Clinical Problems

Related to the **TONGUE**^{*}

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DIGEST

In the physical examination of the head and neck the oral cavity too frequently receives cursory consideration. This article presents an outline of the many local and systemic diseases which may affect one region: the tongue. The fact that numerous generalized diseases are mirrored in various lingual changes is generally appreciated. Few of the lingual signs are pathognomonic. In the constellation with other historic and physical findings, however, the tongue changes will often decide the diagnosis.

Anatomy and Physiology of the Tongue

Anatomy—The tongue is a highly vascular and muscular organ occupying most of the floor of the mouth and forming the anterior wall of the oropharynx. The dorsum is divided into an anterior two thirds and a posterior third by a V-shaped groove, the sulcus terminalis. The dorsum in the relaxed tongue lies just below the occlusal level of the lower teeth. The position of the tongue can be variable and is of importance in tongue habits. Embryologically, the tongue is formed from the fusion of the first, second, and third branchial arches. Persistence of the tuberculum impar, which should normally recede as the two halves of the tongue fuse, results in a congenital lesion

*Adapted from The Pediatric Clinics of North America, November, 1956, pp. 919-33, published by W. B. Saunders Company, Philadelphia. called median rhomboid glossitis. Three Types of Papillae: The squamous epithelium covering the dorsum is specialized and unlike the surface epithelium of other mucosal tissues. On the anterior two thirds three types of papillae are recognized: (1) filiform, (2) fungiform, and (3) circumvallate.

The Filiform Papillae: These contribute to the velvet-like quality of the tongue. They are thin, jagged, epithelial projections, between 1 and 3 millimeters in length, and are generally arranged in rows. These papillae undergo continual desquamation and regeneration. Their presence or absence contributes greatly to lingual coating and smooth tongue. In children the filiform papillae are not as pronounced as in the adult.

The Fungiform Papillae: These are interspersed among the filiform papillae but are more numerous along the side and tip of the tongue. They are mushroom-shaped, larger, and are readily distinguished by their redness. They are not as sensitively attuned to metabolic changes as the filiform type.

The Circumvallate Papillae: Numbering eight to ten, these are arranged in a V-shaped line just anterior to the sulcus terminalis. They are large and flat and are surrounded by a groove containing serous glands and taste buds. The foramen cecum is located at the apex of the V. This represents the embryologic vestige of the thyroglossal duct, a remnant of the early development of the thyroid gland. The Posterior Third: The surface of this part of the tongue contains lymphoid follicles, collectively referred to as the *lingual tonsil*.

Sensory Innervation: The general sensory innervation to the anterior two-thirds of the tongue is mediated by the lingual nerve and the gustatory sense by the chorda tympani branch of the facial nerve. Both general and taste sensation of the posterior third is served principally by the glossopharyngeal nerve and to a minor degree by the vagus. Motor innervation to the entire organ is made possible largely by the hypoglossal nerve.

Physiology—The tongue subserves four functions: (1) taste, (2) speech, (3) mastication, and (4) deglutition.

Taste: The taste buds are to be found in the circumvallate papillae and to some extent in the fungiform papillae. There are taste receptors, however, in other parts of the oral cavity. Though no anatomic support can be found, it is probable that there are four types of taste receptors for sweet, sour, bitter, and salt.

Speech: The phonatory importance of the tongue is readily evident from the observation that the production of many consonants depends upon the proper positioning of the tongue with respect to the teeth and palate.

Mastication: In order for food to be divided and macerated, it must be tossed upon the broad grinding surfaces of the posterior teeth. This is made possible by the extraordinary mobility of the tongue.

Deglutition: The first, and the only voluntary phase of deglutition is made possible when the posterior portion of the tongue containing the bolus of food is depressed while the apex is elevated. This movement hurls the

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TABLE 1.

Diseases Which Produce Macroglossia

Infectious Diseases

Actinomycosis Histoplasmosis Ludwig's angina Smallpox Thrush Tuberculosis Nonspecific glossitis

Physical Disorders

Foreign bodies Jaw fractures Mechanical edema

Chemical Disorders

Plumbism Wasp and bee stings

bolus into the pharynx, and the second stage of deglutition begins.

Clinical Examination of of the Tongue

Methods of Investigation — The tongue can be examined by (1) inspection, (2) palpation, (3) with the aid of a pharyngeal mirror, and (4) Wood's light.

Inspection: The protruded anterior portion of the tongue is easily visualized. To observe the lateral borders, the tip of the tongue is held with gauze and gently retracted to the right and left.

Pharyngeal Mirror: A tongue blade and a pharyngeal mirror are useful for visualizing the posterior aspect.

Palpation: Bimanual palpation is desirable for examining the corpus of the tongue and the sublingual space. This is best accomplished with the tongue in a relaxed position.

Wood's Light: When the mouth is flooded with filtered ultraviolet light, excluding the visible rays, the dorsum of the tongue often displays a redorange fluorescence believed to be due to the presence of porphyrins. In the

Neoplasms Fibroma Hemangioma Lymphangioma Lymphosarcoma Lipoma Neurofibroma Multiple myeloma Papilloma Rhabdomyoma

Cysts Mucocele Thyroglossal duct cyst

Hormonal Disturbances

Cretinism Juvenile myxedema Diabetes mellitus Hypophysial gigantism

Developmental Disturbances

Congenital muscular macroglossia Mongolism Gargoylism

Nutritional Disorders Ariboflavinosis Beriberi Pellagra Vitamin C deficiency

Stress Reactions Angioneurotic edema Erythema multiforme Serum sickness Stomatitis venenata Urticaria

Miscellaneous Disorders Cardiac decompensation Cardiac glycogen disease Progressive muscular dystrophy

TABLE 2. Diseases Which Produce Hypomobility of the Tongue

Infectious Diseases Bulbar poliomyelitis Infective glossitis Sublingual infections

Physical Disorders

Ankyloglossia Traumatic glossitis

Neoplastic Diseases Teratoma

Sublingual neoplasms

Cysts

Thyroglossal duct cyst Sublingual cysts

Stress Reactions Scleroderma

Miscellaneous Disorders

Myasthenia gravis Amyotrophic lateral sclerosis Myotonia congenita Cerebrovascular accident Bulbar paralysis Severed hypoglossal nerve Syringomyelia Cardiac glycogen disease majority of healthy, well-nourished people the fluorescent area extends either over the entire dorsum or at least its anterior half. It has been observed that with papillary atrophy, as is seen in the so-called smooth tongue, there is an absence of this fluorescence. That the various vitamin B fractions seem to influence the restoration of fluorescence is doubtless due to their action in regenerating the papillae. There is, however, no absolute proof at present that reduced or absent fluorescence is always due to a vitamin deficiency. Fluorescence seems to decrease with age even though there is no obvious disease or malnutrition.

Symptoms

By definition, a symptom is *subjective*, and pain is the most common complaint.

Pain—The most important cause for pain is mechanical trauma to the tongue which may develop with accidental biting or other traumatic conditions. Next in frequency are probably systemic and local infective states leading to vesicle and ulcer

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BLE 3.

Diseases Which Produce Color Changes in the Tongue*

ctious Diseases

Karly scarlet fever (W) Late scarlet fever (R) Thrush (R with W curds) Measles (R) Smallpox (R) Fusospirochetosis (R) Gonococcal stomatitis (Y) Nonspecific glossitis (R) Secondary syphilis (W) Histoplasmosis (W)

Physical Disorders

Galvanism (R) Traumatic glossitis (R) Traumatic hyperkeratosis (W)

*The characteristic color change is indicated in code: R, red; W, white; Y, yellow; B, black; G, green; Bl, blue; Br, brown; and P, pale. **Chemical Disorders**

Aplastic anemia (R because of purpura) Sodium perborate (R) Candies (variable) Black hairy tongue (B,Br,Y,G)

Neoplastic Diseases

Fibroma (R) Hemangioma (R) Papilloma (W) Lipoma (Y) Rhabdomyoma (P) Myoblastoma (P)

Cysts

Mucocele (Bl)

Hormonal Disturbances Diabetes mellitus (W)

formation. In some cases lingual pain is described as soreness or burning.

Deficiency Glossitis: When this symptom accompanies papillary atrophy, erythema and enlargement, a deficiency glossitis should be suspected. Such a clinical picture may result singly or in combination with microcytic hypochromic anemia, macrocytic hyperchromic anemia, and vitamin B deficiency states which may in turn be related to gastrointestinal

Diseases Associated WithTABLE 4.Lingual Coating

Infectious Diseases **Chemical Disorders** Scarlet fever Agranulocytosis Diphtheria Black hairy tongue **Fusospirochetosis Neoplastic Diseases** Measles Leukemia Typhoid fever Typhus fever **Hormonal Disturbances** Yellow fever Diabetes mellitus Pneumonia Tonsillitis **Reactions to Stress** Pharyngitis Rheumatic fever Laryngitis Anxiety Acute febrile diseases **Psittac**osis Physical Disorders Traunatic glossitis Mouth breathing **Miscellaneous Disorders** Gastritis Cardiospasm Intestinal and pyloric obstruction **Insuffi**cient mastication of food Renal failure Decreased tongue mobility High intake of soft and liquid foods

disturbances. A protein deficiency, particularly of tryptophane, may induce such a glossitis.

Diagnostic Measures: Since the atrophic changes are not pathognomonic, blood studies, gastric analysis, and an adequate dietary history are necessary to make a diagnosis. In addition, a thorough physical examination should be directed toward finding other confirmatory evidence, such as angular cheilosis, dermatitis, and lassitude.

Diagnosis: The presence of pain becomes important in differentiating deficiency glossitis from (1) geographic tongue, (2) median rhomboid glossitis, (3) black hairy tongue.

Other Symptoms — Fusospirochetosis frequently produces a metallic taste. True abnormalities, however, in the gustatory sense are exceedingly rare. Difficulty with speech and faulty articulation may be associated with macroglossia, hysteria, and various neurologic disorders.

Signs

By definition, a sign is an objective finding. The examination of the tongue should include an appraisal of the following conditions: (1) tongue size, (2) position and mobility, (3) color, (4) coatings, (5) primary and secondary dermatologic lesions, (6) papillary status, (7) grooves, (8) scars, and (9) malformations.

Tongue Size—In the final analysis there are only three possibilities: (1) enlarged tongue, (2) normal tongue, (3) small or absent tongue. Tongue enlargement is by far the most common pathologic condition.

Macroglossia: This condition, also known as enlarged tongue, may be (1) generalized, or (2) localized in its distribution. Thus the entire tongue may be involved, or only one portion. For example, congenital muscular macroglossia is of the generalized type, whereas infection, simple mechanical trauma, or a neoplasm produces enlargment of only a segment of the tongue.

Transient or Permanent Macroglossia: Infectious glossitis, for example, produces swelling for a limited time.

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In contrast, developmental or untreated hormonal disturbances yield a more or less permanent macroglossia.

Congenital or Acquired Macroglossia: The infant may be born with a large tongue or this condition may arise postnatally. Hemangiomas and lymphangiomas are relatively common examples of congenital neoplasms which lead to localized enlargement.

Absolute or Relative Macroglossia: There may be a true enlargement which can be traced to an increase in cellular (for example, rhabdomyoma), or extracellular (for example, edema) factors. Or, the tongue may only appear large because the neighboring structures are small: the tongue may seem large with respect to a small mouth. A relative macroglossia is observed in the young, healthy infant. It is also encountered in mongolism. The diseases of pediatric interest which produce macroglossia are classified in Table 1.

Microglossia and Aglossia: A small tongue (microglossia) and its total absence (aglossia) are exceptionally rare. Usually, in children, a reduced or absent lingual organ is of congenital origin.

Tongue Position and Mobility — The tongue can assume only a limited number of positions: (1) elevation, (2) glossoptosis, or (3) forward thrusting. As already stated, the tongue is capable of a great range of motion. Any consideration of tongue pathology must necessarily include a discussion of (1) tongue elevation, (2) glossoptosis, (3) hypermobility, and (4) hypomobility.

Elevation of the Tongue: This common condition is generally associated with infections and particularly those which develop in the sublingual space. Similarly, any neoplasm or cyst in this region can cause such displacement.

Glossoptosis: In this condition the tongue is displaced in a posterior direction possibly to the point of respiratory obstruction. It may be seen in congenital mandibular hypoplasia.

Forward Thrusting: This habit often follows efforts at breaking a previous thumb-sucking habit, particularly when an open bite has been pro-

TABLE 5.Diseases Producing LingualUlcerations

Infectious Diseases Fusospirochetosis Diphtheria Syphilis Tularemia Actinomycosis Blastomycosis Histoplasmosis Thrush Herpes simplex Herpes zoster Pertussis

Physical Disorders Galvanism Mechanical and thermal trauma

Chemical Disorders

Aplastic anemia Agranulocytosis Simple chemical burn (aspirin)

Reactions to Stress Urticaria

Erythema multiforme Lupus erythematosus Scleroderma

TABLE 6.

Diseases Producing Papillary Atrophy

Infectious Diseases Syphilis Thrush

Physical Disorders Traumatic glossitis

Hormonal Disturbances Hyperthyroidism Cretinism Juvenile myxedema

Nutritional Disturbances Ariboflavinosis Pellagra Celiac disease

Developmental Disturbances Geographic tongue

Miscellaneous Disorders Hypochromic anemia Congestive heart failure duced. The patient will thrust the tongue into the open space each time he swallows. This action obviously perpetuates the dental deformity. Psychotherapy may be required to correct this habit.

Hypermobility: This fairly common neuromuscular sign is usually related to anxiety. Marked tremors on protrusion, however, frequently suggest Sydenham's chorea. This sign may also be present in chemical intoxications (for example, plumbism) and also in hyperthyroidism and rheumatic fever.

Hypomobility: This is invariably significant since, under normal conditions, the tongue is extremely mobile. A painful glossitis, neurologic disorders which involve the muscles of the tongue, and ankyloglossia (tonguetie) all restrict the range of tongue action. The incidence of lingual hypomobility in cardiac glycogen disease is great and should be born in mind. Table 2 presents an outline of conditions producing this sign.

Color Changes in The Tongue — Seldom are color changes the only sign of a disease. Alterations in size, papillary status, and subjective complaints usually add to the picture. With the exception of the white coated tongue, the redness of inflammatory erythema is the most frequent color alteration. Such erythema is either localized or generalized and may or may not be painful. Usually, papillary atrophy accompanies it.

Localized Erythema: Generally the result of physical trauma, a badly broken down tooth, an irritating restoration, or an ill-fitting appliance. Occasionally this erythema is accompanied by a nonspecific glossitis, or, rarely, a lingual abscess. Median rhomboid glossitis is characterized by a smooth, shiny, red, oval plaque located in the midline just anterior to the circumvallate papillae. It is a congenital, benign, asymptomatic lesion. Lingual hemangiomas are characteristically dark blue-red and are of particular interest in pediatrics because they are congenital and are usually recognized in infancy.

Generalized Erythema: This has long been recognized in the various

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No part of this research may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or by any information storage and retrieval system, without permission in writing from the publisher. Visit http://ppnf.org for more information. vitamin B complex deficiency diseases. Pellagra, sprue, ariboflavinosis, and juvenile pernicious anemia are accompanied by a red and painful glossitis. The pellagrous glossitis is described as beefy red, whereas the tongue of ariboflavinosis is magenta. Considerable supportive evidence is required in addition to the tongue findings for diagnosis. The nutritional deficiencies are so frequently multiple that it is seldom that a pure syndrome is recognized.

Other Examples: The pallor of the mucous membranes in anemia, the increased redness in polycythemia and hypertension, and the yellow cast seen in jaundice are examples of the potential importance of this clinical sign. Diseases which present color changes in the tongue are outlined in Table 3.

Tongue Coatings-It should be remembered that the tongue is frequently coated under physiologic conditions, particularly upon arising in the morning. Such coating results from the passive accumulation of bacteria, mucin, food debris, and desquamated epithelium from the filiform papillae. An actual increase in thickness of the papillary epithelium plays a more important role in coat formation than was frequently thought. In support of this is the fact that various dyes painted on the dorsum may require several days before they are removed by the process of desquamation. In healthy persons, chewing, talking, and salivation mechanically cleanse the tongue.

Other Causes of Coating of Tongue: Heavy lingual coatings are observed in children who are habitual mouth breathers or suffer with febrile and other dehydration states. The cause of such lingual coatings is not entirely clear. Drying of the oral mucosa, however, lack of mechanical cleansing of the tissues due to nondetergent diets, local hypoxia, and regional vasomotor changes all probably serve etiologic roles. A theory of retrograde intestinal flow has been advanced to explain the frequency with which tongue coating and alimentary problems are associated but attempts to prove this theory have not been successful.

Intestinal Disorders: Contrary to

popular belief, constipation is not associated with tongue coating, although the vomiting of intestinal and pyloric obstructions invariable produces a thick lingual coat. This is found also in severe cardiospasm with esophageal retention. The underlying mechanism is obscure.

Other Disorders: Coating may be produced on the paralyzed side in various neurologic disorders affecting the hypoglossal nerve. Kidney disease does not generally affect the tongue with the exception of renal failure. In this condition the coating is brown and is believed to be due to the excretion of nitrogenous products in the saliva. Most generally, the color of

TABLE 7. Diseases Producing Fungiform Hypertrophy

Infectious Diseases Scarlet fever Measles

Chemical Disorders Black hairy tongue (filiform hypertrophy)

Hormonal Disturbances Diabetes mellitus

Nutritional Diseases Ariboflavinosis Pellagra

Developmental Disturbances Fissured tongue

the coated tongue is white, but may be changed by foods, tobacco, and medicaments. A summary of the diseases associated with lingual coating is presented in Table 4.

Vesicles—There are four primary epithelial lesions: (1) macules, (2) papules, (3) vesicles, and (4) pustules. Of this group, the vesicle is the most common lesion observed on the tongue. Broadly speaking, vesicles may occur in connection with viral diseases (for example, herpes simplex and chickenpox) and thermal trauma, and as a reaction to stress and to an-

tigenic substances (for example, erythema multiforme). It should be recalled that a vesicle or bulla is thinwalled and that in the presence of the usual intraoral trauma, the vesicle is short-lived. The end result is an ulcer. Thus it is indeed rare to observe vesicle formation anywhere in the oral cavity and especially in a hypermobile and constantly traumatized organ like the tongue.

Ulcerations—There are more than a score of secondary dermatologic lesions (1) nodules, (2) crusts, (3) scabs, (4) excoriations, (5) wheals, (6) fissures, and, most important to this discussion, (7) ulcers. The most frequent cause of lingual ulcerations in children is mechanical trauma. This may occur from tongue biting or irritation from a sharp edge on a tooth. Ulcerations often occur after the ingestion of a hot liquid. It can be observed from Table 5 that there are many disorders accompanied by ulcers on the tongue.

Papillary States—The lingual papillae are highly sensitive structures which change in size under a variety of conditions and can actually grow in a matter of days. Basically, there are two pathologic possibilities: (1) atrophy of the papillae, and (2) papillary hypertrophy.

Atrophy: The smooth or atrophic tongue is always the result of the combined effect of normal or accelerated degeneration and deficient regeneration of the filiform papillae. Although the exact mechanism is not known, tissue respiration appears to be a cardinal factor. A disturbance in the intracellular respiratory enzyme systems is likely at fault in the riboflavin and niacin deficiency states. The pellagrous tongue is fiery red, edematous, and tender. As the condition advances, the filiform papillae atrophy. Tissue hypoxia may also explain the atrophy which occurs in severe cardiac decompensation, and perhaps iron deficiency anemias. It is interesting that both hyperthyroidism and hypothyroidism have been associated with smooth tongues.

Geographic Tongue: This occurs in children, notably in those suffering with mild febrile diseases. It is a benign, usually asymptomatic lesion

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characterized by irregular patches of filiform papillary atrophy surrounded by a gray-yellow border. The atrophic areas migrate continually and therefore produce a "geographic" appearance. It is not related to any deficiency disease and no therapy is required. Table 6 schematizes conditions associated with papillary atrophy.

Hypertrophy: Black hairy tongue (lingua nigra) is the only condition which produces enlargement of the filiform papillae. It is a rare asymptomatic disorder affecting most frequently young adult males. The papillae may overgrow to the extent that they actually tickle the palate. The pigment depends upon the types of chromogenic organisms trapped between the papillae and is usually black, brown, yellow, or green.

Incidence: The incidence of hypertrophy has greatly increased since the advent of antibiotic lozenges. This would suggest an alteration in the balance of normal flora permitting various fungi to flourish.

Treatment: Good oral hygiene should be established and any topical antibiotic agent should be discontinued. In persistent cases the topical application of 10 to 15 per cent salicylic acid may be helpful. A 3 per cent hydrogen peroxide mouthwash will remove discoloration, and the application of a 20 per cent aqueous solution of sodium caprylate may be used as a fungicide.

Other Conditions: The fungiform

papillae are sometimes truly enlarged in ariboflavinosis. The tongue may be slightly edematous and also show prominent fungiform papillae in thiamine deficiency, although this is not a constant or significant finding. The so-called strawberry tongue of scarlet fever is seen in only slightly more than half the cases. An early white tongue coat is noted with prominent fungiform papillae (white strawberry tongue). Three or four days later the coating disappears and a red beefy swollen tongue results (red strawberry tongue). Table 7 summarizes these papillary changes.

Grooves — The median raphe is normally prominent. Usually there are also parallel furrows on both sides of the raphe. When the fissures are irregularly arranged, the term scrotal tongue is applied. Fissuring is generally held to be congenital and may be inherited as an irregular dominant characteristic. Sometimes congenital fissuring is associated with slight macroglossia, prominent fungiform papillae, and geographic tongue. It is of interest that there is a characteristic fissuring of the tongue in mongolism.

Scars—Tongue biting in grand mal epileptic seizures and in nonepileptic infantile convulsions is the most frequent cause of scarring, and preventive measures to guard against this accident should always be taken.

Malformations—Ankyloglossia, fissuring, and median rhomboid glossitis constitute the most common malformations. Median rhomboiad glossitis is relatively rare, the character istics are specific, and its recognition is simple. Nevertheless, many of these cases have been erroneously diagnosed as a lingual neoplasm. It occurs as a slightly elevated red oval patch in the midline of the dorsum of the tongue and just anterior to the circumvallate papillae. It is devoid of filiform papillae, so that its surface is usually smooth and glistening, although it may present a nodular appearance. This condition is benign, congenital, and asymptomatic.

Summary

Pathology of the tongue becomes clinically apparent by way of symptoms and signs. The most important symptom is pain, although there may be alterations in taste, difficulty in speech or swallowing. Signs of lingual pathology are apparent by changes in tongue size, position, mobility, color, coatings, dermatologic lesions, papillary status, grooves, scars, and malformations. Since no one symptom or sign is pathognomonic, it is necessary to fit together the positive subjective and objective findings. These, in combination with an adequate history and physical examination, provide the only means of arriving at an etiologic diagnosis of pathosis in the tongue.

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