all the stamp of authority upon it; and, although compounded of diluted milk, cream, cane-sugar, and a pinch of salt, it is apt to be looked upon as a "fetish" for all the sick and healthy babies in the town of whatever age, and, as it is "humanized," what need is there for human milk?

Some harm has been done to these well-intentioned efforts of the municipalities by enthusiastic friends who have made use of statistics in a way which I cannot think the facts warrant. Thus in one town it was reported that in the year 1900 the rate of mortality among the infants fed from the municipal dépôt was 102 per 1,000, while the infant mortality of the whole town was at the rate of 188 per 1,000; these figures have been published widely to show that municipal feeding saved the lives of children at the rate of 86 per 1,000 during that year. One would be inclined to believe that the infants who were not fed from the dépôt were veritably on the "broad road which leadeth to destruction."

But surely more facts are needed. Of those "fed from the dépôt," how long were they so fed, a week or twelve months? What was the financial position of the parents? Did this class contain any of the infants who only survived their birth a few hours or days, or are these all relegated to the total mortality class? How did the mortality of those fed at the dépôt compare with the infants fed at the breast?

What I think is wanted is a careful investigation of the results of municipal feeding by some skilled observers entirely independent of the officials, and conducted on the lines of the investigations made by a medical committee recently in New York on the work of the charities supplying sterilized milk to the poor in that city.

In writing as I have done I wish to add that it is the "haphazard" methods of administering municipal milk dépôts that I condemn; I most certainly commend the efforts made in Liverpool and Battersea to work in connexion with the medical charities and to keep a weekly record of all the infants being fed from the dépôt.

I am perfectly open to conviction as to the general merits of municipal feeding, but I think more investigations are necessary in order to judge results, and before it can be said that the important problem of how to bring pure, fresh, clean milk within the reach of the poorer classes, and teach them how to use it aright, has been finally solved.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, August 29th, 1903, p. 469.

SOME NOTES ON THE URINE IN BERI-BERI.

By HERBERT E. DURHAM, M.B., B.C.CANTAB.,
F.R.C.S.ENG.

(Beri-Beri Commission of the London School of Tropical Medicine.)

THE following notes are not put forward as being in any way exhaustive, but rather to attract the attention of those who may have other cases of peripheral neuritis under their care (arsenic, alcohol, diphtheria, etc.) How far such different forms of "nerve disease" may be distinguishable by the degree to which the metabolism of the individual may be upset, is a point yet to be determined. It is possible that some aid in establishing diagnoses may be obtained by attention to such matters.

It has long been known, as from the work of Scheube¹ that the excretion of urea and phosphates is much diminished in beri-beri; the figures set out in the accompanying table serve to confirm this point. It will be observed that the figures for urea are extraordinarily low in certain of the cases, so low indeed that it was not until I had made control observations on the healthy, that I was satisfied that there might not have been some error of calculation or of observation. The possibility of some tampering on the part of the patients can be excluded by reference to the proportions of other constituents.

The patients were of three races—Chinese, Tamil (Madras Indians), and Sikh. It will be observed that the Chinese controls gave figures which were comparable to the ordinary physiological standard. Such, however, is far from the case with the control Tamil cases, and it may be doubted whether these men were really in a normal condition, apart from the trivial matters for which they were under hospital treatment. It should be stated² that in the Malay States, as Braddon and others have insisted, the Tamil escapes beri-beri in his home life so completely that cases are but rarely seen in the hospitals. The insusceptibility, or rather non-acquisition, of the disease is even more pronounced in the Sikh, and,

further, usually endures during confinement in gaol, so that beri-beri in a Sikh is most exceptional. In the case of the Tamil, it may be suggested as a possibility that for some reason their nerves are more sheltered from the action of the beri-beric poison, so that they only suffer from a disturbance of metabolism—a "beri-beri sine neuritide." Naturally the control cases are much too few in number to establish such a possibility as a fact, but if it leads to a more complete knowledge of the metabolism of the Tamil, the suggestion will be justified.

DIET.

In respect to diet these control Tamil cases were perhaps on a more liberally albuminous diet during their stay in hospital than in their life outside; for they had 4 oz. of salt fish four times, and 2 eggs three times a week, with vegetables, curry stuffs, and rice. The Chinese hospital patients have 4 oz. of dried fish or meat each day, with rice and vegetables. The diet of the prisoners contained 6 oz. of meat or fish a day in the case of the long-sentenced men, but none in the case of the short-sentenced men. Two cases with extraordinary low urea values (Table Nos. 6 and 17) had been on bread and water for the twenty-four hours during which the urine was collected. The question of malingering naturally has frequently to be taken into account in gaols, and in these two cases the signs of beri-beri were not of severe type, though the diagnosis seemed to be confirmed by the low urea figure, which is below a starvation amount.

UREA.

I did not have the good fortune to examine a case of sudden death shortly before the time of decease, but it is probably not unreasonable to conclude that an individual who is only excreting a fifth or a sixth of the proper amount of urea might be prone to the onset of the sudden death which is so commonly unanticipated in the disease; nor again is there cause for surprise that these two patients complained of not feeling well, although neither cardiac distress nor paralytic symptoms were prominent. They had no albuminuria.

The question arises whether the diminution of the urea is due to a locking up in the tissues, to a deficient removal by the kidneys, or to an actual diminution of output by the effective organs.

It may be stated here that none of the samples tested showed the slightest trace of albumen; this, coupled with the comparatively normal amounts of uric acid and kreatinin, points to the conclusion that the kidneys cannot have been seriously at fault. Moreover, the marked diminution of the phosphates and sulphates confirms the idea that the production is really at fault.

The two following direct observations are opposed to the idea of a locking up of already-formed urea in the body; though the readings are perhaps a little high, it is to be remembered that the sources were *post mortem*; and that there may have been other substances yielding nitrogen with hypobromite.

1. Residue of alcoholic extract of 100 grams of blood after removal of cholin-like bodies, etc., by means of alcoholic platinum chloride, yielded nitrogen corresponding to 0.0308 grams of urea, that is, 0.3 per mille.

2. Residue of 40 grams of pericardial fluid similarly treated gave nitrogen corresponding to 0.0112 grams of urea or 0.28 per mille. Hammarsten³ gives the normal urea content of the blood as 0.2 to 0.15 per mille.

OTHER CONSTITUENTS.

In contrast to the urea the kreatinin, as estimated by S. Johnson's process, may be hardly decreased, indeed it is proportionately increased in nearly all of the cases.

Whilst both the sulphuric and the phosphoric acids show absolute diminution the increase proportionately to urea is more marked in the case of the former than of the latter; it will be remembered that Scheube insists that the phosphoric acid is relatively increased in the majority of cases. Mott and Halliburton have shown that during degeneration nerves lose a considerable amount of their phosphorus, but their degenerated condition can hardly be the direct cause of the diminished excretion in that the phosphoric acid obtainable from the whole of the human nervous system is estimated to be only 12 grams. (Voit.⁴) whilst that of the muscles is given as 130 grams.

The proportions of the conjugated sulphuric acids and of the neutral sulphur were not found to be affected in the first few cases, so that further estimations were deemed to be superfluous.

Several estimations of the purin bodies were made, but

Analyses of Urine.

No.	Nationality.	Condition.	Date.	24 Hours' Urine in c.cm.	24 Hours Urea Grams.	24 Hours' Uric Acid. Grams.	24 Hours' Kreatinin. Grams.	24 Hours' P ₂ O ₅ . Grams.	24 Hours' SO ₃ . Grams.	24 Hours Conjugated SO ₃ . Grams.	24 Hours' SO ₃ after Oxidation. Grams.	24 Hours' SO ₃ of Neutral Sulphur. Grams.	Ratio Uric Acid Urea.	Kreatinin Urea.	P ₂ O ₅ Urea.	SO ₃ Urea.
—	European	Normal (Schäfer's Text-book)	—	—	33.180	0.550	0.910*	3.160	2.010	$\frac{1}{10}$ of total SO ₃	—	$\frac{1}{10}$ of total S	$\frac{1}{88}$	$\frac{1}{38}$ *	$\frac{1}{16}$	$\frac{1}{18}$
†1	Chinese	Healthy	16/10/02	1,180	32.710	0.590	1.670	2.863	1.986	0.163	2.400	0.420	$\frac{1}{10}$	$\frac{1}{18}$	$\frac{1}{11}$	$\frac{1}{18}$
†2	"	"	16/10/02	1,000	26.117	0.790	1.442	2.699	1.450	0.070	1.731	0.271	$\frac{1}{17}$	$\frac{1}{18}$	$\frac{1}{16}$	$\frac{1}{18}$
†3	"	Recent beri-beri	4/9/02	1,280	14.660	Very little not weighed	0.770	1.740	—	—	—	—	—	$\frac{1}{19}$	$\frac{1}{8}$	—
†4	—	"	19/12/02	1,670	11.110	0.105	—	—	—	—	—	—	$\frac{1}{11}$	—	—	—
†5	Chinese	"	16/9/02	1,600	18.660	Less than 0.350	1.020	1.180	—	—	—	—	$\frac{1}{60}$	$\frac{1}{19}$	$\frac{1}{17}$	—
†6	"	Beri-beri ? recent	16/9/02	1,600	4.480	Less than 0.110	0.697	0.620	—	—	—	—	$\frac{1}{15}$	$\frac{1}{8}$	$\frac{1}{1}$	—
†7	"	Recent recurrent beri-beri; critical urination	27/10/02	4,400	15.750	0.280	1.730	0.970	1.060	0.086	—	—	$\frac{1}{10}$	$\frac{1}{1}$	$\frac{1}{17}$	$\frac{1}{18}$
†8	—	"	3/11/02	2,820	27.470	—	2.080	1.950	2.330	0.159	—	—	—	$\frac{1}{13}$	$\frac{1}{14}$	$\frac{1}{15}$
†9	Chinese	10 months' beri-beri	31/10/02	645	8.300	0.480	0.790	0.750	—	—	—	—	$\frac{1}{16}$	$\frac{1}{1}$	$\frac{1}{11}$	—
†10	Tamil, control	Synovitis	19/12/02	1,640	10.850	0.210	—	1.632	—	—	—	—	$\frac{1}{14}$	—	$\frac{1}{1}$	—
†11	"	Ulcus cruris	24/1/03	1,610	9.930	1.050	1.030	—	—	—	—	—	$\frac{10}{99}$	$\frac{1}{16}$	—	—
†12	"	"	24/1/03	1,590	9.350	0.920	1.280	0.860	—	—	—	—	$\frac{1}{10}$	$\frac{1}{1}$	$\frac{1}{11}$	—
†13	"	"Rheumatism"	24/1/03	840	6.740	—	1.020	—	—	—	—	—	—	$\frac{1}{1}$	—	—
†14	"	Fistula in ano	24/1/03	1,200	9.750	0.590	1.240	—	—	—	—	—	$\frac{1}{19}$	$\frac{1}{8}$	—	—
†15	Tamil	Recent beri-beri	8/10/02	1,640	10.060	0.480	0.700	0.860	—	—	—	—	$\frac{1}{14}$	$\frac{1}{14}$	$\frac{1}{11}$	—
†16	"	Recent beri-beri, very oedematous	3/11/02	2,700	8.320	0.590	1.510	—	0.970	0.056	—	—	$\frac{1}{13}$	$\frac{1}{1}$	$\frac{1}{17}$	$\frac{1}{8}$
†17	"	Incipient beri-beri	8/10/02	1,245	4.060	0.540	0.950	0.550	0.305	—	0.410	0.105	$\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{1}$	$\frac{1}{13}$
†18	Sikh	Recent beri-beri	2/10/02	720	9.950	0.030	0.630	0.760	—	—	—	—	$\frac{1}{36}$	$\frac{1}{15}$	$\frac{1}{13}$	—
†19	"	"	8/10/02	1,140	6.110	0.110	1.020	0.580	0.760	—	0.869	0.109	$\frac{1}{26}$	$\frac{1}{8}$	$\frac{1}{16}$	$\frac{1}{8}$
†20	"	"	14/10/02	940	12.080	—	—	1.880	—	—	0.828	—	—	—	$\frac{1}{1}$	—
†21	"	"	27/10/02	1,630	6.650	0.530	0.700	0.590	0.530	0.023	0.633	0.103	$\frac{1}{13}$	$\frac{1}{8}$	$\frac{1}{11}$	$\frac{1}{13}$
†22	"	"	7/11/02	1,600	3.890	0.040	—	—	—	—	—	—	$\frac{1}{108}$	—	—	—

* Kreatinin by zinc method. † Prisoner, long sentence. § Prisoner, short sentence. ‡ Patient in district or general hospital.

owing to various accidents the following only were brought to conclusion. Still they do not appear to be of much importance in the elucidation of the disease.

Estimation of Purin Bodies.

No. (vide Table).	7	21	4	10
Ammonia soluble ...	0.067	Lost	0.037	0.059
Ammonia insoluble ...	0.032	0.009	0.008	0.005
Total ...	0.099	—	0.045	0.064
Uric acid ...	0.283	0.534	0.105	0.210

Some further inquiry was made to determine whether any other substances of interest were to be found. It is to be regretted that the means for the estimation of the total nitrogen were not available. Some attempts were made to get figures for the hippuric acid, but no useful figures were obtained though the substance was readily demonstrable.

Some attempts were made to isolate bodies of a basic nature from the urine. In demonstrating the presence of cholin or cholin-like substance in the blood or other body fluids it was found that the syrupy, highly alkaline residues obtained by alcohol extraction yielded up a substance to benzol which gave a double salt with platinum chloride crystallizing in octahedra from 15 per cent. alcohol. Thus the difficulty of employing sufficiently anhydrous alcohol in so moist a climate was avoided, and the presence of potassium salts could be definitely excluded. The shake-out method with

benzol was consequently applied to several urine samples both before and after alkalization with quicklime. A number of double salts with platinum (gold salts) were only made with the "cholin" of the blood, etc.) were obtained, but, like Mott and Halliburton, I failed to get any octahedra suggestive of cholin. The only substance worthy of mention was one whose platinum compound was of a brilliant orange-vermilion colour, and was soluble in strong alcohol. Marked quantities were obtained from the beri-beric cases, and but mere traces in the few controls. The quantities obtained were not sufficient for further examination. The scarlet body usually crystallized in minute octahedral crystals.

METHODS ADOPTED.

Since in the tropics conditions are not always favourable in respect to materials and apparatus, a few words on the methods employed may be added in order that the value of the estimations may be judged. In all cases the urine was measured and then filtered. It was directed to be passed directly into large stoppered bottles into which a few drops of toluol had been placed to prevent putrefactive changes; the twenty-four-hour periods were arranged from 6 a.m. to 6 a.m. to avoid delay for the like purpose. Albumen was tested for and found absent before proceeding.

A good delicate balance was available, and gravimetric methods had to be adopted in most cases; these had the disadvantage of limiting the number of observations.

Urea was estimated with freshly-made hypobromite solution, the quantity taken being 5 c.cm. and repeated on 10 c.cm., where the read-

ing was very low; with normal urines only 2.5 c.cm. could be used on account of the size of the only available burette. The same measuring cylinder was used throughout, and after draining it was rinsed out with water into the testing tube to avoid loss. Temperature correction to 50° F. and 37.1 c.cm. nitrogen taken as equivalent to 0.1 g. of urea.

Kreatinin.—400 c.cm. of urine were treated after S. Johnson's method, about a teaspoonful of potassic acetate and 100 c.cm. of saturated corrosive sublimate being used. After the immediate filtration the mixture was stood for forty-eight hours and filtered; after this period a precipitation of crystalline flakes occurred. The figures under kreatinin (or better perhaps "Johnson precipitate") are reckoned at 19 per cent. of the mercury compound. It should be added that the figures quoted from Schäfer are the absolute kreatinin estimated by more exacting methods.

Uric acid was precipitated by Hopkins's method with excess of ammoniac chloride which itself had been recrystallized to remove particulate matters; the precipitate was taken direct upon a tarred asbestos wool filter by a suction pump or syringe. After washing with saturated ammoniac chloride and a little spirit, the tubes were left to stand with a few drops of 10 per cent. HCl in spirit, whereby the uric acid was liberated in crystalline form. Later the uric acid was washed with a little water and spirit, and dried eventually in partial vacuum over CaCl₂ and quicklime to constant weight; 100 c.cm. were used for each titration. There did not appear to be any loss in the wash filtrates, as judged by the murexide reaction.

Phosphoric Acid.—The only available mode of estimation was as magnesium pyro-phosphate. A sulphate of magnesia mixture was used, the precipitate being taken up with dilute sulphuric acid, and reprecipitated with ammonia; 200 c.cm. of urine were used in the determinations.

Sulphuric acid was taken with samples of 100 c.cm., which were boiled respectively with 5 c.cm. of acetic or hydrochloric acid (the former subsequently with HCl), and precipitated with barium chloride. The weighings were direct without incineration.

Oxidation for the *neutral sulphur* was done by cautiously adding excess of permanganate day by day until a permanent pink colour was obtained.

The *purin bodies* were doubly precipitated with ammoniacal silver nitrate after removal of phosphates and intermediate removal of uric acid with dilute sulphuric. After reduction of the silver compounds with H₂S they were classified into ammonia soluble and insoluble.

The estimation of sodium, potassium chlorides, etc., was not attempted.

My thanks are due to Drs. Travers, Gimlette and McClosky, of the Malay States Service, for the use of their cases and other help. I am also indebted to the Government of the Malay States for the use of the laboratory at Kuala Lumpur, until they behaved with such discourtesy that it was not possible to remain as a guest of such inhospitable hosts; Captain Rost kindly offered facilities in Rangoon, but unfortunately the season was not favourable for the study of beri-beri. I must also express my gratitude to Dr. F. Gowland Hopkins for much advice.

SUMMARY.

Curtly put, the above notes show that the metabolism in beri-beri is seriously diminished. The amounts of urea, phosphoric, and sulphuric acids may be very greatly reduced; it is suggested that the contributing organs, for example, liver, are seriously disturbed in their output, which is in accordance with the fact that patients complain of illness before the onset of marked symptoms of the involvement of the peripheral nerves.

The conjugated sulphuric acids and the neutral sulphur do not appear to be proportionately diminished.

The purin bodies do not seem to be of great interest in beri-beri; as might be anticipated, they were abundant in the critical urination of a highly oedematous case.

In a number of Tamils, on comparatively liberal diet, in whom no beri-beric symptoms were detected, the excretion of urea and phosphoric acid was found to be remarkably low, though their uric acid was high.

BIBLIOGRAPHY.

¹ Scheube, *Die Beri-berikrankheit*, Jena, 1894. ² Durham, Notes on Beri-beri, *Journal of Hygiene*, 1904. ³ Hammarsten, *Lehrbuch d. physiol. Chemie*, fourth edition, 1899, p. 173. ⁴ Voit, *Hermann's Physiologie*, VI, 1, p. 80.

MYCOSIS FUNGOIDES OR ALIBERT'S DISEASE.

By JOHN REID, B.A., M.D.,
Bandon.

Mycosis fungoides, which was described by Alibert in 1814, is extremely rare in Ireland. The following case may therefore be considered to have special interest:

CLINICAL HISTORY.

The patient was 38 years old, small, stout, and fair-haired. Family history: All healthy and long-lived, but one uncle

suffered from pityriasis rubra. Prior to the first stages her illness she suffered from absolute amenorrhoea for three years. By trade she was a dressmaker, and accustomed to sit sewing for hours at a time—in fact, used to spend almost twelve hours daily plying her needle. Constipation and amenorrhoea, I wish to point out, were well-marked symptoms before the disease manifested itself. When I was asked to see her she presented the following phenomena of the pre-mycotic stage: Round, sharply-defined erythematous patches, with scaling, about 3 in. wide, on both cheeks, and covering anterior tragus of the ear. These patches healed in a couple of weeks. I regarded them at the time as purely eczematous. They left no marks, but shortly broke out again, and also on front of the chest, healing in the centre, and extending at the periphery, and so assuming irregular shapes which are almost characteristic of the disease. Strange to say, there was no pruritus in the area of face affected. This stage lasted three months.

The second stage next came on, and the patches became infiltrated plaques, with nodules over the face, head, back, and chest; they had a reddish-brown colour, and were only slightly scaled. After changing shape for about three months a condition of infiltration of the entire skin took place, from the level of the nipples down the skin became greatly thickened, but not excoriated or fissured. Above the level of the nipples the skin of the chest became fissured, and well-developed nodules soon appeared on face, scalp, and back of neck. The fungoid stage very quickly came on.

The characteristic tomato-like tumours appeared on the face and scalp only. As may be seen from the photograph they vary in size from a bean to a small orange, and are sessile, lobulated, and distinctly circumscribed, flat and ulcerated on the surfaces, and also on the edges where they rub together. I noticed also that a few growths disappeared by cicatrization, the process being helped by pressure of surrounding growing tumours. Pus was common between the tumours. One on upper lid closed the eye, and one on lower lip enlarged it in width to 2½ in. The photographs are absolutely characteristic. The tumours on the face show an almost lepra-like deformity, closing the eyes with their size and weight, producing the leonine brow and the elephantiasis ear.

The variety of ways in which tumours come and go was not apparent here. None of these large tumours disappeared spontaneously, some ulcerated till they became mushroom-like growths, only two or three contracted to a smaller size from pressure, but never absolutely disappearing, and all articulating I might almost say, on the patient moving the muscles of the head and face.