dark brown where degeneration is most marked. The blood sinuses are few and the connective tissue is scanty. There is a tendency to increase in the number of the acidophil cells (figure 3).

The epithelial cells of the glands of batch IV have a whorled arrangement, compact and very closely packed. There are areas of disintegration with dark brown nuclei, and hæmorrhagic patches. The blood sinuses are numerous and the connective tissue is moderate in amount. The acidophil cells as compared with the normal are much more numerous (figure 4).

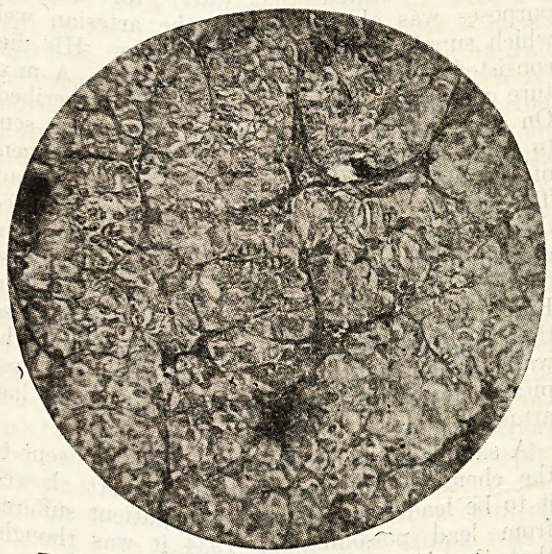

Fig. 5.—Parathyroid gland of animals (rats) on vitamin D deficient diet.

Those of batch V showed the epithelial cells in a state of hypersecretion, being loosely packed with strands of connective tissue in between. The nuclei are mostly round or oval and have taken lighter staining. The principal cells are numerous and are very prominent and the eosinophil cells are very few (figure 5).

Discussion

From the above it is evident that in vitamin A and B_1 deficiency there is a tendency to some degeneration (colloidal in nature) in the substance of the glands, whereas in vitamin C deficiency some amount of disintegration with hæmorrhagic patches is noticed here and there. McCarrison (1917) also noticed similar hæmorrhagic infiltration causing disruption of the polygonal cells in the glands of monkeys on a diet of autoclaved rice and butter (with all its vitamin C content lost). The glands of animals on vitamin D deficiency showed the principal epithelial cells to be hypersecreting. In vitamin B_1 and C deficiency the acidophil cells are also comparatively increased in number.

These histological changes in the glands of animals on deficiency diets proved clearly the influence of vitamins in maintaining the healthy condition of the secreting cells, in the absence

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LEAD POISONING FROM THE LINING OF COPPER OR BRASS COOKING UTENSILS, WITH THE REPORT OF A CASE

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One of my sub-assistant surgeons, a Hindu male aged 40, consulted me some time in June 1936 on account of griping pains in the abdomen with distension and frequency of stools. The stools were said to have been offensive, sometimes liquid and frothy, and two to four a day in number. The pain in the abdomen was of a colicky nature and was relieved by pressure and also by defæcation and was unrelated to food. The distension of the abdomen occurred usually at night and frequently disturbed his sleep. He was easily fatigued and listless and was not able to concentrate much. There was no nausea or vomiting and the temperature had been normal. He said he had had a rather severe attack during the last

(*Continued from previous column*)

of which they deteriorate and disintegrate. McCarrison (1921) is of opinion that 'excess of fat and starchy food together with deficiency of vitamins enhance the susceptibility of the organs to the action of the intestinal anærobes' responsible for the hæmorrhagic infiltration of the diseased parathyroids. In deficiency of vitamin D, however, the glands are found in a hypersecreting condition and the destructive changes are less evident; this is probably to combat successfully the slight lowering of the blood calcium level. All these findings amply corroborate the statement of McCarrison (1921) that 'in the presence of food deficiency the functional perfection of the thyro-parathyroid mechanism is very prone to be impaired' though it was based on meagre data.

Conclusion

Vitamins A, B_1 and C seem to have a profound effect in maintaining the healthy condition of the secreting cells of the parathyroid glands. Vitamin D, on the other hand, appears to promote a resting phase in the glands, in the absence of which the principal cells become hyperactive.

This investigation was commenced in the physiology department of the Edinburgh University and was later on completed in the department of physiology of the Prince of Wales Medical College, Patna.

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two days prior to his seeing me. It was elicited that the condition had been going on since May with remissions, but the attacks had then been mild and consisted chiefly of irregularity of the bowels which tended to be loose and there was a feeling of distension in the abdomen.

Previous history.—The patient suffered from 'enteritis' in 1926, amœbic dysentery in 1928 and datura and lead poisoning in 1934. In the last instance the urine had contained both datura and lead and a compounder was suspected of having administered the drugs. Since then the patient had been perfectly fit.

Personal history.—The patient was a vegetarian and lived chiefly on fruits, vegetables, *chappati* and milk. He was a non-smoker and total abstainer. There was nothing of note in the family history.

Physical examination did not yield anything of note. The patient was thick-set and short and his hair appeared to be prematurely grey. The tongue appeared a little raw. There was no tenderness in the abdomen. Spleen and liver were not enlarged. Heart and lungs were normal. The blood pressure was not taken. Routine examination of the stool and urine did not yield any abnormal result. The question of sprue was considered but was thought to be improbable and it was decided to keep him under observation and treat him with palliatives. He was prescribed gastric sedatives and carminatives and was advised as to his diet. The patient informed me subsequently that just about this time he suffered from pain in the joints, particularly in the wrist and phalangeal joints. But these symptoms were not brought to my notice during the consultation. The condition was very much relieved and nothing further was done. About the beginning of October 1936 all the symptoms recurred with greater intensity. The griping pains became worse and were frequently accompanied by pain in the back and legs. There was marked distension of the abdomen and the patient also suffered from palpitation and precordial pain and insomnia. He was much worried and presented the picture of a neurasthenic. The patient was admitted into Toungoo Civil Hospital on 3rd October, 1936. He stated that since the onset of the illness he had lost ten pounds in weight. He also complained of sore tongue.

The patient was obviously in great discomfort. The tongue was raw in the centre and on the edges with normal areas intervening. There was no blue line. The abdomen was distended. The liver and spleen were not enlarged. The lungs were clear. The cardiac sounds appeared a little weak, otherwise there was nothing abnormal in the circulatory system. The pulse was soft and regular. Blood pressure was 120/85. Neurological examination was negative. Pupils reacted to light and accommodation. All the reflexes were normal but wrist jerk was not tried. Qualitative examination of the urine was normal. Intestinal flagellates were discovered in the stool, but there were no ova or cysts of common intestinal parasites. The patient was kept in bed on milk diet, and a powder consisting of bismuth subnitrate, calomel and Dover's powder was prescribed. The next day the dragging sensation in the legs was worse, and the abdominal discomfort and distension were unrelieved. The patient passed loose stools. As the symptoms pointed to enteritis olive oil was prescribed.

There was only a little relief from the symptoms, and, suspecting that the case might be one of poisoning by lead or some other heavy metal, a careful enquiry was made as to the patient's diet, the source of water supply, etc., but no useful information was obtained. The patient stated that water for domestic purposes was obtained from the artesian well which supplies the Burma Railways. His diet consisted of milk and vegetables only. A mixture containing potassium iodide was prescribed. On 6th October a sample of urine was sent to the Chemical Examiner to the Government of Burma for testing for the presence of lead. The report received on 31st October showed the presence of lead. Meanwhile the symptoms abated fairly quickly and the patient was discharged from the hospital on 9th October. Enquiries were continued and it was discovered that the patient's cook had not been getting the water from the artesian well but from the hospital well nearby for some days prior to his last attack.

A sample of water from this well was sent to the chemical examiner but the report showed it to be lead-free. Though the patient suffered from lead poisoning in 1934 it was thought that the interval of remission of symptoms, lasting for over one and a half years, was too long to render it likely that the old trouble had recurred from re-mobilization of lead from the storehouses in the system.

The enquiry was now directed to the cooking utensils. It was discovered that the patient had bought three cooking pots made of yellow metal, probably brass or copper. Before taking them into use, as is customary in India and Burma, they were lined inside with a white metal normally used for the purpose. These utensils were put into use from the second week of April 1936. One of these was reserved for boiling milk, the major portion of which was consumed by the patient at night, before going to bed. The next consumer was the patient's son aged 11 years who occasionally complained of pain in his wrist. The boy was examined but nothing abnormal was discovered. There was another set of three cooking pots, lined locally during the month of September. I sent the patient to enquire from the persons who lined the pots what they used for the purpose. It was found that they used some white metal or alloy. This substance was used exclusively when the clients pay four annas for a pot, whereas some lead was mixed with this metal in which case the charges were less. The cheapest rate was one anna a pot and very little metal was used. It was admitted that lead was used for the lining of the pots taken

by the patient's cook. Attempts were made to get samples of the white metal and lead pieces for analysis, but these were unsuccessful.

On examining the pots it was found that the lining, which was renewed in September, had practically disappeared within a couple of weeks, whereas it was stated that if the lining material were pure it should have lasted for a much longer period. Milk boiled in one of the pots showed traces of lead. Thus the source of lead in the present case of poisoning was traced to the lining material of the cooking utensils. Milk cooked in one of the pots sent to the chemical examiner (about a month later) on 20th November, 1936, showed no lead, indicating how quickly the lead had been removed by the cooking. Since discharge from hospital the patient had been using aluminium utensils and there had been no recurrence of the symptoms. The urine of both the patient and his son were sent to the chemical examiner on 12th December when no lead was found.

During the first week of February I happened to be in Rangoon and took the opportunity of looking for persons engaged in the occupation of lining brass pots. I found one and after some persuasion he sold me pieces of metal he considered the pure metal used for the lining and another piece which he used in varying proportions when the work had to be done cheaply. The first had a bright silvery appearance, it was difficult to break and did not leave a stain on rubbing it on paper. The second sample was a grey and it marked paper. They were sent to the chemical examiner who reported that the first consisted of pure tin and the latter of commercial lead.

In retrospect one may review the case. The patient had suffered from several attacks of intestinal inflammation including that due to lead poisoning.

In March 1936 he bought new metal cooking pots and, having got them lined, used them from April. In May attacks of colic, abdominal distension and diarrhœa began and these became worse in June. He recovered from the attack, possibly because the lead from the lining had been removed and he had by then excreted the poison from his system. The symptoms recurred with greater severity about the end of September. He then had been using the newly-lined cooking pots for a couple of weeks or so. This time the attack was acute and his admission into hospital apparently saved him from a much worse attack. From the fact that when the patient left the hospital there was little left of the lining of the pots it could be surmised that a large portion must have become dissolved in the articles cooked within a couple of weeks of beginning to use these pots—hence the acuteness and the severity of the symptoms. The fact that the patient was living largely on milk probably prevented him from suffering complications as he was unwittingly administering to himself the antidote with the poison.

A public health problem

From the standpoint of public health this case presents important features. The use of copper or brass utensils for cooking purposes is common in India and Burma. Though aluminium *dekchies* are slowly replacing them they are still being used in large numbers. Before being put into use they are lined inside with what has been discovered to be tin. This gets absorbed or removed in time and the utensils have to be re-lined periodically. The way in which it is done is as follows:—The persons carrying on this occupation in Burma are Indians. When a pot is given for lining it is thoroughly cleaned by scrubbing the inside with straw and sand or a mixture of sand and earth collected on the road side. If the pot is small this process is done by hand. Water is sprayed inside the pot off and on during the scrubbing. Sometimes small bricks are used to scrape the resistant areas. If the pot is large the process is facilitated by making a small pit in the ground into which the bottom of the pot is securely fitted. The cleaning process is then done by a series of to-and-fro circular movements by the feet—the operator in the meanwhile standing inside the pot. After the old lining has been thoroughly removed the pot is rinsed with water and dried. The outside of the pot is rubbed with a paste of mud. I believe this is applied to prevent the surface from being smoked in the process of lining. A fire is made, preferably in an underground stove, with a hand bellows to keep the fire up. When the pot is absolutely dry and sufficiently hot a piece of the metal (tin) is thrown into it and applied thoroughly by rubbing with a piece of tow or cotton-wool whilst the pot is turned round and round with the other hand. The finishing touches are made by rubbing a piece of tin on to any thin or bare patch and rubbing it over with the cloth. The idea behind the lining appears to be two-fold. It enables the inside of the utensil to be kept clean and polished white. It also protects the metal of which the pot is made from being eroded by the material cooked which in several Indian and Burmese dishes contain very acid ingredients. In some cases the pot liners renew the lining cheaply by substituting lead for the more expensive tin or they adulterate the latter with varying proportions of lead. The consequences can easily be imagined. That cases of lead poisoning are not more frequently discovered is probably due to the fact that few patients consult medical practitioners and even when they do so the symptoms of chronic lead poisoning are so vague that the idea of lead poisoning is unlikely to enter the practitioners' mind.

Some years ago the question of the toxicity of aluminium cooking utensils came to the public notice and as aluminium utensils are greatly used in Burma and these are being locally manufactured I have been on the lookout for likely cases of poisoning by this metal,

but so far have been unsuccessful in diagnosing a particular case of poisoning from this cause. The general opinion (Oates, 1932) appears to be that the use of aluminium utensils is practically innocuous. Such cannot be said of any utensil containing lead. Legislation might be effected, prohibiting the use of lead and lead compounds for lining the brass or copper pots used for cooking purposes. An investigation might be made to determine the seriousness of the problem by seeking for cases of accidental lead poisoning from this source. Investigations might also be made as to whether any particular type of cooking dissolves the lead used in the lining. The enquiry might be extended to find out if the tin used in the lining has any toxic or therapeutic effect on the system. It would be interesting to discover whether the men employed in the occupation of lining the pots suffer from the effects of lead absorption from handling the metal and by the inhalation of the fumes that come out in the process of applying the metal to the utensil on the fire. It may be noted that strong fumes are produced and they are regarded by the Burmese people as being injurious to the health.

Comment

The question of the prophylaxis of industrial diseases in India and Burma is not yet properly established on an up-to-date basis. With regard to lead poisoning, no systematic study has been made of its incidence and prevention as an occupational disease. This is partly due to the fact that the industrial and commercial development of this country has been of very recent origin and so there have only been a few occupations in which lead poisoning could occur. So far, no cases appear to have been reported from these sources. We seem to be living in ignorance as to the causes of accidental lead poisoning, which is probably much more frequent than is realized.

Summary

A case of lead poisoning is described in which the poison was apparently absorbed with the food cooked in copper or brass utensils which had been lined inside with tin and lead. The process of lining the pot is described in detail. The danger of using such utensils is stressed.

It is suggested that an organized inquiry might be held into the question of lead poisoning of the workmen engaged in lining these pots and also of the persons who use them for cooking. It is possible that legislation might be found advisable to prevent such poisoning.

My thanks are due to U Chit Thoung, Chemical Examiner to the Government of Burma, for the numerous tests he has performed on my behalf.

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THE CHEMISTRY OF CALCIUM IN TUBERCULOSIS*

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Metabolism

Demineralization, Decalcification

There is a generally accepted hypothesis, warmly supported by the French authors, that demineralization is a common feature in tuberculosis. Much attention has been given to this subject by clinicians as well as biochemists. Attempts have been made to 'remineralize' the patient in order to make good the loss. Sergent, who reviews the early French work, was quite convinced of the value of calcium therapy.

As early as 1877 Senator based the hypothesis of demineralization on the finding that calcium is excreted excessively by the urine in tuberculosis. There were others who supposed that the calcium loss was due to intestinal tuberculosis; while some believed that it is the result of binding of the calcium with toxic products of the tuberculous lesion.

But the fallacy was not found out by the early writers. In the early days of clinical chemistry the data were obtained mostly by the analysis of urine, without calculation of the total intake of inorganic salts, or estimation of the fæcal excretion. To base principles of pathology on such evidence was therefore not only inaccurate but unreliable.

Ott in 1903 was probably the first to study on scientific lines the mineral metabolism in tuberculosis. He calculated not only the total output of minerals, but the total intake also. In his series of pulmonary tuberculosis there was not only no loss, but a plus balance of calcium and magnesium in all cases. In Maver's series of phthisis there was always an increase of calcium excretion in urine characterized by a decrease in the fæces so that, in all his cases, there was a small plus balance of calcium.

Besides these metabolism studies by Ott, Maver and others, tissue analysis of Steinitz shows that there occurs no deficiency of minerals in the tissues of tuberculous animals.

Cantarow (1931) laid much stress on the diffusibility of serum calcium in pulmonary tuberculosis. His findings were that in the active exudative state the diffusibility is greater than in the productive and proliferative type.

According to Krijewsky, serum calcium is higher in tuberculous pregnant women than in normal pregnancy.

Halverson *et al.* (1917) point out that the value of serum calcium depends mainly on nourishment. With adequate nourishment there

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