raids, the number of donors who did not have a good night's rest was too small to be analysed with advantage.

Conclusion

Though the series is not large enough for many definite conclusions to be drawn from it, it would seem that the young faint more than the old and females more than males, and that the incidence is higher among clerical workers than among other groups. With regard to the significance of hunger and fatigue, for example, the smallness of numbers is naturally most obvious at the extremes of the hours between last meal and bleeding and of the hours of work on day of bleeding, where the effects would be most evident. The figures give little information, therefore, concerning the effects of these two factors. There is, however, a strong impression at this depot that hunger and fatigue are contributory causes in the fainting of donors.

It is not improbable that the high incidence of fainting among clerical workers is due to the fact that they are a selected physical type rather than to an occupational effect. It is especially likely that because of conscription the residual male clerical workers are below the normal physical standard.

I should like to thank the nurses at the depot who collected the necessary information from donors, and Dr. W. T. Russell for his help in preparing the figures for publication.

AVITAMINOSIS IN APPARENTLY HEALTHY TRINIDADIAN*^s

BY

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The gross states of avitaminosis seen in the dispensaries (Deane, 1938) and hospitals (Fletcher, 1937) of Trinidad have already been described. There is also a vast amount of subclinical avitaminosis which goes untreated.

This is an account of apparently healthy men who were examined for stigmata of vitamin deficiency when they applied for work in the oilfields. Besides being of academic interest such an investigation has a practical value from the employer's point of view: the worker who suffers from avitaminosis obviously cannot be very efficient; he readily falls sick and has to be looked after, and, when injured, his recovery time is excessively prolonged.

The group investigated is, in a way, selected—by the members of the group themselves. The average work applicant does not present himself for what has the reputation of being a fairly severe medical examination unless he feels that he has a reasonable chance of success. A small proportion of men, though sick, may be driven by lack of money or a nagging wife to seek work; a still smaller proportion with gross lesions may try to slip by what they think may be a cursory examination in the hope of being able to advance fraudulent compensation claims later.

TABLE I

Race	No. Examined	Average Age	Age Limits
East Indian	168	27.99	17-50
Negro	820	28.07	14-64
Mixed creole	16	27.90	21-55
Chinese	7	27.14	21-30

In this investigation 1,011 consecutive applicants were examined. The race and ages of these men are given in Table I.

* Abstracted from an M.D. thesis, University of Cambridge.

Signs of avitaminosis A which appear most frequently in children (e.g., Bitot's spots, xerophthalmia) would not be expected to occur very often at these ages. These men have passed from childhood without suffering from some of the more serious sequelae of avitaminosis (e.g., keratomalacia), and therefore represent a "seeded" group. Those less fortunate have either been carried off by intercurrent disease or been forced to take work in keeping with their poor physical condition. Thus it is further emphasized that the group investigated is a picked one, with health above the average.

Avitaminosis A

Growth Impairment.—Unfortunately a height-weight check was not made. Such cases as were noted under this heading were those of puny physique with a markedly under-developed musculature. Such a condition is obviously not entirely due to continued lack of vitamin A but to other factors (e.g., generalized malnutrition and cachectic states such as ankylostomiasis and malaria). Under-development of this type occurred in 15 (8.9%) of the East Indians and in 3 (0.37%) of the negroes. No instance was found among the mixed creoles or Chinese.

Bitot's Spots.—This sign occurred in one subject—an East Indian aged 23 who had also a rachitic chest, markedly carious teeth, and scaliness of the arms.

Xerophthalmia.—The incidence of xerophthalmia is shown in Table II.

Finely Scaled Skin.—The lesion is labelled thus to distinguish it from a coarsely scaled skin, which is found with vitamin B₂ and probably B₆ deficiency. Usually the skin lesions of avitaminosis A precede the objective eye changes (Mackay, 1937), but in this series one man had a finely scaled skin and Bitot's spots, and another had xerophthalmia with phrynodermia. The scaliness in question manifests itself in the dark-skinned races by a loss of the natural mat surface which is characteristic of a healthy skin in these people. The powdery scales give the

TABLE II

	Race	No. Affected	Associated Deficiencies					Dental Caries
			A	B ₂	B ₆	C	D	
Xerophthalmia	E.I.	2		1			1	1
	N.	3	1	1				3
	M.C.	0						
	Ch.	0						
Finely scaled skin	E.I.	5	1	4	2			3
	N.	19	8	15	5			10
	M.C.	1		1				1
	Ch.	0						
Phrynodermia	E.I.	13		7	2			9
	N.	63	10	36	4			32
	M.C.	1						1
	Ch.	0						
Scrotal dermatitis	E.I.	16	3	11	1	1		8
	N.	36	7	20				14
	M.C.	0						
	Ch.	0						
Coarsely scaled skin	E.I.	18	7	10	6			10
	N.	149	19	45	12			57
	M.C.	1						
	Ch.	0						
Hyperpigmentation	E.I.	10	2	8	1			3
	N.	60	17	42	3			28
	M.C.	1						1
	Ch.	0						
Hypopigmentation	E.I.	8		7	1			3
	N.	49	13	38	5			23
	M.C.	0						
	Ch.	0						

impression that the skin is lighter, but this apparent hypopigmentation must not be confused with a true loss of pigment, which, I believe, is due to vitamin B₂ deficiency. This condition of fine scaliness is seen principally on the forearms, sometimes on the forehead, and less often on the shins and thighs. When seen in the last-named areas it must be distinguished from the effects of certain forms of vitamin B₂ lack. Fine scaliness has also to be distinguished from ringworm and from leprosy. The distribution of this manifestation is included in Table II.

Phrynodermia.—This condition, which has already been adequately described by Nicholls (1938), must in this type of investigation be carefully distinguished from the condition I have termed "pseudo-phrynodermia" (Earle, 1941). Here a toad-skin appearance is produced by a plugging of the pilosebaceous follicles of the wrist and olecranon surface of the elbow by lubricating oil. It occurs in uncleanly negro and East Indian motor mechanics, garage hands, etc., and may be accompanied by a low-grade infection of the hair follicles. Table II shows that phrynodermia is much more frequent than finely scaled skin among both East Indians and negroes.

Other Manifestations.—Other signs which have been ascribed to vitamin A deficiency (nyctalopia; degeneration of respiratory, alimentary, and urinary tract epithelium; lowered resistance to ergot; nerve degeneration) were not seen or could not be determined.

Avitaminosis B₁

This has been held to be a part cause of impaired growth, which has already been dealt with above. The other manifestations of vitamin B₁ lack (loss of tone of the gastrointestinal tract, beriberi) were not seen or could not be investigated.

Avitaminosis B₂

Pellagra.—Mild pellagra occurred in 4 instances (2 in East Indians and 2 in negroes). Stannus (1940) observes that sub-clinical pellagra or pellagroid states may continue for months or even years before being succeeded, in untreated cases, by frank pellagra. Pellagra may be precipitated by: (a) the consumption of large amounts of sugar; (b) increased work; (c) infections such as ankylostomiasis and chronic amoebiasis; (d) snake venom; and (e) cyanogenetic substances such as cassava. Factors (a), (b), (c), and (e) are common in Trinidad, as is the subclinical pellagroid state, yet pellagra is an uncommon condition in the colony.

Angular Stomatitis.—This is held by Nicholls (1938) to be due to deficiency of vitamin B₂ and by Sebrell and Butler (1938) to be a manifestation of ariboflavinosis. Only two members of the series, both East Indians, showed this sign. In one of them scrotal dermatitis was also present.

Scrotal Dermatitis.—Here the scrotal skin is thickened and has a glazed or sodden appearance. The normal scrotal rugae are flattened out and the whole organ may have an enlarged appearance. Pruritus is a frequent feature. The condition has to be distinguished from filarial lymph-scrotum and from ringworm. Its incidence is given in Table II, the percentages being: East Indians 9.53, negroes 4.44. Thus this sign of avitaminosis B₂ is more than twice as frequent among East Indians as among negroes.

Manifestations probably due to Lack of Vitamin B₂

The signs described below are not specially described in the textbooks, but they occur frequently enough, both in the series and in my practice, to make me suspect their being caused by avitaminosis B₂. These conditions are often found in association with tropical ulcer, which Clements (1936) believes to be due in part to lack of vitamin B₂.

Coarsely Scaled Skin.—This sign occurs principally on the lower extremities, and is bilateral in distribution. It is located mostly on the anterior borders, but may also be seen on the calves, knees, and, rarely, on the anterior surface of the lower half of the thigh. The affected skin is coarsely scaled, differing in this way from the fine "branny" scaling, usually of the forearms, which is seen in avitaminosis A. The skin, unlike that in true pellagra, is not especially influenced by exposure to the sun. It may show a slight intensification of pigment, but such hyperpigmented skin is dull in appearance and unlike that described under the heading of "Hyperpigmentation." In some cases this type of scaly skin is found in association with the type of hyperpigmentation and hypopigmentation described below, and it is likely that all three signs are but different stages or manifestations of the same vitamin deficiency. The incidence is shown in Table II.

Hyperpigmentation and Hypopigmentation.—The hyperpigmentation occurs on the tibial surfaces, less often on the knees and lower half of the thighs. It exists in two distinct

types: first, a darkening of the skin, together with a roughening and scalliness somewhat similar to that described in the paragraph above; and, secondly, a condition in which the skin has an atrophic appearance, remarkably like the scalp-skin of those unfortunates who, in the early days of x-ray therapy, sustained an overdose when treated for ringworm of the scalp. The skin in question has a glazed shiny appearance, quite unlike the glossy mat surface of the healthy negro or East Indian skin. The two types of pigmentary change frequently coexist; in fact, although I have placed the two conditions under separate headings, I believe that they have a common origin, and their allocation to one group or the other depends upon which manifestation is the more prominent. Sometimes the two signs are about equal in intensity, and then the ensuing mottled appearance closely resembles the erythema ab igne seen in white races. The margins of these areas of altered pigmentation blend imperceptibly with the surrounding healthy skin; there is no clear-cut line of demarcation. Besides having an unhealthy atrophic appearance, the skin is partially denuded of hair and has a plucked appearance. Total alopecia of the affected areas does not, however, occur. There is slight impairment of the sensation of light touch, but I have noted no other sensory disturbances. On such areas of lowered vitality a slight trauma may produce the so-called "tropical ulcer," or the idiopathic form of tropical ulcer may arise from no apparent cause. The incidence of both hyperpigmentation and hypopigmentation is given in Table II.

Tropical Ulcer.—The work of Clements (1936) suggests that tropical ulcer is closely associated with vitamin B₂ deficiency, the causative organisms being identical with those found in Vincent's disease. King (1940) has suggested that a possible relationship exists between pellagra and Vincent's disease. It is thought that Vincent's disease is also related to a deficient intake or utilization of nicotinic acid and its allied pyridine derivatives of the vitamin B₂ complex. Tropical ulcer, though frequently seen among actual employees, their wives, "keepers" (mistresses), and children, is rarely seen among applicants for work. This is easily explicable: tropical ulcer is a very crippling condition, and a man with this lesion, if passed as fit, might later advance a false compensation claim. It was therefore broadcast that those with ulcerated legs need not apply for work. Those few cases (three in number) which were detected at examination were in men who hoped that a lax examination might allow them to slip through. Typical scars of tropical ulcer occurring on the area of "deficient arterial overlap" (the anterior tibial surfaces, ankle, and dorsum of the foot) were as follows: East Indian, 38 (22.63%); negro, 156 (18.6%); mixed creole, 2 (12.5%); Chinese, 0. The lower incidence among mixed creoles and Chinese may be due not only to the richer dietary enjoyed by these races but also to the fact that they avoid heavy manual work, in the performance of which the traumata that usually initiate the condition are acquired.

Avitaminosis C and D

No evidence of deficiency of vitamin C was seen in the series. An exception is dental decay, but since polyavitaminosis and also calcium lack are concerned in its production it will be discussed separately.

With regard to avitaminosis D, rachitic chest and bony deformity were seen in 3 men: 2 were East Indians, one of whom showed phrynodermia and marked dental caries; the third was a negro who also had advanced dental caries. No signs of this deficiency were found among the mixed creole or Chinese applicants.

Dental Deficiencies

Dental caries was extremely common, and usually involved several teeth; the vast majority of carious teeth were either premolars or molars. The incisors and canines were not often affected. Besides the obvious caries, many gaps in the mouths examined testified to earlier extractions, and completely edentulous jaws occurred in some cases. Irregularity of alignment and tilting of the teeth in relation to the jaw are held to be the result of the deficiency of vitamins A and D; the irregularity is most pronounced in the lower incisors. Nicholls (1938) states that calcium deficiency is also a factor in the production of this lesion.

Dental defects in Trinidadians are due to improper feeding of the pregnant and lactating mother; unbalanced diet, with a deficiency of minerals, in the weaned and artificially fed child; and vitamin lack in the adult. The incidence of dental defect, as Table III shows, is very high.

TABLE III

Race	Caries Alone	Irregularity Alone	Caries with Irregularity	Pyorrhoea Alv. (very advanced)	Edentulous (One Jaw)	No. Affected
E.I. ..	48	9	14	—	2	73 (43.46%)
Negro ..	258	13	18	2	9	300 (36.5%)
Mixed ..	6	—	2	—	—	8 (50%)
Creole ..	2	—	—	—	—	2 (28.57%)
Chinese ..	—	—	—	—	—	—

Summary

Avitaminosis occurring in 1,011 consecutive apparently healthy men applying for work is described.

The racial incidence of the various manifestations is compared.

Lesions probably, though not certainly, due to vitamin lack are described and their incidence in the series tabulated.

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PSYCHOSIS ASSOCIATED WITH VITAMIN B DEFICIENCY

BY

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The case reported below is worthy of publication because it seems to be the first of the kind described in this country, and there may very well be a number of others lying unrecognized in general hospitals.

Case Report

A man aged 52 was admitted to hospital on October 8, 1941, with the following history: 1924, gastro-enterostomy; 1930, operation for adhesions; 1935, partial gastrectomy by Mr. Jennings Marshall at Charing Cross Hospital, for chronic simple ulcer. From that time there had been no further symptoms of indigestion, but in January, 1941, he began to notice a progressive loss of weight, which amounted to a stone. Six weeks later the next-door shop caught fire and "the shock of this put him to bed for six weeks, hovering between life and death." He had diarrhoea and vomiting the whole time and had lost another 13½ lb. in weight. When he got up the left leg from the knee down and the right foot were "dead." He attended an out-patient department for three months, and these symptoms improved somewhat, though he still continued to have a feeling of numbness in the legs.

A complete examination of the C.N.S. showed no abnormal signs other than bilateral doubtfully extensor plantar responses and a severe stammer. The patient said that he had always stammered slightly but that it had been worse of late. No sensory changes and no signs of peripheral neuritis or subacute combined degeneration of the cord were found. A barium follow-through showed no abnormality of the gastro-intestinal tract. Examination of blood films revealed only a very slight degree of secondary anaemia (red cells 4,530,000, Hb 81%). Six days after admission he fell down in the ward and could not get up because of the numbness in his legs; he had to be lifted into bed. On October 20 he suddenly got out of bed

and went into the courtyard, saying he must find a Bible. He said he was the wickedest man in Wales and must be punished for sleeping with his mother by being boiled in oil. He seemed to be slightly disorientated. The following day he was found sitting up in bed, staring fixedly at the middle of the counterpane, repeating monotonously and endlessly, "Oddle, oddle, oddle, oddle, oddle." He would interrupt this monologue to answer questions briefly, but would return to it immediately. He said that he made this noise because he was under compulsion from a spirit beneath the counterpane. Any other noise would do as well, and on request he substituted "Wacka, wacka" for the "Oddle, oddle." He was prepared to talk about his deadly sins and impending punishment, but showed no great interest in the subject. He was clearly orientated and gave no sign of failure of memory or intellectual impairment, carrying out mental arithmetic tests with accuracy and surprising speed. A full neurological examination disclosed signs previously not seen. The stammer, which was pronounced, seemed to have a dysarthric quality; there was definite perseveration in answering questions and executing commands. When put into awkward positions he maintained them in a katatonic way; he also showed echopraxia, clapping his hands repeatedly in imitation of the action once performed by the examiner. He was seen in consultation, and one consultant thought the deep reflexes were increased and the pupils sluggish and the diagnosis possibly one of G.P.I. Dr. William Sargent suggested a confusional state (without confusion) due to vitamin deficiency. Another consultant supported the possibility of a focal lesion in the left frontal or pre-frontal region. The patient continued in this state all day, and until a late hour that night was sitting up in bed in the darkness, saying, "Ay, ay, ay, ay, ay. . . ." This activity was not resumed after a night's sleep, obtained with a powerful sedative, though the patient said that the spirit under the counterpane was still there. Two days later his condition began to deteriorate. The abdomen became very distended with flatus, and the colon was packed with faeces. A lumbar puncture on October 28 showed a normal fluid. From obstinate constipation he passed into a state of severe diarrhoea and became dangerously ill, being put on the danger list. It was noticed that the buccal mucosa was reddened. By November 2 this state was even more marked, and the tongue was bright red, raw, and clean. He was given 100 mg. of nicotinic acid and 50 mg. of ascorbic acid thrice daily. With this treatment improvement was immediate, and by November 8 diarrhoea had ceased, he was taking food well, and was sleeping naturally. His mind was clear, though he could not remember all the mental symptoms of the past. He could recollect, for instance, saying "Oddle, oddle," but not why he had done so, nor anything about the spirit under the bedclothes or being the wickedest man in Wales. On November 29 he was returned home fully recovered. The plantar reflexes still had their same rather abnormal quality and the stammer persisted, although now obviously only a stammer.

Comment

Psychoses of a paranoid and dementing type associated with pellagra have been known for many years. More recently, American authors have drawn attention to the occurrence of neurotic and neurasthenic pictures in sub-clinical states of vitamin deficiency (Ruffin, 1941; Jolliffe, 1941) and to the occurrence of atypical psychotic states, also when characteristic pellagrous symptoms such as stomatitis, peripheral neuritis, and skin changes were notable for their absence (Spies *et al.*, 1938; Cleckley *et al.*, 1939; Sydenstricker and Cleckley, 1941). Frequently loss of weight and ill-defined neurasthenic and gastro-intestinal disturbances are prodromal symptoms. When acute psychoses occur, they are nearly always of the stuporous and delirious type and resemble closely the toxic-confusional psychoses of acute physical illness; they often occur in association with definite physical conditions, such as alcoholism or arteriosclerosis. Frontal symptoms such as spontaneous grasping and sucking movements have also, rarely, been noted and ascribed to a "nicotinic acid deficiency encephalopathy." The case described above