, and were treated by resection of all the bony tissues in the neighborhood. This led to needless mutilation, it is true, but even now while many of the tumors of the jaws admit of conservative treatment, some of the neglected still require the services of a surgeon who can go the route wherever it may lead, be this much or little. Often one does not know at the beginning of the operation just how radical the operation will need to be, therefore, he who begins should be sure that he will be able to finish the job.

Generally speaking in all these features, slow growth, innocent clinical course, years without end, they resemble the mixed tumors of the sali-

vary glands. Happily, in mixed tumors of the salivary glands we have been able to resist the urge to classify each cell type as a separate thing. We look at them as clinical entities, which it is impossible to recognize and accurately to chart the course, because one can watch them, feel them, and wiggle them decades without end before they finally reach a stage of malignancy. Jaw tumors, unfortunately, are more hidden and variable. It is more difficult to estimate the date of their beginning and their rate of growth up to the time the patient presents himself for examination. Because of this the surgeon must, with tumor in hand, be able to supply the missing evidence and figure backward to the probable date of beginning, to wit—infancy, and to proceed with whatever treatment is appropriate. The microscopic examination then becomes a laboratory sport. Simple frozen section examination in the operating room is as apt to mislead as to enlighten. This fact cannot be too strongly urged. The problem is from beginning to end the surgeon's own responsibility.

All the tumors here considered are due to some disturbances of development, and should be regarded as a common group. Nevertheless, certain lesions, perhaps dependent on the stage or type of tissue displaced, run a course more or less characteristic and admit of clinical and pathologic classification. Thus there are pure cysts and cysts complicated by solid tumors of varying cytology. The simple cysts, naturally, are less likely to give trouble than those associated with a solid tumor. Could I free myself from a desire to be orthodox, I should classify all cysts together making but two groups, the cystic and the solid.

An outline such as the following is useful in furnishing pegs on which to hang the several pictures:

I. Dentigerous Cysts
II. Root Cysts
III. Odontomas
IV. Adamantinomas

From the vast literature on the pathogenesis of these various groups one cannot escape the impression that while the fundamental factors lie somewhere in disturbances of tooth development, the details are not yet perfectly understood. However, the details of embryonal misbehavior have only an academic interest for the surgeon. For him certainly this concept is sufficient for his purpose.

Of the several lesions listed in the above classification the adamantinomas are by far the most important. In comparison with them the others are innocent and unimportant. Nevertheless the most innocent of them, if trifled with, may rise up and smite the surgeon.

DENTIGEROUS CYSTS

Dentigerous cysts may be independent of tooth formation or they may include malformed teeth. They are usually single, but may be multiple. These are the simplest form of developmental tumors and are the least likely to be complicated by or merge into more serious disorders. I believe it contributes to clarity to look on these as analogous to dermoids. They form cavities of varying dimensions, and are for the most part smooth walled, and lined with a simple flattened epithelium. Their clinical course is in harmony with this concept. If the cell lining is

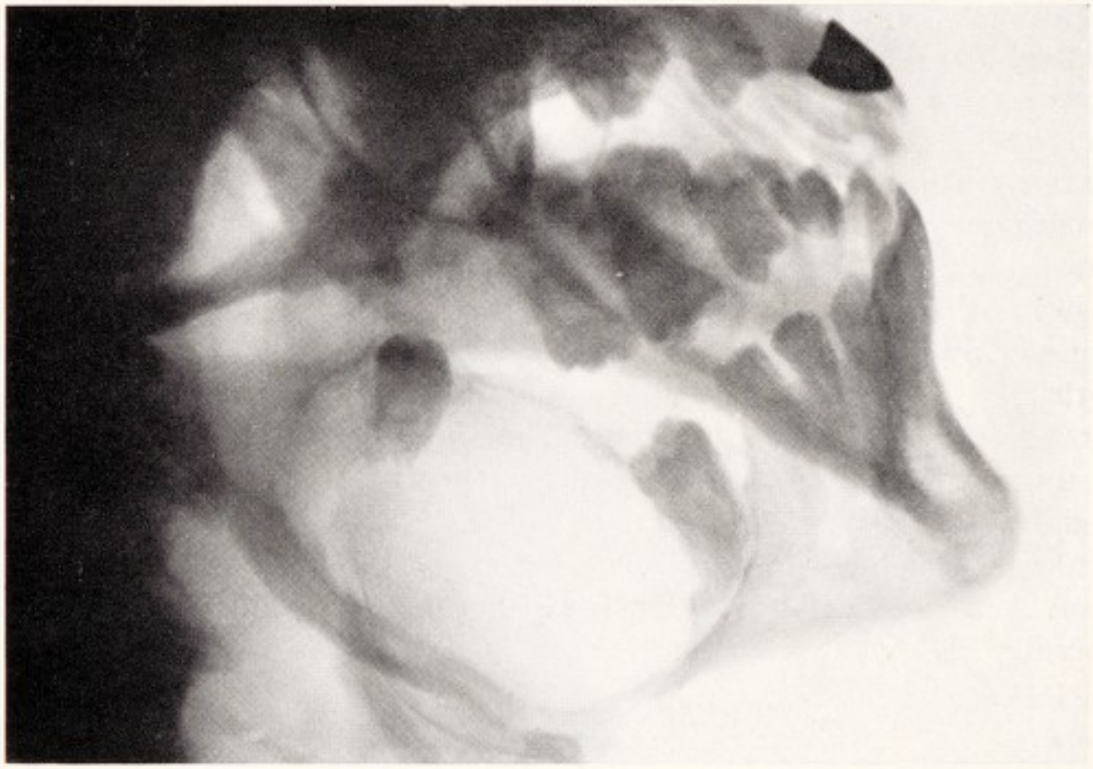

FIG. 146. Dentigerous cyst destroying almost completely nearly the whole of the lower jaw.

removed, or the epithelium is destroyed by chemical means, they stay cured. Again, like dermoids, if a part of the lining escapes destruction, a renewed cyst or a permanent fistula results, which awaits another operation. The relatively benign nature of these cysts is now generally recognized and the common error is to do too little at the operation, and a recurrence is the result. This recurrence, being misunderstood, may incite to an operation that is needlessly radical.

Too timid operating is not the only mistake. I once received in my laboratory a specimen of an entire lower jaw from one articular process to the other. Both horizontal rami were expanded, so that a single cavity

of nearly an inch in diameter was formed. It had been removed by an eminent surgeon of the old school under the diagnosis of sarcoma. Needless to say the tumor was not explored at the operating table.

Such extensive cysts are rare. Usually they are limited to one ramus, the horizontal, most commonly associated with the third molar tooth, forming an oblique or ovoid cavity. Fortunately now, when all tumors of bones are examined by the x-ray, the cystic nature of the tumor is at once apparent. But the x-ray picture should not be accepted without question. The lesion must be inspected at the operating table in order to determine if the interpretation of the plate was correct.

Pathogenesis. In most cases the patient's attention is not attracted to the presence of a cyst until the expanding bone produces a deformity. Sometimes a sense of discomfort in the jaw or some tooth disorder leads to an investigation. Small in the beginning, scarcely demonstrable by x-ray examination, they continue to enlarge, producing a thinning of the bone until it is all but destroyed, though spontaneous fracture does not seem to occur. However, attempts at a conservative operation may lead the luckless surgeon to have nothing left after he has completed his operation (Fig. 146). It is necessary, therefore, in extensive tumors to proceed with caution and chemical destruction or irradiation may be safer than mechanical removal. In most cases, the curetting out or cauterization of the simple lining preserves the continuity of the bone and cures the disease. Any remaining membrane may cause a permanent fistula (Fig. 147), or may lead to the recurrence of an expanding tumor. Such an event, as above noted, should not incite the surgeon to a needlessly radical operation.

Pathology. The expanding bone produces a smooth elevation usually more or less fusiform in outline. If the tumor has attained some size the bone may be so thinned that it crackles under the pressure of the finger, and may be easily penetrated with a needle or a knife. When the cavity is opened the wall is smooth to the touch or elevations made from the roots of the teeth may bulge into it, making a corrugated surface. The curet brings forth nothing but a thin membrane.

The act of wielding a curet is distinctly a study in pathology. The membrane is loosely attached and strips off easily. If it does not, it is a matter of concern. If, in the absence of infection, there is solid or granulation tissue, this should be regarded as giant-cell bearing or even more vicious material, until proved otherwise. Of course, a frozen section will, at once, clarify the histology of one particular small area, but the exact cellular structure makes little difference, because if there is any soft tissue remaining, the operation must continue. The more difficult it is to remove

the cyst lining, the more imperative it is that it be removed. If the bone at such a site is eroded, the possible existence of a tumor mass lying more deeply must be considered. This applies, of course, particularly when the cyst is located in the upper jaw. The failure to regard with suspicion such cysts has caused me more regret than any other lesion of the jaws. When such complications of the walls exist the excision of the tumor-

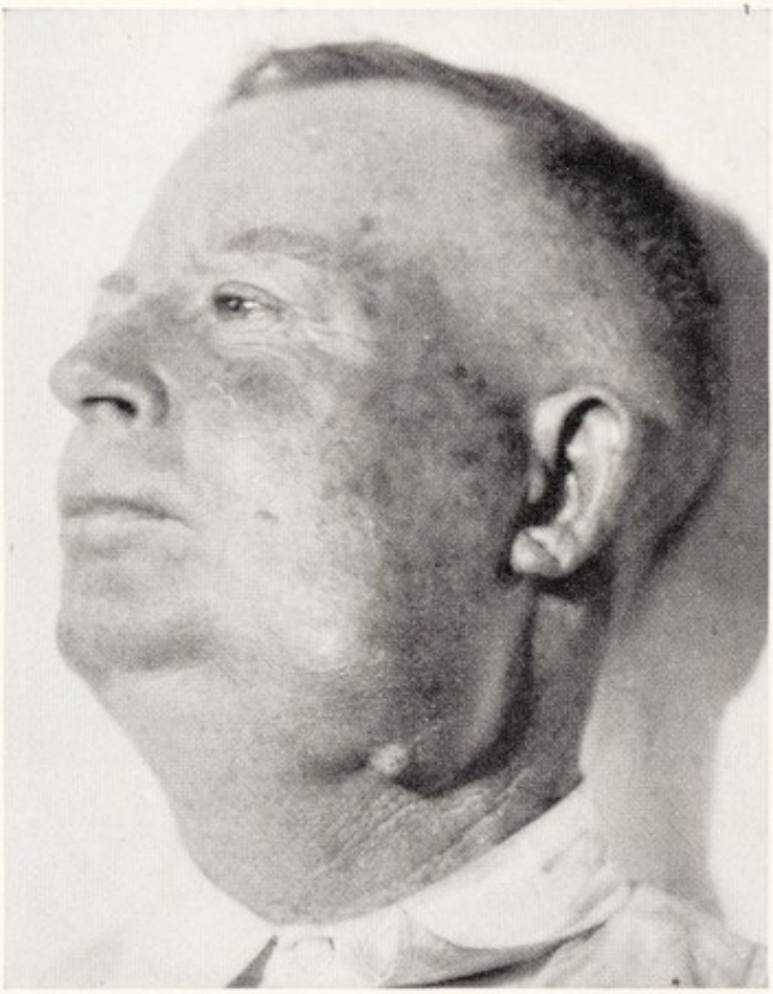

FIG. 147. Draining dentigerous cyst. M., age 57. Had a tumor the size of a hen's egg drained of a clear fluid eight months ago. A fistula formed. Removal of the cyst-bearing area resulted in cure.

bearing area is the safest procedure. This statement applies, of course, only to the cysts of the upper jaw.

Histology. The structure of the lining membrane is confined to a fibrous tissue wall lined with cells, cuboidal or flat, usually arranged in several layers. The occurrence of giant cells or very cellular fibrous tissue is not unusual (Fig. 148), particularly if the lesion has been previously operated on. The discovery of such areas may give one a scare, but if the

bone beneath is smooth after curettage, they need not give one grave concern.

DENTAL ROOT CYSTS

Small cysts attached to the end or the side of a tooth, the size of a pea or larger, are a rather rare occurrence. Most often, nowadays, they are encountered in the course of the x-ray study of the teeth, and they remain in the province of the dentists. This early recognition has made it possible to confuse them with apical granulomas. Being closely associated with the roots of teeth they are apt to be more complicated in

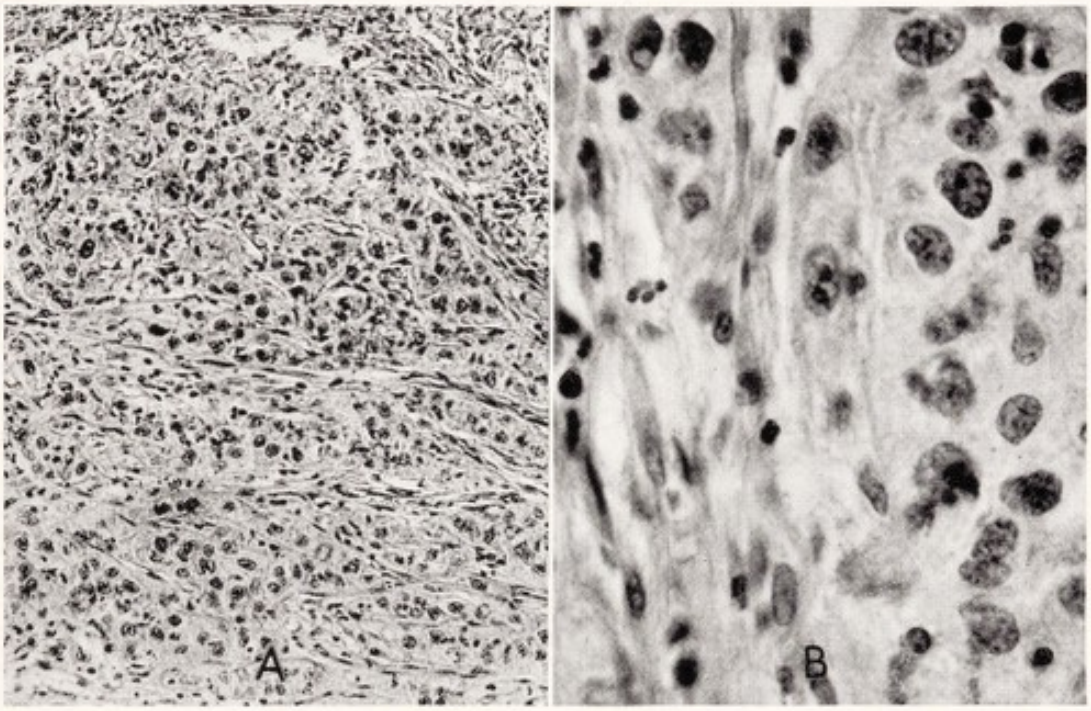

FIG. 148. Slide of a thickened area in the wall of a dentigerous cyst. *A*, Cells arranged more or less in alveolar form with but sparse connective tissue. *B*, The greater part was made up chiefly of palely staining connective tissue in which many epithelial-like cells were intermingled.

structure than the dentigerous cysts, and for this reason deserve a separate classification. On the other hand, they sometimes contain tooth elements or more or less complete teeth, thus aligning themselves with the odontomas.

Other tissues than a fibrous wall with a simple lining may be present. Fortunately, if the cyst wall is destroyed, usually a cure results. Rarely, though too often, the matter is not so simple, particularly in cysts of the upper jaw. If other tissues are present, recurrences should be anticipated by more close observation after operation or better still by a more radical operation at the first sitting. If, in addition to the cyst, the

associated tooth is displaced, either medially or laterally, the lesion should be regarded as potentially malignant and the rongeur instead of the curet should be employed in removing the entire cyst-bearing area. In the upper jaw I do not believe there are any really innocent root cysts. They had all best be removed lock, stock, and barrel. To save the looks of a patient for a time, only to see him die of recurrence, is no triumph.

Pathogenesis. Some pathologists would have these cysts form in granulation tissue about the roots of teeth. To the surgeon this view is not acceptable because epithelial-lined cysts do not occur in inflamed bone elsewhere, and there is no reason why tooth granulation tissue should be an exception. An analogy might be found in Brodie's abscess, but these always remain unchanged and are not lined with epithelium. Furthermore, giant cells are common in the walls of these cysts, and in

FIG. 149. X-ray, after removal, of a mass containing a root cyst.

recurrences, the giant cells may dominate the picture. Furthermore, recurrences, particularly in the upper jaw, may take on a typical adamantine picture. This would suggest that they have a like origin. If one accepts a developmental genesis one is more apt to anticipate a recurrence, the best sort of a stimulus to more thorough treatment.

These cysts, though they have their origin in developmental rests, are not discovered in the earlier stage of development. Formerly, they were not discovered until they expanded the bone. Sometimes they became the size of a hickory nut and remained stationary (Fig. 149) or they may continue to grow ultimately forming tumors the size of a lemon. Even though usually simple in structure they may be the starting point of serious trouble, either from infection or from the development of a malignant tumor.

If discovered by x-ray a small area about the apex or side of a tooth

is the usual finding. When first discovered because of expanding bone the recognition by this is not positive. A tooth may be absent in the affected area, which brands it as a developmental defect. If the alveolar border is expanded a displacement of a tooth may indicate its nature. In this stage the discovery of giant cells may lead to the diagnosis of a giant-celled epulis, despite the presence of a cyst. However, the discovery of giant cells puts a more serious aspect on the lesion.

It seems possible that the development of a new tumor after the removal of one may be due to the formation of another tumor rather than a recurrence. If situated in the same site as the first tumor the differentiation may be impossible, but if the new tumor is situated at a distance, the distinction is simple. In such cysts which contain displaced teeth, the differentiation between cysts may not be possible nor necessary.

Generally speaking, recurrences may be manifest by the production of more cysts, but usually the development of solid tumor tissue made up of fibrous tissue alone or associated with giant cells, makes up the recurrence. Nor is this all. The recurrences may be combined with adamantine structures and the development of these elements may result in limitless extension. These unfavorable results in my experience are confined to tumors situated in the upper jaw.

Pathology. When discovered by the x-ray they present ovoid cysts attached somewhere to a tooth. These, when examined with the curet, produce a membrane. Teeth in a mass or less complete formation may be recovered from the larger cysts.

In larger cysts, the bone over them may be expanded and correspondingly thinned. When the associated tooth is removed and the cavity curetted, ordinarily a simple membrane is recovered but it does not separate as readily as in the dentigerous cysts. Failure to remove all the lining epithelium may result in a permanent discharging sinus or a recurrent tumor. This is to be anticipated if the cyst wall is thicker in some areas than in others or if the bone is invaded. In such cases, the cyst may have extended beyond the cyst cavity and a tumor lying within the antrum may be overlooked, just as happens more commonly in adamantine tumors.

Histology. The lining cells may be cuboidal or high or stratified. There may be other tissue: fibrous, myxoid, and even adamantine areas, thus proving that there is no real dividing line between the two.

ODONTOMAS

Odontomas are closely allied to dental root cysts, in fact may complicate them, but represent a more mature tissue development, though

they vary much in this respect. A single displaced but well-formed tooth represents the slightest deviation from normal. When situated in the lower jaw at or near the site of a missing tooth, it may be regarded as an anomaly of displacement rather than a tumor formation. Almost as much may be said when there is a conglomerate of teeth more or less fully formed. On the other hand, when the tooth misplacement is a part of a cystic process, particularly if there are small fragmentary or illy-formed teeth in the wall of the cyst, the matter assumes a more serious aspect. Lesions located in the upper jaw are much more serious than those of the lower jaw; this fact cannot be too often repeated.

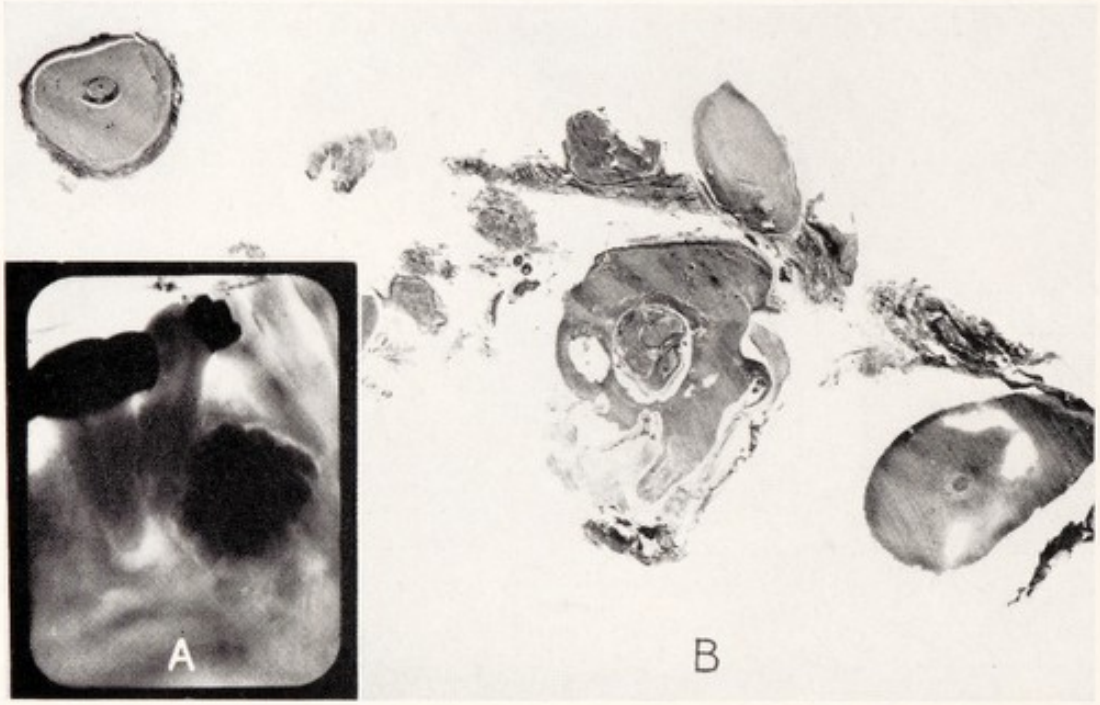

FIG. 150. Odontoma: *A*, x-ray findings showing a conglomeration replacing a tooth; *B*, cross-section of some of the small elements making up the mass.

Pathogenesis. Odontomas are usually small and made up of the enamel of teeth. When they are formed by a conglomerate of teeth, they seldom form a mass larger than a hickory nut and are bosselated. Some contain within their substance a well-formed tooth, or a near perfect tooth may be attached to them, but besides one or more well-formed teeth there may be a mass of material not recognizable in the gross as being of dental origin. They are usually situated at the alveolar border of the lower jaw but may be found in the upper jaw, particularly in the region of the antrum.

Obviously, odontomas are formed by displaced embryonal rests. Usually some dental disturbance of a minor nature leads to their dis-

covery. They may be found at the site of a missing tooth or a supernumerary tooth (Fig. 150). Sometimes, when they are situated about a wisdom tooth, they cause inflammation and fixation of the jaws.

Odontomas are formed usually in relatively young persons and those located in the most common site—that of the wisdom teeth—are most

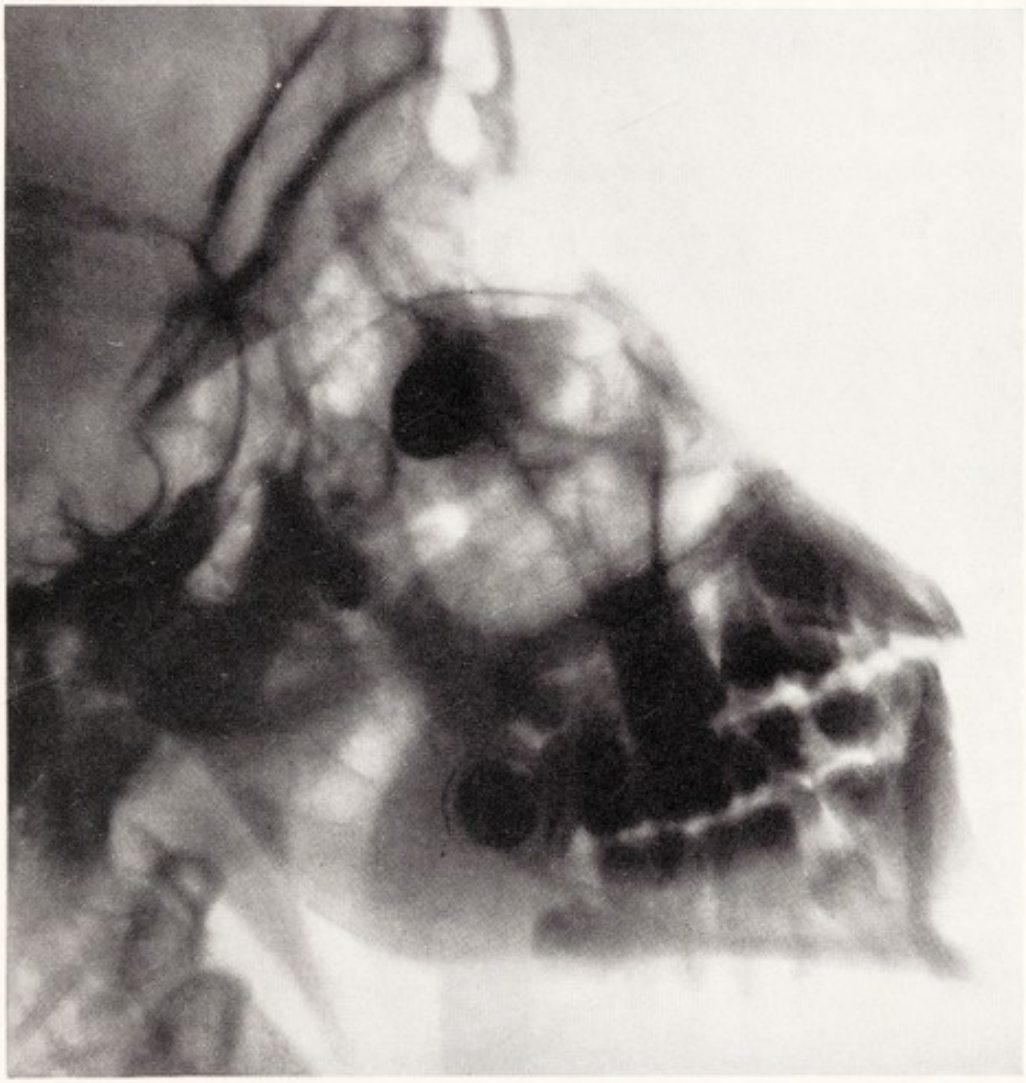

FIG. 151. Odontoma below the orbital plate. This patient was operated on for a root cyst in the lower jaw. A tumor developed in the region of the orbit nine years after the removal of the root cyst, apparently independent.

apt to cause disturbance at the usual time for the eruption of these teeth.

Not all odontomas are located at the site of normal tooth development. Curiously enough they are sometimes located below the orbital plate (Fig. 151).

No matter where situated odontomas may suppurate spontaneously or infection may be caused by unsuccessful attempts at their removal.

In such cases, discharging sinuses may persist. Complete removal usually ends the matter but in the upper jaw the complications may be more serious, even to the development of a malignant tumor. In widely-scattered teeth secondary complications should be thought of.

Pathology. A dense thickening of the jaws represents the physical findings which point to the nature of the trouble. This is confirmed by x-ray examination. Small tumors are sometimes accidental x-ray findings. Dense lobulated shadows displacing a tooth may be easily recognized.

After removal the tissue varies greatly. A single tooth or several teeth represents the usual findings. In less well-formed cases there may be much fibrous tissue in which are imbedded small, hard particles representing pearls.

Histology. Dentine and enamel may be recognized easily enough in decalcified specimens (*B*, Fig. 150) but one only of these tissues may be present. There may be much fibrous tissue made up of large spindle cells surrounding the teeth simulating a fibroma.

ADAMANTINOMAS

These are the most important of the developmental rest tumors. They have their origin in the epithelium of tooth anlages, and it is said, too, that they may develop from the epithelium of the mouth, independently of teeth. They are said to be the most common in the lower jaw but this has not been my experience. The reason for this discrepancy is possibly due to the fact that most of my patients have been previously operated on and tumors of the upper jaw are much more likely to re-form. They are supposed to be most common in mid-life but are by no means rare in adolescence, and there is reason to believe that many begin their development at the time of puberty, though their presence is not detected until several years later. Slow growing as they are, a tumor the size of a lemon or orange must certainly have had its beginning at a considerably earlier date.

In the clinical scheme they have much in common with the mixed tumor of the parotid, both as to rate of growth, their structural complexity, and their ultimate ending in mildly malignant growths. If we knew just a little less of their origin "mixed tumor" would not be a bad name for them.

In structure they are made up of epithelial-like cells, fibrous tissue in which such cells are situated, fibrous tissue made up largely of spindle cells, or pure fibrous tissue. Islands of giant cell tumor tissue and even squamous cell areas are occasionally encountered. They are mixed,

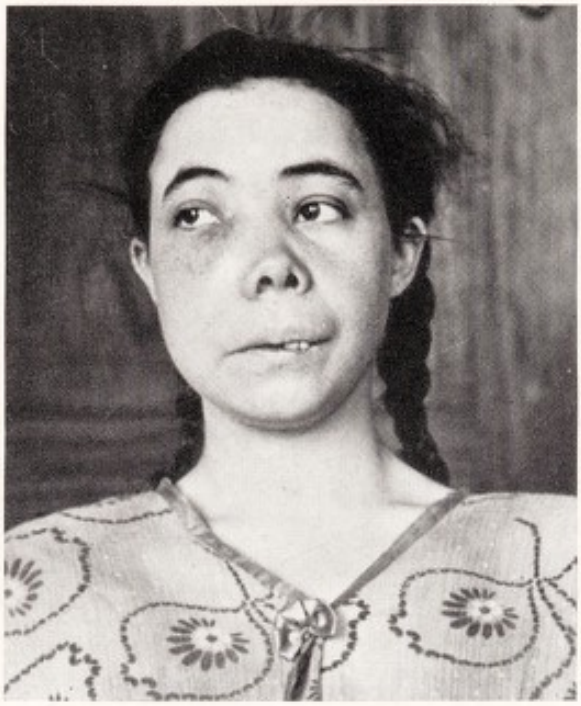

FIG. 152. Adamantinoma. F., age 21. Tumor of jaw six years. The nose was deformed and nasal passage closed. Well 13 years after operation.

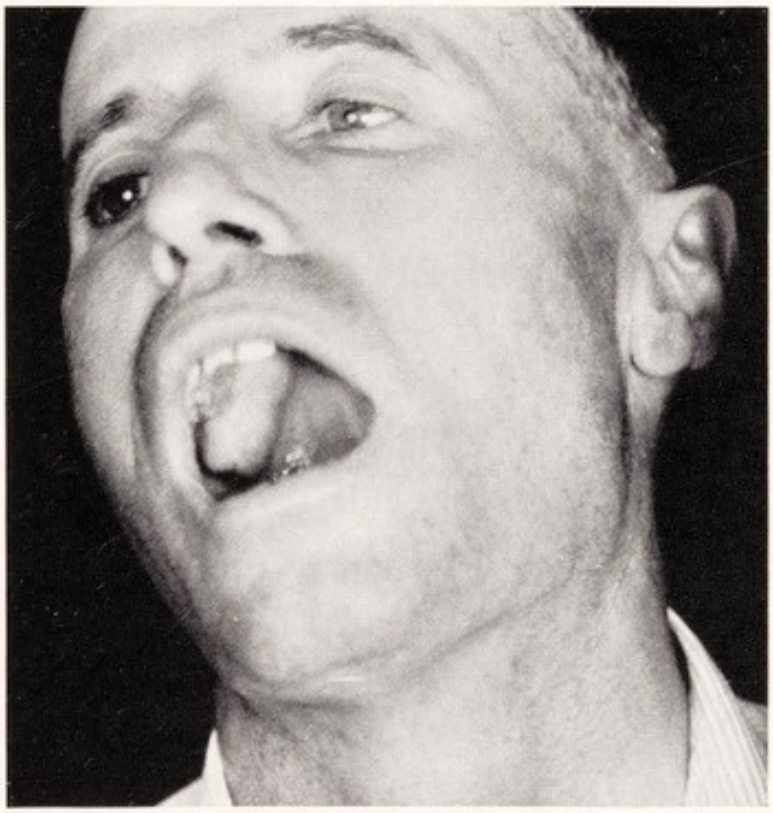

FIG. 153. Adamantinoma. A smooth hard tumor protruded from the alveolar process. The antrum was expanded.

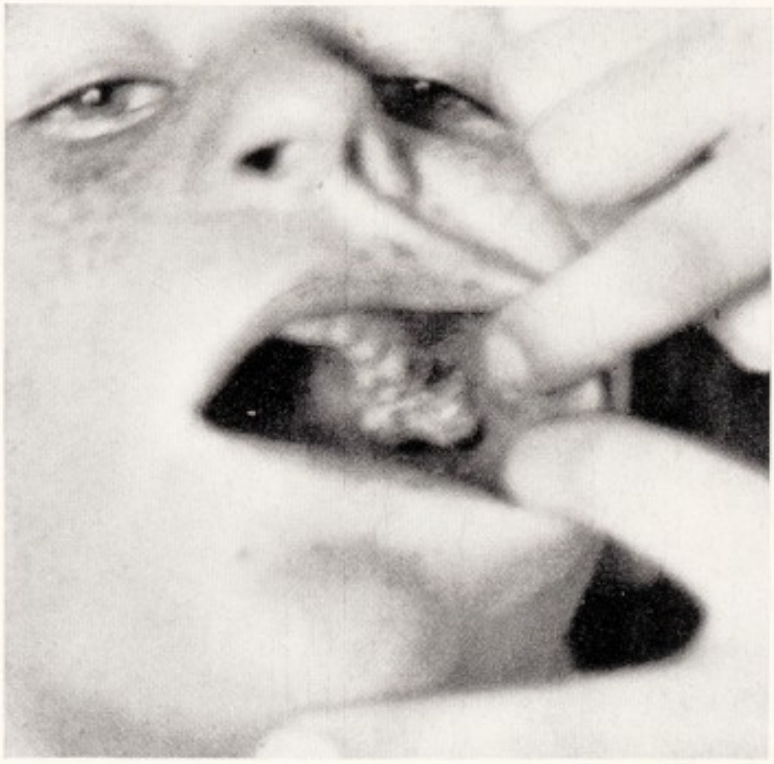

FIG. 154. Adamantine tumor displacing a tooth medially. The top was an epulis. The more serious disease lay in the antrum.

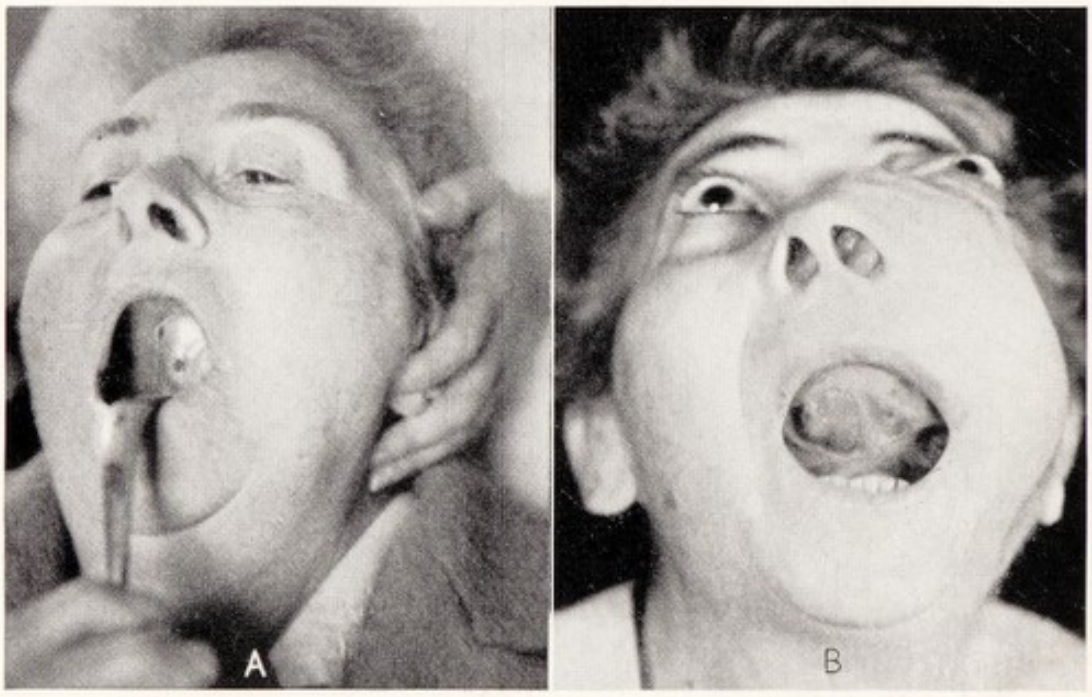

FIG. 155. Adamantinoma of the upper jaw. F., age 63. *A*, When first examined presented half egg shaped tumor of the palate of 25 years' duration. The bone was somewhat eroded. *B*, The patient returned seven years later, seeing double, also red.

indeed, and one cannot know the structure of a given specimen until the whole of it has been examined. It is customary to classify them as adamantinomas when such tissues are present, no matter what other tissues may complicate them.

Pathogenesis. Somewhere about the anlages of teeth the tumor finds its start. Usually they are not discovered until they cause expansion of the bone (Fig. 152). Sometimes a smooth mass protrudes about the alveolar border as the first sign of the disease (Fig. 153). Such protrusions, though small, may cause malposition of the tooth (Fig. 154). When

FIG. 156. Adamantinoma of upper jaw. F., age 75. Twenty years previous had a tooth pulled and a bean-sized tumor developed. It gradually grew until a nodular tumor occupied the alveolar border and half the hard palate.

this is the case it is certain evidence that the important part of the growth lies in the antrum. Though a slide obtained from the protruding part may show nothing but giant cell tissue, if the tooth is displaced, one may be sure that the important part of the tumor lies more deeply and most likely will show adamantinomatous structure. If the antral invasion be overlooked (*A*, Fig. 155), the oversight will be emphasized by the continued growth of the overlooked part in the antrum (*B*, Fig. 155). The same may be true if a mixed tumor be the primary lesion. These may not be sharply differentiated from the mixed tumors of the hard palate. Sometimes only an extended microscopic study may differentiate them, and then not too certainly at that.

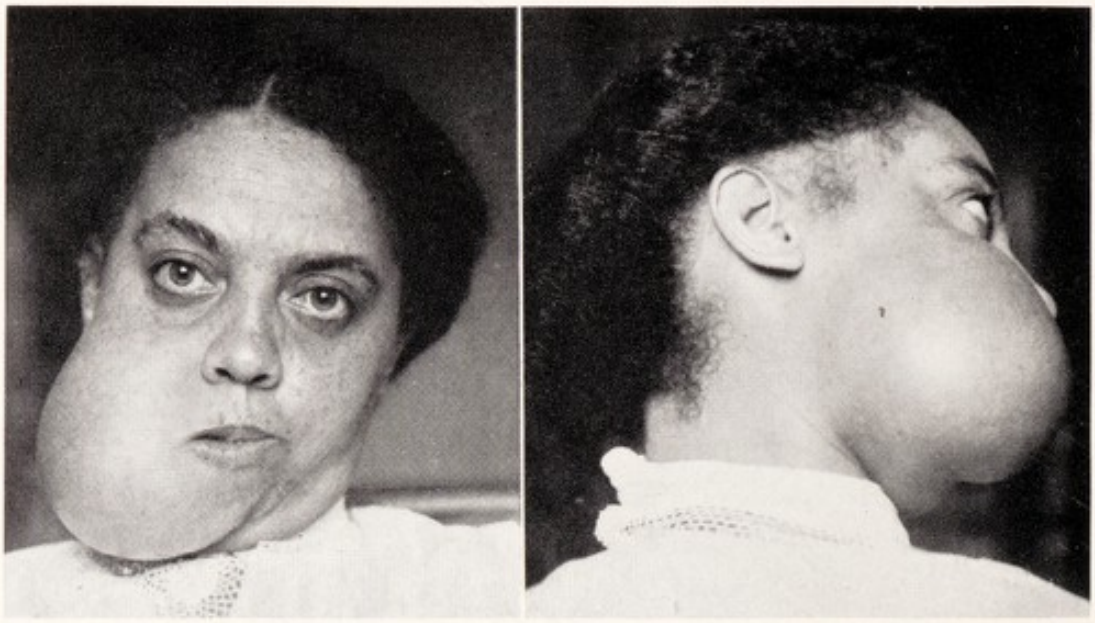

FIG. 157. Adamantinoma. F., age 17. Jaw has been expanding for five years. No disturbance except that due to the deformity. This tumor was removed under the diagnosis of sarcoma.

Not uncommonly the patient tolerates the growth for many years, in one of my cases for 20 years (Fig. 156). Often such long negligence is the result of professional advice. The deformity caused by such neglect

FIG. 158. Adamantinoma. Boy age 15. Jaw began to enlarge three years before, following a blow from a baseball bat. Jaw resection. Lost track of three years later. The specimen from this case reposed on my shelves for a number of years as an osteosarcoma before repeated examinations discovered adamantine tissue.

may be shocking, but the growth may be so slow that little deformity is produced. In rare cases, trauma seems to stimulate their growth.

When the lower jaw is the site of the adamantinoma the growth usually remains small. However, a large expanding growth may be pro-

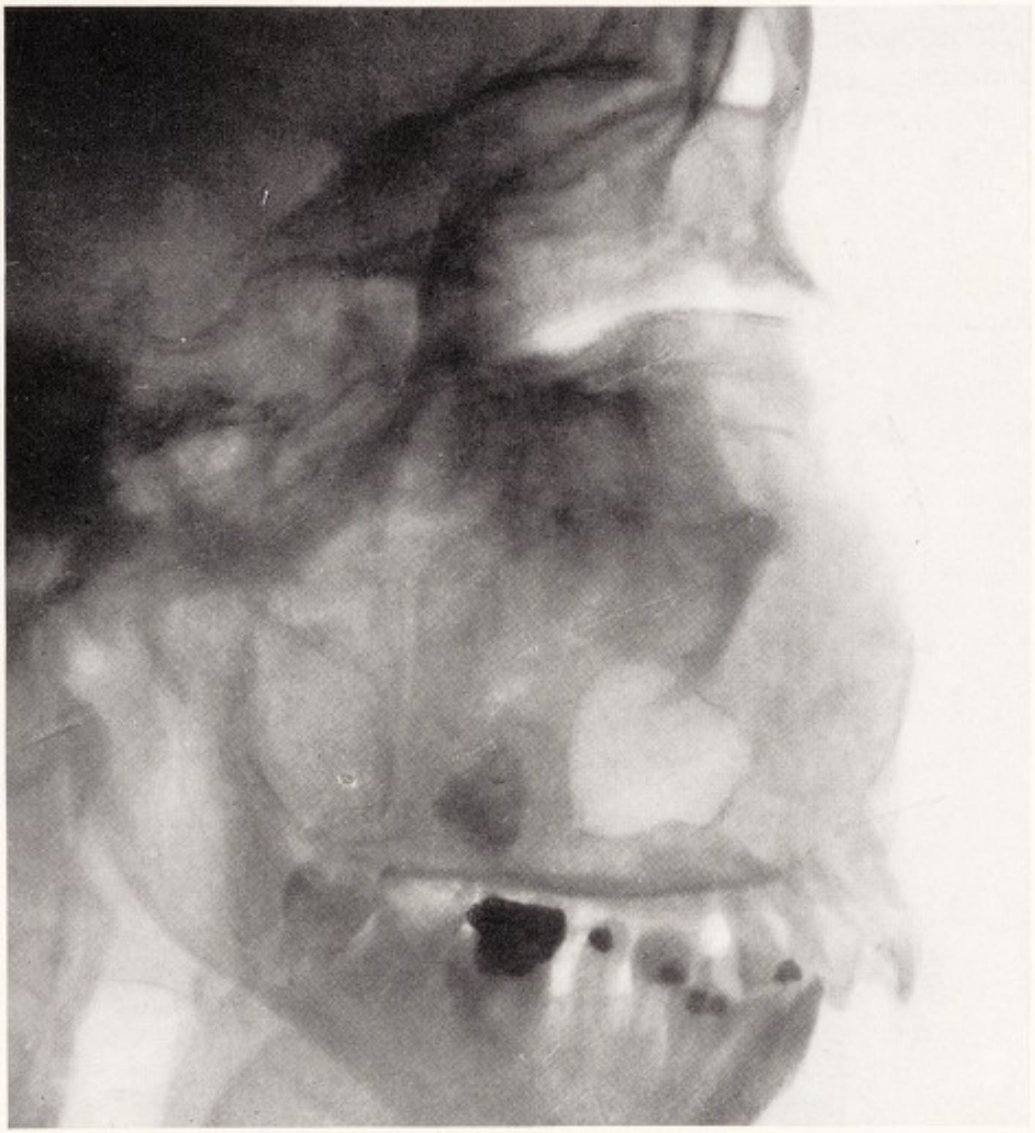

FIG. 159. Adamantinoma. The plate shows a well circumscribed tumor with almost complete destruction of bone but without evidence of bone proliferation.

duced (Fig. 157) limiting by its size the movements of the jaw. Most cases of lower jaw tumors remain small for many years.

The history may be confusing. A trauma may call attention to the presence of a tumor. If rapidly growing, a sarcoma may be thought of (Fig. 158).

The tumors gradually expand and may completely destroy the bone but they do not excite bone proliferation. In their growth in adolescents,

they may cause enormous growth deformity as well as bone expansion (Fig. 159). As a result of this, the conservative removal of the tumor may leave pronounced deformities. However, in such cases the removal of the entire upper jaw, preserving the orbital plate, allows the prosthetic dentist to achieve better cosmetic results than conservative surgery.

When long neglected, as the tumors grow they expand the bones and then may penetrate them, causing invasion of the orbit and even the cranial cavity. Invasion is most likely to follow incomplete removal.

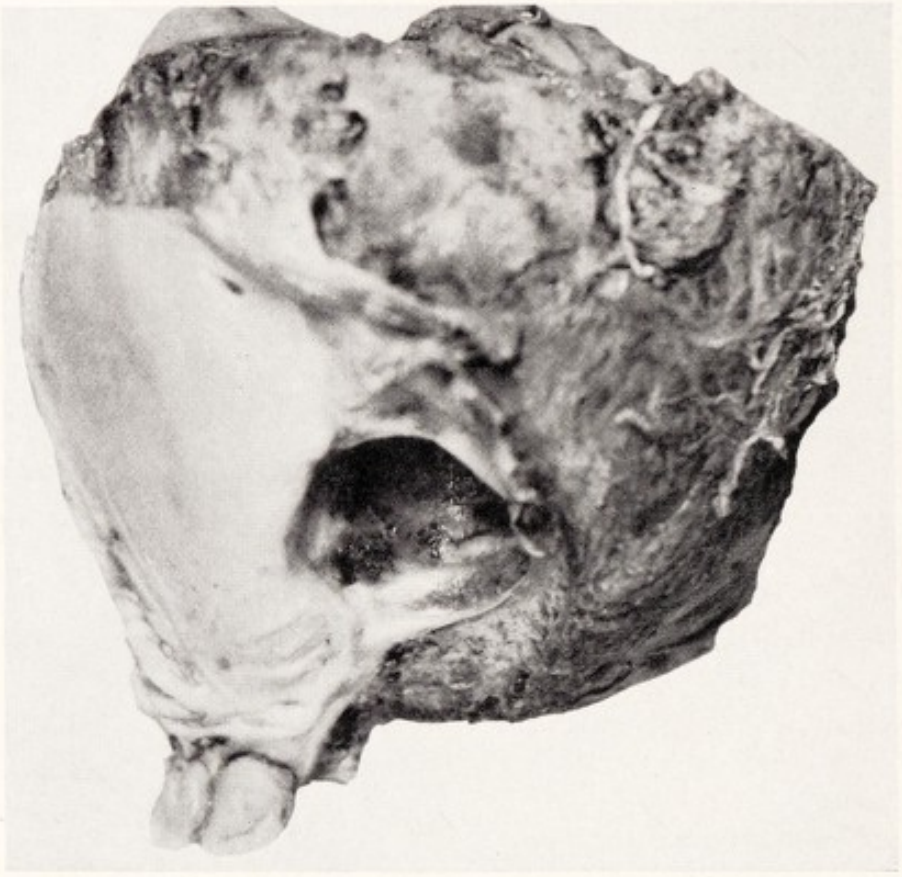

FIG. 160. Adamantinoma of the upper jaw. The growth expanded the antrum but nowhere invaded it.

Naturally the bone farthest away from the site of origin is preserved longest. The result is that the orbital plate is seldom destroyed.

Curettage and cautery cure many of these cases. Recurrences often are again benign and may be controlled by renewed operation, if the patient returns for further operation before irreparable damage is done. Usually the patient does not return, either because the recurrence is not noted, or because of discouragement with the result of the previous operation. Practical considerations, in many classes of society, at least, make it imperative that the surgeon secure all the affected tissue at the first operation.

In the lower jaw the problem is different. If the continuity of the jaw is interrupted, the prosthetic dentist is helpless. Furthermore, recurrences are more quickly recognized and there is less likelihood of extension into regions where it cannot be reached. Conservatism here, therefore, may be pursued to the limit. I have not resected a lower jaw in the past twenty years. Even though the bone is much expanded and defined, bone ridges can be preserved which will retain the contour of the jaw. In adolescents, the outline will improve as development of the patient progresses.

Pathology. Expanding bone is the first sign; the x-ray confirms this finding. Examination seldom goes further than this. The slow growth and the youth of the patient are sufficient to assure a reasonably sure diagnosis. Expanding bone, beyond the range of cysts, just about com-

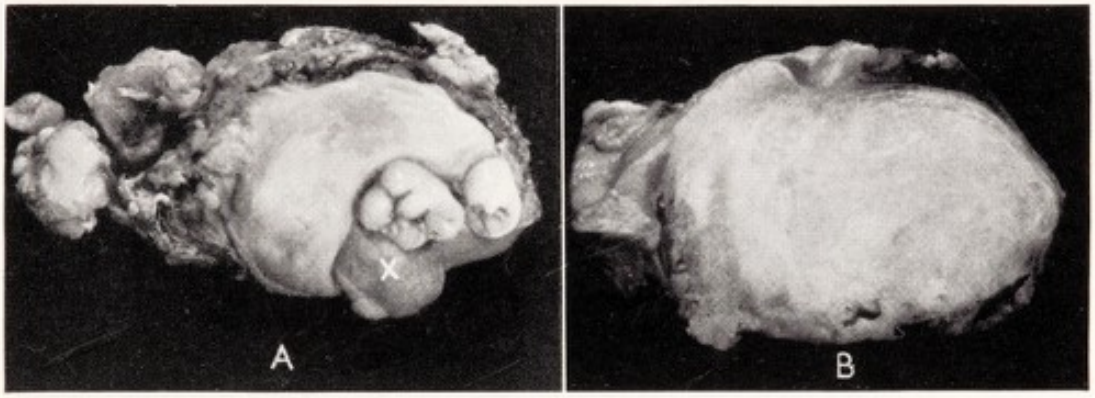

FIG. 161. "Epulis," *x*, about a molar tooth. Adamantinoma above. *A*, Partial resection of jaw. *B*, Tumor in cross-section.

pletes the picture. If the bone has not been perforated, it is possible to secure the complete growth (Fig. 160). If the deformity of the bone be not too great, a conservative operation may be possible. If the deformity be great, a complete resection may be preferable. Prosthetic dentists have achieved a skill which produces better results than conservative surgery. Patients, who so little regard their looks as to allow such large tumors to form, will hardly be much disturbed by the results the surgeon achieves. Such patients usually are not able financially to command the services of such dentists. Therefore, one must consider the esthetic and economic status of the patient before deciding on the procedure to be adopted. The ignorant, too, it must be remembered may neglect the care of their prosthetic apparatus. Charitable organizations may secure the apparatus but they cannot undertake the subsequent care.

In many cases a less radical operation secures the whole growth, but one must be sure that he is removing the whole growth. When there is a

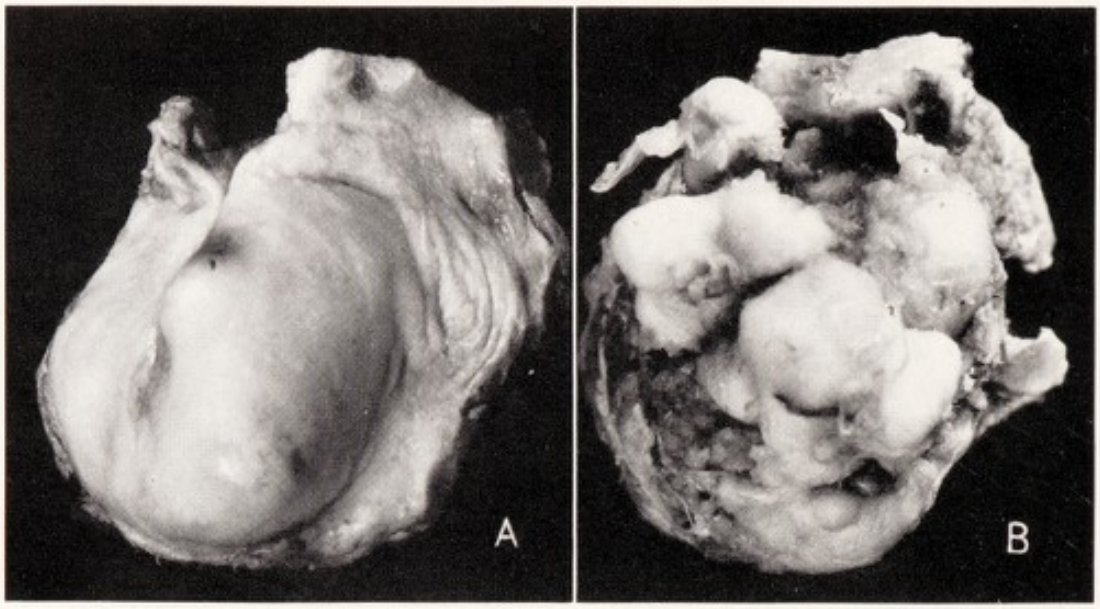

FIG. 162. Adamantinoma: *A*, rounded mass protruding from the alveolar process; *B*, part projecting into the antrum.

protrusion about a tooth (Fig. 161), the chief part of the growth extends into the antrum. The removal of the protruding part hardly would be conservative surgery. Hard rounded tumors of the alveolar border, extending over the hard palate, may be mistaken for mixed tumors and the extension into the antrum be overlooked (Fig. 162). Such tumors may

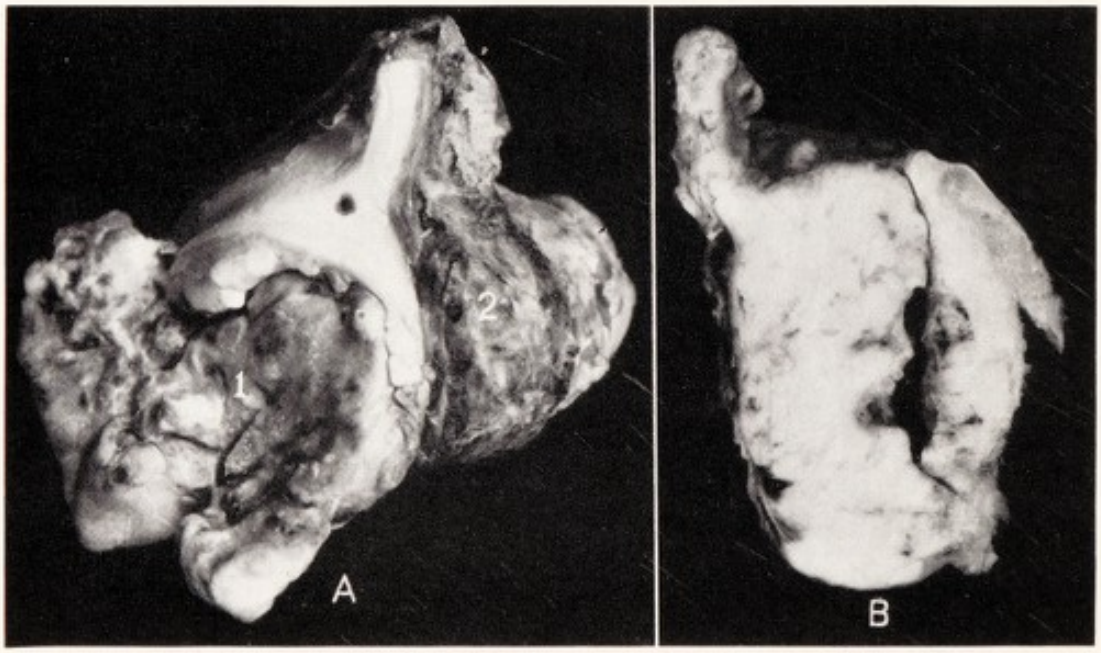

FIG. 163. Adamantinoma protruding through the hard palate. *A*, The mass at the left (*1*) protruded into the mouth and the rounded mass at the right (*2*) extended into the antrum. *B*, Cross-section of same.

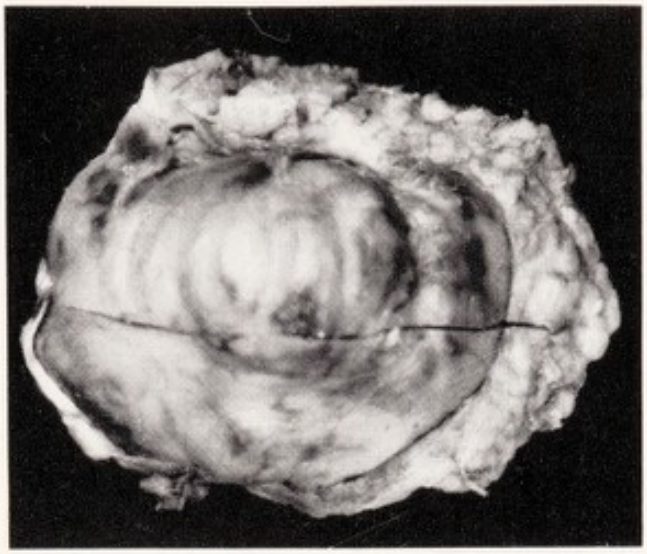

FIG. 164. Adamantinoma of the palate. Removal followed by rapid growth in antrum.

require the removal of the whole of the alveolar process. Cases that have been incompletely operated on may produce dense fungating growths, which are lobulated and do not bleed easily, thus distinguishing them from carcinoma (Fig. 163) even though a biopsy may not.

FIG. 165. Adamantinoma. M., age 83. Had abscessed tooth removed which continued to drain and a tumor developed. The mass is broken down and in part abscessed, forming numerous cavities.

Even when the growth occupies the hard palate away from the alveolar process, there may be an extension into the antrum (Fig. 164). These are likely to be mistaken for mixed tumors of the palate. In fact, the first operation may fail to secure tissues showing adamantine structure. When the tumor mass is a solid pinkish growth without cysts, considerable degree of malignancy must be suspected, a thing usually foreign to the tumors of the palate.

Tumors may become infected after the removal of teeth or incom-

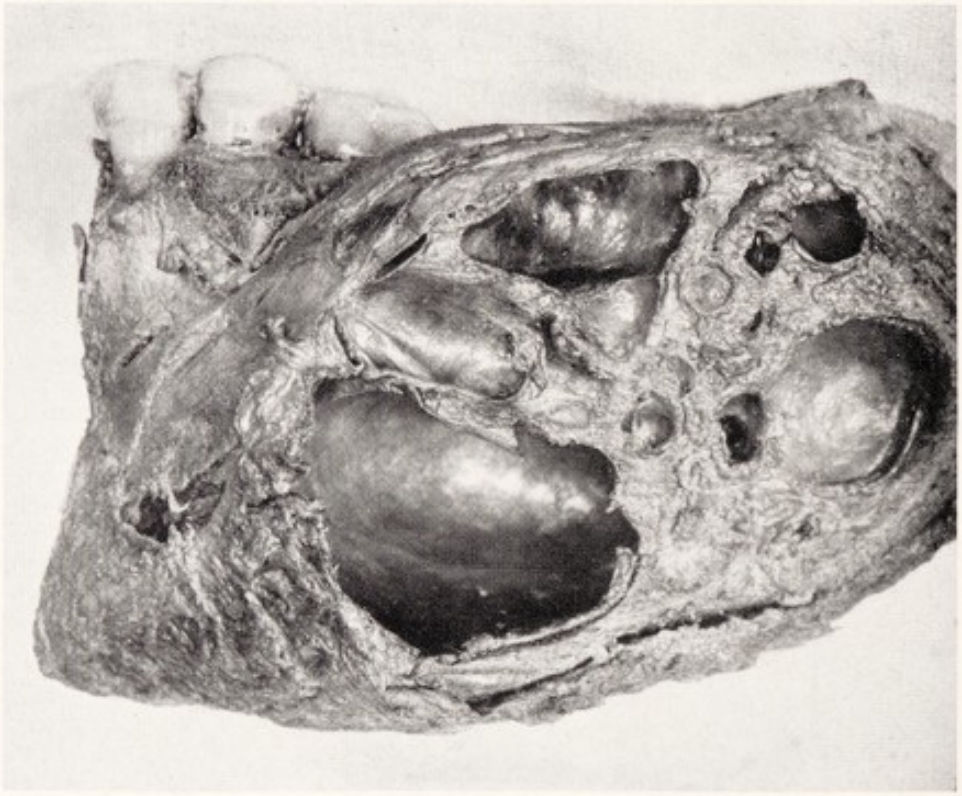

FIG. 166. Adamantinoma. F., age 17. Cystic adamantinoma occupying the whole lower jaw, seemingly making conservative operation impossible, thirty years ago. The bulk of the tumor formed by smooth-lined cavities; specimen removed thirty years ago; results not known.

plete operations, resulting in suppurating cavities, regarded as due to necrosis and the tumor being overlooked (Fig. 165).

On section a variable picture is seen. The tumor may be entirely solid or it may be made up in part of cysts (Fig. 166). The cysts may be of all sizes from that of a pinhead to as large as a walnut. The cyst walls may be smooth, papillary, or divided by septa. The solid portion is usually reddish brown but may be grayish. The septa between the cysts are whitish or bluish.

Once the tumors escape the bone, the extent of the invasion cannot

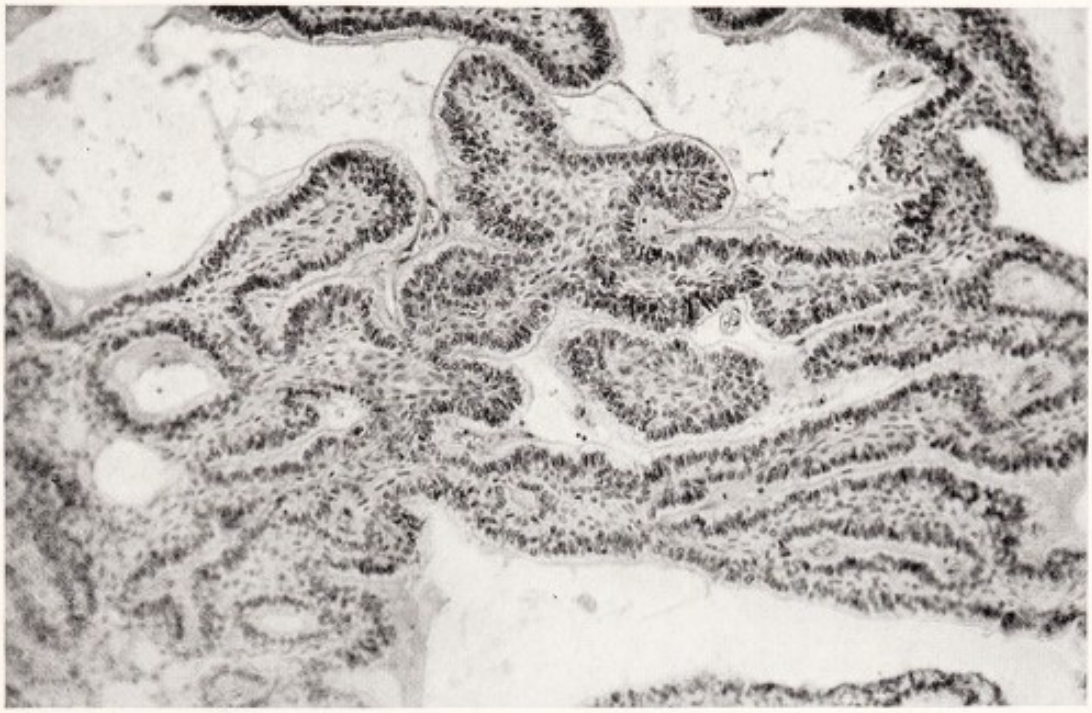

FIG. 167. Adamantine tumor. The cells are arranged in long parallel columns with but little vessel-bearing connective tissue between. The cells are regularly arranged and for the most part cuboidal. Cystic spaces intervene. The connective tissue is distinctly acidophilic like that found in benign papillomas.

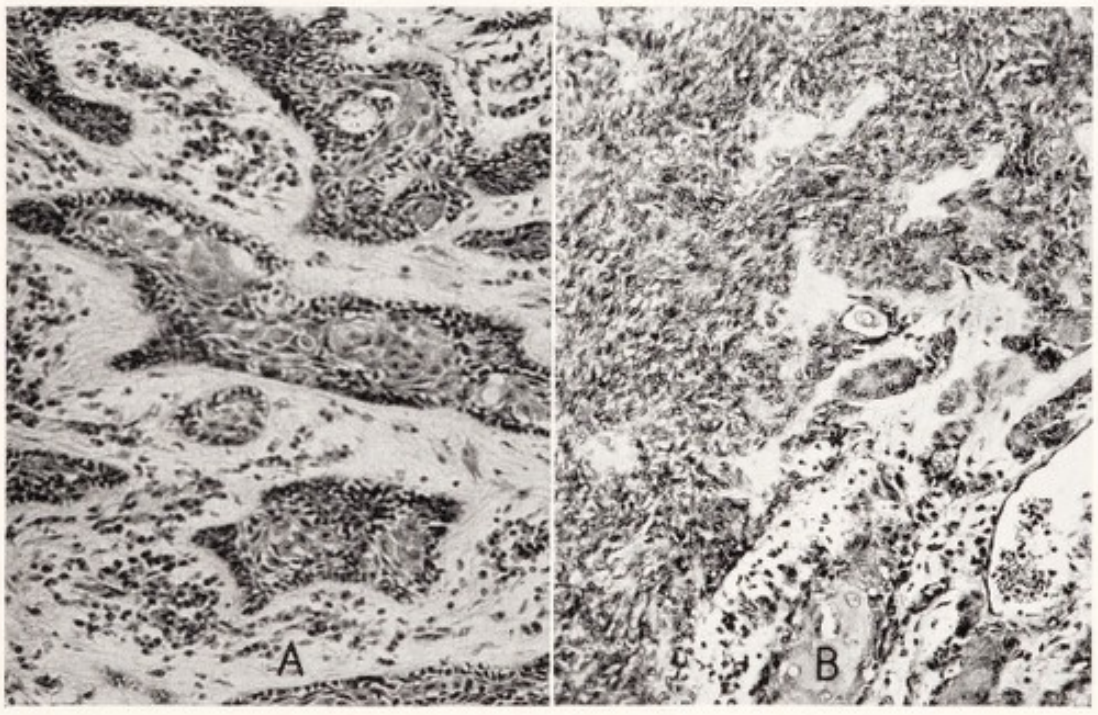

FIG. 168. Advancing adamantinoma: *A*, the cells retain in a measure the column arrangement but there is marked invasion into the adjacent tissue; *B*, all column arrangement is lost and the cells are spindleform. They are more or less diffusely arranged and without alveolar formation. The intercellular connective tissue remains stainless.

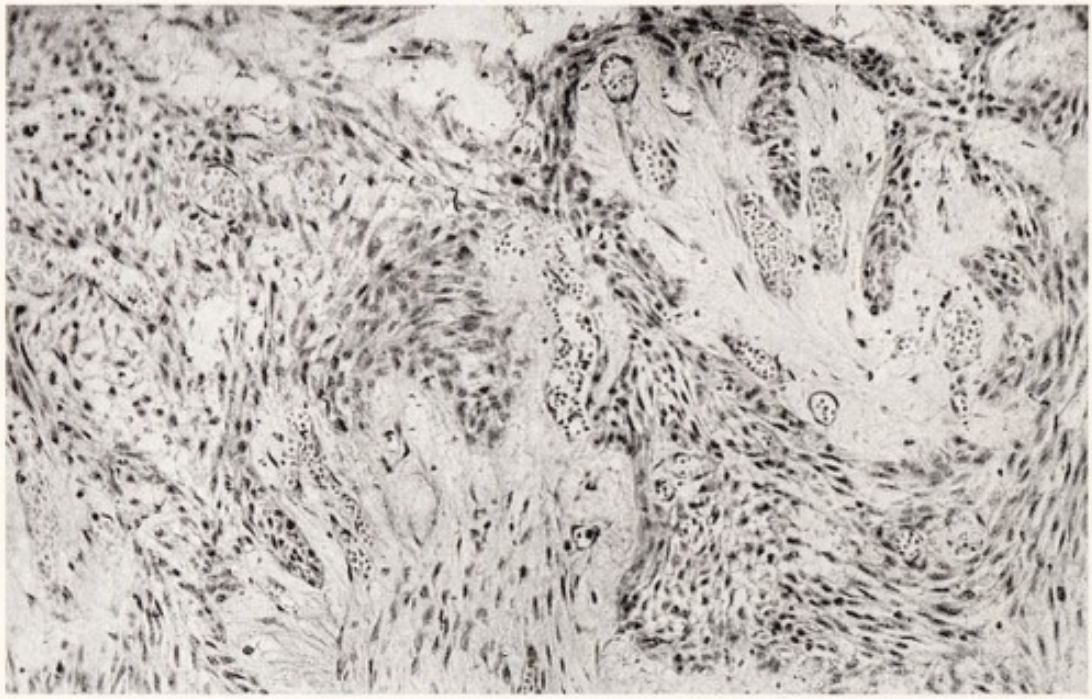

FIG. 169. Adamantinoma of the upper jaw: areas of spindle cell tissue found between areas of more typical adamantine tissue present in other parts of the tumor. The cells are spindleform, varying in size and stainability, the vessels numerous and thin-walled. Some areas are definitely fibrous tissue with but few spindleform cells.

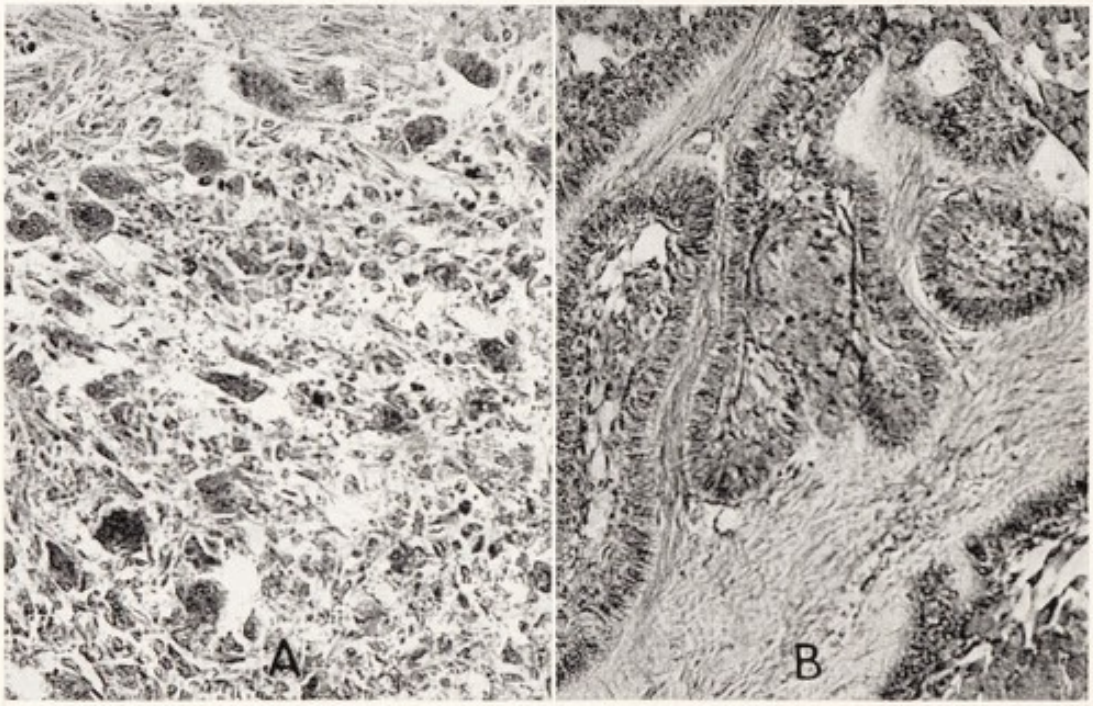

FIG. 170. Adamantinoma of the upper jaw: *A*, typical epulis structure, fibrous tissue and giant cells, staining badly because a diagnostic section had been removed; *B*, typical adamantinoma. The epithelial columns are well preserved, the intervening fibrous tissue stains well without evidence of proliferation.

be predicted. In one of my patients the optic chiasm was destroyed. Invasion of the orbit with exophthalmos is more common. Such pictures tend to discourage overconservatism, particularly in patients who are not likely to present themselves for after-study.

Histology. The ground picture of adamantinomas is made up of long columns of epithelial-like cells with cores of sparse connective tissue. I say epithelial-like advisedly because they lack the invasive potential which they would have if they were truly epithelial. The columns are

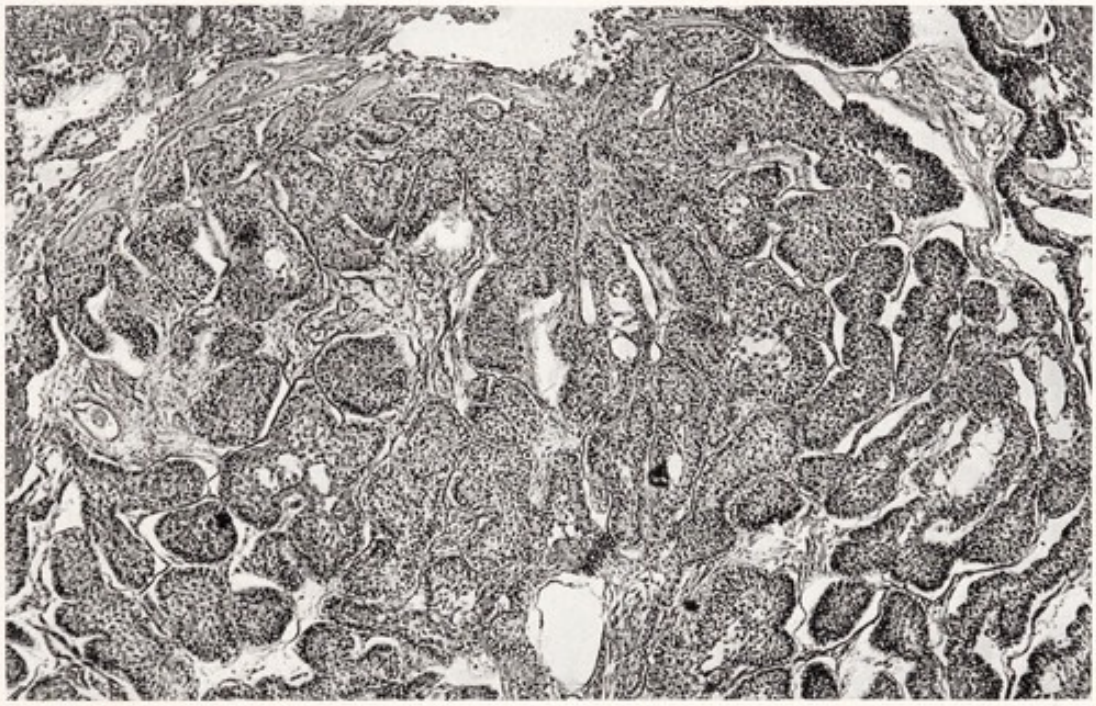

FIG. 171. Adamantinoma of the lower jaw. The cells form solid columns, the nuclei are variable in size and the cell protoplasm somewhat suffused. The connective tissue is sparse in amount and the vessel walls are well formed. These tumors are devoid of any round cell proliferation nor is there evidence of cell division.

stratified cuboidal or columnar (Fig. 167). These may be interspersed with cysts of various sizes filled with débris of degenerated cell columns.

In the lowest degree of deviation from this most innocent stage in which the cells are arrayed in solid columns, the columnar form is lost to a greater or lesser degree (Fig. 168). This is the structure which is found in tumors growing rapidly but is not invading.

Few tumors are made up wholly of such structures. Hard by epithelial columns, masses of fibrous tissue may be found. This connective tissue varies in amount and cellularity. It may make up the bulk of the tumor and the cells may suggest sarcoma (Fig. 169). Bone may be found in such fibrous areas.

The summit of the tumor in many cases is typical epulis (*A*, Fig. 170), while beneath is typical adamantinoma (*B*, Fig. 170). The protruding part of the tumor may be made up of typical epithelioma, the adamantinomatous part lying more deeply. On the contrary the protruding part may be myxoid or fibrous or a combination of these.

In some tumors, particularly those found in the lower jaw, the cells are formed in definite alveoli but they are surrounded by well-staining connective tissue and there are no round cells. In harmony with this structure, such areas are clinically benign (Fig. 171).

If we are to escape hopeless confusion we must regard as adamantinomas all tumors which contain adamantine tissue no matter what the associated structures may be. The clinical course is the same no matter what the admixture.

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CHAPTER IX

Diseases of the Palate

TUMORS of the palate have received but scant attention in the literature. The reason for this is obscure, for certainly they are common enough, and a misdiagnosis is often unpleasant for both patient and surgeon. Those that I have seen in consultation have rarely been correctly diagnosed.

In considering the diseases of the palate those affections which encroach on this region secondarily, notably those of dentigerous origin or invading it from adjacent cavities, have been treated in other chapters and may be ignored here. Of the affections native to the palate mixed tumors are the most important. Angiomas are by no means rare, and may be the most annoying of the lot. Inflammations may be ignored since they are but extensions from the alveolar border. Ulcerations, notably syphilitic, I have seen but one and tuberculosis none. Cancer is rare, but important when encountered.

Listed they appear as follows:

I. Angiomas
II. Exostoses and Ecchondroses
III. Plasmomas
IV. Mixed Tumors
V. Fibromas
VI. Carcinomas

These may be considered in turn, keeping in mind that likely they are all dependent on a congenital displacement.

ANGIOMAS

Angiomas of the palate are not uncommon. On the soft palate they may be diffuse and may be associated with a like condition of the cheek. Of chief interest are the small ones situated in the midline of the hard palate.

Pathogenesis. These may be only millimeters in diameter, but may be a centimeter or two. No matter what their size, because of their abundant blood supply from the underlying bone, they may bleed persistently, aggravatingly, even alarmingly, if injured, or from spontaneous degeneration. Angiomas may be discovered at any age, but are most

common in midlife and in women. They may cause no apprehension because of their presence until they bleed. Only rarely do they become large enough to annoy the patient because of their size.

Because of the passage of the veins through the hard palate, if cauterized when the destroyed tissue is sloughed off, the vessel remains open and persistent bleeding may result. Therefore, the prudent surgeon will coagulate the blood in the vessels rather than destroy them. If injured, they may bleed persistently hours without end. The loss of blood may

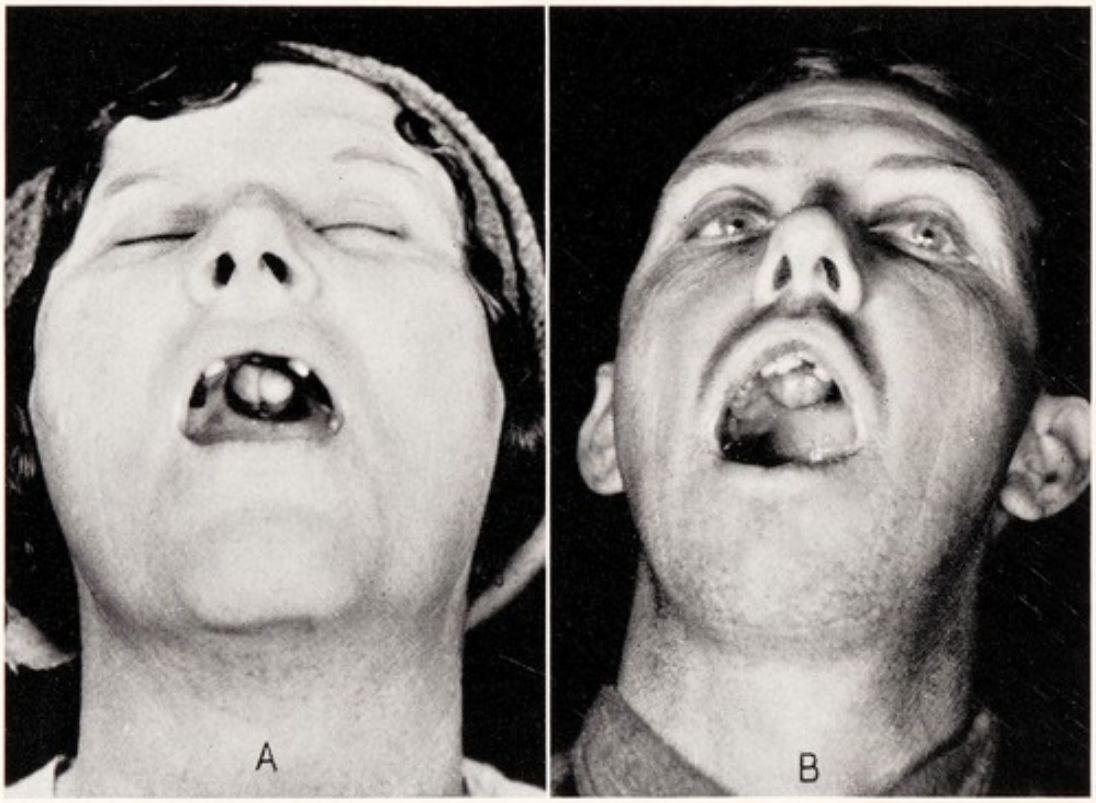

FIG. 172. Exostosis and ecchondrosis of the palate. *A*, Exostosis. Duration 28 years. It is bifid, very dense. *B*, Ecchondrosis. The patient has known of its presence three years. The surface is somewhat bosselated.

become serious unless someone is thoughtful enough to exert pressure over the bleeding point.

Pathology. The areas are circular, situated in the midline, elevated several millimeters above the surrounding surface of the hard palate. They are soft to the touch, compressible, and of a deep red color.

Histology. The structure is that of simple cavernous angioma.

EXOSTOSES AND ECCHONDROSES

These tumors may be formed of pure bone or cartilage; no doubt they belong to the congenital tumors.

Pathogenesis. The patient finds out one day that he has a tumor on his hard palate. Some patients on the contrary are conscious of their existence for many years. These tumors may be astonishingly large when first discovered. They are situated exactly in the midline, grow very slowly, and cause no trouble except, that once discovered, the patient is conscious of their presence. I know of a tumor which has remained the size of a half hazelnut for several decades. It takes many years to gain any considerable size (*A*, Fig. 172). Sometimes they grow more rapidly (*B*, Fig. 172). These are likely to be cartilaginous.

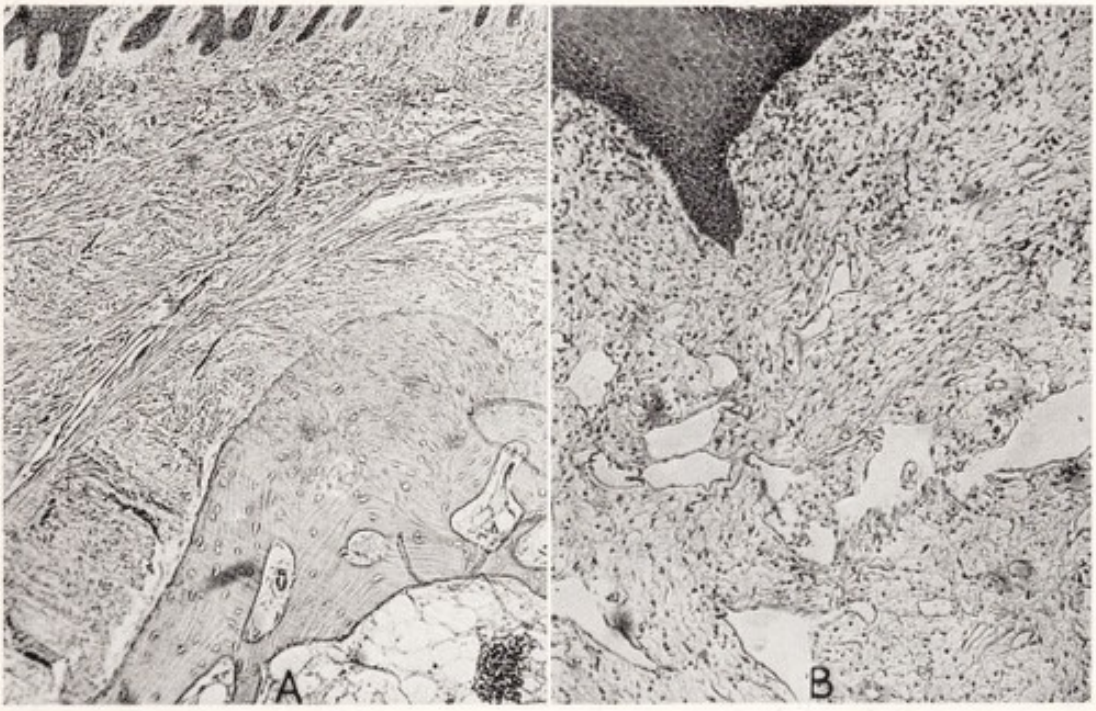

FIG. 173. Exostoses of the hard palate: *A*, stratified epithelium above, beneath which is a layer of connective tissue which covers a base of typical bony structure; *B*, area of myxoid tissue containing many thin-walled vessels. The cells are mostly spindleform, the nuclei deeply staining.

Pathology. The appearance of the tumors is characteristic. One need but feel their smooth hard surface to be convinced of their nature. In some cases softer myxomatous areas are present without apparently changing the nature of the tumor.

Histology. These tumors are covered with an intact epithelium below which may be the bone or cartilage, but usually there is a considerable layer of connective tissue between (*A*, Fig. 173). The cartilage tumors differ only, that instead of bone, they are made up of cartilage. Some tumors show typical myxoid structure in some areas (*B*, Fig. 173), while the remainder of the tumor is of denser structure.

PLASMOMAS

These little tumors are unimportant, except because of their cellularity they may confuse the laboratory man.

Pathogenesis. Like their bony relatives these tumors are discovered fully formed and from that time they remain unchanged, so the story goes. They lie in the midline (Fig. 174), are always small, and seem to remain stationary. However, in rare instances, they grow rapidly and form metastases.

Pathology. The tumors are smooth, round and dense, but not of bony

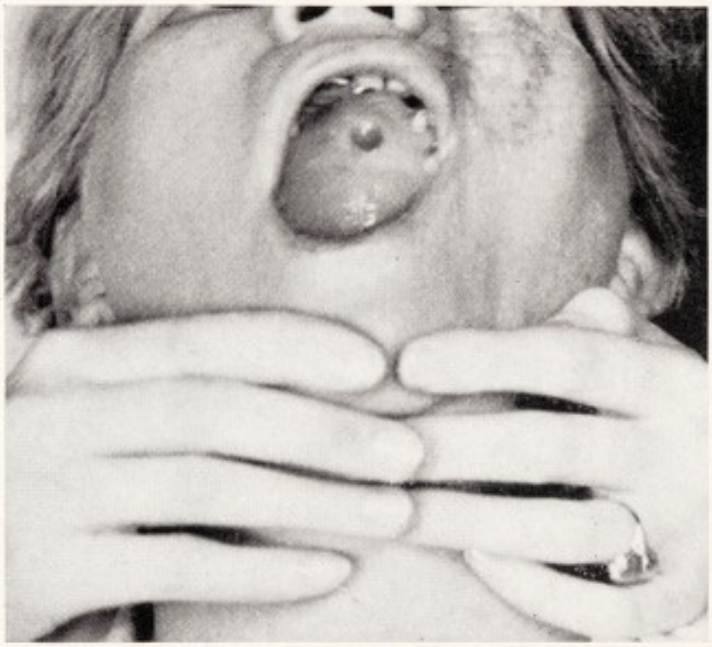

FIG. 174. Plasma cell tumor of the hard palate. A smooth round tumor the size of half a large pea and about the color of one.

hardness. They are of a pale bluish color which distinguishes them from the bony lesions.

Histology. The interest in these tumors is because of the rather surprising makeup of them (Fig. 175). They are composed of a cell mass with but little connective tissue between them. In rare cases when they undergo malignant change the simple structure is lost; they become less cellular, the cells stain less uniformly, the vessels more numerous, and the walls thinner (Fig. 176).

MIXED TUMORS

These tumors present a varying picture. They commonly show bony and cartilaginous areas, but the greater part of the tumor is made up of

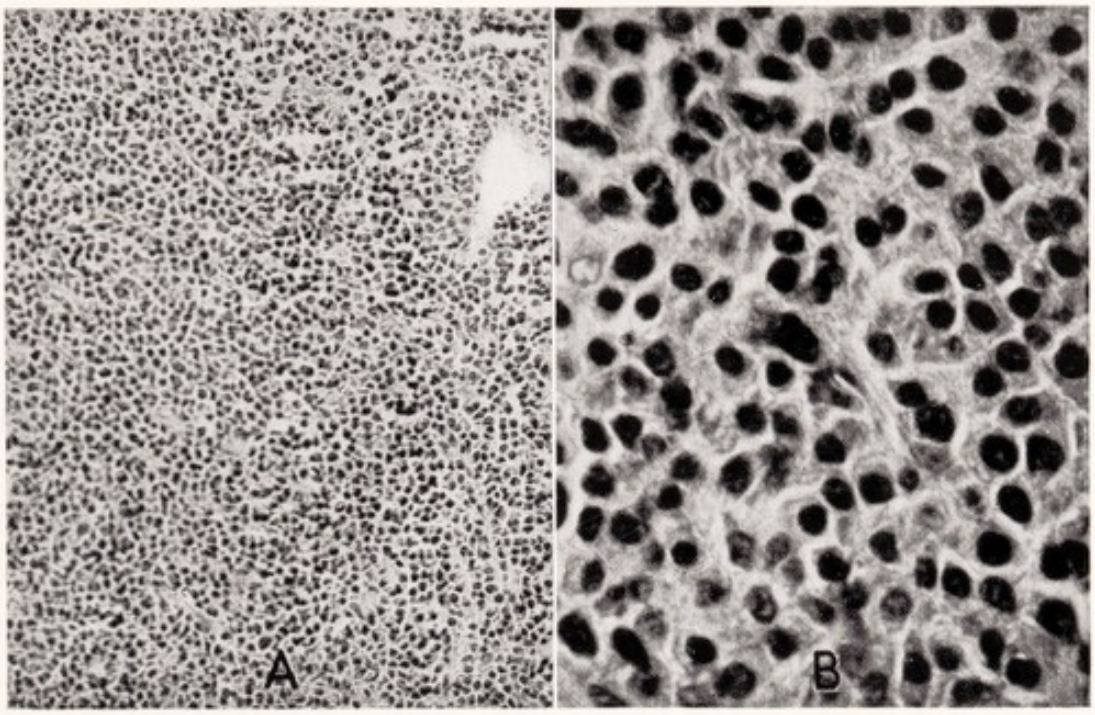

FIG. 175. Slide of a plasma cell tumor of the hard palate: *A*, low power shows a mass of cells without any special arrangement; a few small vessels and a larger one are in evidence; *B*, high power shows the cells with illy defined protoplasm, the nuclei well stained for the most part with but little intercellular connective tissue.

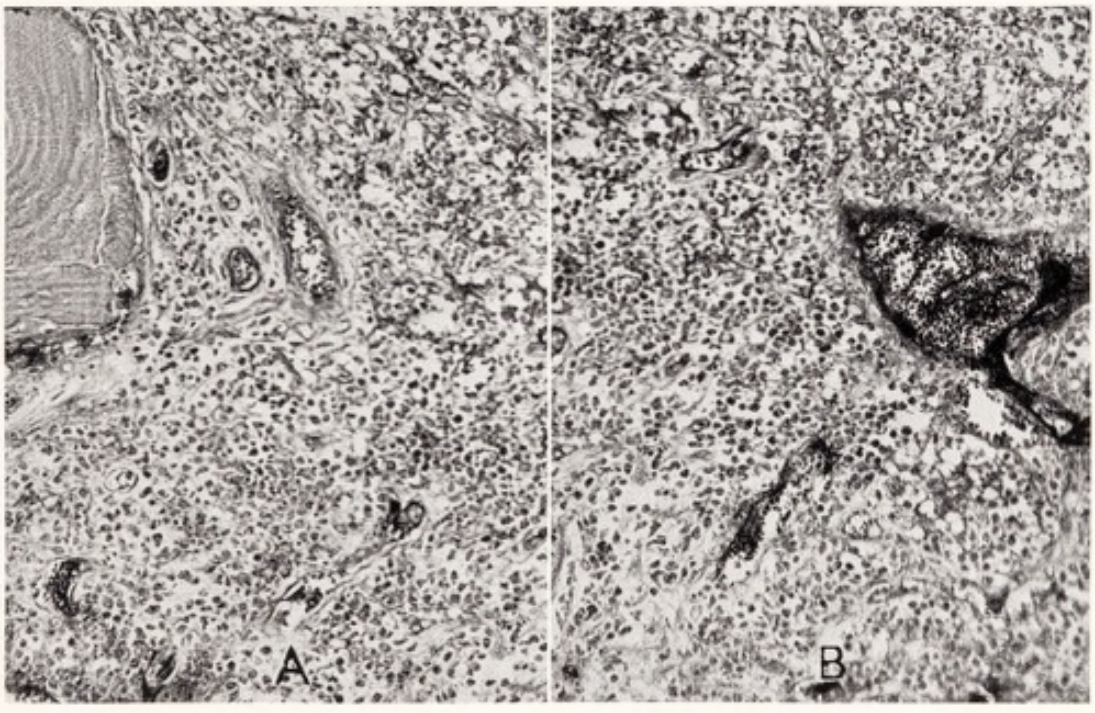

FIG. 176. Metastatic plasmoma: *A*, the uniform size of the cells is lost, the intercellular material is much increased with but little tendency to form definite fibers. There are numerous thin-walled blood vessels. *B*, The protoplasm of some cells is much increased and palely staining. The structure of some cells is indistinct.

a more cellular tissue which closely resembles the mixed tumors of the salivary glands. No matter what the structure, because of a similar clinical course it contributes to clinical convenience to regard them all as of one origin. Those which are composed of pure chondroma and pure osteoma have been considered separately. This is justified because of their invariable sluggish growth. Those tumors, containing cysts, and the fibromas had best be classed with the mixed tumors, because some part of them may take on more active growth.

Pathogenesis. These tumors are usually of slow growth, the patient being hardly conscious of their beginning. They are usually discovered

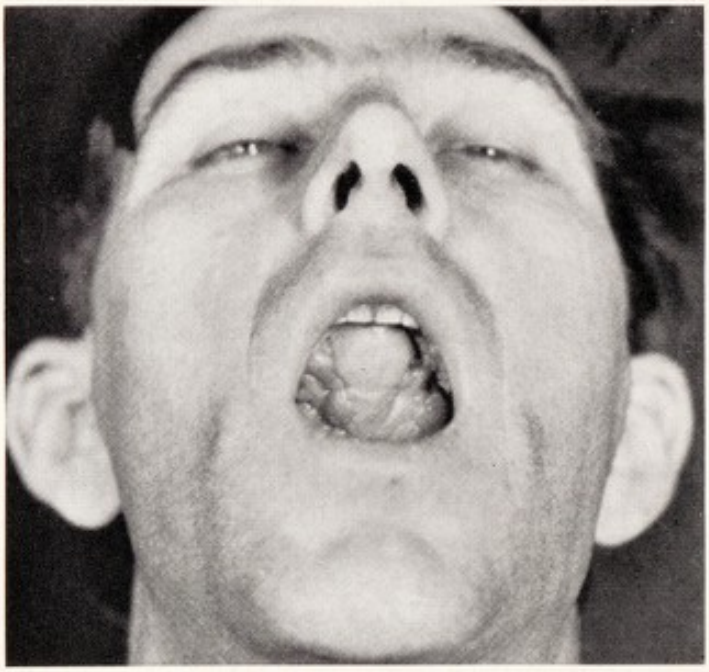

FIG. 177. Mixed tumor of the hard palate. Slightly bosselated. The patient had known of its presence twelve years.

when the tongue detects something foreign on the palate. One patient complained that it interfered with his eating mush; it did not bother with other foods. Curiosity usually sends the patient to the doctor. It is only after they become of considerable size that they interfere in any way with the various functions of the mouth. They are situated for the most part in the midline, but may ultimately extend over a considerable area of the palate (Fig. 177). They may have their point of origin at some other point than the midline. When so situated the possibility of origin in the alveolar process or in the antrum must be considered.

These tumors do not seem to become malignant as do the mixed tumors of the parotid gland, likely because they are not tolerated so long.

Pathology. A smooth-surfaced more or less ovoid nodule greets the examiner (*A*, Fig. 178). They may be densely hard, but usually are firm though elastic and rarely cystic. They are always firmly attached to the underlying bone. The mucosa is not involved, but is closely attached and immovable. Only rarely does the covering mucosa become destroyed, usually then as the result of ineffectual attempts at removal.

The fixity of these tumors is most impressive during their removal.

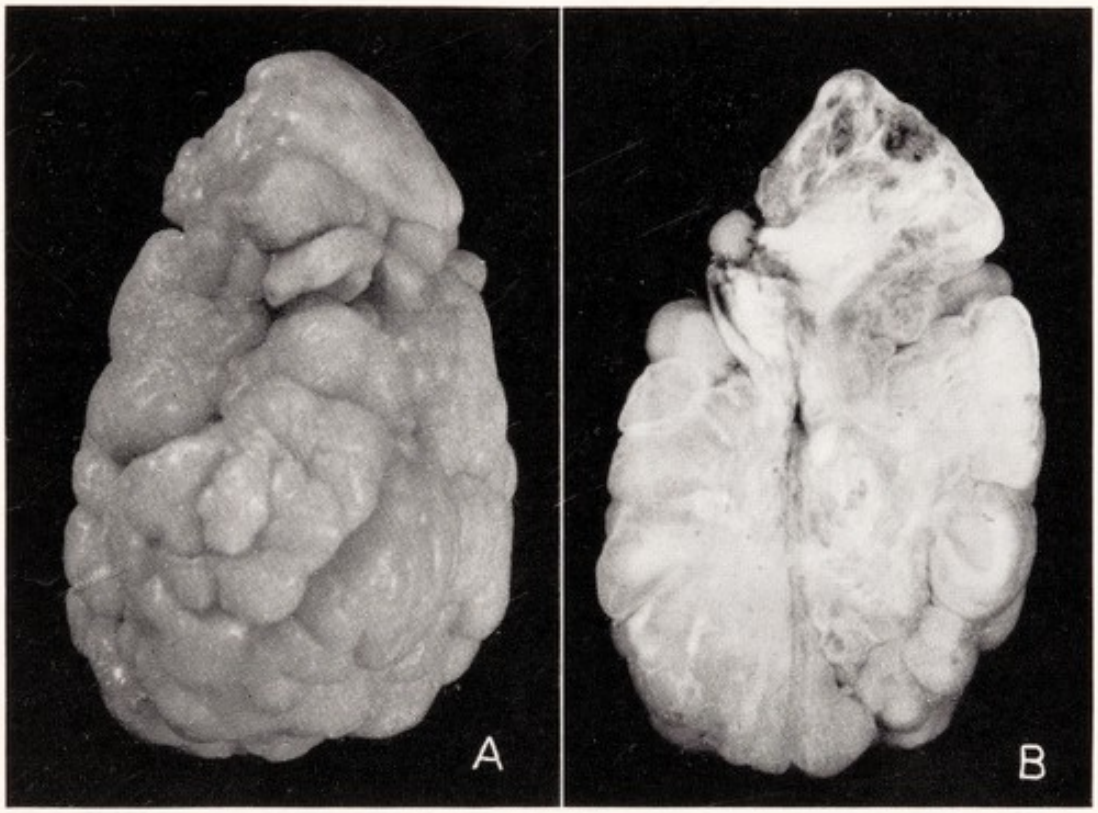

FIG. 178. Mixed tumor of the palate: *A*, surface exposed in the mouth is bosselated; *B*, cross-section showing the mottling of many tissues.

One needs bone instruments to accomplish it. Because of the dense attachment of the covering mucosa this is lost in the struggle.

The cross-section varies greatly; solid bone, pure cartilage, cellular, and cystic tissue may all be found (*B*, Fig. 178).

Histology. As indicated above, pure elemental tissues may be found in some areas. The chief interest centers in the fact that they are really mixed. These quite parallel the mixed tumors of the parotid gland, columns of cells intermixed with bundles of hyaloid connective tissue (Fig. 179). Solid areas of cells are more commonly found than in parotid tumors and myxoid areas are more rare. The cells are commonly found closely associated with vessels warranting the old classification of peri-

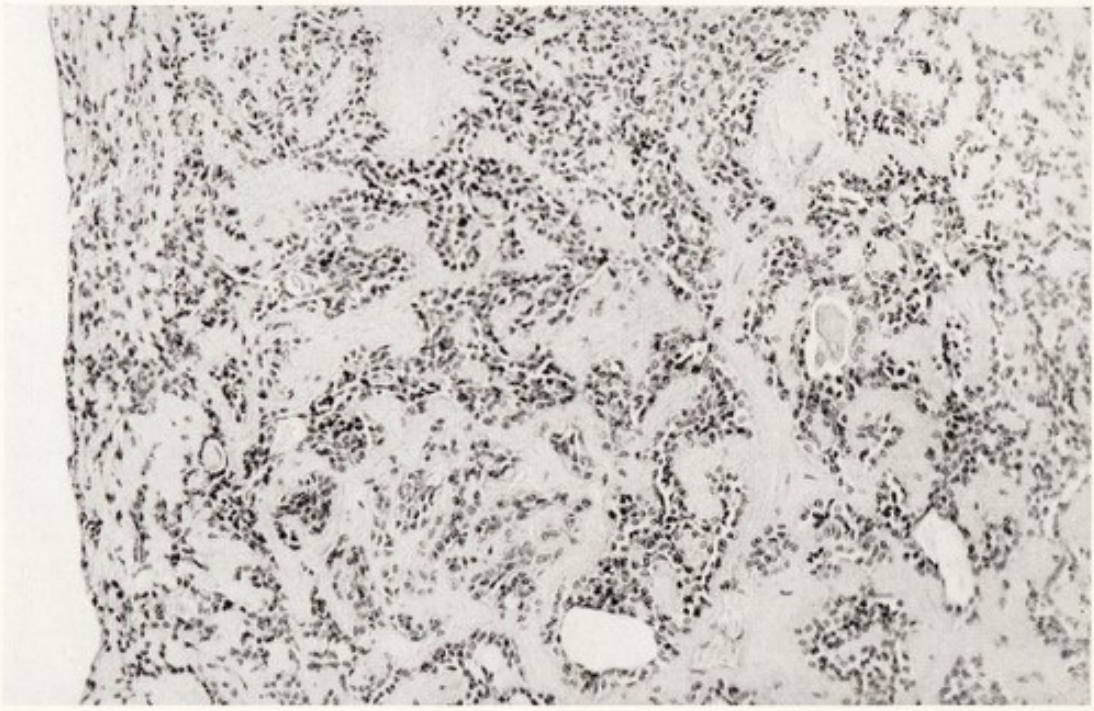

FIG. 179. Slide from a mixed tumor of the palate. Columns of cells make up the greater part of the structure. Many thin-walled vessels are present. The connective tissue is hyaline and dense, staining but poorly. The cells are in many areas arranged along thin-walled blood vessels.

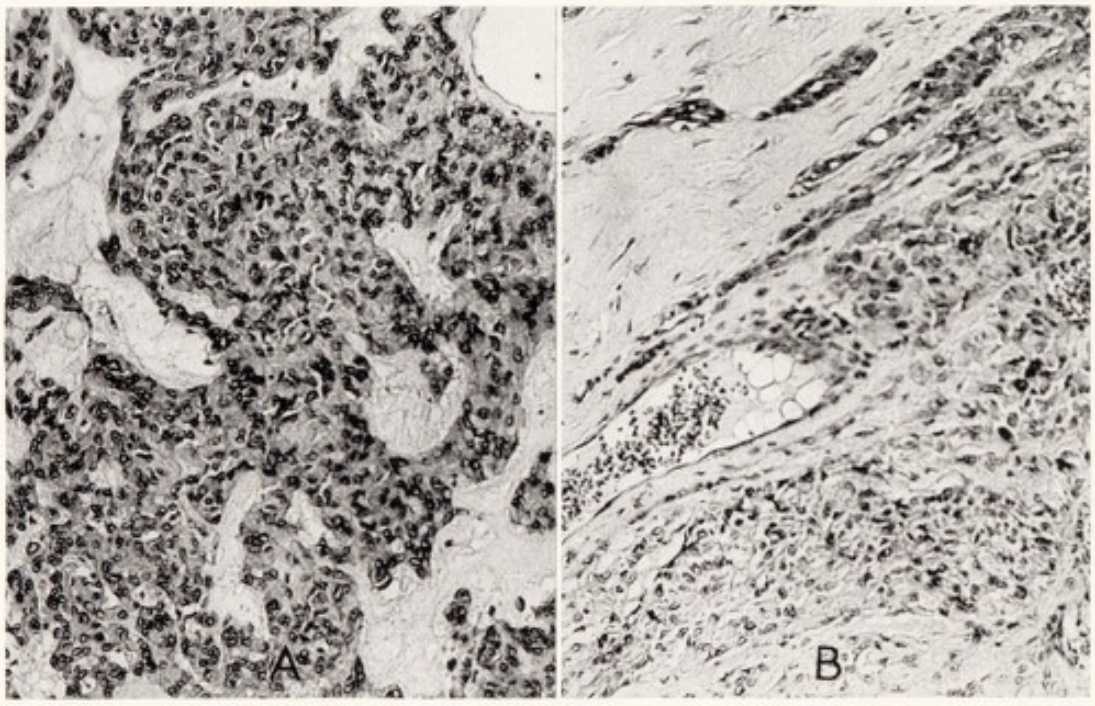

FIG. 180. Slide from a mixed tumor of the palate: *A*, solid masses of palely staining cells. The connective tissue seems degenerated; the close arrangement of cells about the vessels is impressive; *B*, the original cells seem changed into spindleform and in some areas degenerated. The connective tissue is hyaline.

vascular endotheliomas. Some tumors are more cellular, even suggesting invasion (Fig. 180). It is difficult to distinguish the character of the cells in the invaded area; the palely staining character seems to absolve them from any malignant intent.

FIBROMAS

Pure fibromas are less common than the mixed tumors. They seem not to develop at the midline but on a lateral half, and may encroach on

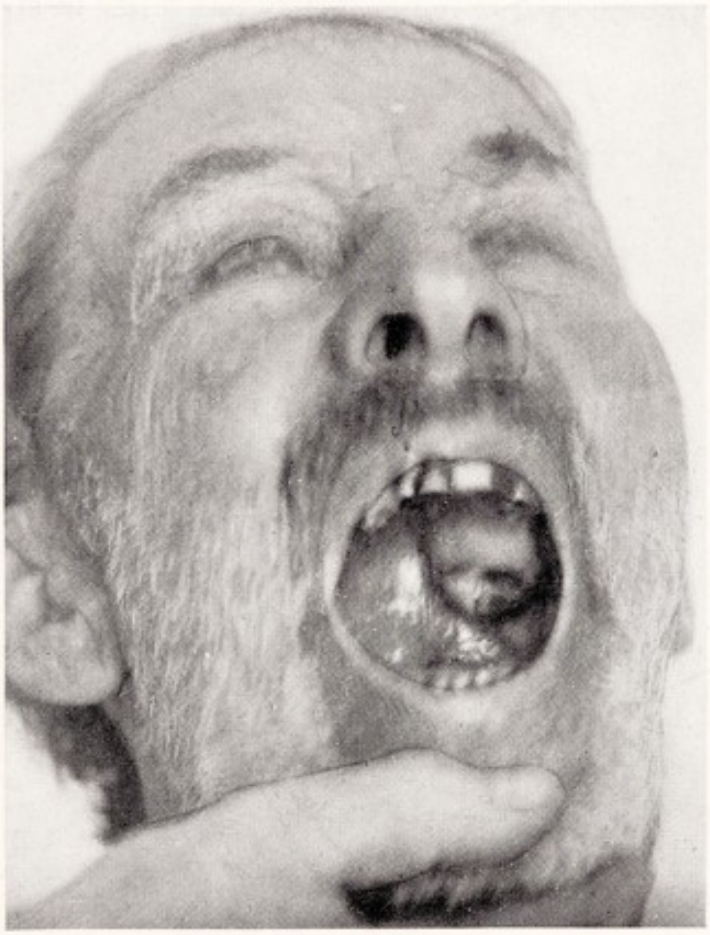

FIG. 181. Fibroma of the hard palate and alveolar process. It formed a rounded tumor extending from the hard palate.

the alveolar border. On this fact depends their importance. They are apt to be diagnosed as fibrous epulides or because of their structure as spindle-celled sarcomas. They are neither. Their clinical course is as innocent as the mixed tumors.

Pathogenesis. They form uniformly rounded or slightly bosselated tumors of slow growth. They seem not to form anything but fibromas (Fig. 181).

Pathology. To the touch they are firm, elastic, and immovable. On section they are densely fibrous.

Histology. The structure is that of a fibroma. The cells form wavy bundles, and are spindleform, the nuclei deeply staining (*A*, Fig. 182). Some areas are distinctly myxoid (*B*, Fig. 182).

CARCINOMAS

Epithelial malignancies confined to the hard palate, away from the alveolar border, are rare but they seem not to differ in structure. Since

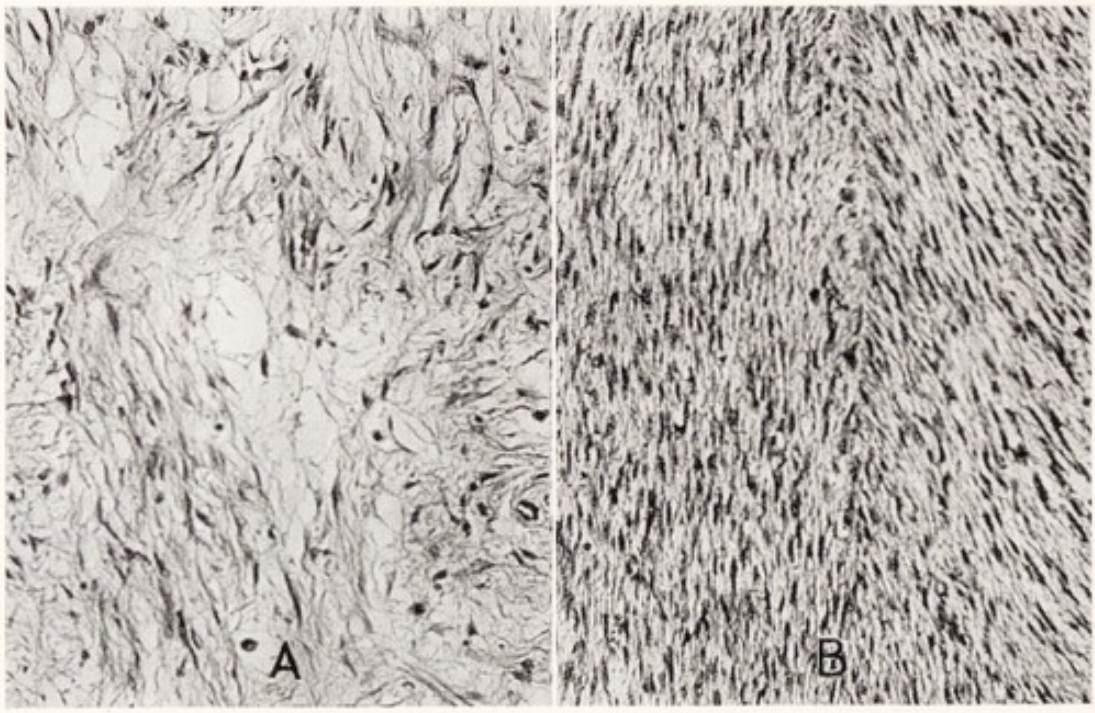

FIG. 182. Fibroma of the hard palate: *A*, the fibers form wavy interlacing bundles and are but faintly staining. The cells are spindleform of uniform size, but vary in their staining reactions. A few show degeneration. The blood vessels are very few and inconspicuous. *B*, More fibroid areas.

they have no tooth sockets to invade they are much less malignant. The hard bone of the palate seems to offer effective resistance to invasion.

Pathogenesis. When they are first seen by the surgeon they have attained a diameter of a centimeter or two and then present the same physical characteristics as the malignant ulcers of the alveolar border. Destroyed by a cautery they seem to stay cured.

Pathology. In feel and appearance they parallel those of the alveolar border. They do not bleed readily, which likely accounts for the fact that patients tolerate them so long before seeking relief.

Histology. The slides present pictures identical with those of the alveolar border.

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CHAPTER X

Diseases of the Nasopharynx

THE diseases of the nasopharynx have been almost completely removed from the field of the general surgeon by specialists in this field. Time was when all major operations were handed on to more bold operative craftsmen. More major operations were done in this region thirty years ago than now. In fact some of the major procedures then done have gone out of use. Temporary resections of the nose to give access to the rhinopharynx are no longer needed. The temporary resection of the upper jaw in order to make tumors of the pharynx accessible, no longer performed, was a real operation and he who could accomplish this feat in twenty minutes required two rows of buttons on his vest and he earned them. Sad as such memories are they are brightened by the fact that the rhinologist with the aid of the radiologist accomplishes more for the patient than we did with our radical operating, and it is much more pleasant for the patient. However, the lesions in this region are still of interest to us because of the relation they bear to other problems.

If one attempts to list all the diseases of the nasopharynx a very formidable array is obtained. If, however, one confines himself to those diseases that present problems in the clinic which require laboratory study for their understanding the number is greatly reduced. This obviously is the course to pursue since the inflammations and deformities are obvious by mere inspection, and present no lesions of pathologic interest.

The lesions which require consideration are chiefly tumorous in character, mostly neoplastic with a few but important congenital lesions. The neoplasms fall obviously into the benign and malignant. The benign are chiefly polypoid in form, which may be divided again into the mucous and the fibrous polyps. These are divisible, if one chooses, according to their vascularity and cellularity. A few are congenital. The congenital lesions fall, for the most part, into the benign group. The malignant tumors, as usual, are divisible into those derived from epithelium and connective tissue, and those of which the origin is not known.

Certain lesions of the accessory cavities may protrude into the nose and are usually obvious in nature, but others may include both cavities. Hence it seems advisable to discuss these lesions in this chapter.

A simple classification leaves a number of conditions which may be grouped as rarities, since they play a much more impressive rôle in the literature than in the clinic due to the propensity of the most of us to parade rarities.

The surgical diseases of the nasopharynx may be classed as follows:

I. Benign Tumors of the Nasopharynx
   - Nasal Polyps
   - Pharyngeal Polyps
   - Papillomas
   - Meningoceles

II. Malignant Tumors of the Nasopharynx
   - Sarcomas
   - Epitheliomas
   - Lympho-epitheliomas

III. Diseases of the Tonsils
   - Hyperplasias
   - Sarcoma
   - Carcinoma

The benign group are mostly simple lesions and acquire interest chiefly because of the need always to keep in mind the possibility of a meningocele.

BENIGN TUMORS

The nasal polyps furnish the most common tumor of the nose. They are hardly neoplasms and the attempt to prove them inflammatory in origin has not been satisfactory. Some of the pharyngeal polyps show a proliferative capacity, which without much display of imagination places them in the group of neoplasms. However, when one speaks of polyps he visualizes a pendent tumor, the nature of which must be declared by a qualifying term.

Nasal Polyps. The nose produces its own type of polyp, the myxoid. Though essentially myxoid in character they may show a considerable admixture of cells which may suggest malignancy, but they remain benign. However, the plasma cell accumulation may be so great as to suggest malignancy, if examined in the laboratory only. Even so they usually remain benign. However, they may show malignant invasion or even form metastasis. Therefore, the prudent surgeon will, in doubtful cases, not make a diagnosis on the microscopic picture alone, but will await the patient's verdict as expressed in the after-course.

Pathogenesis. The nasal polyps begin in the middle fossae almost exclusively. Some believe they are of inflammatory origin, but experience

proves that when the polyps are completely removed they do not return and there is no evidence of inflammation. They are seldom associated with infections of the accessory sinuses.

Nasal polyps are prone to recur unless the base from which they spring is cauterized or the polyp is torn out rather than cut off with the snare. In their persistent recurrence they are characteristic of myxomas, but their reformation must take place in a relatively small pedicle. However, their size is dependent more on the edema which their form and dependent nature engender rather than on cell proliferation. This is easily

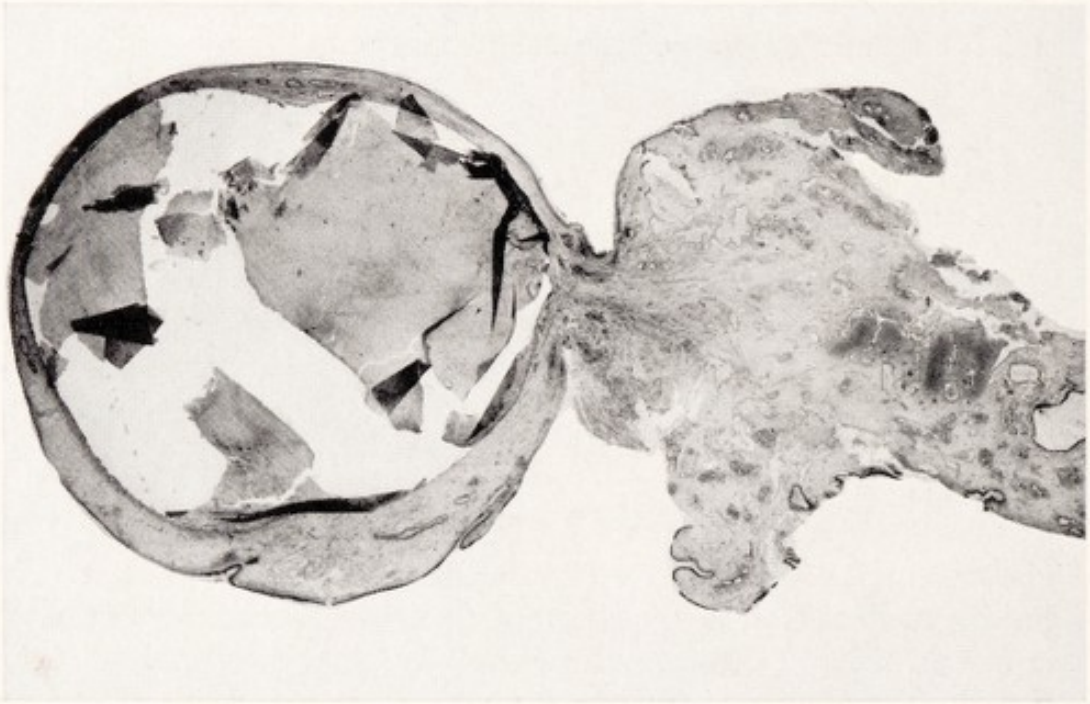

FIG. 183. Cystic polyp resembling a small meningocele. The cyst in the recent state was the size of a marble. Its restricted base and incompressibility distinguished it.

demonstrated by placing them in alcohol after removal. After the water is abstracted there is not much tumor left.

Their chief clinical significance lies in the fact that they fill one or both nostrils interfering with respiration, but do not otherwise interfere with the patient's general health. However, in children the interference with nasal respiration may have important constitutional effects. When they become larger or numerous they may protrude from the nose. More commonly they protrude into the pharynx. If they extend into this free space they may become so large as to fill the entire pharynx and even become visible below the soft palate.

These polyps may remain unchanged for many years if untreated, but if completely removed may recur times without end.

In cases in which malignancy is simulated or actually appears, there is apt to be a serous bloody discharge. Usually, however, these signs are caused by a tumor of an accessory sinus which protrudes into the nose and is mistaken for a polyp. Be this as it may, a serosanguinous discharge suggests the possibility of a serious lesion.

Pathology. The polyps are pinkish white, almost translucent, on inspection. The appearance is characteristic. Examination is required only to determine their number and exact point of attachment, and to exclude a meningocele. Sometimes mucinous cysts form which look very

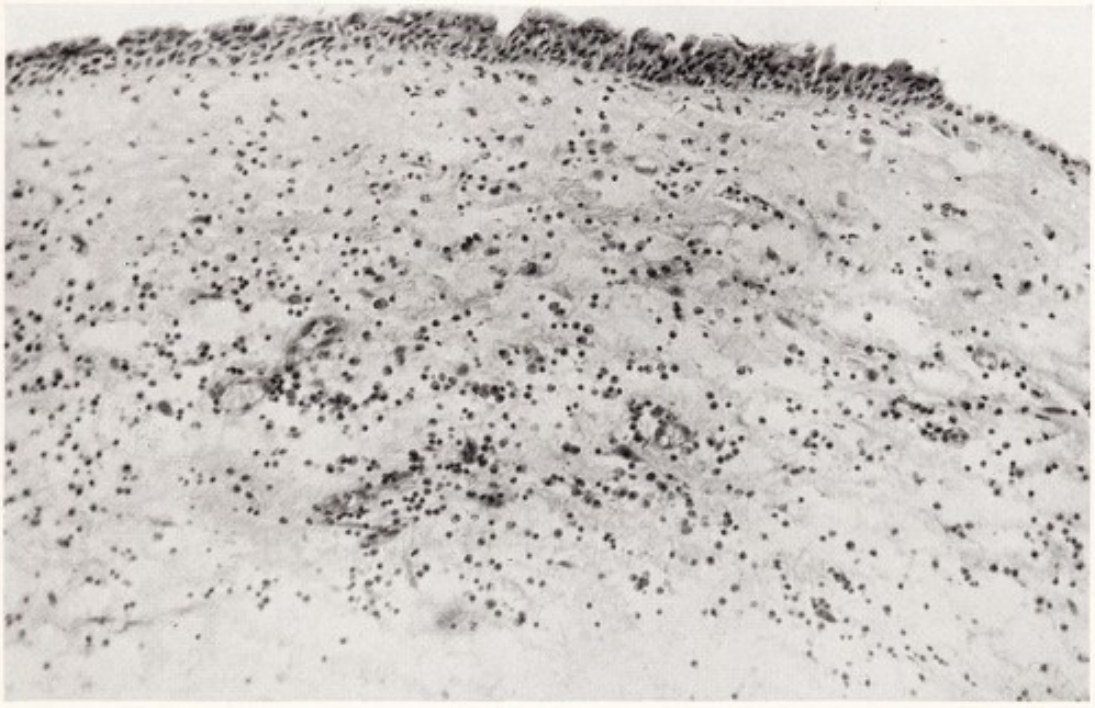

FIG. 184. Slide of a nasal polyp. The surface is covered with several layers of more or less elongated cells. The subepithelial tissue is mucoid, being formed of illy staining fibers. Imbedded in this are many large round cells containing much palely staining protoplasm with distinctly staining nuclei. The remainder sparse with distinctly formed walls.

much like meningoceles, but they are not compressible (Fig. 183). If they are vascular they may be distinctly red in color, and if very cellular they are firmer in proportion.

When removed, the clinical appearance is confirmed. These cysts are so soft that they flatten when placed on a hard surface. Difficulty is encountered when sectioning them for gross inspection upon removal, and after hardening they shrink very much, but the surface is white and usually uniform in color.

Histology. Columnar cells, perhaps several layers, cover the surface. The remainder of the tumor is made up of edematous fibrous tissue with

a varying number of cells (Fig. 184). The vessels may be numerous and prominent. The cells are collected chiefly about the vessels, and may be so numerous as to suggest malignancy, but usually only one part of the polyp is so infiltrated (Fig. 185). However, it is a question if a diagnosis of malignancy should ever be made on the slide alone. If one avoids the use of the high-power lens and examines wide areas, mistakes are not likely to happen.

Pharyngeal Polyps. The pharynx is the habitat of polyps of varying form and structure. The polyps with narrow bases are relatively soft,

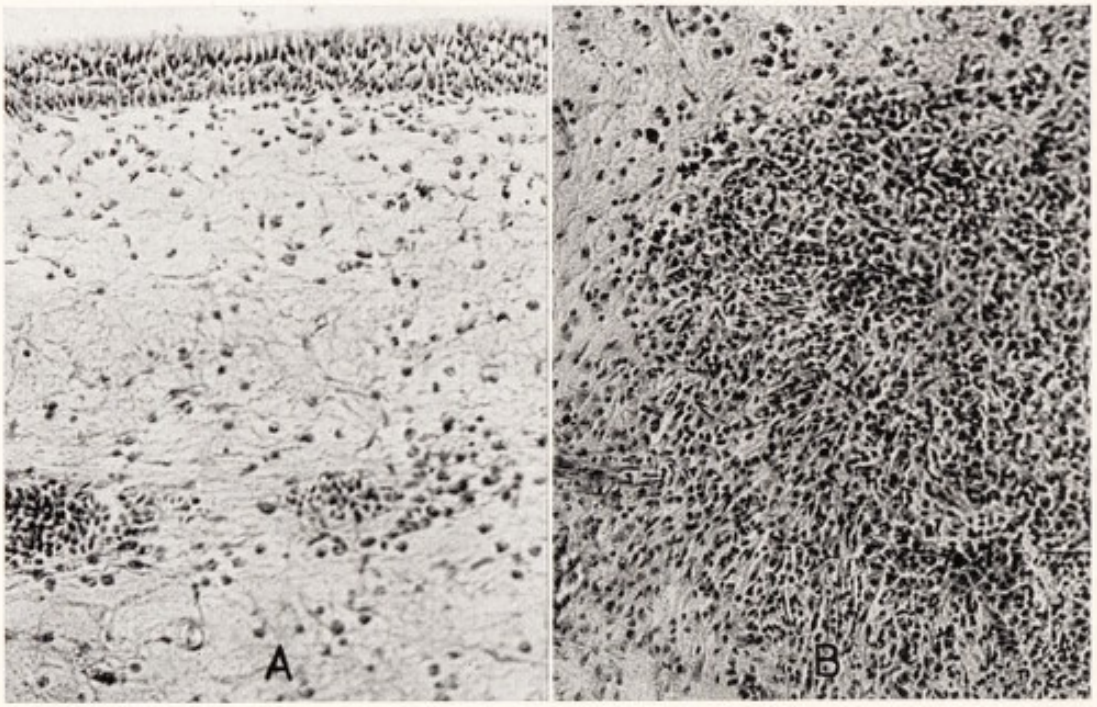

FIG. 185. Nasal polyp in part cellular: *A*, usual structure, the epithelial layer is topped by columnar cells; beneath is mucoid tissue; *B*, cellular area made up of many cells the same in characteristics as those found sparsely in all polyps. They are arranged more compactly and the nuclei are more deeply stained.

with sparse connective tissue, and relatively few cells. These are the counterparts of those of the nose and usually have their attachment in the same region and extend into the pharynx because of the greater space. The broad base type is more densely fibrous and more cellular and has its attachment in the vault of the pharynx.

Pathogenesis. The beginning of the soft mucoid types presumably is the same as the smaller prototypes found in the nose. They are definitely formed before they are discovered, hence, unlike those of the nose, we do not see them when they are small, and seemingly once removed they do not recur. Usually the obstruction these polyps cause to respira-

tion is relatively slight and their protrusion below the soft palate first declares their presence in many cases (Fig. 186). Once formed they persist indefinitely. That they become clinically malignant has not been definitely demonstrated.

The fibrous types are of a different breed. They are much firmer, and are attached to the vault of the pharynx. They may be narrow or broad at the point of attachment. The type with narrow pedicles is most common in women while the harder broad-base type is more prevalent in young males; indeed some observers believe the latter type does not occur in women.

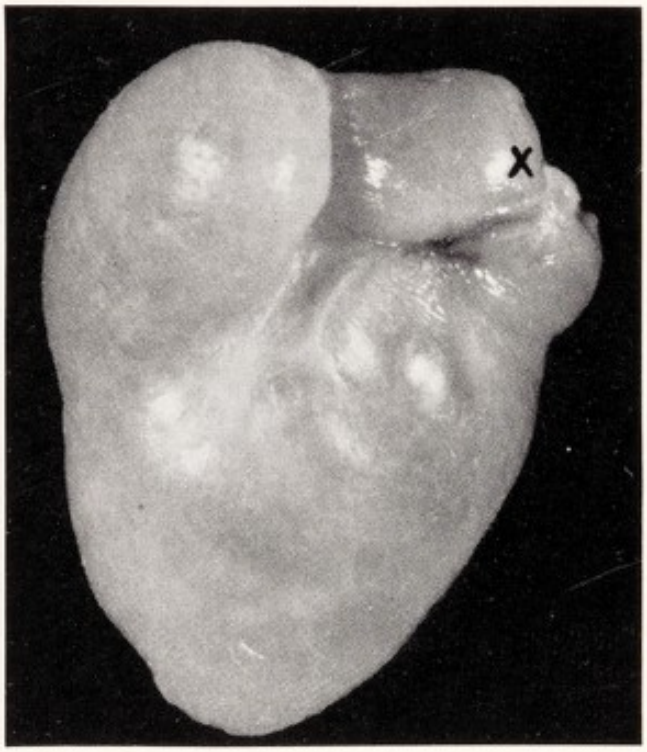

FIG. 186. Large mucoid polyp which projected into the pharynx. The pedicle is at *x*.

The vascularity of the two types differs. The small-based types have a very sparse blood supply and their removal is attended with little hemorrhage. On the contrary, the broad-base types have an abundant blood supply and their removal may be attended by fatal hemorrhage unless adequate measures are taken to control it. It is the broad-base types which were formerly consigned to the general surgeon. Temporary jaw resection was required so that the base could be removed under direct vision and the bleeding area could be promptly packed in order to control hemorrhage. Death from hemorrhage in a specialist's office usually resulted in shunting all pharyngeal polyps to the general surgeon regardless of the size of their bases.

Pathology. Because of their size the extent of the base can seldom be directly observed by posterior rhinoscopy and direct palpation usually is required to determine it. This means of examination likewise gives a knowledge of the density.

Only after removal is the exact extent of attachment of the base disclosed. The base may be relatively narrow (*A*, Fig. 187) or it may be broad occupying considerable area of the vault of the pharynx (*B*, Fig. 187). It is the latter type which may be attended by profuse hemor-

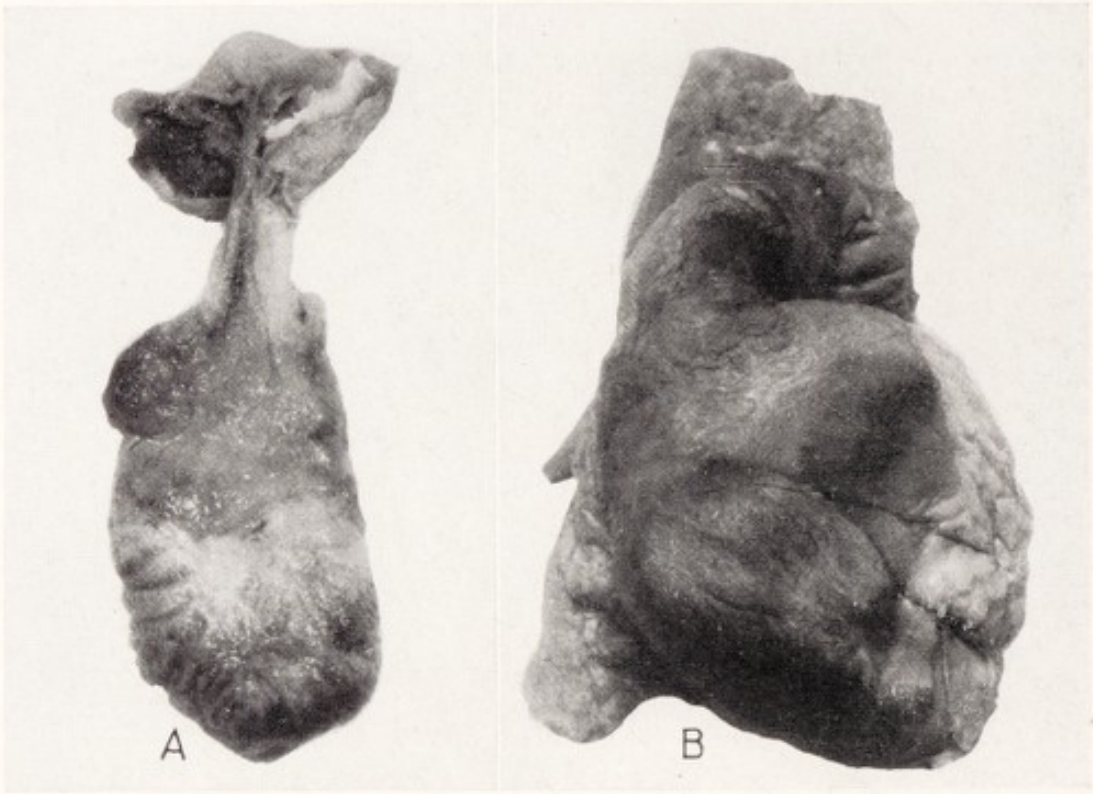

FIG. 187. Fibrous polyps of the pharynx: *A*, narrow-base type removed from a young woman; *B*, broad-base type removed from a young man.

rhage when removed. This is the type for which preliminary resection of the upper jaw is made in order to expose the area of attachment, so that efficient packing can be promptly done. The color is light pink, unless submucous hemorrhages give it a mottled appearance.

The cross-section of the narrow base type shows a myxoid-like pinkish white structure quite like the nasal type, while the broad-base type is reddish and fibrous, therefore firm to the touch.

Histology. The gross appearance above noted indicates the cellular structure. The narrow-base type is myxoid, the connective tissue palely staining, and the cells of the plasma cell type quite like the nasal polyps

above described. These are more likely than the nasal type to show great vascularity. The broad-base type is made up of more dense, more deeply staining, connective tissue and the blood vessels are large and numerous. The cellularity, likewise, varies greatly (Fig. 188). The cells may be elongated, which may give the picture of a spindle cell sarcoma, though clinically benign.

Papillomas. Small hard wart-like tumors are occasionally seen similar to those found in the cheek.

Pathogenesis. These little growths are most common on the faucial

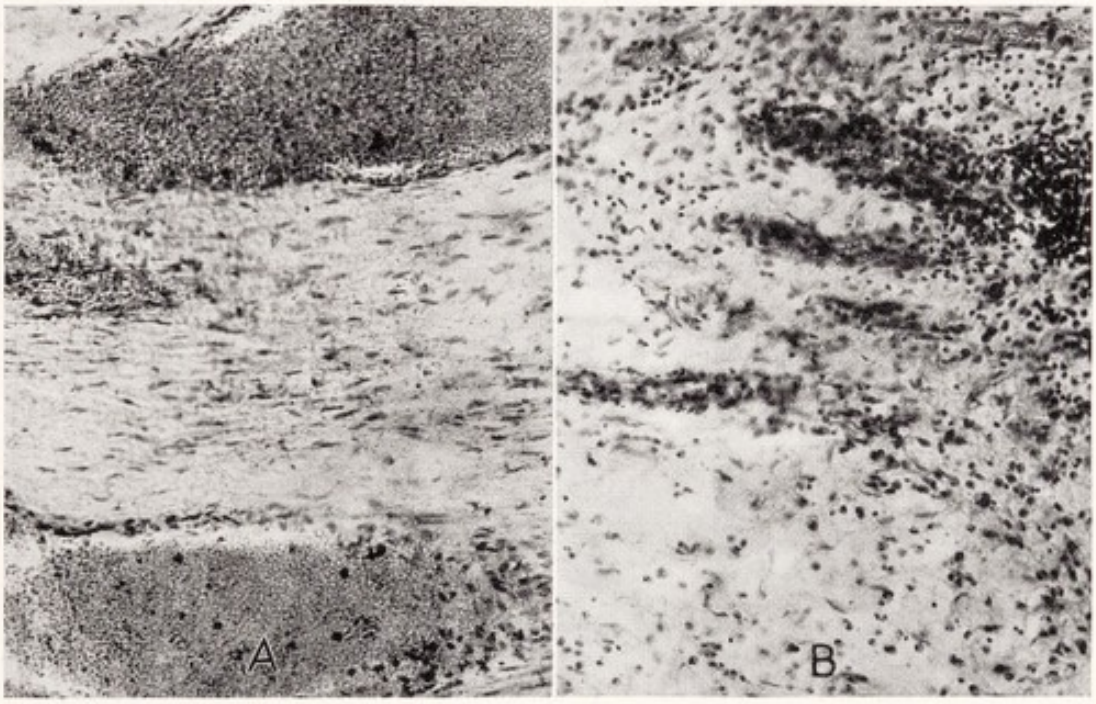

FIG. 188. Fibrous polyp of the pharynx: *A*, the vessels are numerous and large; *B*, the vessels are surrounded by many large round cells the borders of which are indistinct but the nuclei stain deeply. In some areas the cells are spindleform, but for the most part they have ovoid, deeply staining nuclei.

pillars or on the tonsils, less often in the pharyngeal walls. They may be solitary and but little elevated, but occasionally many of them may be scattered about a considerable area. These are sometimes many times longer than broad, having the form of a rooster's spur.

Nothing is known as to their causation. Sometimes the patient is conscious of an irritation of the throat, but more often they are accidental discoveries made during the course of routine examination. These growths seem to undergo no change, but malignancies may develop on their base. Hence they require careful consideration.

Pathology. Their elevation, pale pink color, and firmness on manipu-

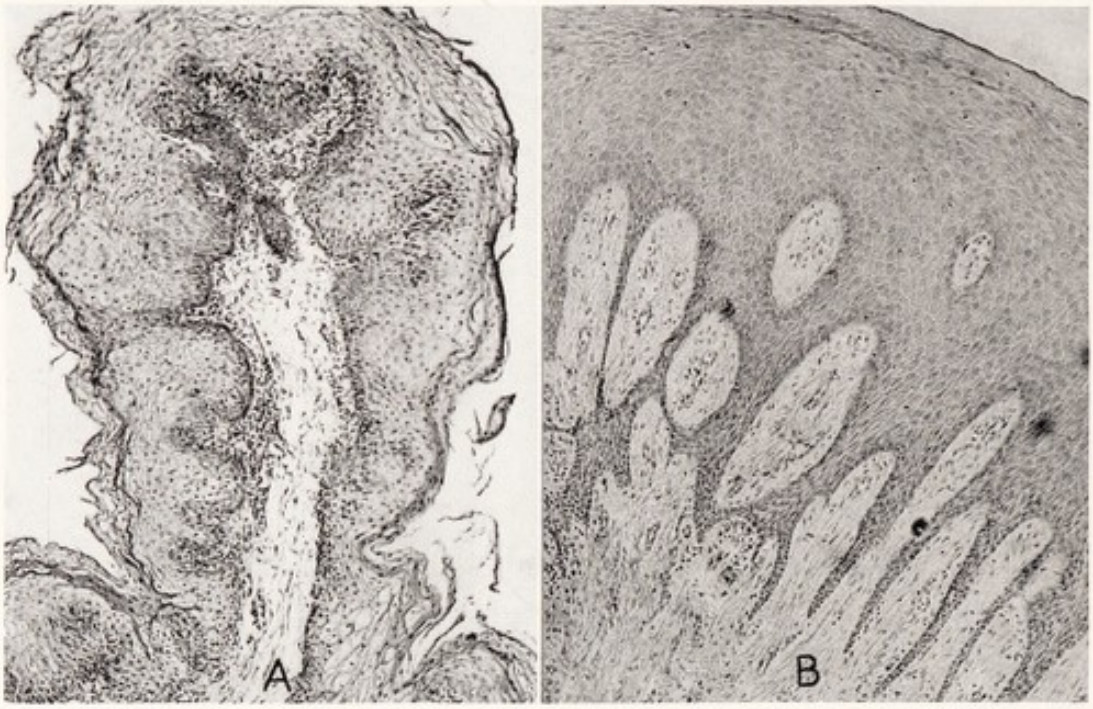

FIG. 189. Section of a papilloma of the pharynx: *A*, only the summit is shown. It is covered with many layers of epithelium the surface of which is desquamating. There is some round cell infiltration about the base. *B*, Round domed nodule showing innocent hyperplasia of the epithelium.

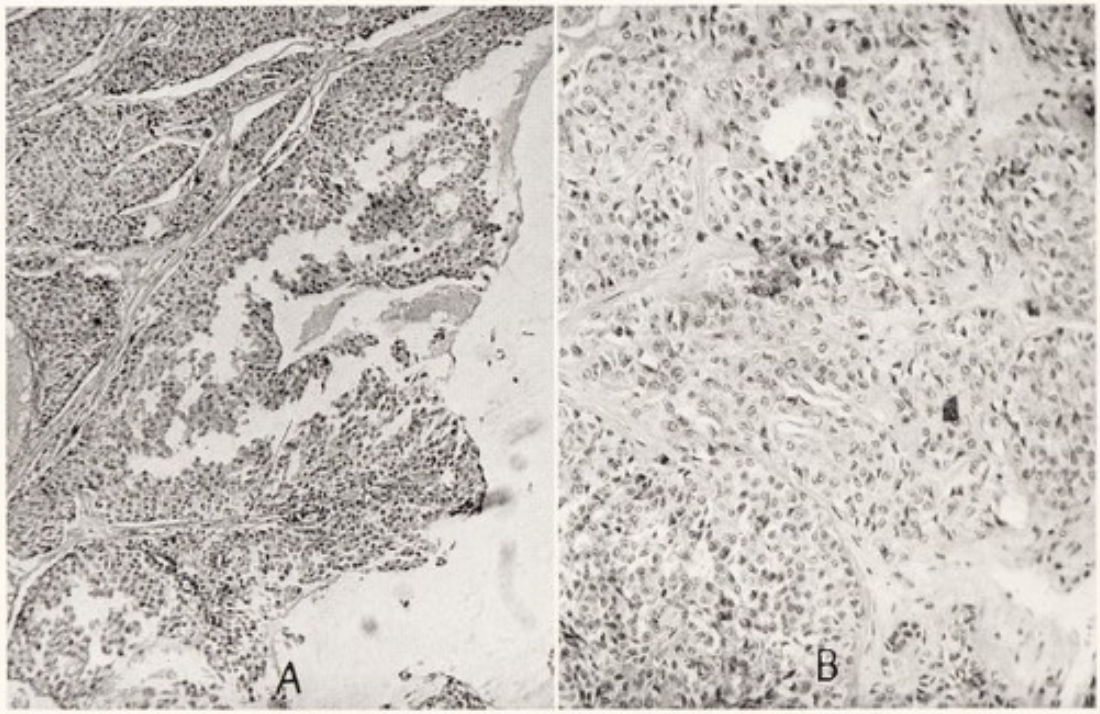

FIG. 190. Malignant papilloma of the pharynx: *A*, the surface presented villiform processes attended by but little vessel bearing connective tissue; *B*, the deeper part was made up of masses of cells with little connective tissue containing a few small blood vessels. All cells were palely staining, many more or less spindleform.

lation declare their nature. Their density is manifest, if they are accessible to direct palpation. When removed they have the consistency of a horny surface wart. If malignancy is beginning, they are less freely tilted about. I have seen but one case. It likely was a soft cauliflower type.

Histology. Because of their density papillomas are unruly on the microtome. The section shows a more or less papilliform surface made up of many layers of deeply acidophile cells (Fig. 189). When malignancy occurs there is invasion of the deeper tissues (Fig. 190), and the surface has the feel of a bladder carcinoma.

Meningoceles. The common types of congenital tumors are meningoceles extending through the cribriform plate directly into the nose.

Pathogenesis. Their appearance suggest polyps, hence their interest. They may appear at the base of the nose, producing a bulge near the inner canthus of the eye. Naturally this makes a very convenient danger signal.

These meningoceles obviously are due to defects in development. They may be apparent at birth or appear soon after. Sometimes they are discovered in later life. They may resemble polyps and, occasionally, their nature is not suspected until persistent drainage of cerebrospinal fluid makes clear their nature. This may be emphasized by the development of a fatal meningitis.

Pathology. The blue color of the meningoceles should suggest their nature. Quite commonly their cystic structure may be suspected when manipulated with an instrument. They are usually compressible.

Histology. The sacs are epidural and one can divine their structure. Those protruding at the canthus of the eye present a thin membrane covered within and without with endothelial cells, unless these have been lost through secondary changes. I have never examined one which protruded into the nose.

MALIGNANT TUMORS OF THE NASOPHARYNX

Three general groups are recognizable clinically; the sarcomas, the epitheliomas, and the lympho-epitheliomas. They form a group of tumors which may be similar clinically and their histologic classification may puzzle the pathologist. Often microscopic examination leaves one confused and the clinical course must be brought in to help decide the problem.

Sarcomas. The primary sarcomas of the nasopharynx are confined chiefly to the pharynx. Those of the nose are usually protrusions from the accessory cavities reaching the nasal cavity when the walls have

been destroyed. Those of the pharynx appear as ovoid tumors protruding from some part of the pharyngeal wall, usually the posterior.

Pathogenesis. These tumors usually reach considerable size before they are discovered leaving little chance to study their beginnings. In the nose their presence is announced by a serous bloody discharge, usually confined to one side. It is possible that they are implanted on a preexisting polyp, but polyps followed over a long period of time, perhaps surviving many recurrences, do not seem to end as malignant tumors.

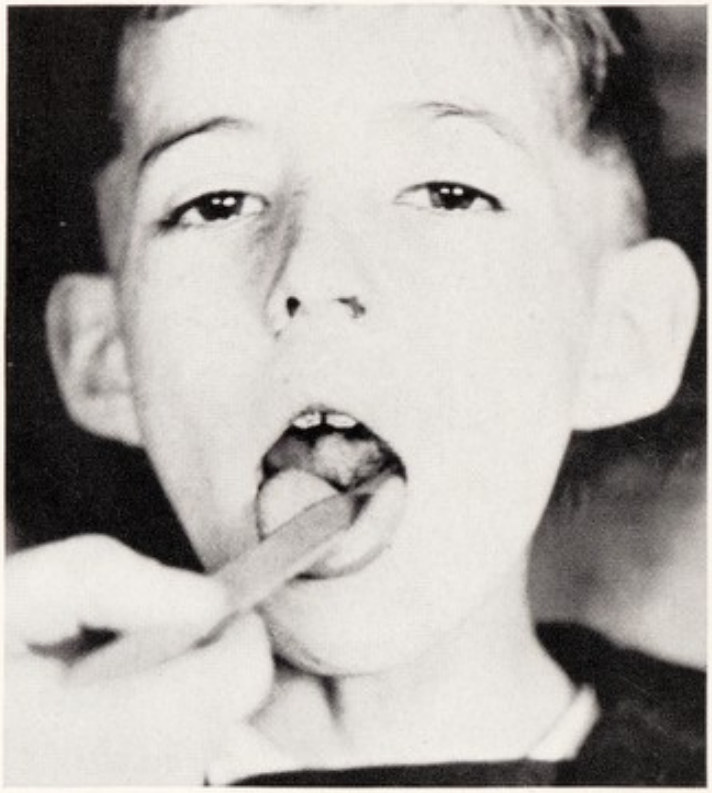

FIG. 191. Sarcoma of the posterior pharyngeal wall. The mass was irregular, the surface being eroded, and was the source of repeated hemorrhages.

The tumors of the pharynx grow more or less spheroidal, tend to ulcerate and bleed. No matter where situated, they recur quickly when removed, ulcerating more than before, and quickly reduce the patient. Pneumonia, rather than local invasion, may be regarded as the normal end.

Pathology. A rounded tumor presenting in the pharynx suggests sarcoma (Fig. 191). To the touch they are elastic and usually show early signs of erosion.

On cross-section the tumor is grayish white.

Histology. Round or mixed cells form the usual picture (Fig. 192). A mere surgeon would hesitate to make a more exact classification. Some-

times they are made up chiefly of plasma cells suggesting a possible origin from a myxoid polyp.

Epitheliomas of the Nasopharynx. Epithelial tumors are rare in the nose, but more common about the tonsils or the adjacent pharyngeal wall.

Pathogenesis. Usually more or less circumscribed tumors develop about the fauces or on the pharyngeal wall. They are of varying rates of growth but the time of beginning is seldom placed more than a few months back.

Pathology. The lesion at first may be slightly movable with the mu-

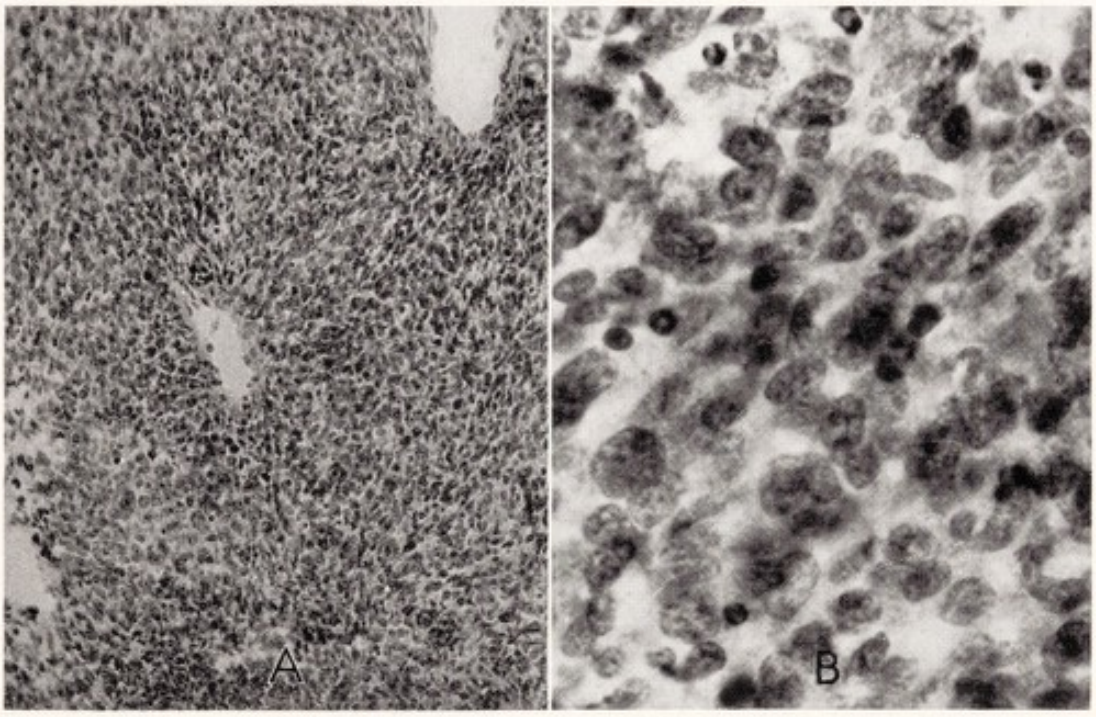

FIG. 192. Sarcoma of the pharynx: *A*, a very cellular structure surrounds numerous vessels with thin walls; *B*, high power shows large ovoid cells with ill-defined borders with large granular nuclei and distinct nucleoli. In some areas the cells are degenerating. The connective tissue is sparse and indistinct.

cosa of the fauces, but is soon fixed. With the extension of the growth they are easily recognized by their cancer hardness.

On cross-section one sees a grayish white mass with the typical dotting of carcinoma.

Histology. The usual picture of carcinoma is in evidence, but the cells may be arranged in columns, such as was formerly suggestive of endothelioma. Now the separation from lympho-epitheliomas is easy; they are unquestionably of epithelial origin.

Lympho-epitheliomas. The course of this type of tumor as it manifests itself in the neck was considered in a preceding volume. It was

noted there that often, even usually, the neck metastases were apparent before the original pharyngeal tumor was suspected, years elapsing sometimes before the primary tumor was demonstrable. Indeed the disease may proceed to a fatal termination without there having been a primary tumor demonstrable, quite a distressing state of affairs to say the least.

Pathogenesis. These tumors are supposed to arise in lympho-epithelial bodies which are alleged to lie beneath the pharyngeal mucosa. At least if they do not lie there some one has prevaricated. The primary location is most common near the fossa of Rosenmüller, but may be anywhere, even in the nasal cavities, so it is said. The tumor is usually small, and only late in the disease do they become large enough to displace the pillars of the pharynx or soft palate. Strangest of all, though they are regarded as epithelial tumors, the mucosa covering them remains intact. Only rarely, apparently, do they become eroded and bleed.

Usually the course of the disease runs for a year or two, death being caused by invasion of the metastases and not from the original tumor. Since they seldom ulcerate, they rarely bleed, and inhalation pneumonia is not common.

It is generally agreed that these tumors are radiosensitive. This evidently is more strikingly true of the original tumor than of the metastases in the neck. In fact the pharyngeal tumor may not recur after irradiation, but the metastases proceed to a fatal termination. The point is the patients all die from the disease despite the radiosensitivity, and the salutary reaction to treatment is not productive of ultimate salutary results to the patient, no matter how much it may delight the radiologist. That is to say, the patient dies from the disease.

Pathology. The tumor on inspection is a flat or ovoid tumor when first discovered, the size of half a hazelnut, seldom as large as a whole walnut. It is elastic to the touch, the only tumor allegedly of epithelial origin which does not present the cancer hardness on palpation.

Since these tumors are radiosensitive they are no longer subject to operative removal. Even in the old operative days the surgeon seldom removed them in such a shape as to admit of satisfactory gross inspection.

Histology. The characteristic feature is columns of large palely staining cells of uncertain border imbedded in lymphoid cells. That the cells are epithelial one would not have suspected unless told. The cells are ill-defined, the nuclei often imbedded in indistinct, ill-staining syncytial masses. There is little evidence of alveolar formation such as one is accustomed to see in epithelial tumors. The more remote the metastases

the more confused the arrangement becomes until no one can say whether they are epitheliomas or sarcomas, or something else. Connective tissue stains, notably with silver, produce a suggestion of alveolar formation which is not in evidence with the usual stains. In this they resemble the melanomas. The close association with the lymph cells is noteworthy.

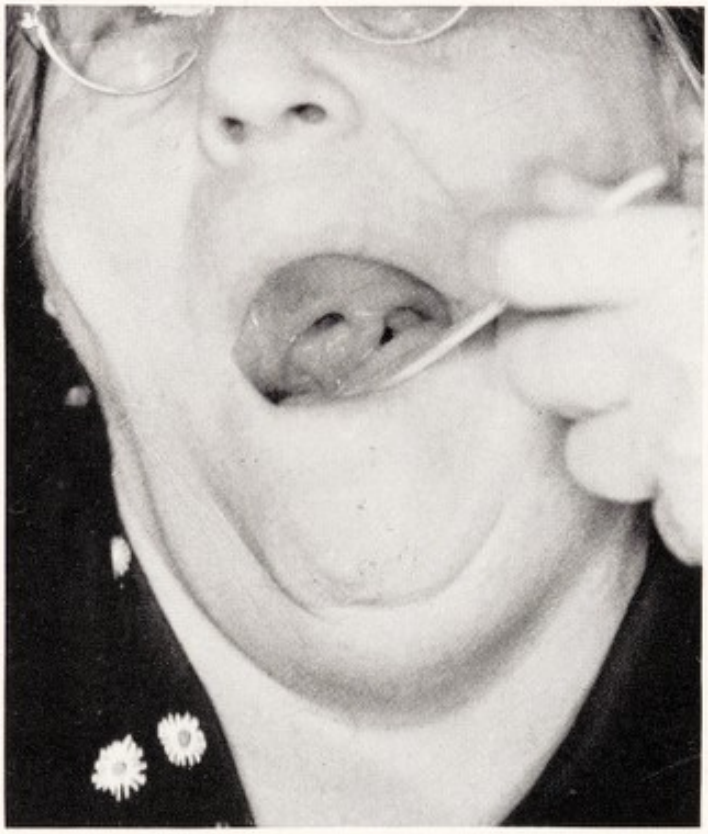

FIG. 193. Hyperplasia of the tonsil. It protrudes as far as the uvula.

In this they resemble the papillary cystic tumors of the lymph glands, since the lympho-epitheliomas sometimes show cystic areas.

DISEASES OF THE TONSILS

The tonsils are the seat of several varieties of tumorlike lesions. Sometimes simple hypertrophies may assume such proportions. Usually, however, these are sarcomas or carcinomas.

Hyperplasia of the Tonsil. It is not uncommon in children to see tonsils so huge that they nearly come in contact with each other. In adults such large tonsils are unusual, and if unilateral, suggest lesions of great moment.

Pathogenesis. What induces one tonsil to undergo pronounced hyperplasia is not known. That they may do so and continue to be innocent for prolonged periods is proved by clinical experience. Yet one dare

not assume a benign lesion until microscopic diagnosis declares them innocent and even then one had best watch his patient for a few years before rendering a final judgment.

Pathology. The tonsil on palpation is elastic, but the chief characteristic is that the whole organ is movable on its environment (Fig. 193.) In other words it has not invaded the capsule (Fig. 194).

Histology. Extensive hyperplasia of lymph tissue, nothing more (Fig. 195).

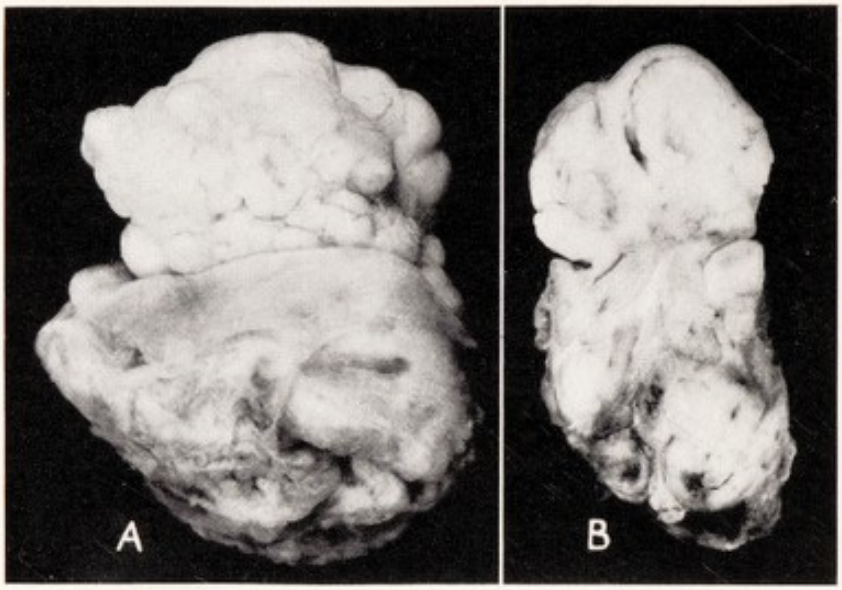

FIG. 194. Hyperplasia of the tonsil. It was removed together with its pillars. *A*, These are seen to be uninvolved. *B*, The cross-section shows lobulations of lymphoid tissues.

Sarcoma of the Tonsil. Sarcomas of the tonsil present the same features and the same clinical course as like growths of the pharyngeal wall.

Pathogenesis. These sarcomas are of relatively rapid growth, and by the time they reach the surgeon, have invaded their environment. They produce a characteristic bulging at the angle of the jaw, which permits a diagnosis at a glance. The point of bulging is higher than one sees in the lympho-epitheliomas (Fig. 196). These growths formerly were subject to radical operation; of course without result, to the patient or surgeon; only the surgical pathologist profited. I have not removed one in twenty years.

Pathology. The organ is uniformly large, bulging, and fixed to the surrounding tissue without metastases to the adjacent lymph glands.

Histology. Microscopic examination showed a structure quite like the sarcomas of the pharyngeal wall.

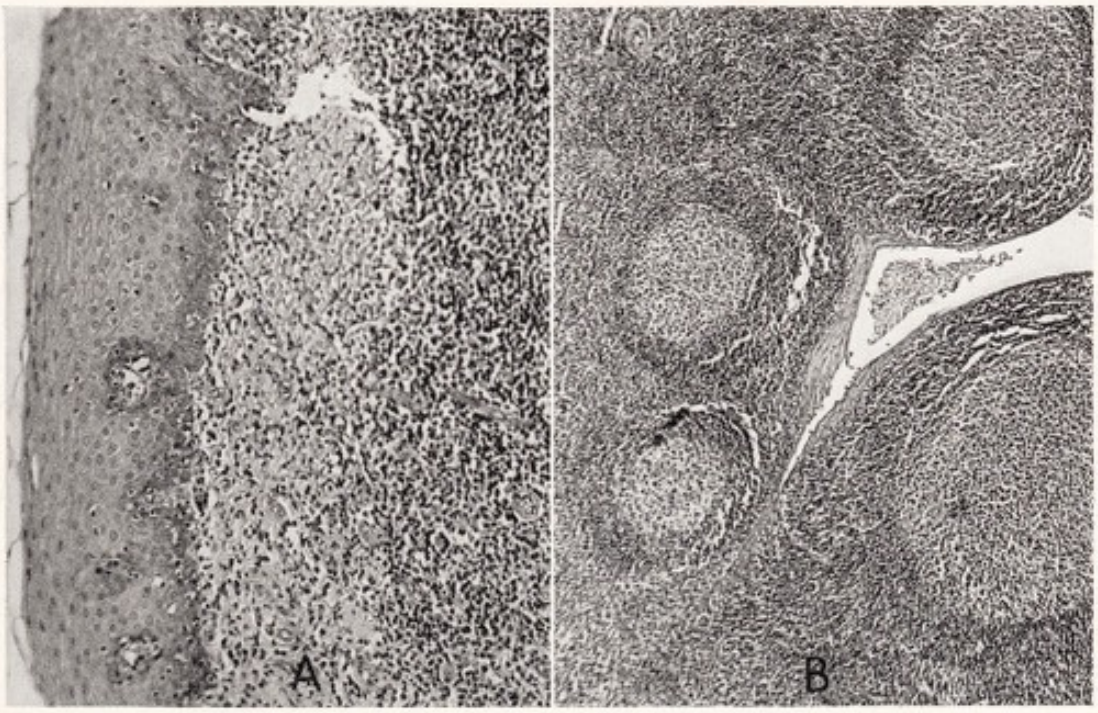

FIG. 195. Hyperplasia of the tonsil: *A*, the surface epithelium is stratified squamous celled without notable thickening. Beneath is the connective tissue infiltrated with cells. *B*, The bulk of the tumor was made up of lymphatic tissue composed chiefly of germinal centers but without any notable deviation from the normal.

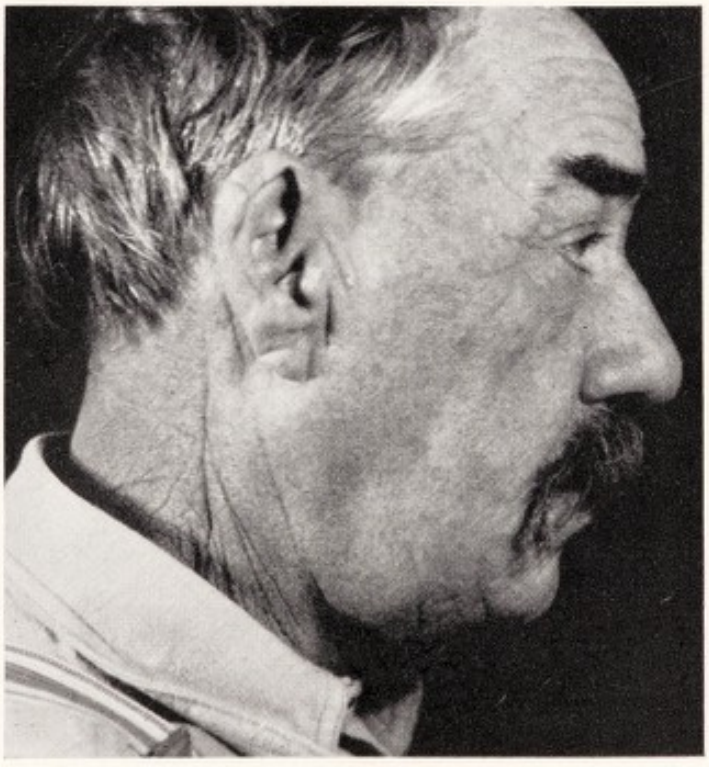

FIG. 196. Bulging of sarcoma of the tonsil. It is located over the angle of the jaw.

Carcinoma of the Tonsil. These are more rare than the sarcomas.

Pathogenesis. Relatively superficial ulcerations when first observed in most cases carcinomas tend soon to invade their environment, but less rapidly than the sarcomas. Usually they do not cause bulging at the angle of the jaw as sarcomas do. On the contrary, invasion of the lymph glands takes place fairly early. Carcinomas kill by interference with nutrition, hemorrhage or pneumonia.

Pathology. The ulcerous lesion is cancer-hard to the touch. One has but to remember the possibility of a syphilitic lesion. Usually the presence of lymph glands in the neck is evident.

Histology. The microscopic structure is that of a very cellular carcinoma.

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CHAPTER XI

Inflammatory Lesions of the Jaws

INFLAMMATORY lesions of the jaws, save for granulomas about the teeth, are not common. This is fortunate for the surgical pathologist, because there is no other lesion of so little interest. All the reward he has for removing an area is just another piece of dead bone. Furthermore, the clinical phases are so plain that there are few chances for surprises in the laboratory. The clinical phases in fact are so unmistakable that there is little need for x-ray examination. In fact the use of x-rays is more apt to mislead than to help in the diagnosis of bone inflammation, and this is particularly true about the jaws, where the securing of a good picture is so difficult. In the diagnosis of tumors the problem is, of course, different.

Local necroses following injuries, notably compound fractures, are occasionally encountered but they have little interest for the surgical pathologist because the relationship is so obvious. Lingering necroses following the removal of infected teeth furnish the largest contingent of cases belonging in this chapter. Enterprising dentists even dispute this ground with the surgeon, as they should. Local necrosis of a considerable area of bone is middle ground and when involving a considerable area usually falls to the general surgeon. The diffuse infectious osteomyelitis, happily rare, is about all that is now assigned to the general surgeon without protest.

These several lesions may be listed as follows:

I. Root Infections
II. Local Necroses
III. Osteomyelitis

While these are, or at least may be, interrelated, it is convenient to discuss them as entities.

ROOT INFECTIONS

The importance of infections of the teeth was appreciated by Benjamin Rush but it is only in recent years that the full import has been recognized. Like most infants in science, it has received more attention than its importance warranted. Not only were demonstrable lesions

attacked but the possible existence of lesions not demonstrable was assumed and the teeth extracted. The argument seemed (or seems) to be that the teeth, not being absolutely necessary to life, may be removed and thus exclude a possible source of the trouble. The philosophy is that of the tax gatherers, if an individual has anything not absolutely necessary for existence, it should be removed. Women at the menopause were particularly the victims of this easy philosophy. Endocrine in nature, as their joint troubles mostly are, the removal of teeth could not possibly have any influence except to make the portly matron better fitted to play the rôle of grandmother. For the spinster there is no consolation at all.

Pathogenesis. Just why infections occur about the roots of teeth is not definitely understood. It is easy to say that the tooth decays and bacteria lodge in the tissues about the beleaguered teeth. This does not reach ultimate causes. Dental hygiene is supposed to combat the tendency of the teeth to decay. This is good esthetics but poor pathology. The general disturbance of health of the individual is the fundamental factor and all the brushing possible will not remedy a defective metabolism.

It is common for writers, when they get to the end of their string, to say that this is not the place for a detailed consideration of this subject, and then go on to prove why this is the case. Yet at the same time such an alibi has its merits, since it is no more transparent than to hide lack of knowledge in a flood of words.

Just what do infected tooth roots do to their possessors? The removal of teeth does not influence acute rheumatic process in adolescents as does the removal of tonsils in children. For one thing root infections are not common at this stage of life. In more chronic pains, the removal of tooth abscesses sometimes is followed by relief. It is the less localized disturbances that are particularly likely to be influenced by such removal. Be this as it may, a demonstrable root infection cannot be regarded as an asset to the patient and its removal is mandatory. Whatever improvement may follow is naturally attributed to such removal. However, the discovery of a root infection should not stop the study of the patient because there may be something else wrong. At least, if the removal of a few teeth does not help, it does not follow that the removal of all the teeth will most certainly do so. Time was when if a female complained, the ovaries were removed, then came the mammary glands as victims to the slaughter. The furor of tooth pulling seems to be subsiding and the problem is being handed back to the dentist where it belongs and he treats only lesions which he can see. Here, of course, the x-ray is of the greatest value in diagnosis. It is not a problem for the general surgeon.

Granulomas about the gums still come to the general surgeon and this is as it should be because of their relation to neoplastic diseases.

Pathology. Due to an infection of a tooth root, granulation tissue forms. Actual pus may form. This lesion is demonstrable by the x-ray as a rarefied area about the apex of the tooth. When a tooth so affected is removed, a granuloma may be attached to the root. This remains attached because the peridental membrane is involved in the process. The granuloma is loosely attached and is easily removed. If a

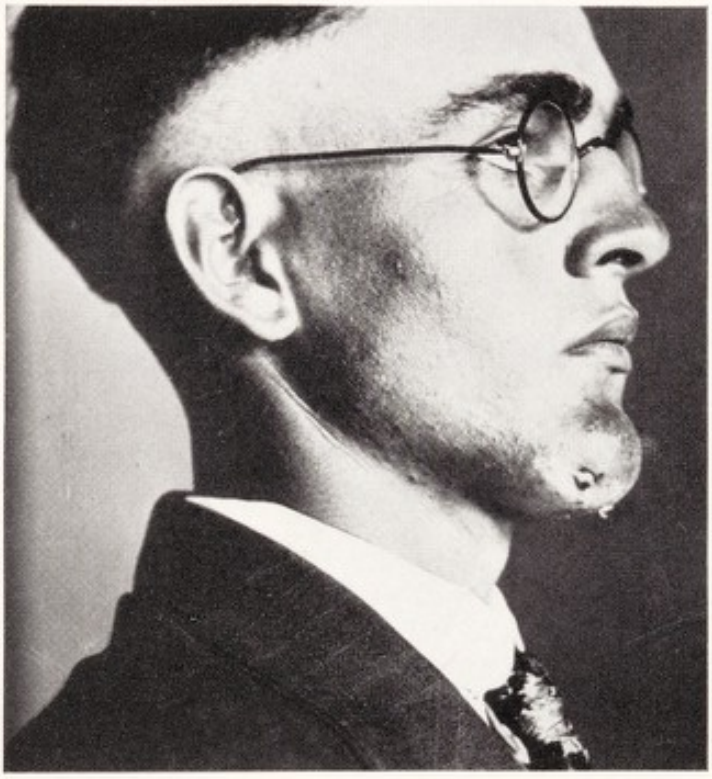

FIG. 197. Sinus on the chin due to a root infection about a bicuspid tooth. The lesion was of some years' duration, as is evident by the scarring about the drainage tract.

tooth abscess continues, the bone about the tooth may become infected and, by a process of necrosis, the bone is perforated and a superficial abscess results (Fig. 197). These lesions are characteristic.

Histology. The granuloma does not differ from granulomas elsewhere except perhaps that, owing to the location, vessels are more sparse than in a surface granuloma (Fig. 198). The attachment to the tooth is very slight. The tooth itself may show more or less necrotic change.

LOCAL NECROSIS

Localized lesions of bone still are the province of the general surgeon unless there be an oral surgeon about to dispute his right. The most com-

mon source of such infection is an aggravated root infection above described, either spontaneous or following the extraction of a tooth. Malerupted teeth not infrequently start a ruckus in their own peculiar way. The most common site is about unerupted wisdom teeth.

Pathogenesis. There is one obvious clinical factor in such cases, the patient comes at once to the point. He has a pain in his jaw. In confirmation of this expressed opinion, in most cases he, uninvited, proceeds to demonstrate the evidence. This may be a swollen gum, possibly a swollen cheek, even a closed eye (Fig. 199). If long neglected, an abscess

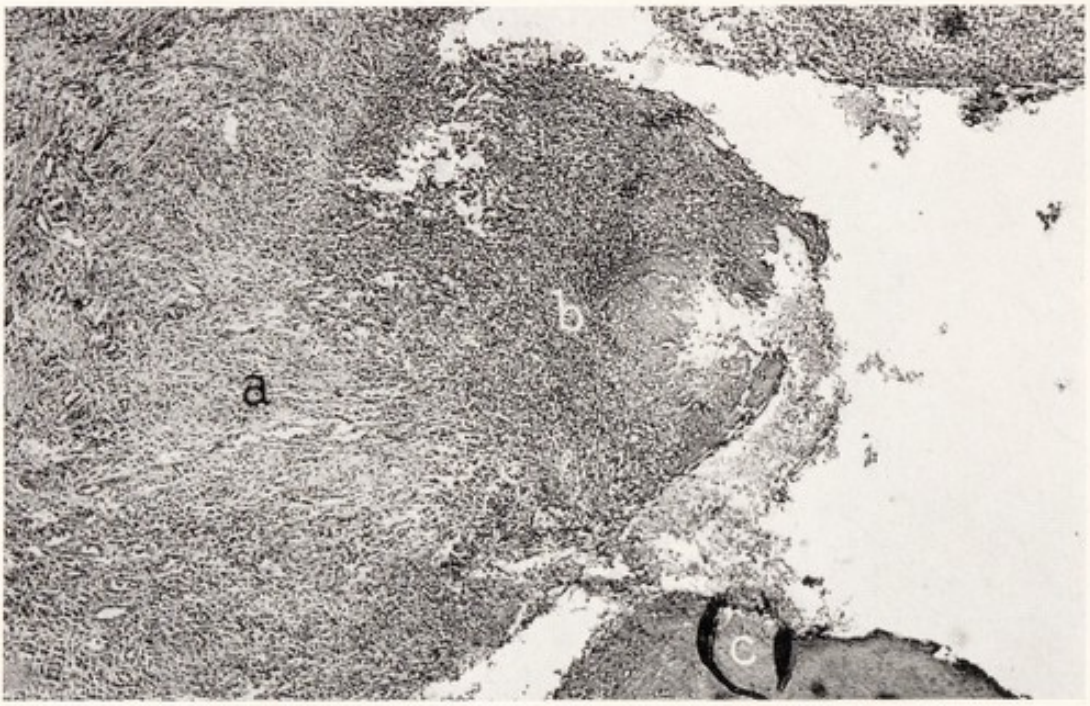

FIG. 198. Granuloma about a tooth root. The abscess is of some duration, as is made evident by the fibrous tissue formation: *a*, fibrous tissue area; *b*, round cells in process of degeneration, the area of actual abscess; *c*, tip of tooth root.

may already exist to emphasize the point the patient is trying to make.

With the discharge of pus the chief pain is relieved and the subsequent course is dependent on the damage done the bone. The necrosed bone must be removed by the surgeon. Because of the region involved, the surgeon desires to remove the lesion as early as possible and in operating early the exact extent of diseased bone is difficult to determine. This leads to repeated operations.

In rare cases the infection may be so extensive that the jaw muscles become fixed and the patient is unable to open his mouth. Worse still, pus may extend down the deep fascia of the neck and reach the medias-

tinum with fatal results. These grave cases usually follow malerupted wisdom teeth, not previously the seat of root abscess. For this reason any irritation on the part of an erupting wisdom tooth forfeits its right of tenure.

Pathology. Just infected bone, nothing else. The local involvement at once declares the nature of the lesion. Not all jaw sinuses are due to local jaw necrosis, however. Actinomycosis is a possibility.

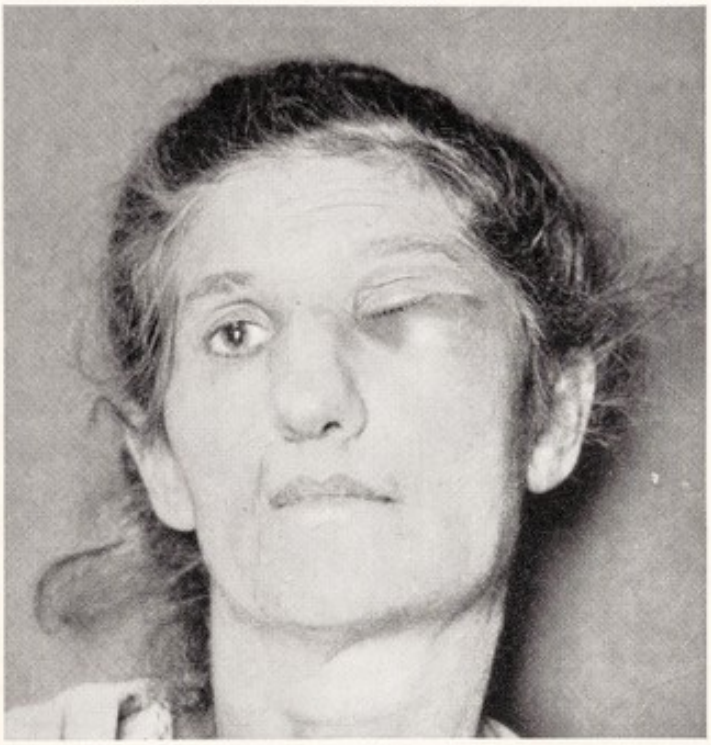

FIG. 199. Swollen cheek due to the presence of bone necrosis of the upper jaw.

Histology. The necrosed bone has no peculiarities. Granulation tissue and bone particles complete the picture.

ACUTE OSTEOMYELITIS

Acute osteomyelitis is discussed more fully in the volume on *Surgical Pathology of Diseases of Bones*, Lippincott's, 1931, p. 46.

Acute bone infections may be but an exaggeration of the preceding, starting from an infected tooth or after the removal of a tooth. In some cases, however, the lesion begins as a hyperacute process involving much of the jaw. This is particularly true of the upper jaw in children. In those cases it parallels the acute myelitides in the long bones in adolescents.

Pathogenesis. The milder progressive cases begin as a localized process, developing more or less as a local osteomyelitis. An abscess may

develop, invading the gums or the soft parts beyond. In a short time the next adjacent area, too, becomes involved; this loosens and is removed. The process may be repeated until all the teeth of one side become affected. In rare cases the other side of the jaw may become likewise affected. Accompanying the local process there is a varying degree of constitutional involvement, fever, high leucocyte count. One can, from the local reaction, gain some idea of what the course will be. If there is a notable sepsis, one may be sure that the invasion is extensive and the area of bone necrosis is likely to be great. In such cases the surgeon may anticipate subsequent events by removing all the teeth on one side, tunneling the entire alveolar process, followed by vigorous chemical sterilization. In these cases the body of the jaw is preserved and after the alveolar infection is controlled, the lesion quickly heals.

In acute osteomyelitis the process is more impressive. With high fever, possibly rigors, even convulsions in children, the entire jaw is swollen without especial involvement of any particular tooth. The periosteum becomes involved and soon elevated. Extensive periosteal pus invasion follows, demanding drainage. With this the acute stage is passed. In time the entire bone is extruded. The entire upper jaw, saving only the orbital plate, may come away as one piece. The lower jaw, being more massive, requires a longer time. Usually before this occurs an involucrum has formed so that the contour of the jaw is maintained after the dead bone has been removed.

Pathology. The pathogenesis as related above tells the entire story. In the localized form, the loosening tooth tells the serious nature of the process. Exploration may reveal a localized abscess. In the diffuse type, general edema may be all that can be discovered. The degree of this may reveal the formation of pus. The signal for the removal of the necrosed bone is demonstrable mobility.

Histology. This differs in no wise from that of osteomyelitis in general. In the localized type, there is usually a mixed culture. In the diffuse type, the staphylococcus is usually the offending organism, so it is said.

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CHAPTER XII

Diseases of the Larynx

THERE are still laryngologists who are not sufficiently surgically minded to be trusted with the major diseases of the larynx. So long as so many of them will pull off the tops of tumors with forceps for therapeutic or diagnostic purposes, they should be under the supervision of general surgeons. I have seen so many errors, and I still see them, due purely to this unsurgical practice. It is a reflection not so much on talent but on method. There is one celebrated case that illustrates this: A distinguished member of a royal family had something wrong with his larynx. A laryngologist bit off a piece with a forceps for microscopic examination. It was examined by a pathologist, quite a pathologist in his day, namely Rudolph Virchow. He found only an inflammatory reaction. A general surgeon took a look and said it was cancer. He felt of it. The patient died of cancer. The pathologist diagnosed only the piece that was given him. It so happened that he was given a piece from the wrong place.

I was once sent a patient for laryngectomy under local anesthetic. The diagnosis was made by a most eminent authority. The diagnosis was cancer. The patient was not sent to me as a surgeon. I was not supposed to use my head, just my hands. It was supposed that I was quite handy with local anesthetics. I did a laryngectomy under local anesthesia. The organ removed, I surreptitiously used my head. It was syphilis.

Repeatedly I have seen patients who had had something wrong with their talking apparatus. Laryngologists looked and saw nothing. They waited until they did see something. Then it was too late. My prize patient was a person of some consequence. He was examined by an even dozen laryngologists, several of national reputation. They saw nothing. He developed a gland in the neck. I block dissected his neck and found a gland definitely malignant. There was no recurrence in the neck, so that it is evident I did my job well. The position of the gland was such that the primary malignant growth could only be in the larynx. I wanted to look in and confirm my diagnosis. Not until three years after did the growth in the larynx become large enough to be seen by the laryngoscopic mirror. Then everybody could see it, but it was too late. Such a slowly-growing cancer would have been pie to the general surgeon, and for a

laryngologist for that matter, who would have split the larynx so that a good direct look could be obtained.

These few typical cases are sufficient excuse for the general surgeon to keep himself informed on the intralaryngeal growths. A neck lesion may indicate to him that there is something wrong in the larynx and one had better take a look. There is no excuse for not taking a look. Splitting the larynx is such a simple procedure under local anesthesia that there is no excuse for remaining in doubt.

The surgical diseases of the larynx are so few that it is no great task for the general surgeon to keep reasonably well informed. They may be listed as follows:

I. The Inflammatory Diseases of the Larynx
II. The Granulomas of the Larynx
III. Nonmalignant Tumors of the Larynx
IV. The Malignant Tumors of the Larynx

Generally speaking each of these diseases is characteristic on direct inspection at operation so that a diagnosis is easy and certain.

INFLAMMATORY DISEASES OF THE LARYNX

Inflammatory lesions of the larynx become of surgical importance only when the swelling is sufficiently great to endanger the lumen of the organ.

Pathogenesis. Surgical lesions of an inflammatory nature are usually transmitted to the larynx from the neighborhood. Major infections of the neck are the cause in the majority of cases but local causes may be active, notably following trauma. The history of sudden onset is usually sufficient to permit a working diagnosis of inflammation. The indication is promptly to eliminate the primary lesion if such is found. If the mechanical interference is at all notable, an artificial opening allows a direct inspection and relieves the threatening sign while the inflammatory lesion is subsiding.

Pathology. Swelling, just swelling, is all one can see if the patient is dyspneic. Because of the difficulty of direct examination and the urgency of the condition, one must depend more on what he hears than on what he sees. If there is no disease of the neck to account for the inflammatory edema and the difficulty persists, the possibility of a primary lesion should be suspected, which admitted, the infection must be thought of.

Histology. The few tissues I have been permitted to examine showed only inflammation.

GRANULOMAS OF THE LARYNX

Only two granulomas need be here considered, tuberculosis and syphilis.

Tuberculosis. I worked for a time in a large foreign clinic where tuberculous ulcers of the larynx were an everyday observation. Naturally they were almost exactly as common in the deadhouse. In this country, in rural communities at least, the lesion is rare. I write, therefore, from memory rather than sustained experience.

Pathogenesis. In most cases there is a co-existent pulmonary tuberculosis. Even if there is no demonstrable lesion the character of the patient suggests the possibility. The slow onset and the age of the patient adds additional evidence.

Pathology. The early lesion may be covered by inflammatory reaction, an edematous swelling. At this stage the associated constitutional conditions lend probability. One has to exclude syphilis. Once ulcer forms, the diagnosis, under the conditions mentioned, is easy. The superficial ulcer, gray base, over-hanging edges and the moderate swelling about it, is all but pathognomonic. If the lesion is so situated that it is accessible to the palpating finger, there is little doubt; the lesion is soft. This is particularly impressive if the lesion is exposed to direct inspection, a procedure wholly justified if it is the major lesion. Even in the presence of pulmonary tuberculosis the treatment of the local lesion with the electrocautery is the best possible treatment besides assuring a positive diagnosis.

Histology. The slide shows the typical lesion, if one finds the right place. It is possible to examine only an inflammatory area.

Syphilis. Syphilis of the larynx comes to the general surgeon only when it is suspected of being something else, as was the case above related. In the early stages swelling may be the only symptom. A swelling without apparent cause must always be suspected of being syphilis.

Pathogenesis. If emergency operation is demanded and only swelling is found the diagnosis becomes highly probable, particularly in certain types of individuals. An apparent perichondritis makes it all but certain. Occasionally one finds like conditions due to constitutional causes. If one's grandmother is the patient, naturally one would think first of nephritis. I once saw a patient who developed acute edema of the larynx following the taking of a teaspoonful of the oil of wintergreen. This was puzzling until one got the history. It was necessary to do a tracheotomy before one could get a history.

Pathology. In perichondritis and the gummatous stage, there may be

only a generalized swelling. The nature of it may not be definite. Once an ulcer is found, the peculiar form suggests the diagnosis. Tuberculosis is never so regular in the ovoid outline and usually is more superficial and is usually less acute in onset. It is distinguished from cancer by the overhanging border and its softness, if accessible to the palpating finger. Of course the laboratory will likely confirm the diagnosis. If not, perhaps a therapeutic test will.

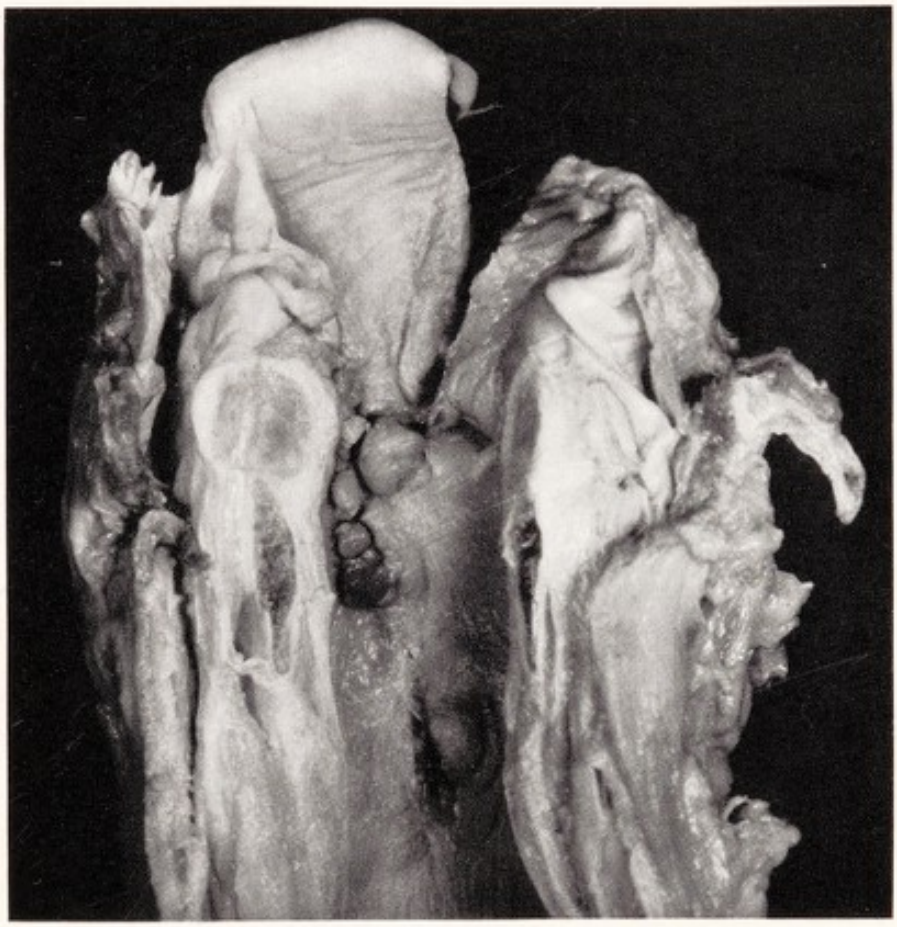

FIG. 200. Multiple polyps of the larynx and trachea. The patient died of suffocation after leaving the hospital after undergoing thyroidectomy.

Histology. If the lesion is not tuberculosis nor cancer and has many thick-walled vessels surrounded by many round cells, likely it is syphilis. Of course if the specific organism is identified, the diagnosis is certain. For this one must ask the laboratory man. The microscopic diagnosis is seldom a task for the surgeon.

NONMALIGNANT TUMORS

If one reads over-much this subject is confusing. If he has had experience it is simple. He remembers but two tumors: papillomas and polyps.

Papillomas. These are the commonest tumors of the larynx but they seldom come to the general surgeon. They are simple and long-suffering and will ultimately disappear under the kind, any kind in fact, ministration of the laryngologist.

Pathogenesis. Unfortunately over-use of the vocal cords does not appear to be an etiologic factor. They just come. Huskiness of speech is the chief symptom with a sense of discomfort when the size or situation causes irritation. The slow growth and the age of the patient may give a preliminary suggestion.

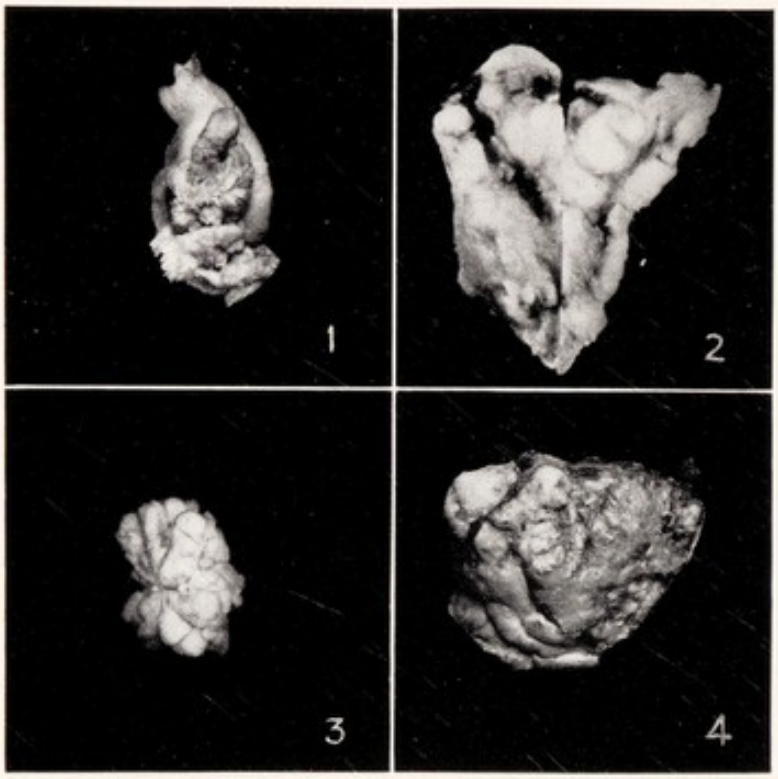

FIG. 201. Tumors of the larynx: *1*, early papillary cancer of the larynx; *2*, nodulated epithelioma; *3*, benign bosselated papilloma; *4*, diffusely infiltrating cancer.

Pathology. They may appear as individual warty-like tumors or they may appear in clusters covering a considerable surface. If composed chiefly of fibrous tissue, they may be rounded (3, Fig. 200). They are most likely to be situated on the cords but may extend on other surfaces.

Histology. The lesions are made up chiefly of a piling up of epithelium. In all cases the base must be available for examination because the top may be made up of simple epithelium while the base is invaded.

Polyps. This is an unusual situation for these tumors as compared with the nasopharynx but because of their location they may become

suddenly of great importance since they may cause occlusion of the respiratory tract.

Pathogenesis. It is likely some inflammation starts them growing. We may assume this because no other cause is known, and we may draw conclusions from like growths higher up, the nature of which we do not know over much.

Pathology. Pale pinkish rounded tumors, I reckon, should be visible with the mirror. Sometimes they are situated so low that they escape

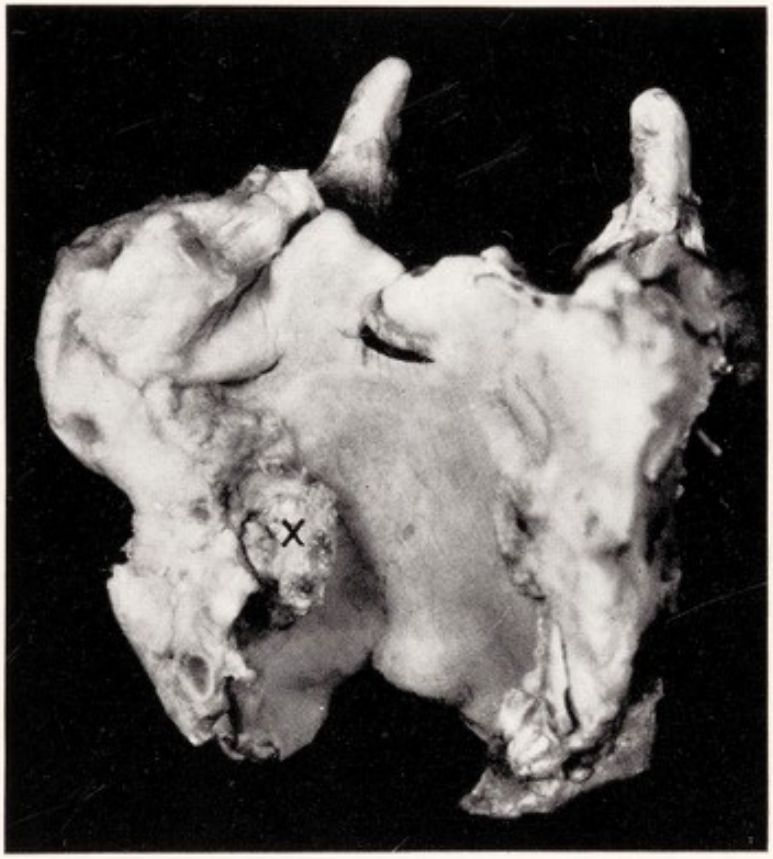

FIG. 202. Cancer of the cord extending to the adjacent soft parts.

vision. So far as I am concerned they have become visible only at operation, or at autopsy. When exposed to view, the diagnosis is easy. It seems possible that inflammation or circulatory reaction may cause them to become edematous and produce suffocation. At least we had a patient who died in bed after leaving the hospital following a thyroidectomy (Fig. 201).

Histology. The structure is that of polyps elsewhere. The few that I have seen showed a greater cellularity than the nasal polyp. This may have been due to associated circumstances that caused the tissue to be-

come available. An extended histologic study does not seem to have been made.

MALIGNANT TUMORS OF THE LARYNX

I need consider here only the intrinsic tumors of the larynx. They are the only kind that offer any prospect of cure to the operating surgeon. By so confining myself, the field is confined to the epithelial malignancies. At least so far as my experience goes this excludes the sarcoma group of

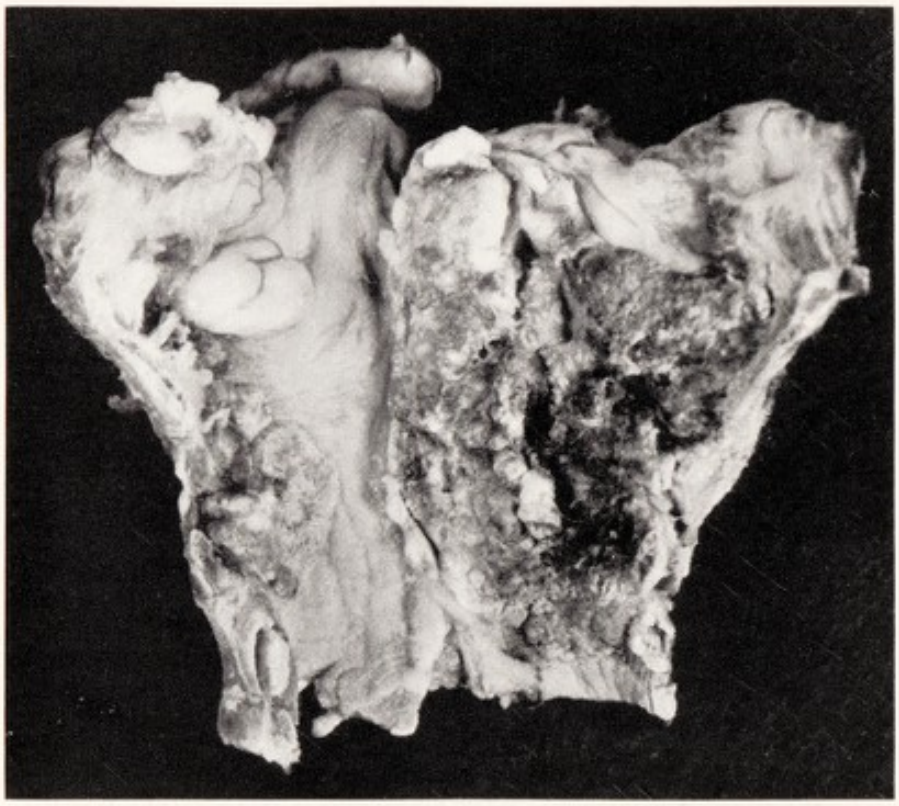

FIG. 203. Carcinoma of the larynx. The greater part of the laryngeal ring is involved but the growth does not extend beyond the cartilage.

tumors. These all come from the neck and invade the larynx secondarily, usually as a terminal process. Intrinsic sarcomas reported lack definiteness.

Carcinomas. The intrinsic epitheliomas of the larynx range along with cancers of the big gut as furnishing the best prognosis after radical operation. Discovered early and completely removed, they furnish a pleasing number of lasting cures, this too without sacrificing the larynx. But the operation must be done early, usually before the diagnosis, viewed through the mirror, is positive.

Pathogenesis. What proportion develop on preceding papillomas

cannot be positively determined. The history of some cases suggests that they are not rare. The initial symptoms are much the same, just laryngeal irritation. There seems to be no other early sign. Obstruction to respiration is, of course, always a very late sign. It establishes not only the diagnosis but also the prognosis. Pain may be classed in the same category, certainly if the pain is due to invasion. Lymph gland metastasis naturally falls in the same class. Nevertheless the isolated invasion

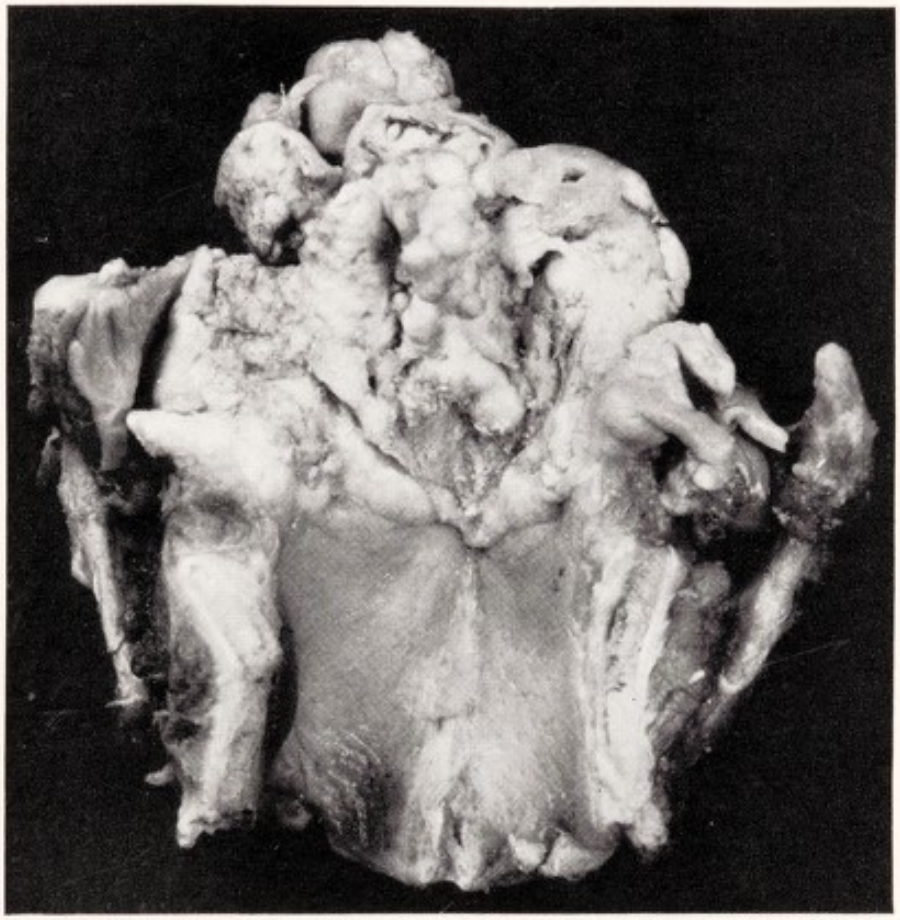

FIG. 204. Cancer of the larynx invading the surrounding tissue, completely destroying the epiglottis. Should have been recognized as inoperable.

of the lymph gland located just beyond the horn of the hyoid bone may be the first positive sign. In my experience these have been found long after intralaryngeal irritation should have led to exploration. A good prognosis is possible only when the lesion is discovered so early that local excision is possible. When the lesion is so advanced that a laryngectomy is needed, the prognosis is always bad. It is bad when it develops or extends to the fauces. There, flanked by the cartilage, the prognosis is best. This structure seems to furnish a certain resistance to extension.

Pathology. On inspection, early carcinoma presents an irregular wart-like elevated lesion (1, Fig. 201). If early, it is confined to one small area. If they develop in the chink, they may attain some size before they are visible. The structure may be wart-like, showing little invasion. These

FIG. 205. Extensive invasion of laryngeal cancer. Tracheotomy was followed by pneumonia.

are the least malignant. Nodulated tumors come next (2, Fig. 200). Ulcerations which extend without any evidence of limitation to the surrounding tissue are more malignant (4, Fig. 200). More diffuse lesions extending to the adjacent soft parts require laryngectomy (Fig. 202). Operation is technically possible when there is considerable invasion of the soft parts (Fig. 203). Once the growth has extended to the uvula

and pharynx, though they may be technically operable, the prognosis is bad (Fig. 204).

When there is associated lymph gland metastasis, the disease is really inoperable. If one must operate, the neck should be blocked out first and the primary tumor removed a week later. If both are done at one sitting, the chances of a diffuse neck infection is very great.

It is amazing what degree of invasion is possible, compatible with life, before the patient comes for a terminal tracheotomy (Fig. 205). Tracheotomy in such cases is to rescue the patient for the time being, only to

FIG. 206. Early malignancy of the epiglottis apparently developed on a diffuse papillary growth.

have him die of a renewed obstruction later. One has the hope, of course, that a terminal pneumonia may rescue them from a worse fate.

Intratracheal growths may complicate cancer of the thyroid gland. One must think of such a possibility if dyspnea persists after a thyroidectomy for malignancy. I have reported one such case (*Diseases of the Thyroid Gland*, Fig. 144, p. 252, ed. 3, 1935, Mosby Co., St. Louis).

Histology. The microscopic structure may show a predominating papillary structure if seen early (Fig. 206). Diffuse invasion may appear in the primary growth or may follow the extension from the less vicious papillary form. The most malignant type begins as an ulcerous lesion and rapidly invades the adjacent tissue.

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