Chapter Nineteen

The Clinical Evaluation of Nutritional Status

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Dentists, in general, have not availed themselves sufficiently of the simple but valuable tool of clinical inspection to obtain insight into the adequacy of the nutritional state of their patients and to identify the relationship of nutrition to some types of oral pathology. Although more time-consuming, the taking of a brief dietary history when a patient is first seen or when suspicious clinical signs appear also will be rewarding.

The dentist should become particularly familiar with the clinical signs suggestive of nutritional deficiency and should look for them routinely in his patients. It is convenient to do so, as he has ample opportunity to examine closely and under good light those body areas in which such signs are most likely to be detected: the hair, eyes, lips, mouth and skin of the face, neck and arms. No special equipment is required, and the patient need not disrobe. It should be very clear, however, that these tissues can respond to stimuli in only a limited number of ways; hence, the signs are seldom diagnostic of nutritional deficiency, merely suggestive of the problem. Suspicions aroused by the clinical examination must be verified by medical and dietary history and biochemical tests.

Clinical examination of the individual for signs suggestive of nutritional deficiency is an integral part of all nutrition surveys. The U.S. Interdepartmental Committee on Nutrition for National Development (ICNND) (1963) has had worldwide experience in this field and has developed a manual of procedures. A more concise document on the subject has been prepared by a committee of experts convened in 1962 by the World Health Organization (1963). Both include methods of collection and evaluation of dietary, biochemical and anthropometric data, as well as clinical data. Much of the information compiled in population surveys can be used in the appraisal of the nutritional status of patients, but interpretations may not be fully reliable.

MEDICAL HISTORY

A detailed medical history for diagnostic purposes is outside the role of the dentist, but he should inquire of the patient about the presence of diseases which may influence the interpretation of clinical signs of nutritional deficiency or be pertinent to the dentist's handling of oral problems of the patient. These will include acute and chronic infections and disorders such as diabetes, hypertension, heart disease, and allergy to foods or drugs. Other diseases that may condition secondary malnutrition are shown in Table 21-1, Chapter Twenty-one.

CLINICAL EXAMINATION

Each patient should be looked at as a whole before examination of specific areas, for general evaluation will enable the dentist to ascertain whether a person is grossly overweight or underweight, or has excessive pal-

I-373 En: The Science of Nutrition and its Application in Clinical Dentistry. Ed. Abraham E. Nizel. 2nd ed. Philadelphia, and London. W.B. Saunders Company, 1966, p. 235-246.

FIGURE 19-1. *Flag sign of poor nutrition.*

lor, generalized skin lesions or other indications of unsatisfactory health that may possibly be related to diet. At this stage the height and weight of the patient should be obtained and possibly skinfold thickness measured at selected sites.

Once the patient is seated in the dental chair, the following order of examination will be convenient:

General inspection
Hair
Eyes
Face
Neck
Arms
Lips
Tongue
Teeth and gums

(The only additional examinations made routinely by physicians when they are making clinical nutrition surveys are of the skin of the legs and feet, inspection or palpation of the ankles for edema, with determination of the presence of normal knee and ankle jerk reflexes, and a tuning fork test for vibratory sense. Rarely do these add any pertinent information except among rice-eating peoples or alcoholics, in whom altered reflexes due to thiamine deficiency may be encountered; very mild cases of kwashiorkor also show lesions in the lower extremities.)

The following signs have proved useful in the assessment of nutritional status, although it must be emphasized again that they are largely nonspecific, and most are more reliable indicators when their frequency is assessed in population groups than in a single individual. Excellent color illustrations of most of the lesions described will be found in Jolliffe's recent book (1962) and will supplement the black and white illustrations of this chapter.

HEIGHT, WEIGHT, AND SUBCUTANEOUS TISSUE

Although much can be told about the status of grossly underweight or overweight individuals by simple inspection, standard weight for height is a more reliable means of discrimination (Appendix VI). Similarly, a gross estimate of subcutaneous fat can be obtained by pinching a double fold of skin over the outer surface of the upper arm (midtriceps region), although skin calipers provide a more precise estimate.

HAIR

Protein Deficiency. Protein malnutrition causes the hair to become light in color, fine, dry and brittle. Characteristically, the hair of persons with severe protein deficiency can be pulled out of the scalp without discomfort; whole tufts of hair come out readily by the roots. Since the hair is brittle, the tips also break off easily. In persons known to have brown or black hair, the color change is readily detected; without this knowledge it could be thought to be of genetic origin. The hair of Negroes becomes reddish in cases of protein deficiency. In blond persons with normally fine hair, the change is difficult to detect with certainty.

Color Changes. In assessing hair changes, the bleaching action of the sun and the care given the hair must be taken into consideration. Color change resulting from exposure

to the sun generally will involve the upper layers of hair, while that caused by protein deficiency will affect deeper as well as superficial layers. Use of grease and oil and frequent washing or lack of it complicate interpretation. Individuals exposed to alternate periods of very poor and good nutrition may develop transverse light and dark hair bands; this is known as the flag sign. Children recovering from the severe protein malnutrition of kwashiorkor often develop this sign as normal hair grows out (Fig. 19-1).

EYES

Xerophthalmia. Both vitamin A and riboflavin deficiencies are known to affect the eyes. The xerophthalmia due to avitaminosis A begins with a dryness of the bulbar conjunctiva, as evidenced by loss of the light reflex, lack of luster and decreased lacrimation. It may proceed to keratomalacia, which is a softening of the cornea leading to ulceration, perforation, rupture and destruction of the cornea. The final result is a scarred, opaque cornea and a sightless eye.

Bitot Spots. Bitot spots often are associated with vitamin A deficiency, although they cannot be regarded as specific to it (Fig. 19-2). They appear as frothy, irregular, white or light yellow spots from one to several mm. in diameter, most often on the conjunctiva lateral to the cornea. They look as if they could be wiped away but are beneath conjunctival epithelium. A small Bitot spot may consist of only a few tiny air bubbles visible in the triangles, especially the outer ones. Both photophobia and the inability to see in dim light may be due to vitamin A deficiency.

Circumcorneal Injection. The area in the eye where the sclera changes to the cornea is called the limbus. The circumcorneal injection which is seen in riboflavin deficiency consists of penetration of the corneal limbus and branching of the subconjunctival arterioles that normally terminate within 0.5 mg. of the limbus (Fig. 19-3). This proliferation and congestion of the blood vessels in the sclera and their extension into the clear corneal tissue is by no means pathognomonic of riboflavin deficiency. Moreover, excessive exposure to sunlight, smoke, dust and other irritants is a recognized conditioning factor even when riboflavin deficiency is the underlying cause. Riboflavin deficiency may also

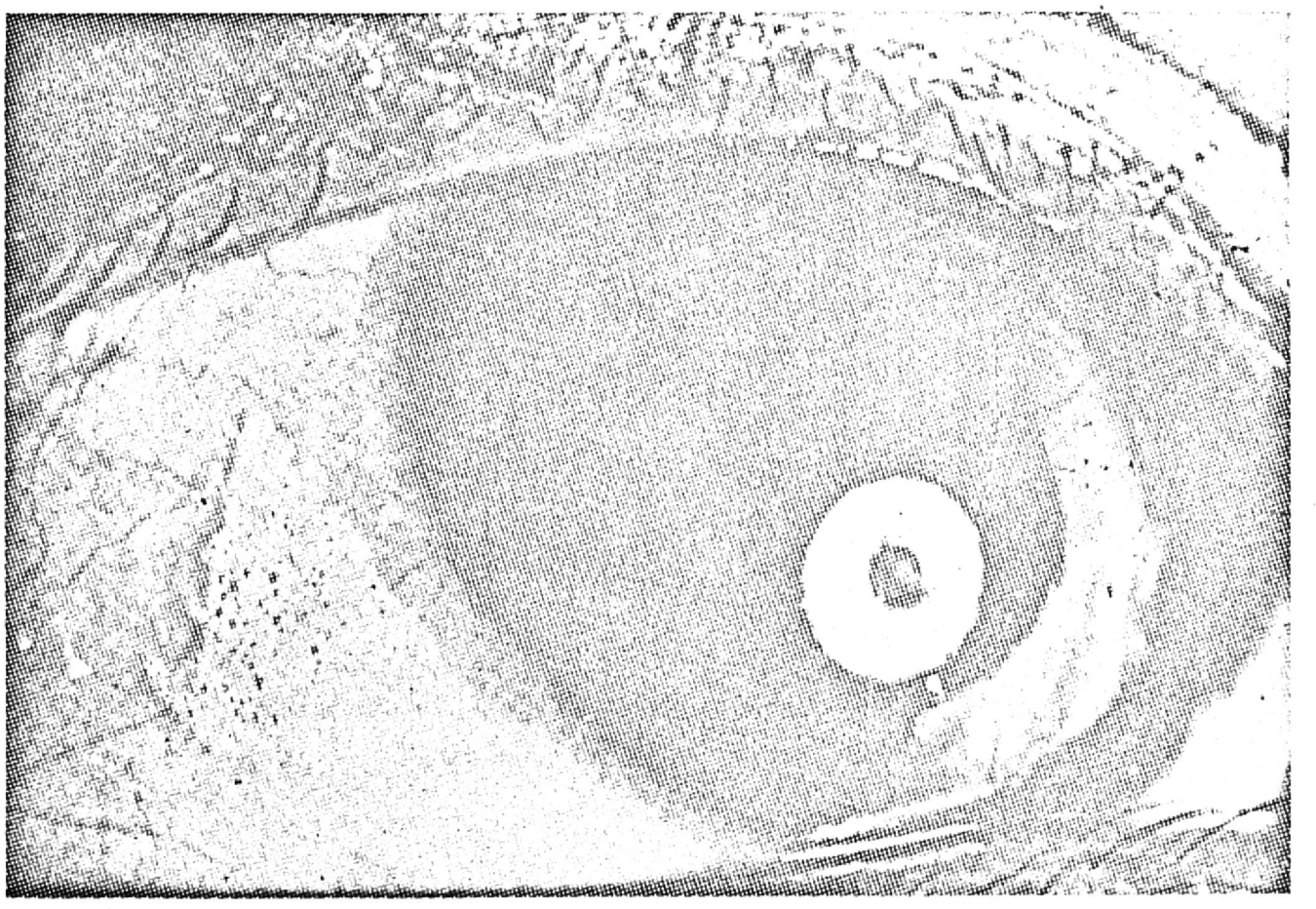

FIGURE 19-2. *Bitot spot.*

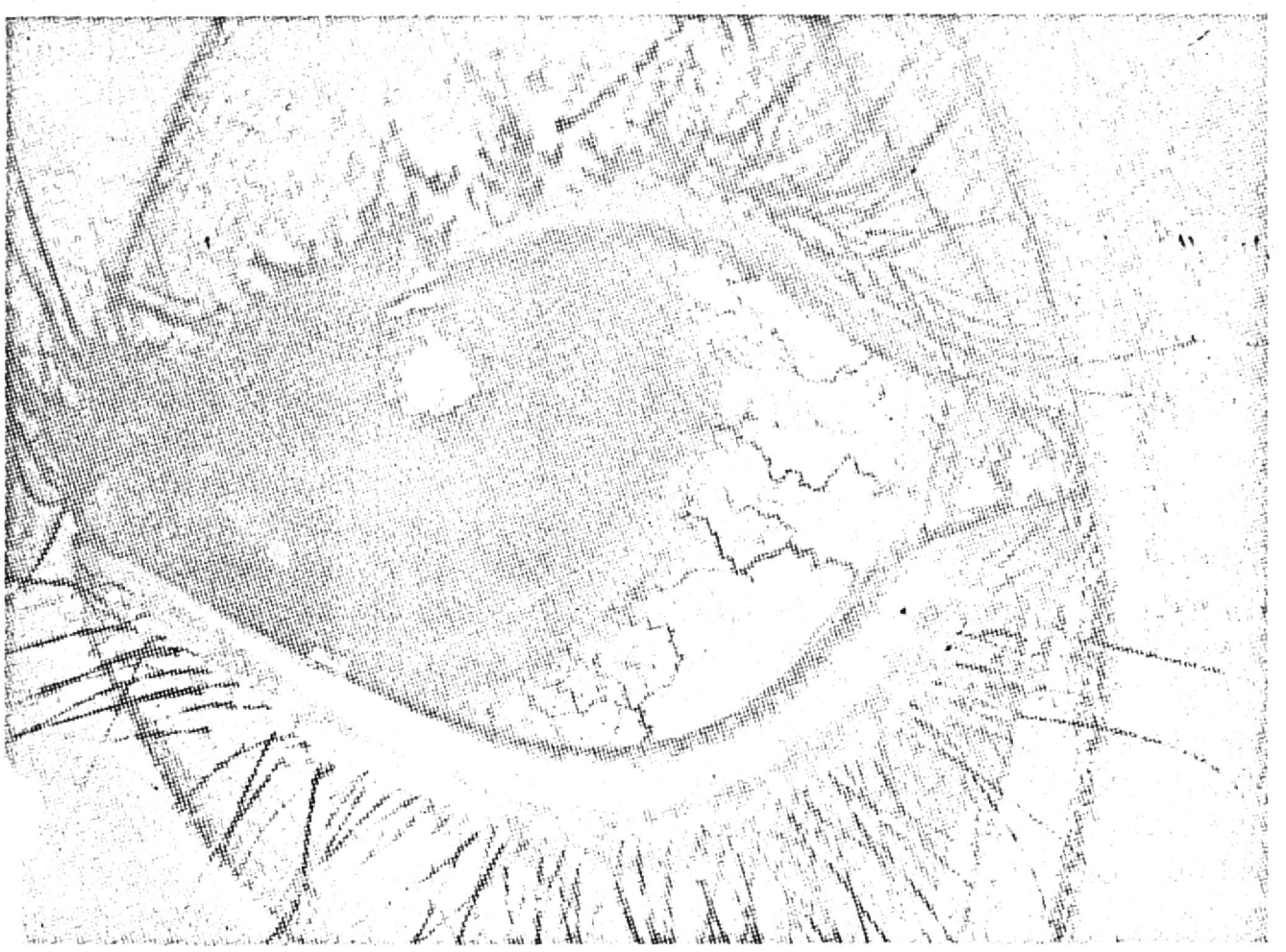

FIGURE 19-3. *Circumcorneal injection.*

FIGURE 19-4. *Dyssebacea of nasolabial fold.*

produce a moist, red lesion of the external angle of both eyes.

Signs of Non-nutritional Diseases. There are a number of other signs in the eye which may be confusing to the examiner but which have no nutritional significance. Corneal scars, usually gray in color, are generally due to infectious conjunctivitis. Tiny hemorrhages at the end of the subconjunctival arterioles, as well as an increased vascularization and pigmentation of the exposed part of the conjunctivae, are very likely due to irritating environmental factors. Pinguecula are very small, white or yellow subconjunctival cholesterol or lipid deposits which may be confused with Bitot spots. A pterygium consists of a fleshy, red growth which encroaches from either angle of the eye upon the cornea and eventually may cover the pupil. Its cause is unknown.

SKIN

The areas of the skin most affected by nutritional deficiency usually are accessible to examination because they are also those most exposed to the environment. Thus, the skin of the face, neck, arms and legs, and the skin over pressure points such as the elbows, knees and ankles are most likely to show positive findings in nutritional deficiencies.

Dyssebacea. Dyssebacea is the clinical term used to designate a series of disturbances of the sebaceous glands characterized by increased oiliness and dermatitis, fissuring and exfoliation. The lesion of this type already mentioned as seen at the external angle of the eye in riboflavin deficiency is also found at the nasolabial fold (Fig. 19-4) and behind the ears, and may occur in other skin folds of the body. The nasolabial lesions should not be confused with acne in adolescents or with the irritation from nasal discharge in small children. For positive identification, the lesion must be red and humid.

Xerosis. Xerosis or generalized dryness of the skin is characteristic of vitamin A deficiency but difficult to evaluate in the individual because the appearance of the skin is influenced so much by bathing habits and exposure to dust and sunlight. Follicular hyperkeratosis is characterized by keratinization of the hair follicles to give a goose flesh appearance which is not altered by brisk rubbing. Fully developed lesions consist of symmetrically distributed, rough, horny papules formed by keratotic plugs projecting from hypertrophied hair follicles; they make the skin look and feel like coarse sandpaper (Fig. 19-5). Traditionally, the disease is associated with vitamin A deficiency and more recently in India with essential fatty acid deficiency. However, in Central America where it is exceedingly prevalent among schoolchildren, investigators have been unable to demonstrate any relationship between this lesion and either dietary vitamin A or fat.

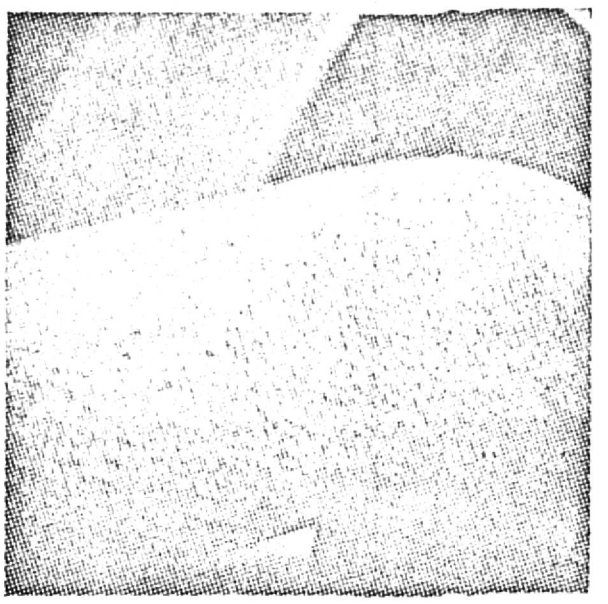

FIGURE 19-5. *Early follicular hyperkeratosis resembling "goose flesh." (Jolliffe, N., Tisdall, F. F., and Cannon, P. R.: Clinical Nutrition. Paul B. Hoeber, Inc.)*

Ascorbic Acid Deficiency. It is important to differentiate follicular hyperkeratosis from the perifolliculitis of ascorbic acid deficiency (Fig. 19-6). The latter consists of perifollicular congestion, swelling and, eventually, hypertrophy of the follicles. Only in this late stage is the skin rough to the touch. Other skin changes caused by severe ascorbic acid deficiency include petechiae, purpura, hematomas and increased capillary fragility with trauma easily caused by the blood pressure cuff, shoes or an accidental bruising. Again, it must be emphasized that there are many other causes of the phenomenon.

Pellagra. Cutaneous lesions of pellagra are variable because of differences in the degree of deficiency, original skin color and conditioning factors but are sufficiently char-

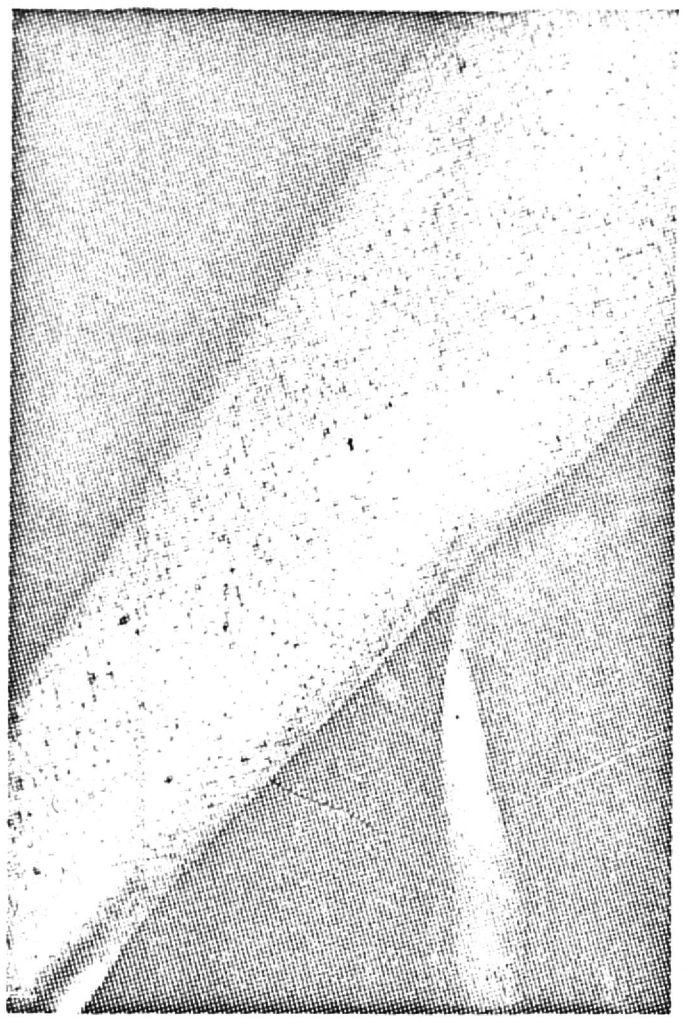

FIGURE 19-6. *Perifollicular hemorrhages on the leg of a boy, age 16, with scurvy. (From the Merck Report, May, 1956, Merck and Co., Inc., Rahway, N. J.)*

acteristic to be diagnostic in many instances. The most common chronic form is thickening, inelastic, fissured and deeply pigmented skin in areas especially exposed to sunlight or over pressure points. Eventually, the skin in affected areas becomes dry, scaly and atrophic.

Erythema upon exposure to sunlight is an acute manifestation, with subsequent vascularization, crusting and desquamation out of proportion to the precipitating exposure. Also, a common acute but highly nonspecific reaction is redness, maceration, abrasion and superimposed infection in intertriginous areas. Heat friction and poor personal hygiene are major conditioning factors. The symmetrical distribution of pellagrous skin lesions over exposed areas and pressure points is the most characteristic feature.

Edema. In severe protein deficiency the skin of the legs, arms and thighs shows marked pitting edema (which is a sine qua non of the diagnosis) and a dermatosis characterized by hyperkeratosis, hyperpigmentation and desquamation. Although often similar in appearance to the lesions of pellagra, they are not limited to the exposed areas and characteristically extend to the thighs and lower trunk.

NECK

Casal's Necklace. Casal's necklace is a pellagrous dermatitis following the neckline when a low-necked dress or open shirt collar is habitually worn outdoors by persons on a niacin deficient diet.

Goiter. Endemic goiter resulting from a deficiency of iodine available to the thyroid gland usually can be detected by inspection of the neck with the head thrown back, and in case of doubt evaluated by simple palpation. If the goiter is visible with the head in the normal position, its pathological significance is beyond doubt, and it is Grade II according to the classification of the World Health Organization (Fig. 19-7). Grade III goiter is one easily recognizable at a distance. The difficulty in this superficial examination lies in the fact that in thin individuals not all thyroid glands that become visible with the head thrown back can be classified as being goiterous. As a rough guide, the size of a lobe of the normal thyroid can be taken as that of a lima bean about the size of the thumbnail of the person being examined. When the volume of the lobe exceeds this by 4 or 5 times, the goiter is classified as a Grade I or higher.

MOUTH

The mouth is one of the areas most sensitive to nutritional deficiencies, but the changes are nonspecific, confusing and difficult to evaluate. Pallor of the lips and mucous membranes, like pallor of the skin and fingernails, may be a consequence of anemia, but its clinical appraisal is so subjective as to be of little value except in severe cases. The angular stomatitis which may be a consequence of riboflavin deficiency also has been mentioned (Fig. 19-8). Angular scars may be the result of past episodes of acute ariboflavinosis but also may be wholly non-nutritional in origin. Even loss of teeth which produces closed vertical dimension or closed bite may

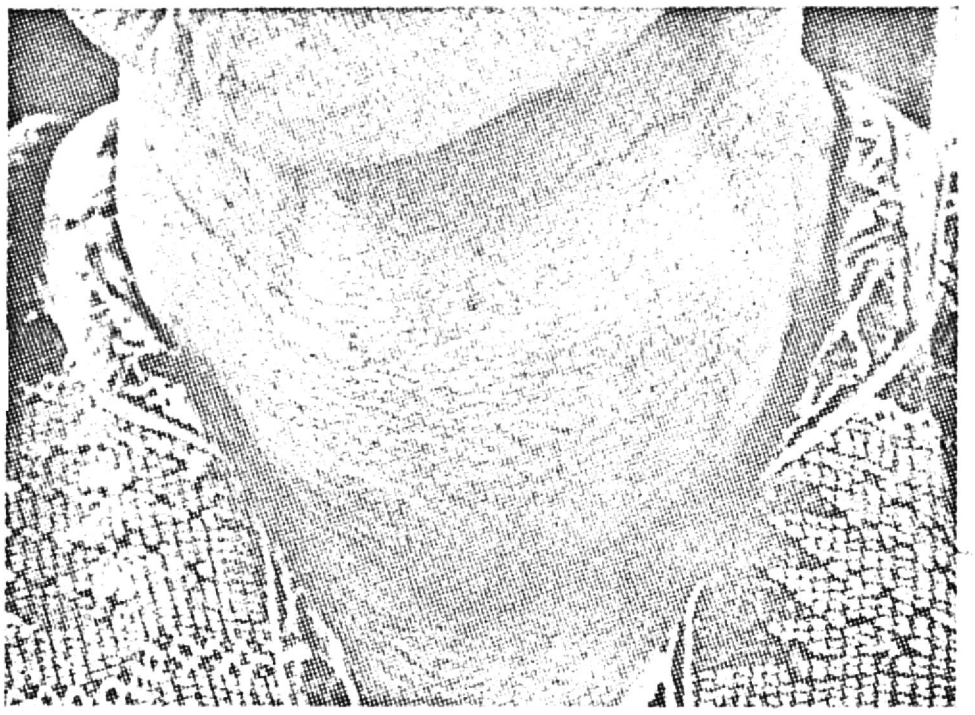

FIGURE 19-7. *Goiter.*

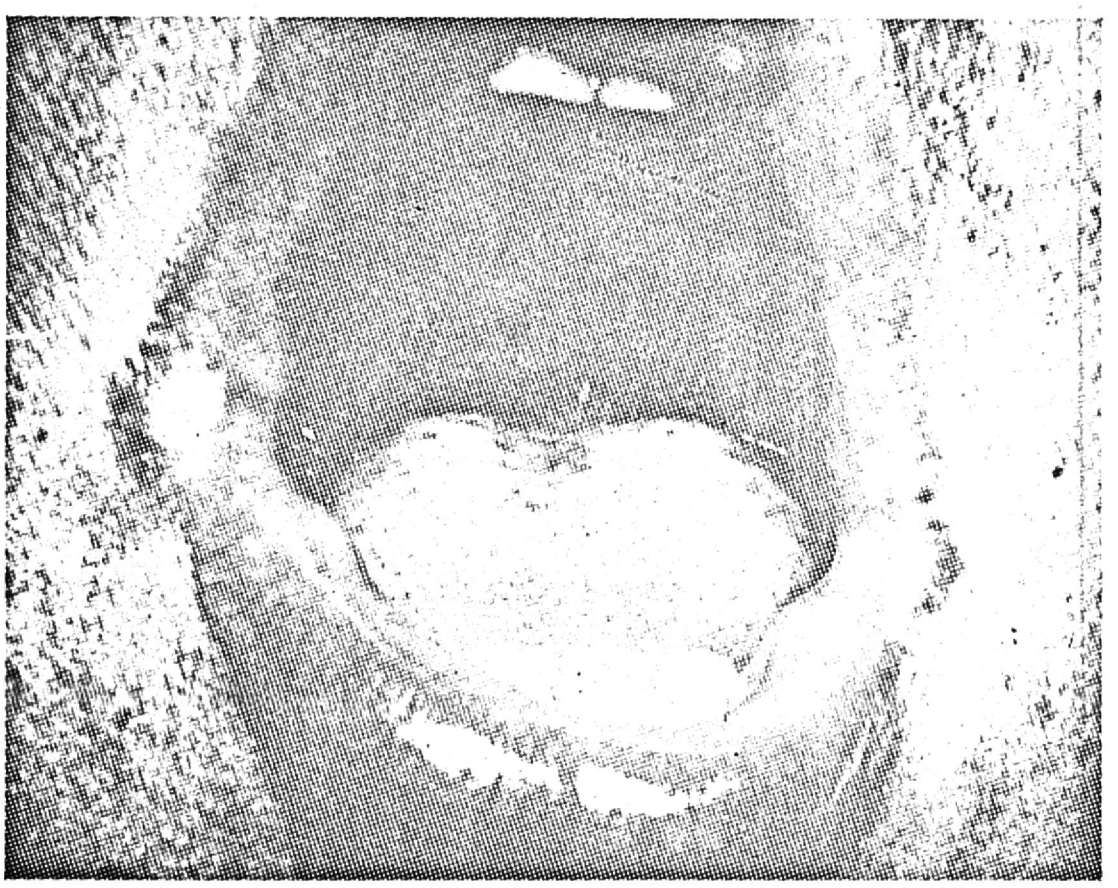

FIGURE 19-8. *Angular stomatitis.*

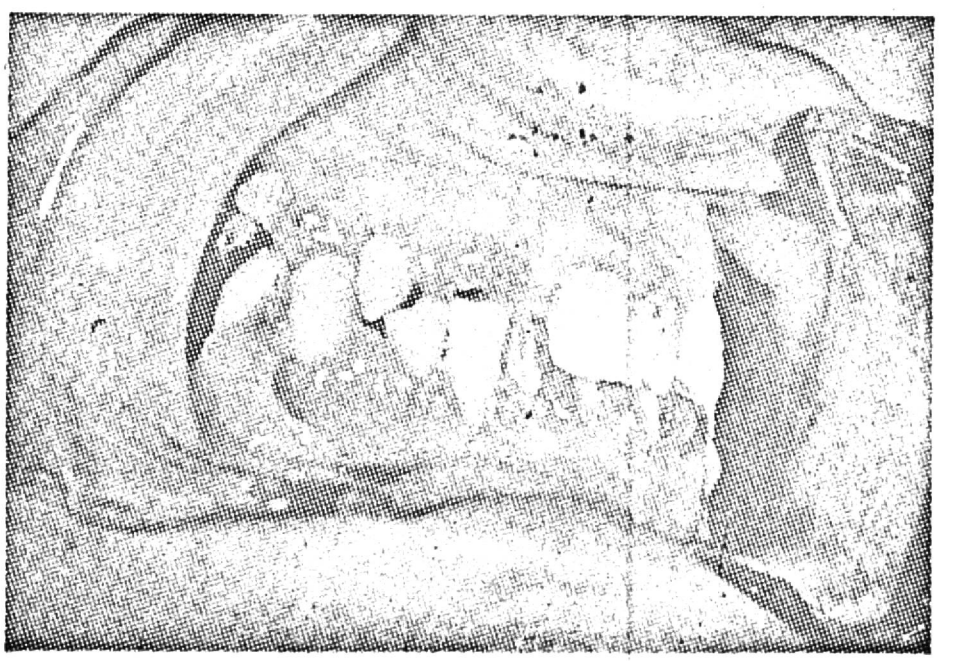

FIGURE 19-9. *Scorbutic gingivitis. (Courtesy, Dr. G. Shklar.)*

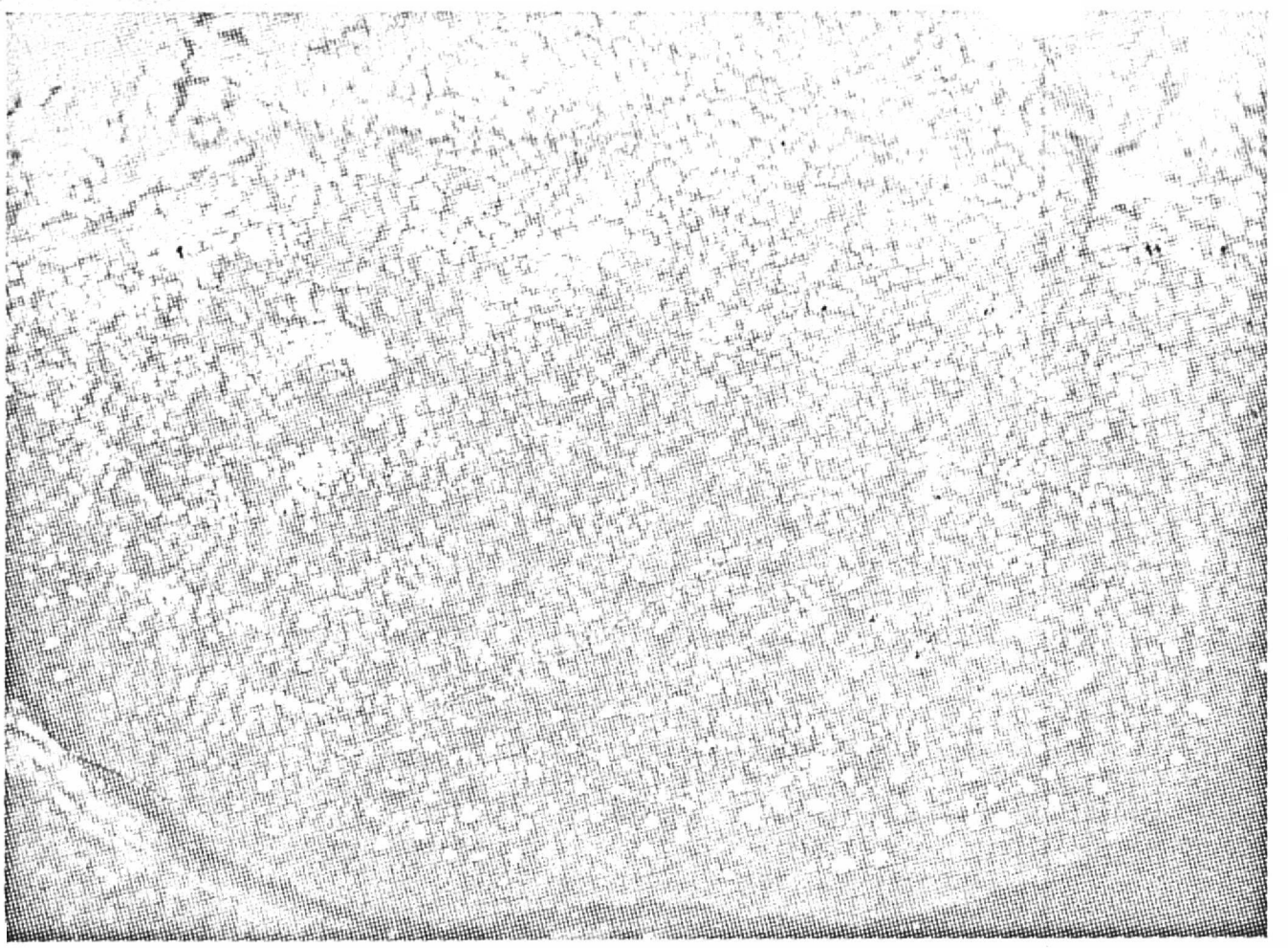

FIGURE 19-10. *Hypertrophy of tongue suggestive of vitamin B-complex deficiency.*

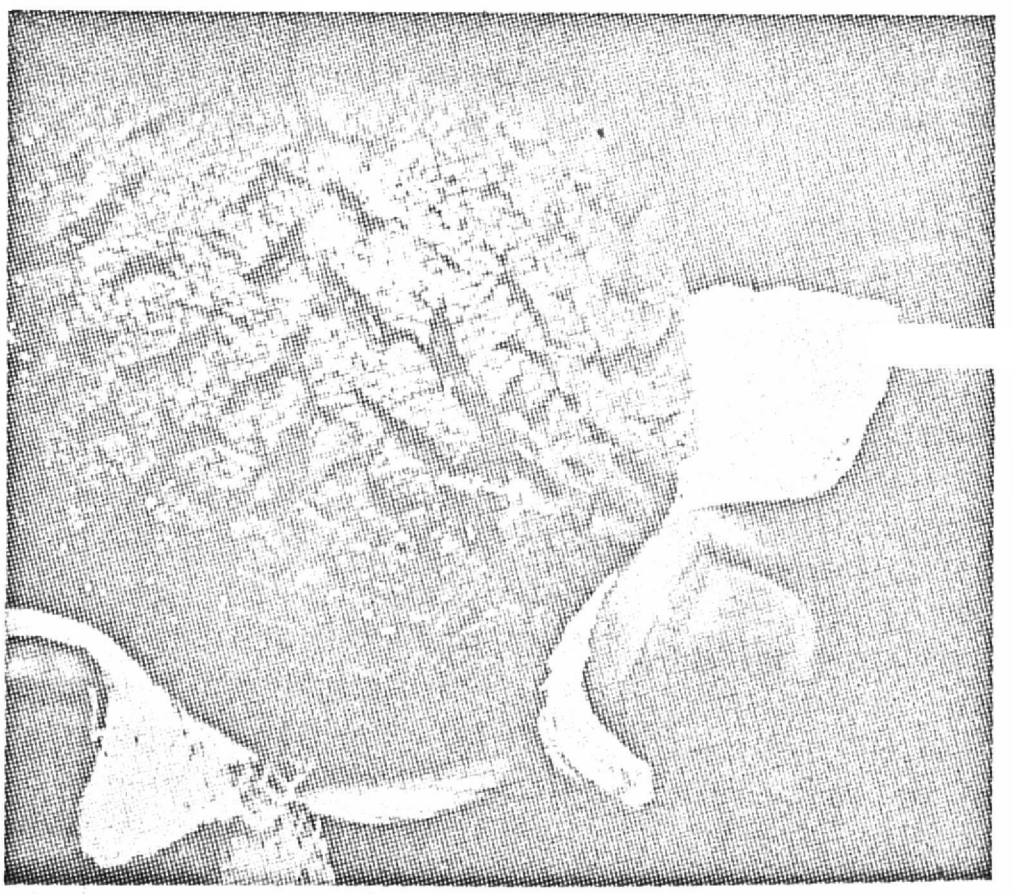

FIGURE 19-11. *Scrotal tongue.*

result in maceration of the lips and corners of the mouth.

Gingivae. The gums may reflect any of a variety of nutritional deficiencies, but even the dentist will have difficulty differentiating the changes from those resulting from accompanying local oral irritants. This is especially true because neglect of diet and of oral hygiene are common in the same individual. Gingivitis is reported to be common in people whose diets are deficient in ascorbic acid, but most attempts to prove such a relationship by ascorbic acid therapy have been failures. On the other hand, engorged, dark red and bleeding gums are almost pathognomonic of scurvy (Fig. 19-9). (See Chapter Thirteen.)

Both marginal and generalized gingivitis have been stated to be associated with various other nutritional deficiencies, including vitamin A, niacin and riboflavin, but without proof. The problem is that many factors may play a causal role simultaneously so that the experimental correction of a single factor does not necessarily effect a cure.

Tongue. Marked hypertrophy of the filiform and fungiform papillae of the tongue is more frequently seen in populations in which B-complex deficiencies, particularly of thiamine and riboflavin, are present (Fig. 19-10). Papillar atrophy is sometimes related to niacin or iron deficiency, although any disease causing severe anemia will have this effect. It is for this reason that nutritional megaloblastic anemia results in a smooth tongue. A markedly furrowed, so-called scrotal tongue is a possible manifestation of vitamin A deficiency (Fig. 19-11).

Impression of the teeth on the borders of the tongue may be due to the edema resulting from protein deficiency as well as to defective dentures and a variety of other causes. When edema is the cause, there is also likely to be evidence of edema of the extremities.

Nutritional changes also occur in the color of the tongue, although they are usually difficult to evaluate. A reddening of the tongue which is generalized or limited mainly to the distal third may be associated with the deficiency caused by sprue. It affects first the tip and lateral margins but may progress to include not only the entire tongue but all of the oral mucous membranes as well. A beefy red glossitis resembling raw beefsteak probably represents most frequently a deficiency of niacin, but other B-complex vitamin deficiencies may be involved. Riboflavin deficiency is probably most often responsible for the purplish discoloration known as magenta tongue. Patchy areas of paler discoloration give rise to the name geographic tongue, which has no nutritional significance.

TABLE 19-1. CLINICAL SIGNS MORE OR LESS SUGGESTIVE OF NUTRITIONAL DEFICIENCIES

Deficiency	*Suggestive*	*Possibly related*
Vitamin A	Xerosis of skin Xerophthalmia Keratomalacia Nonpurulent conjunctival injection Photophobia Night blindness	Bitot spots Follicular hyperkeratosis Conjunctival thickening Fungiform papillary hypertrophy of tongue Marginal gingivitis Scrotal tongue Circumcorneal injection
Riboflavin	Angular stomatitis External angle lesion of eye Nasolabial seborrhea Canthal fissures	Circumcorneal injection Papillary hypertrophy of tongue Red tip of tongue Magenta tongue Angular scars
Niacin	Pellagrous dermatitis Red tongue	Gingivitis
Thiamine	Absence of ankle jerk	Papillary hypertrophy of tongue
Ascorbic acid	Gingival injection and swelling Gingival bleeding Subcutaneous petechiae, folliculitis	
Iodine	Endemic goiter	
Iron	Papillary atrophy of tongue	Pallor of mucous membranes
Protein	Hair changes: color, texture, easily pluckable Edema (children) Lingual edema	Retarded eruption of primary teeth Malposition of teeth

Teeth. Although discussed in more detail in Chapter Twenty-nine the relationship between nutritional deficiency in infancy and early childhood and caries susceptibility, retarded eruption and malposition of teeth should be mentioned here. Malposition of

primary and secondary teeth resulting from retarded bone development is sometimes the result of early protein deficiency.

In many underdeveloped countries a so-called hypoplastic line across the upper primary incisors has been described, into which, in time, a yellow or brown pigment is deposited, followed by the development of caries on the labial surface of the teeth. Subsequently, the teeth break off along this line. A nutritional or febrile insult in the neonatal period is a probable cause.

Fluorosis is characterized by mottled enamel and chalky white patches distributed over the surface of the teeth. In some cases, the entire tooth surface may be dull white, giving an unglazed appearance.

The signs suggesting nutritional deficiency are easier to evaluate and more specific in children than in adults. In adults lesions from other causes, such as aging and repeated trauma, and confusing signs such as pinguecula, pterygium, and geographic tongue are more likely to be present. The clinical signs which suggest more or less strongly the possibility of a nutritional deficiency are summarized in Table 19-1. It should be clear from the foregoing that clinical examination alone will rarely be sufficient to establish a definite diagnosis. A medical history is useful and so are dietary and biochemical data.

DIETARY HISTORY

The techniques of obtaining quantitative data on nutrient intakes by interview or diet records methods, although beyond the scope of this discussion, have been described in Chapter Twenty-eight and also delineated elsewhere (*FAO*, 1953). The dentist can and should obtain qualitative dietary information which could shed light on the probability that signs of nutritional deficiency will be encountered and on the cariogenicity of the habitual diet. For example, an individual who drinks milk regularly is not likely to be deficient in riboflavin, and one consuming abundant citrus and other fruit and juice will not be deficient in vitamin C. In fact, a person consuming a diet which is normal and balanced is not likely to be deficient in any nutrient. In such a case it is quite wrong to try to interpret minimal changes in the skin, eyes or mouth as a consequence of some nutritional deficiency and to suggest vitamin supplements. On the other hand, if questioning reveals that a patient is an alcoholic or food faddist, signs of nutritional deficiency may well be present. In the first case, the signs are likely to be those associated with B-complex vitamin deficiencies, and in the second, nutritional anemia. Similarly, the questioning may reveal a heavy consumption of candy and sweet desserts which are exacerbating the problem of dental caries.

The dietary questioning need not be involved or time-consuming when the diet is relatively good, but when it is poor, time and skill are required to obtain reliable information and to draw proper conclusions. For more detailed work on quantitative dietary estimates, the services of a dietitian or nutritionist should be enlisted by dentist and physician alike.

BIOCHEMICAL TESTS

Just as a single picture may be worth more than a thousand words, so a single biochemical determination, if accurate, may decisively confirm or deny the nutritional origin of an uncertain complex of clinical signs. Here again, biochemical measures vary in their reliability and specificity. Serum levels of vitamin A and ascorbic acid are quite useful, but levels approaching deficiency may be present for some time before lesions appear. Total serum protein and serum albumin are decreased markedly in protein deficiency but only when the deficiency has progressed to the point at which it is clinically evident. The ratio of urea to creatine has been recommended recently as a measure of the loss of lean body mass in protein-calorie malnutrition. For the B-complex vitamins, blood serum levels are relatively insensitive, and measurement of thiamine, riboflavin, and niacin excretion in 24 hours or in timed urine samples is recommended. Anemia is best diagnosed by determination of plasma hemoglobin and hematocrit accompanied by examination of a thin blood smear for red cell size. It should be noted that most biochemical

TABLE 19-2. TENTATIVE GUIDE TO THE INTERPRETATION OF SELECTED BIOCHEMICAL DATA USEFUL IN THE APPRAISAL OF NUTRITIONAL STATUS*

	Deficient	*Low*	*Acceptable*	*High*
Plasma protein: gm./100 ml.	< 6.0	6.0 – 6.4	6.5 – 6.9	≧ 7.0
Serum albumin (electrophoretic method): gm./100 ml.	< 2.80	2.80– 3.51	3.52– 4.24	≧ 4.25
Hemoglobin: gm./100 ml.				
Men	<12.0	12.0 –13.9	14.0 –14.9	≧ 15.0
Women (nonpregnant, nonlactating; ≧13 yr.)	<10.0	10.0 –10.9	11.0 –14.4	≧ 14.5
Children (3-12 yr.)	<10.0	10.0 –10.9	11.0 –12.4	≧ 12.5
Hematocrit (PCV): per cent				
Men	<36	36–41	42–44	≧ 45
Women (nonpregnant, nonlactating: ≧13 yr.)	<30	30–37	38–42	≧ 43
Children (3-12 yr.)	<30.0	30.0 –33.9	34.0 –36.9	≧ 37.0
Plasma ascorbic acid: mg./100 ml.	< 0.10	0.10– 0.19	0.20– 0.39	≧ 0.40
Plasma vitamin A: μg./100 ml.	<10	10–19	20–49	≧ 50
Urinary thiamine: μg./gm. creatinine	<27	27–65	66–129	≧130
Urinary riboflavin: μg./gm. creatinine	<27	27–79	80–269	≧270
Urinary N-methylnicotinamide: mg./gm. creatinine	< 0.5	0.5 – 1.59	1.6 – 4.29	≧ 4.3

* From Manual for Nutrition Surveys. 2nd ed. Bethesda, Md., Interdepartmental Committee on Nutrition for National Defense, National Institutes of Health, 1963.

measures show only the present situation so that chronic nutritional lesions are possible even in patients with normal nutritional biochemistry.

Table 19-2 provides a guide to the interpretation of biochemical measures of particular value in appraising nutritional status. It should be noted that a vitamin deficiency lesion could be present as the result of a previous deficiency even when biochemical examination at the time of an office visit gives normal values. A short period of improved nutrient intake could obscure biochemical values before a lesion caused by a deficiency has had time to heal.

The Dentist's Responsibility with Respect to Nutritional Advice

The use made by the dentist of information regarding the nutritional status of the patient necessarily varies greatly with the situation. When anemia is suspected, the patient should be urged to see his physician for more detailed diagnostic procedures and treatment. This should be the case also when severe deficiency disease of any kind is present. On the other hand, advice can be given appropriately by the dentist when there is obvious excessive use of cariogenic foods, evidence of imbalanced diets likely to lead to difficulty, or minimal suggestive clinical signs coupled with compatibly poor dietary habits. In between lies a spectrum of conditions for which the need for referral will depend upon the nature of the situation, the training and experience of the dentist, and the diagnostic and consultant facilities at his disposal.

In mild cases involving oral lesions under his direct observation, the dentist may be justified in recommending dietary changes or vitamin supplements as a therapeutic trial before deciding on the need for outside consultation. Because of the systemic ramifications of any frank nutritional disease, however, and the importance of precise diagnosis of the etiology of anemias, the dentist always should obtain medical consultation or be sure that the patient sees his physician when these entities are identified or strongly suspected.

SUMMARY

The dentist is in a favorable position to observe, without inconvenience to his patients or disruption of his routine, nearly all the areas in which signs suggestive of nutritional deficiency are commonly encountered. These areas are the hair, face, mouth, neck and skin of the arms and legs. The lesions found are generally not diagnostic but only

suggestive of possible nutritional deficiency; they are seldom cues to nutritional deficiency alone but instead to a variety of conditioning factors acting upon weakened tissue.

Clinical lesions in these areas, however, usually serve to alert the dentist to nutritional factors which may be influencing adversely the oral tissues or the general health of his patient. Qualitative information on dietary habits, as well as a medical history, and laboratory examinations made at the same time may sufficiently confirm the clinical impression to justify dietary counseling or medical referral, depending upon the nature and severity of the suspected condition.

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