

SPECIAL ARTICLE

PIGMENTATION OF THE BUCCAL MUCOSA

By

R. KUFFER and J. M. SOUBIRAN

Pigmentation implies the normal or pathological accumulation of a pigment. By the word pigment, we mean not only organic endogenous pigments which may be normal or abnormal, soluble like the biliary pigment or insoluble like melanine, but also the exogenous pigments, organic and mineral insoluble coloured substances. It is thus that the "bismuth pigmentation" has been spoken of for a long time and cutaneous argyria has been studied under the category of the melanodermias. This classification, though not very satisfactory from a theoretical point of view, has the merit of being rather practical and we shall adopt it to study the pigmentations (or, the colourations, as some may prefer to call them) of the buccal mucosa, produced by the insoluble organic and other pigments. We shall confine ourselves to the study of the deep colourations, having a more or less definite brown, grey, blue or black tint, thereby eliminating the yellowish or whitish blemishes and the colourations due to the biliary pigment which pose very different diagnostic problems.

These pigmentations are very commonly seen, but paradoxically they have been given very little attention: firstly because the patient who ignores them never consults the doctor for his observation and secondly because they often pass unnoticed. If the buccal pigmentation is detected accidentally, with the exception of a major melanodermic syndrome, the physician has the tendency of attaching little importance to it, since the etiological problem is difficult and the evolution is most often benign and asymptomatic. We shall see however, that this is not always the case and that the finding of a greyish speck on the buccal mucosa may lead to the diagnosis of a malignant buccal melanoma which is the most malignant of tumours.

The classification of the buccal pigmentations poses difficult problems. In fact it is difficult to consider successively the different pigments since in the majority of cases it is melanine which is involved in rather varied affections such as Addison's Disease, the pigmentation due to nivaquine and even the hemochromatosis where melanine plays a more important role than hemosiderine. The symptomatological classification is equally difficult since the various affections produce clinical picture which are often closely similar.

We shall therefore adopt a rather arbitrary procedure and study successively:

1. The false pigmentations.
2. The genotype pigmentations.
3. The endocrinal pigmentations.
4. The dysmetabolic pigmentations.
5. The pigmentary and pigmentogenous dermatoses.
6. The toxic and medicamentous pigmentations.

7. The pigmentations due to oxydation of keratine.
8. Tattooings.
9. The buccal melanomas.
10. The pigmentations of unkown origin.

I. THE FALSE PIGMENTATIONS

An attentive examination would help to eliminate a certain number of colourations of the buccal mucosa which should not be mistaken for pigmentations :

—The superficial colourations, seen chiefly on the dorsal surface of the tongue due to the various food stuffs, tooth paste, or drugs (liquorice, carbon etc.).

—The varicosities of the inferior surface of the tongue, the telangiectasis and the angiomias should generally be easily recognized.

—The mucoid cysts have a bluish appearance; they are usually found in case of certain large exteriorised maxillary cysts.

—A brownish colouration may be observed in the mucosa covering certain myeloplastic tumours.

—A blackish sub-gingival tartar should not be confused with a gingival margin.

II. THE GENOTYPE PIGMENTATIONS

There exists a racial pigmentation of the buccal mucosa which is transmitted as a dominant characteristic. Usually of a homogenous slate gray colour, at times blackish or chamois, it chiefly affects the gingival vestibular region, circumscribing for some distance the crown of the teeth in bands or plaques which more or less regular, more rarely as lenticular specks. We may also find coloured plaques, illdefined, having a reticulate appearance, on the palatine mucosa and the internal surface of the cheeks and the lips, less often on the muco-cutaneous surface of the lips which then assumes a bluish violet appearance. The affection of the tongue is exceptional.

The pigment responsible for this colouration is melanine which is found in great abundance in the basal layer of the epithelium, as also in the Malpighian bodies and the melanophores of the papillary dermis.

This pigmentation which has no pathological significance is found in the Black Races, very frequently in the Yellow Races and at times also among certain White populations of Asia and Africa; Arabs, Turks, Persians, Indians, etc.; Central and Eastern Europe (Hungary, Rumania, Balkans and Gypsies) and the Iberian peninsula. It is also found in France in the S. W., Central and Eastern (Bourgogne) regions, where it represents a sequelae of the grand invasions (Huns, Moors).

Finally, famillal pigmentations of this type are found quite often in subjects who are most often brown, more or less bronzed, all of their ancestors appearing to be Parisian, Britons or Alsatian. This is why we prefer the term genotype pigmentation to that of ethnic pigmentation.

III. THE ENDOCRINAL PIGMENTATIONS

I. Addison's Disease

We should firstly recall that the melanic pigment is produced by special cells found in the basal layer of the epidermis, namely the melanocytes. These cells, which elaborate and excrete the pigment, may be considered as glandular cells forming a specialised "cytocrinal" system (Masson), since the excretion takes place not into the internal milieu, but into the malpighian cells and the melanophores. This melanic system is related to the endocrinal system through the intermediary of the pars intermedia of the hypophysis which secretes the MTH of the melanotropic hormone. The hormone acts not on the melano formation, but on the melano dispersion; thus a fragment of the skin of a frog placed in a solution of MTH turns brown, not by stimulation of the melano formation but by dispersion of the granules melanine in the cytoplasm of the melanocytes. The Addisonian pigmentation is hence explained by an augmentation of the MTH secretion in the absence of the hypophyseal inhibition by the suprarenal corticosteroids.

The muco-cutaneous melanoderma is one of the cardinal signs of the disease. The buccal pigmentation may be the first to be observed and remain isolated for several months. The appearance is very similar to that of the genotype pigmentation. It manifests as patches which usually appear on the internal surface of the cheeks, the palatine mucosa and the mucosa of the alveolar margins. Their pattern reveals all possible forms; round isolated patches, irregular patches with definitely limited contours or coalescent patches forming more or less homogenous layers and more or less widespread. They are more abundant and more widespread in the supple and free mucosal zones, whereas they are less numerous and smaller in the fibro-mucosa. The mucosa is dry, smooth and dull red in colour.

Classically, these patches are slate-coloured, i.e. a dark greyish-blue, almost black in places. In fact according to the density of the pigment they take on various tints ranging from a slate grey to maroon, or even to a coffee colour (BATAILLE AND PAYEN).

The pigmentation regresses under the effect of a substitutional hormone therapy, together with a regression of the other signs of the disease. Often the mucosal pigmentation disappears before the cutaneous melanoderma, which may therefore be considered a test of the efficacy of the treatment. However, the muco-cutaneous melanoderma may be resistant to the treatment and persist after the disappearance of the other signs. Mechanical, chemical and thermal irritations play an important role and subjects using dentures are apt to have a deeper and a more tenacious pigmentation.

The histological examination of the buccal mucosa of these patients shows an abnormal abundance of melanine in the basal layer without any other anomaly; in particular, the melanocytes are not increased in number.

2 Other endocrinal pigmentations.

The muco-cutaneous melanoderma may be observed, though with a much lesser frequency, in the course of a certain number of affections:

- chronic tuberculosis) “false addisonism”).
- Basedow’s disease.
- primary hypophyseal tumours.
- Cushing’s syndrome.
- diseases treated for a long time with A. C. T. H.
- the suprarenalectomised subjects, etc.

The mucosal pigmentation, whenever it exists, is of the Addisonian type. In this group are also included certain diffuse pigmentations occurring in the course of chronic infections; kala-azar, amoebic dysentery. In these cases, the pigmentation does not usually affect the mucosae.

On the other hand, in the melanodermias of the vagabonds, where pediculosis, multiple nutritional deficiencies and suprarenal insufficiency co-exist, one often finds pigmented patches on the buccal mucosa resembling those of Addison’s disease.

IV. DYSMETABOLIC PIGMENTATIONS

1. Ochronosis.

It is a very rare affection characterised by a bluish or blackish colouration of the cartilages, ligaments and fibrous tissue due to a disturbance in the metabolism of tyrosine and phenylalanine, having a recessive genotype transmission. It is generally accompanied by an alcaptonuria.

2 Haemochromatosis

It is a hereditary disease brought about by a marked disturbance in the metabolism of iron and characterised by a hepatic cirrhosis with diabetes and melanoderma (bronzed diabetes). Classically, the pigmentation is diffuse, homogenous without hyperpigmented macules, dirty grey with a metallic reflection, not affecting the mucosa. In fact, in 16–20% of cases (WORINGER and LAUGIER), one finds slate grey or ochre colour patches on the internal surface of the cheeks. Prussian blue lenticular macules may also be noted on the gums.

The diagnosis is confirmed by the elevation of the serum iron level and the appearance of sclerosis with hepatic siderosis on a puncture biopsy of the liver. Curiously, the pigmentation of the skin and the mucosae is due more to a melanic overloading of the basal epidermis rather than to pigments derived from hemoglobin (hemofuchsin and hemosiderine).

Besides the primary hereditary form, there also exist various secondary forms (post transfusional, in the course of alcoholic cirrhosis, hemolytic anemias).

The pigmentations described in chronic malaria may also be grouped under the hemochromatosis since they are derived from the products of disintegration of the hemoglobin.

3. Localised hemosiderosis

Brownish or slate grey buccal patches may be observed following haemorrhagic stomatitis, purpura, hematomas, epulis, etc. leading to difficult diagnostic problems as regards differentiation from the melanomas. However, dermal depots of hemosiderine coloured blue are observed by means of specific tests.

V. THE PIGMENTARY AND PIGMENTOGENOUS DERMATOSES

1. Pigmentary urticaria

Buccal patches have been observed in the course of certain cases of pigmentary urticaria. It manifests as dermatosis characterised by brownish or blackish cutaneous macules, giving the body a striped appearance, and having the property of becoming urticarial and pruriginous after rubbing or pressure. Histologically, we find under an epithelium the basal layer of which is abnormally rich in melanine, a dermal infiltration, the metachromatic granulations of which are demonstrated with Unna's Blue.

2. Lichen Nigricans

Buccal lichen nigricans is relatively rare, compared to the frequency of lichen planus. It appears as more or less rounded patches rather well circumscribed, at times assuming a reticulate appearance, black in colour, affecting the lips, tongue, internal surface of the cheeks and the soft palate. In almost all the cases a careful examination would reveal white lichen plaques superimposed upon the pigmented macules. Biopsy would demonstrate the characteristic appearance of the lichen with the presence of an abundant melanic pigment in the dermal melanophores. The sub-dermal or sub-basal detachment (Max-Joseph fissures) seems to be particularly frequent in this variety.

3 Acanthosis nigricans

This affection manifests a villous appearance of the tongue and a papillomatosis of the lips and cheeks, but these lesions are rarely pigmented.

4. Nigricant Syphilide

MILIAN pointed out the syphilitic origin of certain pigmentary patches particularly in cases of associated leucoplasia.

VI. THE TOXIC AND MEDICAMENOUS PIGMENTATIONS

I. The Metals

(a) Lead (chronic saturnism).

This is a professional intoxication, the buccal signs of which appear relatively late. These are:

—The Burton's Line having a bluish colouration, situated on the gingival mucosa outlining the dental crowns. It predominates on the lower gum in the region of the incisors and the canines.

—Gingivitis and porrhoea are frequently associated, as also a painful swelling of parotid glands.

—The slate grey patches of GUBLER may be observed on the internal surface of the cheeks.

Other clinical signs should be looked for: pallor, asthma, lead colics, polyneuritis, as well as the para clinical signs: anemia, an increase in the red cells having basophil granulations, increase of the plasma lead and the coproporphyrines III in the urine.

(b) **Bismuth.**

The bismuth pigmentation is noted in all patients receiving treatment with injections of the bismuth salts. It is rare after the ingestion of the carbonate or the sub-nitrate of bismuth. It appears 2 or 3 weeks after the commencement of the treatment. Having a slate blue or a blackish blue colouration, it affects:

—the gums, forming a marginal line at the level of the dental crown, more marked at the roots and in the carious teeth:

—the tongue, where it assumes a punctate form at the summit of the papillae, and appears as vertical striae on the margins;

—on the internal surface of the cheeks, the lips, and the palate, etc., forming large tattooed plaques.

Most often, the pigmentation remains isolated, not contra-indicating the continuation of the treatment and disappearing a few weeks after its termination.

At times, ulcerations appear resulting in a more or less intense bismuth stomatitis, the signs of which differ slightly according to the salt utilised; with the soluble salts, it is a late stomatitis having a sudden appearance; with the liposoluble salts, there is an early localised ulceration preceding the pigmentation.

(c) **Mercury.**

In the chronic professional intoxication with mercury, there usually appears a greyish silver gingival border, which is broader and lighter in colour than the Burton's Line. The gums are swollen and assume a fungoid appearance. The teeth, loose, assume a grey or black colouration (the mercurial teeth of LETULLE). The tongue slightly edematous, there is emaciation, digestive disturbances and intentional tremors resulting in awkward movements and slurring of speech.

(d) **Antimony**

Rarely, after a course of emetine, one may observe a fungoid or necrotic gingivitis with a black margin.

(e) **Silver**

Cutaneous argyria is much more frequent than the stomatitis. A brownish gingival border may at times be observed, resembling the genotype pigmentations.

(f) **Iron**

It gives rise to a reddish brown pigmentation.

(g) Copper

Very rarely it produces a greenish border with fungoid and haemorrhagic gums.

(h) Nickel

Gives rise to a greenish blue colouration.

2. The synthetic anti-malarials

Though first noted by the malarialogists, the dyschromias due to the synthetic anti-malarials have become much more frequent since the extension of the indications and of these medications. They are observed whatever be the substance utilised (Nivaquine, Quinacrine, Atebine, Flavoquine). Not being of a serious nature, independent of the dosage utilised, reversible, they are not an obstacle to the pursuit of the treatment.

The mucosal affection selectively attacks the palatine region where one finds the existence of symmetrical pigmented plaques, well defined, affecting the anterior two-thirds of the palate, having a slate blue colouration.

The possibility of blackish gingival and labial patches, and a yellowish brown pigmentation of the sclera has also been pointed out.

The pigmentation of the skin, having a slate brown colouration, chiefly affects the nail bed of one or several fingers or toes. Due to the transparence a brown line is seen transversely, 2-3 mm. in breadth, more distinct when pressure is exerted on the free border of the nail; at times, pigmented patches are also observed on the medial surface of the legs and in the perineal region. Conversely, a decolouration of the hair of the scalp and the body may be seen, at times partial and localised.

These hyper-pigmentations would be due to a hemosiderinic and melanic overloading in the corium and the papillary dermis within the pigmentophagic histiocytes (WORINGER and LAUGIER).

3. Myleran

The muco-cutaneous melanoderma appears only after a prolonged administration of the drug (3 years according to MATHE and LAUFER), and particularly in brunettes:

—On the mucosal surface of the lips, the pigmentation appears as round or oval plaques, arranged parallel to the labial border, having a bistre colour. In the muco-cutaneous region, the patches are more scattered, fine and linear.

—On the dorsal surface of the tongue, the pigmentation is mottled, On the ventral surface it is clearer, chamois coloured and diffuse.

—On the floor of the mouth, bistre coloured patches are situated symmetrically behind the salivary caruncles.

—The palate and the internal surface of the cheeks show a diffuse pigmentation.

—The gums are not affected.

The pigmentation also affects the skin (the upper eyelids, the latero-cervical regions, flanks, inguinal fold and the external surface of the thighs.).

Histologically, one finds the presence of enormous quantities of melanine in the basal layer and the papillary dermis.

VII. PIGMENTATIONS DUE TO OXYDATION OF KERATINE

Only a single affection of this type exists as regards the buccal mucosa: the black villous tongue. It is a relatively frequent affection characterised by a brown or black colouration of the median and posterior parts of the dorsum of the tongue. It is rarely accompanied by subjective signs and a sensation of a "foul mouth" usually attributed to a bad stomach.

"Two varieties of black tongue have been noted:

"—**The black villous or hairy tongue** induced by a brown colouration of the extremities of the hypertrophied filiform papillae of the villous tongue. It is the pigmentation of the corneated substance itself which gives this colouration, as the black ichthyosis. The duration of the affection is very prolonged, extending over a number of years with remissions and fresh attacks.

"—**The black medicamentous tongue** resulting, in a large number of cases, from the action of oxydising products; mouth washes and make up with oxygenated water or other oxydising medicaments. But here again, it is the terminations of the filiform papillae, normally, hypertrophied which manifest the affection, anterior to the lingual V, or developed upon a saburral tongue. At times a black tongue is noted after sucking or ingestion of antibiotics.

"The difference between these two types of black tongue appears to us rather artificial. In both the cases it is suggestive of a reaction of the keratine of the filiform papillae. Oxydation appears as the direct frequent cause of the reaction in the second variety, but it is probably the same mechanism which intervenes in the black villous tongue. Fungi have been thought to be responsible and several varieties have been isolated (glossophyton, saccharomyces linguae pilosae; candida albicans, tropicalis, krusei). Although these fungi are common saprophytes of the tongue, their role has not been excluded." (DEGOS).

VIII. THE TATTOOINGS

A certain number of buccal pigmentations are due to the inclusion of coloured foreign bodies in the tissues. These tattooings may be accidental, professional or therapeutic (by the last, we mean the accidental inclusions of foreign bodies during the course of dental treatments).

(a) **Accidental tattooings.**

The diagnosis is easily made. Inclusions of clinkers, tar, and gravel may be seen following road accidents.

The inclusions occur in cutaneous wounds of the lips and cheeks, at times also in wounds involving the mucosal surface. In the subsequent months, a blackish pigmentation, mottled or reticulate is found to develop around the cicatrices.

Gun shot wounds may also give rise to small black lenticular macular tattooings. When the shot is fired at a very close range, the powder particles give rise to a mottled tattooing around the orifice of entry of the projectile.

(b) **Professional tattooings.**

It may be observed in tar and cement workers, stone cutters, etc.

(c) **“Therapeutic” tattooing.**

This is the most frequent. It is frequently suggestive of grains of carborundum and black vulcanite detached from the abrasive discs. These grains are retained in the mucosal wound. Rarely, one may find the inclusion of a fragment of a broken disc.

Also, very frequently it manifests the inclusion of fragments of silver amalgam, either as the result of the fall of the debris of a filling into an extraction wound or the sub-gingival compression of a neighbouring filling, or exist beyond the apex of the filling paste containing grains of amalgam. The blackish grey pigmentation appears slowly, at times at the end of several years and may spread itself as oily specks. The clinical appearance may simulate a malignant melanomas, but radiography usually detects the presence of opaque particles.

Finally, carbon tattooings are seen which may be due to the use of a denture material which is now abandoned or from smoke, which was the case in a patient who had the habit of piercing the pyrrhoic abscesses which he frequently presented, with a needle flamed on a match.

IX. THE BUCCAL MELANOMAS

The melanomas so frequent on the skin are not exceptional on the buccal mucosa, though the equivalents of the numerous cutaneous varieties have not yet been described with regard to the mouth. Schematically, one distinguishes the malignant melanomas or the naevo cellular naevi from the malignant melanomas or the naevo carcinomas. It should be remembered that it is not the presence of melanine which characterises these different tumours, since some of them are even completely devoid of melanine, (the achromic melanomas) but a particular cell, the melanoblast, an embryonic melano forming cell is solely capable of giving rise to these tumours, whereas the physiological adult melanocyte never gives rise to them (DUPERRAT). It should also be noted that in the case of a melanic tumour, a partial biopsy is formally contra-indicated and complete excision should always be followed by a histological examination of the tumour.

I. Benign melanomas

There exist only a few observations published on the benign melanomas of the buccal mucosa. The statistics of CHAUDHRY in 1958 recorded 105 cases of malignant buccal melanomas. Now, the malignant melanomas of the skin are infinitely rarer than the benign melanomas. If one agrees with DUPERRAT that all malignant melanomas are the result of the degeneration of a benign melanoma, at times clinically latent, it may be deduced that the frequency of benign melanomas of the buccal mucosa is much greater than is generally believed,

(a) The lentigo.

Though rather frequent on the muco-cutaneous region of the lips, it is quite rare on the mucosa where it does not always manifest a well circumscribed lenticular appearance as on the skin.

Disseminated melanic patches may be seen on the internal surface of the lips and the cheeks, at times salient, with a bunching of lentigos on the exposed surface of the lips and around the mouth. One should then look for the association of a digestive polyposis which may affect the stomach even up to the rectum, and is at times the cause of abdominal pains, melaena and intussusception, giving rise to the Peutz-Touraine-Jeghers Syndrome. The digestive polyposis is capable of undergoing a malignant degeneration.

Histologically, one may find a melanocytic hyperplasia of the basal layer, as in the ephelides, but there is always in both the cases a melanoblastic junctional layer. Often there is the appearance of a junctional naevus, with or without a thecal production in the papillary dermis.

The lentigo is capable of degenerating into a naevo carcinoma if it is exposed to traumas.

(b) The verrucous naevus.

The verrucous naevus is very rare. It is the equivalent of the soft cutaneous wart. Histologically, one may find either a dermal naevus without junctional activity, at times of the neuro-naevus type, or a mixed naevus with junctional activity, malignant degeneration being more frequent in the latter variety. The discovery of such a tumour in the mouth necessitates a systematic excision, due to the risks of inevitable traumas.

(c) The circumscribed pre-cancerous melanosis of Dubreuilh

The circumscribed pre-cancerous melanosis of Dubreuilh had been observed in the buccal mucosa. It appears in adults 40-50 years old, manifesting a circular polycyclic patch, well circumscribed, having a polychromic appearance, since the colouration varies from one spot to another, the basic brown colour being marked by deeper patches, with at times a few areas of non-pigmentation. The surface may be smooth or verrucous. The histological appearance shows an intimate blending of the naevic and the epithelial cells and epithelial hyperplasia with a marked inflammatory reaction of the dermis which contains an enormous quantity of melanine agglutinated in large masses. Degeneration is very frequent, but the naevo carcinoma supervening on the melanosis of Dubreuilh appears to be less grave than the other varieties.

(d) The melanotic progionoma

The melanotic progionoma or the pigmented ameloblastoma is a maxillary tumour, extremely rare, which we mention here just to complete the classification, though it is not a true melanoma.

2. Malignant melanomas.

The malignant melanomas of the buccal cavity are rare tumours, but of a severe malignancy. Very often they are discovered late when they have already given rise to ganglionic metastasis, and the prognosis is then almost always fatal, all the more since they are frequently subjected to irregular therapeutic trials.

For this reason, it is rare to observe in the mouth the cancerisation of a pre-existing benign melanoma, the latter having remained unnoticed in most cases, and tumour thus presents as a "malignant melanoma from the outset."

The condition is most frequently localised to the hard palate (about 51%); next in frequency come the alveolar processes (20%) and the soft palate; the other localisations are very much rarer.

The diagnosis is easy if the tumour is frankly pigmented: it appears as a small round tumour, never attaining the dimensions of the epitheliomas or the sarcomas, having a more or less grey, blue or black colouration, soft in consistency, not tender, bleeding easily on contact. The surface may be smooth or nodular. At times soft nodular formations exist around the principal tumour. Besides, there may be an irregular ulceration manifesting a greyish nodular appearance bleeding on the least contact, surrounding the crown of the loose teeth.

When the adenopathy exists it consists of lymph nodes, never very large, firm, mobile, without peradenitis.

But usually, there may be more difficult cases where the pigmentation may be only at the periphery or even away from the tumour, the latter being only very slightly pigmented or even completely achromic. One should be careful not to mistake this appearance for a co-existing epithelioma with an ethnic pigmentation since it may result in a catastrophic biopsy. It should be stressed that this distant pigmentation does not necessarily manifest the extension of the tumour but may only correspond to a simple migration of the pigments secreted by the dermal melanophores.

Finally, there are cases where the pigmentation being extremely discreet is recognized only if it is systematically looked for, and others where the tumour is completely achromic, simulating a cyst, an angioma, an epulis or even a buccal neof ormation

To sum up, there is almost always the typical appearance of the junctional naevo carcinoma: the malignant melanoblastic cells invade the epithelium from below upwards, and the dermis from above downwards. The cells present numerous abnormalities; the mitoses are abundant and atypical. It is classical to distinguish the forms with the round so-called "epithelioid" cells from the varieties with the fusiform cells, the prognosis of which is more favourable. In fact, the two types are often represented in the same tumour.

In very rare cases, one comes across a naevo carcinoma manifesting a complete integrity of the junctional zone. Lastly, one may mention the recent observation of exceptional case of a malignant neuro-naevus of the superior maxillary.

X. THE PIGMENTATIONS OF UNKNOWN ORIGIN

It should be remembered that one still frequently comes across a buccal pigmentation which cannot be classified under any of the preceding groups. In olden times, these cases were simply considered as syphilitic, even if their serology was negative and they denied all venereal contacts. The problem is in reality much more complex, perhaps some of these cases may be explained by a metallic impregnation of the mucosa by electro-galvanism when there exist in the mouth fillings and dentures of various metals. Other may perhaps be some unknown naevi or endocrinopathies.

We have observed that buccal pigmentations are frequent, that they may co-exist with a cutaneous pigmentation or be isolated. To detect them, other than in those cases where they are particularly marked, it is necessary to carry out a systematic examination of the mouth under good technical conditions and in good light. Their etiological diagnosis may necessitate a certain number of complementary examinations.

The study of these pigmentations should not be neglected, since though some of them do not have any pathological significance or are absolutely benign, others which are not necessarily the most visible may reveal the presence of a highly malignant melanoma.—*LA REVUE DUE PRATICIAN, Vol. 14, No. 21.*

IMPORTANT TO OUR READERS

We receive many enquiries from both old and new subscribers to supply them with back numbers of the *INDIAN JOURNAL OF DERMATOLOGY AND VENEREOLOGY*. We usually run out of stock due to heavy demands. Hence all our Subscribers and Patrons are kindly requested to intimate the non-receipt of this bi-monthly Journal to the Managing Editor by the fifteenth of the succeeding month of publication. *INDIAN JOURNAL OF DERMATOLOGY AND VENEREOLOGY* is published always in last week of February, April, June, August, October and December during the year.

PLEASE MENTION YOUR SUBSCRIPTION NUMBER IN ALL YOUR COMMUNICATIONS WITH US
