Comparative Mortality in Cities of Bengal, Bombay, Madras, North-Western Provinces, and Punjab having more than 50,000 inhabitants.

BENGAL (AUGUST 1884).

CITY OR MUNICIPALITY.	Area in acres.	Population.	Number of persons to an acre.	Death-rate per 1,000 of population for the month.	Actual death-rate per 1,000 of population.
Suburbs ...	14,413	251,439	17	Returns not received.	
BOMBAY (MAY 1884).					
Bombay ...	14,052	773,196	55	Returns not received.	
MADRAS (MAY 1884).					
Madras ...	17,164	406,117	23	Returns not received.	
NORTH-WESTERN PROVINCES (JUNE 1884).					
Moradabad ...	1,650	67,387	40	3·0	36·0
Bareilly ...	2,785	103,160	37	2·6	31·2
Shahjehanpore ...	5,625	69,892	12	2·7	32·4
Meerut	401	60,948	151	3·1	37·2
Koel	400	62,443	156	4·5	54·0
Muttra	1,146	55,016	48	6·7	80·4
Furruckabad ...	2,551	74,872	29	2·9	34·8
Agra	14,425	138,094	9	2·8	33·6
Cawnpore	2,389	151,444	63	4·2	50·4
Allahabad	19,747	150,338	7	1·4	16·8
Goruckpore ...	2,920	57,922	19	2·1	25·2
Benares	3,141	208,083	66	1·1	13·2
Mirzapore	3,376	85,362	25	2·1	25·2
PUNJAB (FROM 1ST TO 28TH JUNE 1884).					
Delhi	1,437	117,363	81	4·5	54·0
Umritsur	807	44,216	178	3·4	40·8
Lahore	461	97,208	218	2·5	30·0
Peshawar	500	50,292	118	1·9	22·8

ABSTRACTS & EXTRACTS.

A MEMORANDUM ON THE "COMMA-SHAPED BACILLUS," ALLEGED TO BE THE CAUSE OF CHOLERA.

BY SURGEON-MAJOR TIMOTHY RICHARDS LEWIS, M.B., *Assistant Professor of Pathology, Army Medical School.*

With a view of studying the phase which the cholera question has now entered upon, in consequence of the publication of the results of the investigations of the German Cholera Commission in Egypt and India, I availed myself of the opportunity which the present vacation at the Army Medical School afforded of proceeding to Marseilles, where the disease has been prevalent since the end of June. Sir Joseph Fayrer was so kind as to enlist for me the valuable assistance of Dr. Le Roy de Méricourt, Médecin en Chef of the French Navy, who, in various ways, did his utmost to further my wishes. Dr. Marroin, the Chief of the Sanitary Department in Marseilles, was so good as to introduce me to the authorities of the Pharo Hospital, where the cholera cases are treated, and where, with the permission of the principal medical officer, Dr. Trastour, I was able to renew my acquaintance with the disease, and to collect material for studying afresh the microscopy of the intestinal discharges.

Before, however, referring to the results of my own observations, it will be convenient to epitomise the published history of the German Commission; to point out the salient features of the results of their investigations in Egypt and in India; and to make a few brief comments on such of the circumstances and conclusions as appear to call for notice. Shortly after the arrival of the Commission in Egypt, Dr. Robert Koch reported, on behalf of himself and his colleagues, that no special micro-parasites had been discovered in the blood, the lungs, the spleen, the kidneys, or in the liver in cholera, but that the intestinal mucous membrane was permeated by certain bacilli which nearly resembled in size and form the bacilli found in glanders. As is well known these bacilli are straight, and are, in fact, uncommonly like the ordinary microphytes associated with decay. Dr. Koch also states in connexion with this subject that he had, previous to proceeding to Egypt, found similar bacilli in the intestinal mucous membrane of four natives of India, but that he had then looked upon them as due to merely post-mortem changes. When he came to Egypt, however, and found these same bacilli in the intestines of perfectly fresh cases, he felt that an important link was furnished towards establishing the identity of the disease in Egypt with Indian cholera.

It is highly probable that the specimens from India which Dr. Koch had examined were those which were sent, at the request of the Imperial Health Department in Berlin, by the Sanitary Commissioner with the Government of India. These consisted of numerous dry-cover glass specimens of blood which I had collected from several cholera patients, and of portions of the viscera of four natives who had died of the disease. All these were examined by me before they were despatched, and portions of each were reserved for further study. I had heard nothing further of them, but the publication of the remarks above referred to in Dr. Koch's Report of September 17th, 1883, from Alexandria, recalled them to my mind, and I was glad to infer that my own negative results had been confirmed in Berlin. As already observed, no importance had been originally attached to the organisms which were present in the intestinal mucosa. During the last six months I have examined hundreds of stained microtome-sections of these four, and of other specimens of cholera intestines in my possession, and have found that when the mucosa is infiltrated with microphytes at all they are either micrococci, bacteria, or long-oval, and straight bacilli.

In the report of the Commission, dated Calcutta, February 2nd, 1884, Dr. Koch, however, announces for the first time that the specific bacillus of cholera is curved or comma-shaped, and not straight, so that apparently it had become necessary to abandon the microbe first fixed upon. Assuming that the four specimens from natives of India which had been examined by Dr. Koch were those which passed through my hands, the evidence they furnish seems to be in accordance with this view, as in not one of them have I been able to detect any invasion by unmistakable "commas," though at least one of the specimens may fairly be characterised as abundantly infiltrated (in the manner described by Dr. Koch) by straight (and as I prefer to call them) putrefactive bacilli. Judging from my own experience, therefore, any extensive infiltration of the intestinal mucous membrane in cholera by comma-shaped bacilli must be exceedingly rare; and this, I believe, is likewise the experience of the members of the late French Cholera Commission, MM. Straus, Roux, and Nocard, whose acquaintance I had the pleasure of making at M. Pasteur's laboratory on my return through Paris.

Whilst at Marseilles I had, as already stated, opportunities of observing numerous specimens of choleraic excreta, and found that comma-shaped bacilli were, more or less conspicuously, present in all of them, though in some instances more than one slide had to be examined before any could be satisfactorily detected. It may also be mentioned that some of the discharges in which these organisms were present manifested an acid reaction when tested with litmus paper. As Dr. Koch himself remarks, the proportion which the comma-shaped bacilli bear to other organisms in the dejecta varies greatly. In some instances only one or two specimens are to be found in the field of the microscope, while in others they are very numerous, and Drs. Nicati and Rietsch (who are at present engaged in the study of the disease at Marseilles) were so kind as to show me a specimen of choleraic material they had obtained from the small intestine in which the "commas" existed almost to the exclusion of all other organisms. This is a condition, however, which, I understand, is exceedingly rare. On the

other hand, I have seen samples of choleraic dejecta in which totally different organisms prevailed to a like exclusion of others; and in one instance at Marseilles spirilla of various sizes and forms were the most conspicuous of the micro-organisms present. So far, therefore, the selection of the comma-shaped bacilli as the *materies morbi* of cholera appears to be entirely arbitrary.

Dr. Koch and his colleagues have adduced no evidence to show that they are more pernicious than any other microbe; indeed, as a matter of fact, the sole argument of any weight which has been brought forward in favour of the comma-shaped bacillus being the cause of cholera is the circumstance that it is more or less prevalent in every case of the disease, and that the German Commission had not succeeded in finding it in any other. With regard to the suggestion that the cholera process may in some way favour the growth of these bacilli, and that these are not necessarily the cause of the desease, Dr. Koch remarks, in the report from Calcutta above cited, that such a view is untenable, inasmuch as it would have to be assumed "that the alimentary canal of a person stricken with cholera must have already contained these particular bacteria; and seeing that they have invariably been found in a comparatively large number of cases of the disease both in Egypt and India—two wholly separate countries,—it would be necessary to assume, further, that every individual must harbour them in his system. This, however, cannot be the case, because, as already stated, the comma-like bacilli are never found except in cases of cholera."

Had Dr. Koch and his colleagues submitted the secretions of the mouth and fauces—the very commencement of the alimentary canal—to a careful microscopic examination of the same kind as that to which they have submitted the alvine discharges, I feel persuaded that such a sentence as the foregoing would not have been written, seeing that comma-like bacilli identical in size, form, and in their reaction with aniline dyes, with those found in choleraic dejecta, are ordinarily present in the mouth of perfectly healthy persons.

[Since this memorandum was submitted I have observed, that Dr. Koch states, in his recent address on the subject, that after his return to Berlin he had examined, amongst other things, secretions of the mouth for comma-shaped bacilli, but had found none; and, further, that he had consulted persons of much experience in bacterial researches as to whether they had ever seen such organisms, and was told that they had not. It may be of assistance to future observer if I give the dimensions of half-a-dozen comma-shaped bacilli, as found in each of the following: (*a*) In the alvinc discharges of three cholera-affected persons; (*b*) in the small intestine of a person who had died of the disease, and in whom they existed almost to the exclusion of other organisms; (*c*) in a cultivation of them in agar-agar jelly; and (*d*) in the secretions of the mouth of three healthy persons, ranging from four to fifty years of age. The measurements were made with the valuable assistance of Mr. Arthur E. Brown, B. Sc. Lond, under a magnifying power of 1000 diameters, a Powell and Lealand's $\frac{1}{16}$th of an inch oil-immersion lens, with a wide angle condenser, being used.]

Length and Width (in Micro-Millimetres)* of Comma-Shaped Bacilli.

Number.	In Choleraic Material.				
	Alvine discharges.			Intestinal Contents (Autopsy	Cultivation in Agar-agar Jelly.
	I.	II.	III.		
	μ μ	μ μ	μ μ	μ μ	μ μ
1	2·4×0·40	2·0×0·60	1·1×0·25	2·0×0·40	1·6×0·40
2	2·6×0·40	2·5×0·65	1·8×0·35	1·2×0·40	1·4×0·60
3	2·0×0·50	3·2×0·70	2·0×0·60	1·5×0·45	1·8×0·50
4	2·2×0·45	3·0×0·70	3·0×0·70	1·3×0·60	2·0×0·50
5	2·8×0·35†	2·5×0·60	2·2×0·50	2·1×0·50†	2·6×0·45†
6	1·5×0·35	2·0×0·50	1·6×0·40	1·2×0·50	1·1×0·35

Number.	In Secretions of the Mouth in Health.		
	I.	II.	III.
	μ μ	μ μ	μ μ
1	2·0×0·50	1·4×0·35	1·5×0·50
2	1·3×0·35	2·0×0·40	1·3×0·50
3	1·6×0·40	1·7×0·40	1·0×0·30
4	1·2×0·35†	1·3×0·45	1.2×0·40
5	2·2×0·65	2·1×0·50	2·7×0·50
6	2·0×0·40	2·8×0·40†	1·4×0·55

* One-Micro-Millimetre (μ) = ·001 millimetre[= $\frac{1}{25000}$"],
† S-shaped Comma-bacilli.

There is no difficulty in putting this statement to the test; and to any one acquainted with the methods ordinarily adopted for staining and mounting fungal organisms of this character, no special directions need be given. The procedure followed by me to demonstrate these "commas" in the saliva is precisely that adopted for finding them in the dejections. A little saliva should be placed on a cover-glass (preferably in the morning before the teeth are brushed), and allowed to dry thoroughly, either spontaneously or aided by a gentle heat. The dry film thus obtained should be floated for a minute or two with one or other of the ordinary solutions of aniline dyes adopted for such purposes, such, for example, as fuchsine, gentian-violet, or methylene blue. The cover should then be gently rinsed with distilled water, and the film re-dried thoroughly. The preparation may now be mounted in dammer varnish or Canada balsam dissolved in benzol, and should be examined under a $\frac{1}{12}$th or $\frac{1}{16}$th of an inch oil-immersion lens.

As in choleraic discharges so in the saliva, the number of comma-shaped bacilli will be found to vary greatly in different persons, and at different times in the same person. Sometimes only one or two "commas" will be seen in the field, at others a dozen may be counted, and, occasionally, little colony-groups of them may be found scattered here and there throughout the slide.

It may be remarked in passing, and as bearing upon what has been already said regarding the general absence of comma-shaped bacteria from the intestinal mucosa itself, that they do not appear to manifest any special tendency for attacking the decayiug epithelial scales of the mouth, but that, on the contrary, they are for the most part found free in the fluid, the epithelium being studded with other bacterial forms.

Persons who have not been in the habit of examining dried saliva-films will probably be surprised at the number and variety of the organisms which are, more or less, constantly to be found in the mouth, and specially at the number of spirilla with which the fluid is generally crowded.

The alvine discharges in cholera sometimes swarms with precisely similar spiral organism, and, indeed, as has long been known, the fluid exuded into the intestines in this disease is peculiarly suitable for the growth of these and allied microbes. But, so far as my own experience—dating from 1869—of the microscopic examination of such a fluid goes, all the microphytes ordinarily found in it are likewise to be found, to a greater or less extent, in the secretions of the mouth and fauces of unaffected persons. And with reference to the comma-like bacilli found in cholera, to which such virulent properties have been ascribed, I shall continue to regard them as identical in their nature with those ordinarily present in the saliva until it has been clearly demonstrated that they are physiologically different.

A limit has now been put to the tenure of the professorships at the Army Medical School at Netley. Hitherto the professors were allowed to continue in office as long as they pleased. Now it has been decided that the term of office will be seven years; but in the case of civil professors this may be extended for a second term of seven years.

FEBRIS EXANTHEMATOSA ORIENTALIS, OR BERIBERI FEVER.*

BY NORMAN CHEVERS, C.I.E., M.D.,

President of the Epidemiological Society of London.

I am led by a careful enquiry into the ætiology of beriberi, and especially by a review of the history of the epidemic which visited Bengal and Mauritius in 1877 and 1878, to regard this malady as a specific fever, *sui generis*; and, consequently, to be of opinion that physicians will remove it from its present anomalou sposition in our nomenclature of disease, as the last and least defined in the roll of general diseases, and place it next to scarlatina, with which fever it has certain marked features in common.

We are told† that the word beriberi is derived from the Hindustani name of a sheep (*bhere*), from a fanciful notion that persons affected with the disease walk like a peculiar species of that animal, *i. e.*, with a kind of jerking motion; but the name by which it is known to the Gentoos of the coast, *Ooboo-waioo*, simply means rheumatism combined with dropsical swelling.‡ The writer adds, "This disease, though endemic in many situations, frequently also appears as an epidemic after the setting in of the rains, or from July to the close of the year, when fevers, dysenteric complaints and cholera also break out, to a greater or less extent annually.

Morehead justly objects to the unscientific vernacular designation of this malady; but the name beriberi has been so long accepted that it would now be useless to attempt to abandon it entirely, although our present knowledge of the ætiology of the disease renders it needful that we should modify it—as by calling it "beriberi fever."

The form of acute general anasarca, generally recognised under the name of beriberi, occurs as an endemic and as an epidemic. Its principal endemic habitats are Ceylon and portions of the Madras Presidency. According to Hamilton, the limits of the Indian *endemic* were the Malabar coast and that tract of country which extends from Madras to Ganjam, being confined to these parts, and extending no farther inland than forty miles. Later observation has considerably extended this endemic area, the limits of which it may have transgressed in recent times. Its ascertained *epidemic* range is exceedingly wide; its field occupying a very large expanse of the globe's surface—the South Pacific Ocean and its islands; the southern part of Australia and Mauritius in the southern hemisphere, and, in the northern hemisphere, as high as the Kasia hills, above the parallel of 25°. If we are to regard the epidemic which invaded Lower Bengal, in 1877, as true beriberi, as I think we must, the influence of this malady, when it becomes epidemic, may be expected to extend, in a plain country, as far as the sea breeze does, *i. e.*, about 250 miles inland—Calcutta standing 110 miles, and Shillong some 140 miles from the sea. Both places suffered in the recent epidemic.

Beriberi is most common among natives, and it attacks half-castes. European soldiers have often suffered; their officers occasionally, but far less frequently. It spares neither age nor sex.

Hitherto the scientific definition of beriberi has always appeared to be one of the most obscure problems in ætiology. Probably feeling this, the College of Physicians have, in their nomenclature, placed beriberi next to general dropsy, but have refrained from giving it any definition. Although its manifestations are numerous and complex, I think that, on carefully studying its phenomena, we cannot fail to arrive at the conclusion that true acute beriberi is not, as some have thought, a group of diseases, but a malady quite distinct from all others—a fever of varying type, but characterised by a very marked individuality. According to their opportunities of observing it, and to the type of disease with which each authority has been most familiar, authors have assigned various causes to this malady. Thus (1) Ranking held that it is renal anasarca; (2) Malcolmson viewed it mainly in its paraplegic and scorbutic aspects; (3) Morehead considered it to be an expression of scorbutic cachexia; while (4) others have regarded it as an outcome of paludal cachexia. Each of the leading authorities on the subject adduced very cogent facts in support of his opinion. I think, however, that an analysis of the characters of the epidemic disease which first appeared in Calcutta in 1877, and Mauritius in 1878, enables us to declare (5) that *acute beriberi is an exanthematous fever, hitherto best known by its sequelæ—acute general anasarca, and frequently but not invariably morbus Brightii and paraplegia.*

I shall review these five doctrines *seriatim*, save that I shall give precedence to the consideration of the fifth.

Fifth.—Acute Beriberi an Exanthematous Fever characterised by general dropsy, effusions into the serous sacs, and other grave sequelæ.

I have mentioned above that, in 1877, a disease having all the characters of acute beriberi made its appearance in Calcutta, and became rather extensively prevalent. No European is proved to have been affected. Eurasians and Armenians suffered. A very full account of this outbreak will be found in the *Indian Medical Gazette*, *passim*, for 1880, as contributed by various good observers, who were not at all unanimous regarding the nature and origin of the malady. None but those practically well acquainted with beriberi in its homes can judge with absolute certainty whether we ought to agree with one or other of the only two Calcutta men who had seen beriberi. Dr. Coates, who had observed what was called beriberi in the Madras regiment at Cuttack, and who holds that the new Calcutta disease is not identical with beriberi, and Baboo Rammay Roy, who had recently treated some 500 cases of veritable beriberi in the Madras District of Coimbatore, and had, since his return, seen some 150 cases of the new disease in and around Calcutta, who asserted that "they were identically the same malady." My own conviction, arrived at after a very full survey of the whole question, clearly is that the Calcutta disease was true acute beriberi.

In early accounts of beriberi we frequently meet with such entries as these: "The disease commenced with fever, followed by swelling and numbness of the feet and legs;" "Had fever last night, skin dry, pulse excited." In the following quotation the italics are mine: "In detailing cases of beriberi, the cause supposed or probable should be first given, stating whether it was preceded by, or followed any epidemic visitation of disease in the district or neighbourhood, such as cholera, *fever* or dysentery; and also distinguishing particularly the *acute form of beriberi, attended with increased vascular excitement*, and coming on as a primary affection, from the chronic form which is frequently the sequela of rheumatism, *fevers*, &c."*

"Beriberi occurs both in an acute and chronic form; the acute form being usually attended by fever of an intermittent type, and of four or five days' duration, whereas the chronic form is more frequently the sequela of rheumatism or of fever, either of the intermittent or remittent form."†

In an abstract given by Malcolmson of nineteen cases of men, admitted with various diseases, who subsequently became the subjects of beriberi, five were admitted for "fever," and the eleven who were admitted for the four diseases—phlogosis, two; rheumatism, five; diarrhœa, one; anasarca, three—probably had commencing beriberi when they went into hospital. Malcolmson, and many who followed him, threw over this disease a darkness which has never yet been dispelled, by commencing their description of its symptoms at the beginning of the end. I cite Malcolmson's words (the italics are mine), "It (beriberi) usually *commences* gradually with a feeling of numbness, sense of weight and slight weakness and stiffness below the middle of the thighs." We might as well attempt to describe splenic cachexia by saying, "It usually *commences* with a pain and sense of weight in the left side." They fell into the great error of not enquiring strictly into the preceding fever. Doubtless their patients, not being properly questioned, did not enlighten them on this material point. Natives pay little attention to a single attack of fever, but usually seek relief when œdema and still graver symptoms occur. The severity of fever varies greatly in the exanthemata, and so does the amount and character of the exanthem, especially in the dark races. Judging by the light which the recent outbreaks in Bengal and Mauritius afford us, I am confident that Malcolmson, and others, erred in overlooking the fever and its exanthem, and by regarding the complications as the disease. In the epidemic which made its appearance in Calcutta in

* Read before the Medical Society of London, March 31st, 1884.

† "Madras Topographical Reports," Russel Condah, p. 89.

‡ Those who may be desirous to trace out fully the etymology of beriberi will find ample data in Fayrer's article, "Quain's Dict.," p. 104, and in vol. ii., O. S. of the *India Journal of Medical Science*, p. 343.

* Editorial remarks, *Madras Medical Journal*, p. 472, vol. i. for 1839.

† "Madras Medical Topographical Report," Northern Division, p. 90.

1877, the initial fever and its exanthem were frequently overlooked, but it was generally noticed that the attack was ushered in by febrile symptoms of a few days' duration. It must be borne in mind that a malarious taint is to be suspected in the constitution of every Calcutta native; but it is clear that this fever was not the common paludal fever of Bengal. Many observers stated that there was no periodicity. Elsewhere, we read of evening exacerbations and morning remissions. In February, 1880, Dr. McLeod said that "fever sometimes preceded and sometimes succeeded the œdema, and sometimes did not occur at all. It seemed to be of quotidian type when present, and well-marked rigors ushered in the attacks."

Being a general practitioner, Dr. Chambers had an advantage over nearly all of those who observed the disease, in seeing cases from the commencement, while they, being consultants, were doubtless, in most instances, called in late. Indeed, Dr. Harvey remarked that, although he had seen many cases, he had not been able to have any one case under observation throughout. Dr. Chambers is the only reporter who says much of the cerebral conditions. He noticed marked head symptoms. He speaks of "intense frontal headache," and insomnia, and says that "when the disease is ushered in with great severity, there is a tendency to stupor and wandering delirium, generally confined to one particular subject. The patient is, however, rarely insensible, but is always roused up from the stupor and delirium by being loudly spoken to; sometimes furious delirium and violence occur."

Frequent mention is made in the reports to an exanthem, thus, in a girl expressly stated to have escaped fever, Tumeez Khan says, "The limb swellings were stated to have been preceded by intolerable itching of the whole body, and the subsequent appearance of a rash all over it, and which, from the very vague description given, I think to have been of an urticarious kind." In 1879, Dr. Juggobundoo Bose had two cases in which a redness of the skin, like scarlatina, preceded the swelling." In February, 1880, Dr. McLeod noticed, from large experience, that "the skin had been carefully examined, and in only one case was any abnormality found. This was a claret-coloured discolouration of the legs, disappearing on pressure," But he also stated, doubtless from the patients' own imperfect accounts of what had occurred to them, that the œdema "was preceded by burning and painful sensations, which seemed to be confined to the skin and soft parts." In a case of Dr. Nicholson's "there were purpuric spots." In March, 1880, Dr. Chambers, in describing the leading symptoms, mentions a "sensation of burning heat in the body, as though the skin were rubbed over with ground chillies, preceded or followed by repeated sensations of chill; a scarlet efflorescence from the beginning of the disease, especially confined to the face, neck, upper part of the thorax, and the extremities, or petechial spots with circumscribed measly eruptions on face or trunk, or large purpural or mulberry patches. In infants a marbly, mottled, purplish or red tinge of the skin, especially of the extremities, neck and cheeks; a flushed puffy countenance, with suffused glistening eyes." These observations become very noteworthy when we are told by Dr. Lovell that, in 1878-79, the Mauritius epidemic of "acute anæmic dropsy," as it was termed, was ushered in by "slight fever, and in most cases by a rubeolar skin eruption, disappearing under pressure, sometimes ending in petechiæ or phlyctænæ and desquamation." In Calcutta, the fever was succeeded by general anasarca. The œdema was by some described as being "hard and brawny." It usually commenced in the lower limbs, sometimes in the hands, spreading more or less to the trunk and head. "Œdema," said Dr. McLeod, "was doubtless the most constant symptoms." "The œdema," said Dr. Harvey, "is general, not only throughout the connective tissue of the muscles, but the connective tissue of solid and visceral organs, in every cavity of the body, is bathed in fluid.'

Dr. Harvey noticed that "in two cases there was an appearance of paralysis of the lower limbs, as if from effusion of serum into the spinal canal, and in a good many more there has been great difficulty in walking, apparently not due to paralysis, but to the local condition of the limbs."

Tumeez Khan's expression, that, in one of his cases, there was inability of locomotion owing to excessive tension and stiffness of the lower extremities," appears to represent correctly the symptom in question in many cases. Dr. Chambers notices the occurrence of epistaxis in several instances. Capillary bronchitis was frequently present; pneumonic consolidation in some cases. Death commonly resulted from large, but apparently not inflammatory, effusions of clear serum into the pleuræ and pericardium. Consequently, there were palpitation and great præcordial distress and orthopnœa. Anæmic murmurs were frequently present. Dr. McLeod, speaking from large observation, stated that "death where it occurred would seem always to be sudden and associated with dyspnœa." Many early writers described the suddenness of death in beriberi. Malcolmson and McLeod use nearly the same words. The former wrote, "there is oppression and weight at præcordia, dyspnœa on slight exertion, diffused and irregular pulsation in the cardiac region." . . . . "The patient is often found dead in bed, or sinks after several fainting fits or throbbings of the heart." The urine was often noticed to be high coloured, scanty (not always) almost suppressed, but there was no albuminura or other evidence of Bright's disease. In a solitary case, which I think goes for nothing, there was "a small quantity of albumen." In some cases the urine was of very low specific gravity. Here and there a case is reported to have commenced or terminated with diarrhœa, or there was diarrhœa at the height of the disease, or dysentery later on; but, in most cases, there was constipation; in several agonising pain in the lower bowel in defæcation. In many cases, in persons who were not sailors, the state of the gums gave evidence of scorbutus. In some the gums were healthy. In examining a considerable number of Bengalis, we may always expect to find that some are scorbutic, but it does not appear to be shown that scurvy was generally present among these patients. Still, Dr. McLeod observed that "in a few cases the gums were rather swollen and bled when the teeth were cleaned." "Neither an anæmic nor scorbutic condition appeared to be an essential precursor of the malady, though a certain degree of both dyscrasiæ seemed to be developed in its progress."

Most of those who observed the disease at its outbreak, in 1877, met with many cases which did well. In 1880, there were numerous deaths. In February, Dr. Cayley reported four deaths, and two persons in a dangerous condition, in one family. In that month, Dr. McLeod, Health Officer of Calcutta, said that the town returns showed 266 cases and 51 deaths, and the suburban 364 cases and 163 deaths. It was observed by Dr. Harvey and, others, that the disease was not confined to the poor and ill-fed; rich and well-to-do people, well-fed and living in well-raised and very dry houses, were quite as freely attacked as the poor, living in damp huts on the ground level. "The disease," says Dr. McLeod, "lasted for two or three months in well-developed cases."

Much was said about the contagiousness of this disease, but no evidence of contagion is given, beyond the frequent occurrence of several cases in one family—which, standing quite alone, is, of course, no valid proof of the agency of contagion—and one very suspicious case mentioned by Dr. Harvey. Dr. Chambers, who was one of my most esteemed pupils and house physicians, considered that this "new disease" was relapsing fever. He does not appear to have found support from any other observer. He shows that a tendency to relapse marked several of his cases; but I cannot think that his patients suffered from true relapsing fever. I think that he saw an exanthematous fever; but he appears to place his fever cases apart from those which, he considered, resembled beriberi.

This epidemic was observed at Dacca and Sylhet, and at Shillong in the Kasia hills.

There does not seem to have been any appearance of the disease in Calcutta after the hot weather of 1880.

Between November, 1878, and June of the following year, a similar epidemic prevailed in Mauritius. It was stated to have caused 729 deaths Dr Lovell, the chief medical officer, sent a report on this disease, which he termed "acute anæmic dropsy," to the Calcutta Medical Society, in July, 1880. I have already spoken of the exanthem which generally characterised this malady. In the worse cases, there were hydropericardium, hydrothorax, and ascites. Signs of scorbutus are not mentioned. "Europeans were entirely exempt." "The patients, generally Indians, were very anæmic, the blood watery, with marked diminution of the red blood cells, and increase of leucocytes and granules." "The urine was seldom albuminous." Enlargement of the spleen was not found to be a feature of the disease. Opinions of medical men were divided regarding the contagiousness of this malady; the majorit were opposed to the view of contagion.

A report which appeared in the first volume of the *Madras Quarterly Medical Journal*, page 70, shows, that late in the year 1829 beriberi, which had previously been unknown in

that locality, attacked the Madras sepoys at Singapore, while "*the convicts remained without a single patient with the complaint.*" True beriberi prevailed in the criminal prison at Singapore in 1875-76-78-79, and 80. *It did not spread among the community at large.* No European or native female prisoner suffered. In 1878 the death-rate was 16·20 per cent. of admissions, and rose to 20·63 in 1879. There is no mention of fever or eruption. Rheumatic pains and paraplegia were frequent symptoms. The great serous cavities, peritonæum, pleuræ, pericardium, were filled with serum. Scorbutic symptoms appeared in some cases. *The urine was scanty, high-coloured and albuminous.* In a leading article in the *Indian Medical Gazette*, for September, 1880, Dr. McLeod observed that "as regards symptoms and mortality, the Calcutta disease presented a sort of transition between the pure beriberi of Singapore, with rheumatic and paralytic phenomena and albuminous urine, and the acute dropsy of Mauritius, which presented many of the features of a contagious exanthem."

I must repeat that my own inference from the evidence is, that the "new disease," as it occurred in Bengal and Mauritius, was an exanthematous fever. Its mode of invasion, when epidemic, would alone declare beriberi to be a fever. No one who has studied the natural history of fever will believe that the epidemic disease which, as we have seen, invaded Lower Bengal in 1877, and Mauritius in the following year, was either paraplegia or scorbutus, or consider that it was renal anasarca.

First.—Beriberi regarded as Renal Anasarca.

In a valuable but very rare volume,* "The Proceedings of the Hyderabad Medical and Physical Society," Mr. J. L. Ranking published, in 1853, a series of reports, tending to show that the previously obscure disease, beriberi, depended mainly upon the existence of a form of *morbus Brightii*. Mr Ranking said that his limited experience led him to adopt the opinion that beriberi is primarily and essentially a renal disease; that deterioration of the blood is caused by lesion of the kidney, and that the "numbness," staggering gait, or more confirmed paralysis of motion (pointing to affection of the spinal cord), the "irritable" condition of the heart, the endo- or pericardial "inflammation" [?] and effusion (which latter is generally the immediate cause of death) are secondary affections. He showed that in sixteen cases of beriberi, the urine was more or less albuminous in eleven: in eight, the microscopic elements characteristic of kidney disease existed. Microscopical examination was made in these eight cases only; so that, in every case of well-marked beriberi which fell under Mr. Ranking's notice, the microscopic evidences of kidney disease were discovered when sought for. In nine, there was general anasarca; in one, the anasarca existed with ascites; in two, there was delirium; in one, coma; in fourteen, numbness without actual anæsthesia; in two, impaired sensibility with numbness; in six, there was more or less loss of motor power; in seven, symptoms of pericardial and endo-cardial affection; in five, signs of œdema of the lungs; in two, hepatitis; in two, nausea and vomiting; in two, diarrhœa. It was noticed by Malcolmson that there were certain points of resemblance between beriberi and *morbus Brightii*. This resemblance forced itself upon Mr. Ranking's attention before he had access to Mr. Malcolmson's work. Upon commencing the examination of the urine of all beriberi patients, it was found that, although the quantity excreted, the reaction, specific gravity, colour, &c., differed according to the stage and form of the affection, the microscope revealed the presence of blood-discs and epithelial scales. In two cases there were also tube casts, and mucous and organic globules. The urine was often coagulable by heat and nitric acid, though never to a high degree albuminous. He represented that, in the earlier stages of beriberi the state of the urine points only to simple congestion or sub-acute inflammation of the kidneys. He found it to have a deep red colour, owing partly to the admixture of blood, partly to concentration; a specific gravity at or somewhat above the natural standard. A deposit of lithic acid and lithate of ammonia sometimes occurred, and abundant and large crystals of lithic acid. These morbid conditions of the urine will, Mr. Ranking considered, be found to precede the anasarca, numbness, and other symptoms of spinal affection. Unless the state of renal congestion, or sub-acute inflammation, upon which it depends, can be overcome, the anasarca continues, effusion "into the spinal cord" [?] takes place, the heart becomes involved, and effusion into the pericardium and pulmonary œdema carry off the patient. The urine remains scanty, in such cases, to the end, or is even totally suppressed. In more chronic cases, or in individuals who have struggled through the acute stage, the urine was found to alter in character—it increased in quantity, but decreased in density—it seldom, however, reached the healthy standard even as to quantity—it acquired a pale opalescent colour—had an alkaline reaction, a specific gravity of 1,010, and coagulated more or less by nitric acid and heat. "Here," said the author, "we have all the characters of the urine of 'Bright's kidney.'" "The alkaline reaction is due doubtless to the existing lesion of the spinal cord which, although in itself a secondary affection, reacts upon the urinary organs, the mucous membrane of the bladder especially causing an increased secretion of mucous, or even pus, and consequent alkalinity of the urine. The microscope still reveals blood-discs; a great abundance of epithelial scales with mucus or blood globules entangling crystals of triple phosphate, or, if any deposit occurs, it is made up of mono-basic or bi-basic phosphates, amorphous phosphate of lime and mucous."

Others who observed beriberi nearly at the same time, noticed the renal complication.

In the middle of August, 1852 (dates are self-evidently of great importance in the correlation of cases of beriberi, as showing agreement or difference in type in the same or in other epidemics of the disease), Dr. George Mackay* admitted to hospital, at Rangoon, a Madras sepoy, who died, in about 26 days after sickening, with typical symptoms of beriberi. "The urine presented a copious deposit of mucus, with altered blood-discs and crystals of the triple phosphate." Many other cases of a nature similar to this came under Dr. Mackay's notice during the same (second Burmese) war, but none in which the symptoms so nearly resembled those of beriberi. His attention in those cases was early directed to the state of the urinary organs. "Epithelial scales, altered blood-discs, crystals of the triple phosphate being almost invariably met with; in some cases oxalate of lime crystals, and in one or two tubular casts were observed."

The "Hyderabad Proceedings," in which Mr. Ranking's paper appeared, also published a case by Mr. Maillardet. Dr. Van Someren also discovered "casts of uriniferous tubes, epithelial scales, altered blood-discs and octohedral crystals of the oxalate of lime."

We have here descriptions, by three observers, of the conditions of the urine in (epidemic?) beriberi as the disease presented itself within a short space of time—apparently a period of a few months—on the continents of Madras and Burmah. Although the above facts, being published in the *Indian Annals of Medical Science*, must have come before many observers in India, I do not find that Ranking, Van Someren, Mackay, or any one else recorded other cases of albuminuria, cast desquamation, &c., in beriberi. Was this because, during that particular epidemic and that one only, the kidneys suffered nearly as they do in the cognate but very distinct (from epidemic acute beriberi) exanthematous scarlatina fever? At any rate, regarding beriberi as a fever of this genus, I am quite prepared to find that, when the exanthem is checked by the "chill," so frequently mentioned as a prominent exciting cause in this disease, albuminuria may occur. It is clear, however, that albuminuria was not a marked feature in the late Bengal and Mauritius epidemic, although it is to be regretted that none of the reports which I have seen take any notice of the microscopic condition of the urine.

It will have been noticed that in the Singapore outbreak, which commenced in 1875, "the urine was scanty, high-coloured and albuminous."

Undoubtedly the incidence of beriberi is especially directed to those who are the subjects of renal disease. It has always been observed that a very considerable proportion of the victims of this disease are drunkards. Intemperate European soldiers are liable to this malady; their officers are far less so, although they have not always escaped. Christie says, in his report on beriberi: "I have remarked that a very great proportion of the patients seized with this disease were men accustomed to lead a sedentary and debauched life, such as tailors, shoemakers, &c., who, when working at their trade, are often excused the duty of the field, and by their double earnings are enabled to

* I only know of one copy, possessed by my friend Dr. Waring, C.I.E. I gave extracts from its contents in the "Bibliographical Record" attached to No. 2 of the *Indian Annals of Medical Science*, in 1854.

* *Indian Annals of Medical Science*, No. 5, p. 349.

procure a larger quantity of spirits than the other men." He adds, "I have never met with an instance of this complaint in a woman, an officer, or a boy under twenty." I was told at Aden, about ten years ago, that European soldiers posted there, who are temperate, generally escape; but that, when a man becomes a sergeant, he is not unlikely to take to free-living, and then probably is attacked with beriberi. At the same time it is clear that, while beriberi sometimes resembles Bright's disease, it by no means always assumes that character, Certainly it is not produced by the ordinary causes of renal anasarca; and, if some of the cases of beriberi, such as those observed by Ranking, Mackay and others, may be designated as examples of this form of general anasarca, ordinary renal anasarca is in no sense beriberi. The paraplegic element of beriberi is not characteristic of common *morbus Brightii*.

Second.—Beriberi viewed in its Paraplegic aspect.

One of the leading sequelæ of beriberi being anasarcous and dropsical effusion into the areolar tissue and serous cavities, the occurrence of spinal paralysis, in various degrees, as one of its complications, is clearly accounted for. As I have shown, in describing the Bengal epidemic, the stiffness of the lower limbs is frequently due to the presence of hard œdema, not to spinal paralysis; but, in many cases of beriberi, distinct impairment of motor and sensory power in the lower limbs has been observed.

In an abstract of Malcolmson's prize essay on beriberi, Sir G. Ballingall says:—"In dissecting patients who have died of this disease, serous effusions, more or less extensive, into the cellular substance as well as into the great cavities, would seem to be the more prominent appearances; and, in addition to this serous effusion, there often exist in the lower region of the spine, about the origin of the lumbar and sacral nerves, symptoms of congestion; and, in one very remarkable case, of which the details are given by Mr. Malcolmson with an illustrative engraving, an effusion of reddish coagulable lymph had taken place on the posterior surface of the theca, at the fourth dorsal vertebra, and the same in the region of the sacrum."

Although the form of chronic paraplegia, termed by Bontius and other old writers on the diseases of the East "barbiers," is, as shown by Henry Marshall, quite distinct from beriberi, it can hardly be doubted that much of that which was formerly regarded as barbiers was, in reality, beriberi with spinal paralysis.

Lathyrism, paralysis of the lower limbs caused by habitual feeding upon the *dâl* of the *Lathyrus Sativus*, prevails extensively in Upper and Central India, especially near Allahabad and in Upper Scinde. M. Proust has observed it in the Jurjura mountains of Algeria, and it is stated to be far from uncommon in France. A recent writer having stated that lathyrism and beriberi are identical diseases, M. Pierre Marie * has undertaken to prove that such is not the case. It appears to me not improbable that the writers have confused beriberi with barbiers. At p. 113 of the *Indian Annals of Medical Science*, No. 23, Dr. James Irving has contrasted the symptoms and other conditions of lathyrism and barbiers. I should have considered beriberi and lathyrism as little octogeneric as Monmouth and Macedon—beriberi being a very acute disease, lathyrism, as it occurs in India, a chronic one. But, since the question has arisen, M. Marie's paper calls for notice from medical men working in those districts, far separated as they are, in which lathyrism and beriberi are most common; lathyrism prevailing far inland, beriberi occurring almost entirely within the influence of the sea breeze. It appears singular that, in giving long and minute descriptions of the conditions of the lower extremities in the contrasted diseases, M. Marie appears to have overlooked œdema in beriberi.

The loss of power in the lower limbs, so frequently noticed in beriberi, appears to need clear definition. In what cases is it dependent upon spinal lesion, and in what others is it due to hard œdema of the extremities and debility? With regard to the former cases, one point ought to be cleared. Sir Joseph Fayrer has suggested the probability that filarious disease may be in some way concerned in inducing beriberi.† A form of spinal paralysis in the horse, *kumree*, which prevails within the Madras area of beriberi, and in Eastern Bengal, has been ascribed by Twining and others‡ to the presence of entozoa in the cord. Do spinal lesions in beriberi ever arise from this cause?

Undoubtedly, although spinal paralysis is recognised sequel of beriberi, it cannot be considered that beriberi is, in any sense, paraplegia.

Third.—Beriberi regarded as an expression of Scorbutic Cachexia.

Marked evidences of scorbutus are so often noticeable as prominent symptoms in cases of beriberi, that at least one of our best Indian observers has considered that beriberi is essentially a form of scurvy.

Wishing to give fair play to this, as well as to all the other leading theories upon the causation of beriberi, I may cite the following rather striking example of this coincidence, as observed by Tumeez Khan, in Calcutta, in 1878, and others, which I find in the *India Journal of Medical Science*, vol. v., N. S., p. 75, as occurring *circa* 1839 on a passage from Java.

1839.

A Dutch barque arrived in the Madras Roads, from Java, on the 12th May, having left Malacca, where she lay only three or four days, all being well, on the 7th April. The crew were Javanese, who had made objections about coming to the Coromandel coast, as they said "many of their countrymen had died of a similar disease on former voyages."

"About twenty days before reaching Madras, many of the crew were seized with swellings and numbness of the legs and feet, but yet were able to perform their duty; when they arrived within 50 or 60 miles of the coast, and within the influence of the land winds, in three cases the symptoms became much aggravated, they were unable to walk, dyspnœa and pain in the præcordia supervened, and they died the day after the ship arrived."

"They were a wretched looking set, and were very badly fed, their rations consisting only of rice, and of that a very limited quantity. The water was bad, and they were on very short allowance.

1878.

A Mahomedan ship-captain of one of the Arab sailing vessels which ply between Calcutta and Jeddah, a native of some part of the Coromandel coast, his son, and a third member of his family—both said that they had returned hale and hearty from a trip to Jeddah, the third person had died previous to Tumeez Khan's visit.

A short time after their arrival in Calcutta "both were nearly simultaneously attacked with febrile symptoms, which lasted for a few days, and were followed by œdema of the lower limbs, to which succeeded cough and dyspnœa" (the son had hæmoptysis). The "œdema extended up to the loins." When visited, they had "orthopnœa, palpitation and inability to lie down." It is not said whether the father and son died, but they probably died.

"Both had spongy and discoloured gums."

In connection with the above observations, all who are desirous to investigate the nature of beriberi ought to study Morehead's chapter on this disease. His data are mainly derived from histories of "beriberi as it occurred among the Lascar crew of the ship *Faize Allum*, in May, 1853, when in Lat. 10 north, within sixteen days' sail of Bombay, and from an account of an outbreak of "epidemic ascites," or "peritonæal dropsy," in thirty of the crew of H.M.S. *Juno*, on her arrival at Sydney from a lengthened cruise among the islands of the Pacific, in January, 1856.

The native subjects, four cases of beriberi, from the ship *Faize Allum*, whom Morehead treated, were doubtless scorbutic; the men of the *Juno* were probably so. Morehead does not appear to have met with any other cases of beriberi. He considered that "beriberi, more particularly in its acute form, occurs usually in persons favourably circumstanced for the development of a scorbutic taint, and subsequently exposed to cold, dry or moist winds, or to lying on the ground wet with rain or dew." With the judicious circumspection, which characterises all his reasoning, he observes that to him "it seems that beriberi is a general dropsy, and that, in regard to each instance the question ought to be—what is the pathology of this case of general dropsy?" He adds, "generally it will be found that a scorbutic diathesis and external cold or wet are the determining conditions."* Dr. Peet adopts this view without qualification. He says, "When individuals who are scorbutic are exposed to cold and alterations of temperature, they are not unfrequently attacked with general dropsy. It has been customary to designate the disease now referred to as beriberi."† Here is, I think, another example of the common error of regarding as cause and effect two circumstances which are merely associated as frequent coincidences. It is to be borne in mind that beriberi is often epidemic, which scorbutus cannot possibly become. There was no beriberi among the scorbutic mariners of last century; and in many cases of sea scurvy, which I have treated, there was not the very slightest

* *Progres Medical*, No. 43. An abstract of this paper is given at p. 606, *Medical Times and Gazette*, vol. ii., for 1883.

† "Quain's Dictionary," p. 105.

‡ *Vide Calcutta Med. and Phys. Trans.*, vol. i.

* *Op. cit.*, pp. 707-8.

† "Principles and Practice of Medicine," p. 570.

evidence of its existence. Beriberi occurs in some localities notorious for scurvy, as Aden ; but it does not prevail in certain of those districts of Hindustan—as the province of Behar, and in Scinde—in which scurvy is extremely prevalent.

Scorbutus, as we have already seen, occurs in Lower Bengal, but not so extensively as in some other Indian districts. The recent Bengal epidemic frequently attacked the scorbutic, but it was noticed that many of the sufferers from the epidemic were free from evidences of scorbutus. I think that, when we state the fact that wherever beriberi prevails, it especially selects the scurvied, we are free from the error of supposing that acute beriberi is an expression of scorbutic cachexia, although scorbutus should be looked for in all cases of beriberi, and be treated, when present, as a grave complication.

Fourth.—Is Beriberi, as some have considered, a form of Malarious Cachexia ?

I think that this question must be answered in the negative. Beriberi prevails in localities, such as Aden and Mauritius, where the paludal poison does not abound in great intensity. Until 1877, the Delta of the Ganges, that hot-bed of marsh influence, was never visited by this pest as an epidemic, and has never generated endemic beriberi. I believe that, up to the time of my leaving Bengal, eight years ago, it was never suggested that beriberi could originate there. Considering that a vast number of sailors pass through the Calcutta hospitals, I think it noteworthy that only one not unquestionable case of this disease, in a half-caste seafaring-man, made its appearance in my wards. No one acquainted with the natural history of the marsh poison would, on studying the outbreaks of beriberi in Calcutta and Mauritius in 1877 and the following year, consider that there could have been any interchange of paludal influence between these two widely separated localities. Nevertheless, seeing how generally and how often gravely prevalent malarious cachexia is in most of the regions which beriberi visits, the necessity of searching for it, as a most serious complication, must never be overlooked. Few constitutional states can be regarded as more perilous than that of the multitudes of unfortunates who, being the subjects of the combined cachexiæ of marsh-poison and scorbutus, are attacked with beriberi.

I think that we are justified in concluding that beriberi is not a malarial fever, but that the victims of malarious cachexia are especially liable to suffer from its attacks.

Treatment.—If I am right in the firm belief which I hold that true beriberi is an exanthematous fever which, although wholly *sui generis*, pursues a course which, in more than one of its leading actions, much resembles that of scarlatina, it is evident that here our first object should be, as it is in scarlatina, to endeavour to see each case *ab initio*, and to prevent the occurrence of renal and other visceral mischief, anasarca and dropsical effusions into the serous cavities. If it could be my duty to have charge of military and jail hospitals within the endemic area of beriberi, every European soldier, sepoy, and prisoner, would be warned to seek relief from my assistants *immediately upon being attacked with even the very slightest symptom of fever*. I would myself examine all fever cases early, within twelve hours if possible, carefully searching for the exanthem on the lighter skins, and considering that, if I frequently discovered it there, its presence might be suspected in those of darker complexion. Then the chief indications would be :—To admit all fever cases as in-patients ; to avoid the occurrence of that CHILL so frequently assigned as the cause of general anasarca in beriberi ; to promote healthy cutaneous action and to subdue fever ; to discover and deal with pre-existing renal and other visceral disease, and with malarial and scorbutic cachexia. After remaining for two or three days under observation, the cases of ordinary intermittent fever would explain themselves, and would then demand less close attention. When patients do not seek relief until general anasarca has become established and, being too weak to go about, are desirous to come to the hospital as a refuge, the same principles of treatment ought to guide us. With the deepest respect for the memory of a great Indian physician, I cannot agree in the validity of his maxim, guarded as it is, "remove the dropsy by purgatives or diuretics, being guided to the use of the one or the other by the state of the pulse." I would no more give direct diuretics in the anasarca of beriberi than I would in that of scarlatina. My great effort would be to obviate renal congestion in all possible ways, and to make the work of the kidneys as easy as possible. I would not give anything more drastic than the mildest laxatives in these cases, liable as they are to end fatally by diarrhœa or dysentery. If we can only remove the other complications of beriberi, the anasarca will undoubtedly disappear as constitutional power becomes re-established. No purgative or diuretic will, except by promoting exhaustion, touch a pericardial, arachnoid or pleural effusion which threatens life.

I believe that, fifty years hence, the futile and destructive practice of endeavouring to remove dropsical effusions by the use of drastics and direct diuretics will be viewed as one of the most barbarous therapeutic errors of the present century.

I think that the whole subject of acute and chronic beriberi, especially as they occur in the Madras Presidency and in Ceylon, ought to be thoroughly re-investigated by sound pathologists. Work out the subject of beriberi, is one of the best pieces of advice that can be given to a young physician entering upon a career in the Madras Presidency.

The field of observation is large. In 1881, there were 102 cases of beriberi, with twenty deaths, among the native troops in the Madras Presidency, in a strength of 30,394. No cases occurred in the Bengal and Bombay Presidencies.

Especial care should be taken to ascertain the precise nature of what is termed the "chronic" form of beriberi. Are these cases of malarious and scorbutic cachexia, not allied to true beriberi, or are they examples of repeated attacks of beriberi and consequent ill-health? Christie observed that, "men who have once had the complaint are the most subject to it in future." In the epidemic and acute form, light is needed with regard to the precise type of the fever, the character of the exanthem, the range of temperature, the chemical and microscopical conditions of the urine, &c. All observers have noticed that, although the œdema of the lower limbs is hard, the serous effusions, arachnoid, pericardial, pleural, and peritonæal, the pressure of which tends to cause death in beriberi, are distinctly *non-inflammatory*. A little sifting would clear this question, enabling us to judge whether we ought or ought not to draw off the fluid from the pleuræ and pericardium, and with what prospect of success.—*Medical Times and Gazette.*

ASCITES FROM LIVER DISEASE TREATED BY PILOCARPINE.

Ascites caused by cirrhosis of the liver has not been very amenable to treatment up to this time. Most physicians have therefore come to the conclusion that paracentesis was the only resort. Drastics and diuretics had to be abandoned as inefficient ; and, in fact, there seemed to be little hope left for this class of sufferers. Harley and Roberts allege that repeated paracentesis promises good results ; while, on the other hand, there are those who have never accomplished anything by puncture. Mackenzie reports two cases as cured by means of flannel bandages, tightly wrapped around the abdomen. Siegnit combines with the bandaging faradisation of the abdominal muscles. From all the different reported cures, one can readily understand that the treatment of ascites caused by disease of the liver has been quite unsatisfactory, and most of the cases have been treated on the expectant plan.

During the year 1880, the author (a writer in the *Mittheilungen des Vereins der Aerzte*, signing himself T.) treated a case of this kind by all the remedies that have been recommended in the books without any results. Finally paracentesis was resorted to, but with each operation matters grew worse, and the quantity of liquid steadily increased. It occurred to him to administer pilocarpine immediately after an operation to prevent or give other course to the serous exudation that each time was emptied into the peritoneal cavity. The patient received twice daily 15 milligrammes (¼ grain) of pilocarpine for six consecutive days, with the effect of causing profuse diaphoresis and discharge from the salivary glands. Stimulants (whiskey, 60 grammes [℥j]) were given the patient to counteract the weakening action of this medication. From the administration of the first dose the ascites diminished, and the patient was finally discharged from the hospital as cured. Three months later the patient was examined, but did not discover a symptom indicating disease of the liver or ascites.

Another case the author treated in conjunction with Dr. Haas. The diagnosis was an enormous ascites caused by cirrhosis of the liver. The swelling was so great that it caused dyspnœa, for the relief of which paracentesis was made a few times and 20 to

30 litres (5 to 8 gallons) of fluid were removed. The effect was not lasting. Finally, the respirations ran 38 and the pulse 118 per minute; the urinary secretion was entirely suppressed. After a consultation it was determined to employ large doses of pilocarpine, since the case seemed hopeless. The patient received twice daily 0·01 gramme (gr. ⅙) of pilocarpine in a large dose of whiskey, besides some solid food. This drug did not seem to act so favourably in this case, diaphoresis being very scanty. Two weeks later, after another puncture, the dose of pilocarpine was increased to 2 centigrammes (gr. ⅓) and followed by a large quantity of whiskey, with the result of causing profuse sweating. Powdered jalap with cream of tartar was given in conjunction for the constipation. The patient gradually recovered, when he was discharged, and he was as healthy as before.

If the good results obtained in these two cases do not indicate much, they may cause other observers to try the pilocarpine treatment. Paracentesis often does not only do no good, but seems to do harm. Both these patients were very much weakened, and it seems almost incredible that they could tolerate 2 centigrammes (⅓ grain) of pilocarpine twice a day; but possibly the large doses of stimulants given shortly after did much to obviate the depressing effects of the pilocarpine. —*London Med. Record*, July.

Fothergill on the Treatment of Bronchitis.—After laying down the usual principles of treatment—relaxant expectorants, especially ipecacuan, in the dry stage, and stimulant ones in the succeeding stages—Dr. Fothergill enters into those additional principles which have arisen out of recent investigations into the physiological actions of drugs. People die of bronchitis either because the respiratory centre fails, and they cannot breathe any longer, or because the cardiac centre fails, and their heart cannot beat any longer. Therefore, as Dr. Alison used to say, endeavour to obviate the tendency to death. Digitalis is a cardiac stimulant. Atropia and strychnia stimulate both the respiratory and cardiac centres; one to two minims of liq. atropia may be given thrice a day in combination with spirit of ammonia and aniseed oil. Dilated pupil and dry throat need be no bar to the continuance of the remedy. Strychnia may be given thus: Ammon. carb., gr. v.; tinct. nux. vom ℥x.; tinct. scillæ ʒss; infusion serpentariæ. ʒj. Ter in die. The only objection to this is that strychnia sometimes stimulates the bladder unpleasantly in elderly people. In severe cases the strychnia may be pushed till it produces twitching of the limbs. Where there is a dilated right ventricle and intermittent pulse, digitalis is especially indicated. Dr. Fothergill's experience is against opium. Ammonium bromide, in half-drachm doses, with camphor mixture and tincture of hyoscyamus is the best sedative to employ. In the cirrhotic form of bronchitis, simulating phthisis, atropia should be given for night-sweats, sulphate of copper for diarrhœa, and careful *feeding*, but not *overfeeding*, which may be fatal. When dropsy comes on free catharsis by compound jalap, or elaterium with compound scammony should be employed. If there is gouty or contracted kidney, tapping the swollen legs often gives great relief. If the dropsy is from venous congestion only, tapping is worse than useless. In the early stage of intercurrent valvular disease, do not give digitalis; it is spurring a newly-lamed horse; give rest till the acute stage is over.

The Treatment of Acne.—Dr. Piffard, the well-known American dermatologist, considers that, in the treatment of acne, internal measures are more important than external applications. For the common form (*acne vulgaris*), he says that the sulphide of calcium, given in small doses, is the most trustworthy agent that we possess. Next in usefulness he ranks the bromide of arsenic, which should be given in doses of from the $\frac{1}{100}$ to the $\frac{1}{50}$ of a grain. It is best administered in alcoholic solution, well diluted. For a local application in acute cases, he recommends an ointment of belladonna; and in subacute and chronic, green soap or some mercurial ointment.

Connection of Acute Diabetes with Pancreatic Disease.—Dr. Duffey, in a paper read before the Academy of Medicine in Ireland (*Brit. Med. Jour.*, March 1884, p. 608), reports a case of diabetes occurring in a farm labourer, aged 24, the duration of which was two months. There was a sudden onset of abdominal pain, vomiting, and diarrhœa, a week before death, which was preceded by coma. At the *post-mortem* examination nothing remarkable was seen, with the exception of the condition of the pancreas. This gland was hypertrophied, and felt extremely hard and indurated; on microscopic examination, this was proved to be due to carcinomatous infiltration. A very interesting paper on 'Pancreatic Diabetes' is recorded in the *London Medical Record*, 1881, p. 150. Vide *Medical Digest*, sec. 336 : 1.

Eczema Capitis.—In the ordinary eczema of the head in children, so commonly met with in dispensary practice, after two or three thorough cleansings, the daily application of the following salve nearly always suffices to obtain a rapid and lasting cure:—

℞ Acid. Salicylic ... ... gr. x.
Tinct. Benz. ... ... ℥ xx.
Vaselini ... ... ʒ j.
M. ft. ung.

On other parts where a soft, easily melting salve such as this is not suitable, or where a firm dressing or a drying effect is desired, the following paste should be rubbed on:—

℞ Acid. Salicylic ... ... gr. xix.
Vaselini ... ... ʒ j.
Zinci Oxidi, Amyli ... ... aa. ʒ ss.
M. leniter terens, fiat pasta.—*Edinburgh Medical Journal*, September 1883.

Elephantiasis treated by Electricity.—M. M. Silva-Aranjo and Moncorve communicated their first note on this subject to the Academy of Sciences of Paris (1880) and to the Academy of Medicine (1881). More than a century ago Henley treated successfully a case by electricity (Alard, *De l' Inflammation des Vaisseaux Absorbans (sic) Lymphatiques dermoides et souscutanés*, Paris, 1824, p. 589). When Silva-Aranjo employed electricity for the first time in 1879 for a case of elephantiasis of the scrotum, he neither knew of this case nor of those of Beard and Rockwell (Tibbits's *Handbook of Med. and Surg. Elect.*, London, 1877). For the galvanic currents the authors employ a battery of 40 to 60 elements of Trouvé (sulphate of copper), placing the negative pole on the different points of the affected part, the positive pole on the sound skin, more or less near. The duration of each application varies from five to thirty minutes. For faradisation they use an apparatus of Trouvé, and employ the two methods together or separately in the same patient and in the same séance. In very severe cases they have had recourse to electrolysis with all Listerian precautions. Their conclusion is that electricity, if it do not constitute a means of infallible cure, applicable to all cases of elephantiasis, represents the best therapeutic means known at present. M. Besnier, from whose paper in the *Annales de Derm. et de Syph.*, March 1884, we take the above, adds that judgment must be suspended concerning the *permanence* of the cures by this treatment. Trials of this treatment have been successfully made in the service of Professor Oliveira Feijaô, of Lisbon, and are in course of execution at Vienna, in the division of Professor Kaposi, by Riehl.—*Medical Record*, July.

MEDICAL SOCIETIES.

THE CALCUTTA MEDICAL SOCIETY.

The Seventh Meeting of 1884 was held at the Medical College on Wednesday, the 10th September, Dr. Harvey, President, in the Chair.

Dr. Sanders exhibited a child suffering from

A PECULIAR NERVOUS AFFECTION.

The child, a Hindu female, aged 2½ years, is somewhat idiotic, and has been suffering for the past four months from slight hydrocephalus; but it is only within the past few weeks that the child has exhibited the peculiar movements to which Dr. Sanders directed attention. During the time the child is awake she constantly thumps her temples with both fists simultaneously, and with such great force as to contuse her forehead as well as her knuckles.

The skin over the knuckles and that part of the child's forehead which is being so constantly struck has become very much thickened. The spasm begins with a grimace, then the child's arms are abruptly pronated, the hands closed firmly