On the evening of the twenty-second day 20 cubic centimetres of antistreptococcic serum were given per rectum, repeated next day, and again on the twenty-seventh, thirtieth, and thirty-fourth to thirty-ninth days. During this time the pulse-rate varied between 100 and 130 per minute with profuse sweating and occasional vomiting. On the thirty-fifth evening following delivery I found the abdomen distended, tympanitic, and tender, more especially in the left hypochondrium, where a hard lump could be felt, moving slightly on respiration and exhibiting the characteristic notch on its anterior border. Being uncertain as to the presence of pus in what was obviously the enlarged spleen and also whether that organ was adherent to the anterior parietes or not I advised the continuance of the antistreptococcic (polyvalent) serum. This was given daily until the forty-first day, when I saw her again with Mr. Vinter and his partner, Mr. R. J. Percy Thomas. The spleen had increased in size; it no longer moved on respiration, but still retained its notched border and the parietes superjacent were slightly reddened and œdematous.

Under chloroform a three-inch vertical incision was made along the centre of the left rectus sheath; the areolar tissue was found to be œdematous and on pushing a pair of sinus forceps through the peritoneum a quantity of horribly foul-smelling pus of a dirty yellow colour, mixed with greyish-purple sloughs of splenic tissue, escaped. A large rubber drain-tube was passed into the cavity of the spleen and stitched to the skin incision. The temperature rapidly fell and in 48 hours was normal and remained so during convalescence, while the pulse-rate soon reached 84. The number and bulk of sloughs that came away were extraordinary, but very little bleeding occurred. The cavity closed at the end of three weeks.

The interesting features of this case seem to be: (1) the spleen was the only organ or part affected; (2) though swollen it never lost its characteristic outline, but ceased to move on respiration; and (3) the large amount of splenic tissue lost in the form of sloughs and yet there are now (May, 1907) no apparent blood changes. I regret that no microscopical or bacteriological examination of the discharge was made, but the stench was so overpowering that a specimen would have nauseated even the most hardened pathologist.

With regard to the pathology of this case it is probable that septic thrombosis of the veins at the placental site took place and from there an embolus was carried through the heart and pulmonary circulation into a branch of the splenic artery. As throwing a light on the reason why the spleen became the "seat of election" for infective embolism and infarction, a history was given that three weeks previously to the birth of her boy the patient fell from a table, striking the left side of her chest so violently across the back of a Windsor chair that the latter was broken by the force of her impact. It is therefore probable that a hæmatoma was caused in or around the spleen which subsequently became infected in the manner above outlined.[1] The patient has never resided abroad nor suffered from enteric or other fever, nor had she valvular disease of the heart. Abscess of the spleen occurs in the tropics during malaria or yellow fever, also in the course of enteric fever, septicæmia, pyæmia, and particularly in ulcerative endocarditis.

The following references in recent medical literature are appended. 1. Nolan,[2] in describing a case of abscess of the spleen, states that only five successful results are recorded after operation. His case is very similar to the one I have just described. A woman, aged 25 years, six weeks after confinement became ill and feverish. Enteric fever was suspected. Sudden acute pain in the left side was followed by dyspnœa and a pleuritic effusion was found, together with a greatly enlarged but *not* tender spleen. A vertical incision along the left linea semilunaris was made; a cavity was reached from which about a litre of dull brown pus, with a faintly sweet smell, escaped. The peritoneal cavity was not opened. The wound was plugged with gauze and uninterrupted recovery followed. It is doubtful whether this patient suffered from enteric fever or whether the abscess followed puerperal infection; in either case an infective embolus would be the proximate cause. This paper is referred to in the *British Medical Journal Epitome*, April 14th, 1894, para. 295, and also quoted by Mayo Robson in the *Medical Annual* for 1895, p. 453. 2. Black.[3] The patient was a sailor, aged 21 years, who two years previously had contracted yellow fever; the tumour was incised yielding three and a half pints of bluish yellow pus; the cavity was plugged and healed by granulation after secondary suture with complete recovery. 3. Mayo Robson in the *Medical Annual* for 1904, p. 651, refers to a case reported by Dr. G. Riolo in the *Riforma Medica* of April 24th, 1902, of a splenic abscess complicating malaria successfully treated by incision and drainage. 3, 4, and 5. Mr. Robson also refers to a case under his own care and cites a paper by Dr. A. L. Staveley in the *Annals of Surgery* for June, 1903, on Splenic Infection, reporting two successful operations. 6. Harrington[4] reports two cases both following typhoid fever. 7. Moynihan[5] refers to seven cases of splenic abscess treated by operation up to 1900 and three cases recorded since by Murphy, Karewski, and Eberhart.

With regard to the treatment it appears to me better to wait until adhesions to the anterior parietes have formed and then perform splenotomy with drainage rather than traverse the unguarded general peritoneal cavity to undertake splenectomy, an operation sufficiently severe in an uninfected patient with its amount of shock and risks of hæmorrhage, immediate and remote.

Plymouth.

1 Cf. Moynihan's Abdominal Operations, p. 646.
2 Weekblad van het Nederlandsch Tijdschrift voor Geneeskunde, March 10th, 1894.
3 Brit. Med. Jour. Oct. 17th, 1896, p. 1116.
4 THE LANCET, Nov. 11th, 1905, p. 1393.
5 Abdominal Operations, p. 646.

RICE AND BERI-BERI:

PRELIMINARY REPORT ON AN EXPERIMENT CONDUCTED AT THE KUALA LUMPUR LUNATIC ASYLUM.

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DURING the year 1905 an epidemic of beri-beri broke out in the Kuala Lumpur Lunatic Asylum. Commencing in February, it reached its height in July and August, declining somewhat towards the end of December. Out of 219 lunatics treated in the asylum during the year 94 persons were affected, of whom 27 succumbed to the disease. The chief constituent of the rations supplied to the inmates of the asylum was uncured (Siamese) rice, and in view of the fact pointed out by Dr. Braddon that beri-beri occurs chiefly amongst communities with whom such rice is the staple article of diet it was decided, with the sanction of the Government, to place half the lunatics on cured (Indian) rice. The Government readily gave its consent and the experiment was commenced on Dec. 5th, 1905. The result up to Dec. 31st, 1906 (i.e., one year and 26 days) was that 34 out of 120 persons fed on uncured rice suffered from beri-beri and 18 died, whilst among 123 patients dieted on cured rice there were no deaths from beri-beri and only two cases, both of whom were suffering from the disease on their admission to the asylum.

1. By uncured rice is meant the ordinary white rice, sold in the Federated Malay States as Rangoon or Siam rice, which is eaten by all classes except Indians and Malays. This rice after being harvested is taken to the mills, where it is husked and cleaned before being sold to the rice merchants. Cured rice is brownish in colour and forms the staple of diet of Indians and Ceylonese. The great difference between this rice and the uncured variety is that the former is boiled and dried before being milled. The Malays keep their rice stored unhusked and pound and winnow it when they wish to cook it. A. White rice is stored unboiled after being husked. B. Cured rice is stored after being boiled and then husked. C. Native Malay rice is stored unhusked. Except for the difference in the rice the two parties—those on cured and those on uncured rice—received the same kind and the same amount of rations. Excepting the rice the food-stuffs for all patients were prepared together in the same kitchen and cooked in the same cooking pots.

2. The lunatics are housed in two exactly similar buildings on opposite sides of a quadrangle surrounded by a high wall. On Dec. 5th all the lunatics at that time in the hospital were drawn up in the dining shed and numbered off from the left. The odd numbers were subsequently domiciled in the ward on the east side of the courtyard and no alteration was made in their diet, they were still supplied with the same

uncured rice (Siamese) as in 1905. The even numbers were quartered in the ward on the west of the quadrangle and received the same rations as the occupants of the other ward, with the exception that they were supplied with cured (Indian) rice instead of the uncured Siamese variety. The following is the ordinary diet scale of the lunatic asylum: fresh meat, four ounces four times a week; fresh fish, 5⅓ ounces two times a week; salt fish, 5⅓ ounces once a week; vegetables, eight ounces daily; curry stuffs, 1⅓ ounces daily; and cocoanut oil, ⅔ of an ounce daily. Uncooked rice: Siam, 28 ounces to be supplied as per sample for uncured rice ward; Bengal, 28 ounces to be supplied as per sample for cured rice ward. At the commencement of the experiment all patients showing unmistakeable symptoms of beri-beri were removed to the district hospital, which is two miles distant from the asylum. On Dec. 5th there were 59 lunatics in the asylum; of these 29 were put on cured rice and 30 on Siamese rice. The next patient admitted to the asylum was admitted to the Bengal rice ward, and the one admitted after him to the uncured rice ward, the next to cured, and so on alternately to the end of the year.

3. The knee-jerks of all patients in the asylum on Dec. 5th, 1905, were noted on that date and of those admitted subsequently on the dates of their admission. The presence or absence of œdema was noted at the same time.

Patients admitted to Siamese (Uncured) Rice Ward.

Knee-jerks average	... ...	86	(21 developed beri-beri).
,, absent	... ...	11	(2 ,, ,,).
,, impaired	... ...	7	(1 ,, ,,).
,, increased +	...	10	(5 ,, ,,).
,, ,, + +	...	4	(3 ,, ,,).
,, ,, + + +		2	(2 ,, ,,).

22 patients in the Siamese rice ward had some œdema on admission.

Patients admitted to Bengal (Cured) Rice Ward.

Knee-jerks average	79
,, absent	28*
,, impaired	2
,, increased	5
,, ,, ...	6
,, ,, ...	3

* Two had beri-beri on admission.

32 patients in the Bengal rice ward had some œdema on admission.

4. By June 20th many cases of beri-beri had occurred amongst the patients in the east ward who were eating uncured rice, whereas no cases had occurred in the west ward, the inmates of which were dieted on cured (Indian) rice.

5. In view of the theory so strongly advocated by Sir Patrick Manson that beri-beri is a place disease, it was thought possible that the east ward was infected. Therefore on June 20th the patients were transposed, those on uncured rice being moved to the west ward and those on cured (Indian) rice transferred to the east. From June 20th to Dec. 31st no beri-beri developed amongst the patients on cured rice although they were living in a ward where beri-beri had been rife amongst the lunatics who were fed on uncured (Siamese) rice.

6. From Nov. 3rd, 1905, until April 11th, 1906, all persons showing symptoms of beri-beri were transferred to the district hospital, as it was considered that this would be beneficial to those suffering from this disease and also might possibly prevent the spread of it within the institution. In all 13 persons suffering from beri-beri were sent to the district hospital of whom nine died.

7. In April, as no patients on cured rice had developed beri-beri, it was thought that it might benefit those already suffering from the disease to withdraw them from the uncured rice diet and place them on the cured or Indian variety. Between April 11th and Dec. 31st ten cases of beri-beri were transferred to the ward occupied by patients on cured (Indian) rice and were supplied with that rice instead of the uncured variety which they had been taking when they developed the disease. All these ten patients recovered.

It is worthy of note that although patients actually suffering from beri-beri were put to live amongst the lunatics fed on cured rice none of the latter developed the disease. This, as far as it goes, is opposed to the theory of Dr. C. W. Daniels that beri-beri is a parasite disease, probably protozoal, conveyed by the agency of bugs or lice. Neither does the above result conform to Dr. Hamilton Wright's theory that beri-beri is an infectious disease communicated from patient to patient by means of the excreta. In this connexion it should be mentioned that in September, 1905, it was noticed that the lunatic asylum was infested with bugs, and at the suggestion of Dr. Daniels some of these were collected and put on monkeys, mice, and guinea-pigs. Lice from the head of a beri-beri patient were used in the same way. Later the bacteriologist made an emulsion of some bugs taken from the lunatic asylum and injected it beneath the skin of an orang-outang. None of the animals showed any signs of beri-beri. The bugs in the asylum were to a great extent got rid of before the commencement of 1906 by means of hot water, sun, perchloride of mercury, and tuba root, but they still are, and, I am told, always have been, present in the old building.

8. With regard to the general management of the lunatics, the two batches of patients are both fed in the same shed but at different times and at different tables, those patients on cured rice diet commencing their meal when the patients on Siamese rice have finished. The two batches of patients are only separated at night and when eating their meals; at other times they associate together in the courtyard and are employed outside the asylum in working parties. Those patients who were sane enough and strong enough physically to be employed in working outside the asylum, in the hospital grounds, or in the Guinea grass field opposite the asylum enjoyed no special immunity to beri-beri but suffered equally with those patients not so employed. 12 out of 34 persons affected with beri-beri belonged to the outdoor working party.

9. The uncured (Siamese) rice supplied to the asylum was of excellent quality (No. 1) and much better than that to which persons of the class from which the patients are drawn are accustomed outside the asylum. No mouldy or stale rice was given. The cured or Indian rice was of No. 1 quality. It was not Province, Siam, or Rangoon rice cured in Penang, but the small, round-grained Indian rice cured in India. The rice was cooked in the usual Eastern manner.

The following is the procedure. It is first washed with cold water in galvanised-iron buckets. It is then thrown into a shallow iron pan or kwali half full of hot water, which is placed over a wood fire. In about 20 minutes the rice has swelled and has taken up all the water (a little earlier in the case of the Siamese rice and later in the Indian variety). It is then stirred round twice with a large spoon or chabok and the burning wood is taken out from under the kwali, only glowing charcoal being left. When the rice has become soft, which occurs about 20 minutes later, the rice is taken out with a large spoon, care being taken not to disturb the thin sheet of burnt rice, or krak, which is caked over the surface of the kwali. The cake of burnt rice is afterwards scraped out and given to the fowls, pigs, &c. The two varieties of rice were both cooked in the same manner by the same cook (a Chinese). It is worthy of note that the culinary methods followed by Tamils and other consumers of cured rice differ slightly from the Chinese procedure. Tamils cook their rice with a larger proportion of water and when it commences to boil they pour off the supernatant fluid, whereas the Chinese allow the rice to take up all the water and pour none off. This is an important fact if the theoretical poison in rice which is supposed to cause beri-beri be soluble in water. Throughout the course of the experiment at the asylum a separate kwali, or cooking-pot, was used for each kind of rice and no uncured rice was cooked in the kwali set apart for the preparation of Bengal rice. Similarly different spoons, &c., were used for stirring each kind of rice and also for serving it out to the patients. Two sets of plates were provided, one for the patients on cured rice and another for the patients on Siamese.

10. It has already been mentioned that 10 patients who had been eating uncured rice and had developed beri-beri were transferred to the Bengal rice diet and recovered. The admission of these patients caused some overcrowding in the Bengal rice ward. Four men were therefore transferred from the cured rice ward and put on a diet of uncured rice. These men were not selected in any way, but the first four names on the Bengal rice list were taken. They were apparently healthy and had been on a diet of cured rice since

Dec. 5th, 1905. The following is a list of these four patients and the result of their transfer :—

No.	Name.	Transferred to uncured rice.	Result.
1	Salleh.	24/4/06	Remains healthy.
2	Foo Lim.	,,	Developed beri-beri on 7/7/06. He had had beri-beri 10 months before.
3	Hee Chong	27/4/06	Developed œdema and some weakness.
4	Qual Kiam.	23/6/06	Developed beri-beri 27/8/06 and died on 24/9/06.

No conclusion can be based on the above, the numbers are too small; but one cannot help being struck by the fact that 10 patients suffering from beri-beri transferred to Bengal (cured) rice all recovered, whereas of four apparently healthy men transferred from Bengal to Siamese (uncured) rice two developed beri-beri, one of whom died.

11. At the commencement of the experiment on Dec. 5th, 1905, there were 59 patients remaining in the asylum; 29 of these patients were placed on a diet of cured rice, whilst 30 remained on Siamese rice. From Dec. 5th, 1905, to Dec. 31st, 1906, 90 lunatics were admitted to the Siamese rice ward, 17 of them developed beri-beri; whilst of the 30 remaining on Dec. 5th 17 developed beri-beri before Dec. 31st, 1906. During the course of the experiment four lunatics were admitted to the asylum who were already suffering from beri-beri. They were admitted on the following dates: Dec. 10th, Jan. 23rd, March 19th, and June 5th. Two of them were put on Siamese and two on Bengal rice. The majority of the cases of beri-beri occurred amongst lunatics who had been in the asylum for a considerable time.

	Cases.
Cases suffering from beri-beri on admission	4
Cases occurring amongst lunatics during their first month in the asylum	6
Cases occurring amongst lunatics during their second month in the asylum	7
Cases occurring amongst lunatics during their third month in the asylum	8
Cases occurring amongst patients who had been in the asylum more than three months	20
Total	45

In all 36 persons suffered from beri-beri. One patient had three attacks and seven patients two attacks. 28 had one attack. Whether the eight patients who were attacked with beri-beri more than once during the experiment actually suffered from a fresh "infection" (if one may use the word in this connexion) or whether the subsequent attacks were of the nature of relapses it is impossible to say, owing to our lack of knowledge of the actual cause of the disease.

Of the 36 persons who suffered from beri-beri 16 died during their first attack; ten were transferred to the cured rice ward, and all recovered; of the ten patients who remained on Siam rice two were discharged from the asylum before being attacked a second time and eight had relapses.

Died during first attack	16
Transferred to Bengal ward during first attack and recovered without relapse	10
Discharged from asylum before being attacked a second time	2
Remaining on Siam rice and suffering from second or third attacks of beri-beri	8
Total	36

12. The mortality of the lunatics on either diet was much the same except with regard to beri-beri :—

Cause of death.	Number of deaths (Bengal rice).	Number of deaths (Siam rice).
Beri-beri	0	18
Mania	6	4
Other causes	4	2
Dysentery	11	11
Total	21	35

Summary.

13. *A.* Amongst 120 patients on uncured rice there were 43 cases of beri-beri (two admitted with the disease) and 18 deaths. Amongst 123 patients on cured rice there were two cases of beri-beri, and these both had the disease on admission. *B.* Ten lunatics actually suffering from beri-beri who were placed on a diet of cured rice all recovered. Of 26 patients suffering from beri-beri who were not put on a cured-rice diet 18 died. *C.* None of the ten lunatics suffering from beri-beri who were placed on a diet of cured rice had a relapse. Of the 26 patients suffering from beri-beri who were not transferred to a cured-rice diet 16 died during the first attack and the remaining ten all developed beri-beri again with the exception of two who were discharged, being no longer insane. The latter have been lost sight of and whether either of them subsequently relapsed is unknown. *D.* Four lunatics who had been on a diet of cured rice for more than five months and were apparently healthy were transferred to a diet of uncured rice. Two of these patients developed beri-beri within three months.

14. In an experiment of this nature the personal factor always comes in question. It may, therefore, not be out of place to state here that at the commencement of the experiment the opinion was held by myself that rice was neither directly nor indirectly the cause of beri-beri. It was fully expected that the patients on Bengal rice would suffer from beri-beri to the same extent as those who remained on the Siamese variety and that the result of the experiment would be a refutation of the rice theory. With this in view precautions were taken to provide separate cooking utensils, plates, &c., for each set of patients in order that the upholders of the rice theory might not be able to point to any possibility of contamination of the Bengal rice with the poison which is supposed to be present in uncured rice. Contrary to expectation, the result of the experiment, so far as it goes, is to prove the truth of Dr. Braddon's contention that uncured rice is the cause of beri-beri. It remains to be proved whether the cause of the disease amongst the eaters of uncured rice is a poison contained in the rice or whether there is something essential to the human economy which is supplied by the cured rice whilst it is absent in the uncured. Takaki and the Japanese school still hold that a deficiency of proteids in the diet is the cause of beri-beri. As yet it has, unfortunately, not been possible to obtain analysis of the two kinds of rice; but when this is done it will probably be found that the cured rice contains a larger quantity of proteid matter than the uncured. If this be the case the deficiency of proteid matter in the diet may be the actual cause of the disease, or, what is more likely, the lack of nutriti vematter in the rice may induce a condition in the patient which renders him an easy prey to some external agency—bacterial or protozoal—which is the actual cause of beri-beri. It is well known that certain conditions of the body render it specially liable to the attacks of beri-beri—e.g., syphilis and the puerperium. In 1904 there were eight cases of beri-beri amongst women admitted to the General Hospital, Kuala Lumpur; all these occurred amongst women in the puerperal state.

Conclusions.

15. Uncured rice is, either directly or indirectly, a cause of beri-beri, the actual cause being either (1) a poison contained in the rice; (2) deficiency of proteid matter, the disease being due to nitrogen starvation; or (3) uncured rice does not form a sufficiently nutritive diet and renders the patient's system specially liable to invasion by a specific organism, which is the cause of beri-beri.

Postscript.—Throughout the course of the experiment no doubtful cases have been noted as beri-beri. Many cases of slight œdema, abnormal reflexes, &c., may have been of the nature of abortive attacks. The following record of an examination of the knee-jerks and of the presence or absence of œdema amongst the patients now in the asylum may be of interest. The examination was made on May 19th, 1907. The results were as follows: the knee-jerks of patients on Siam rice (May 19th, 1907): present, 13; absent 18 (in 5 absent on admission); increased, 6; total, 37. Knee-jerks of patients on Bengal rice (May 19th, 1907): present, 23; absent, 8 (in each case absent on admission); increased, 5; total, 36. With regard to œdema, patients on Siamese rice 10 out of 37 had it and patients on Bengal rice 4 out of 36. Sixteen of the 37 patients on Siamese rice were admitted to the asylum before the beginning of 1907 and consequently

had been on this diet for a considerable period; in eight of these 16 cases the knee-jerk was absent and in three it was increased, while in five œdema was present. In all those eight cases on Bengal rice in which the knee-jerks were absent they were noted as being absent on admission. Three were cases which had been transferred to a Bengal rice diet after having developed beri-beri whilst on a Siam rice diet. Two had been treated for beri-beri in other hospitals. Two gave a history of previous attacks. In one case the cause of absence of reflex is unknown. Since the end of December, 1906, up to the present date (May 19th, 1907) 12 cases of beri-beri have developed amongst the patients on a Siamese uncured rice diet. No patients on Bengal (cured) rice have suffered from the disease. The 12 cases occurred as follows:—

During the first month of residence in asylum	0
During the second month ,, ,, ...	3
During the third month ,, ,, ...	4
After three months' residence in asylum	5
Relapse cases	0
Total	12

Thus from the commencement of the experiment in December, 1905 (excluding patients actually suffering from the disease on their admission to the asylum), there have been (41 + 12) 53 cases of beri-beri amongst the patients on Siamese rice and none amongst those on a Bengal rice diet.

Kuala Lumpur, Federated Malay States.

Medical Societies.

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.—The annual provincial meeting of this society was held on June 15th at Bedford, Dr. R. H. Coombs being in the chair.—Dr. C. O. Hawthorne opened a discussion on Acute Rheumatism in Childhood. He referred to the considerable change which had taken place in the professional view of the age incidence and to the special characters which distinguish the disease in childhood, especially the frequency of cardiac disease. This he attributed partly to the absence of restraint of the child's activities resulting from the comparative escape of the joints, and he advocated the practice of informing the parents of rheumatic families of the risks involved even in the apparently trivial illnesses of their children. He thought that there was too ready a tendency to apply the term "rheumatism" to isolated events, the nature and origin of which were obscure. He also discussed the curative and prophylactic treatment.—Dr. W. Ewart suggested that the glandular condition of early childhood was less prone to favour retardation in the joints and the child could rid himself of disease in the joints and it would find a seat elsewhere. He suggested calomel and antiphlogistic remedies in the treatment of endocarditis.—Dr. A. Morison referred to erythema nodosum, which, he said, was not limited to rheumatic subjects; it was generally preceded by tonsillitis at an interval of from 10 to 14 days. He looked upon this as its incubation period.—Dr. A. C. Hartley (Bedford) said that in that town, on the banks of a river, with a damp climate, it would be expected that rheumatism would be very common, but his experience of 14 years did not bear this out.—Dr. G. A. Sutherland thought that it was a mistake to prolong rest in bed after active endocarditis had passed away, which was frequently done owing to the reappearance of a murmur. He referred to cases of recurring subacute rheumatism where salicylates failed; he considered that there was some concealed source of infection which was not discovered.—Dr. E. Cautley read a paper on Cerebro-spinal Meningitis. He mentioned the different varieties and their relative age incidence as an aid to diagnosis; also the relationship of the sporadic to the epidemic variety, the means of diagnosis, including lumbar puncture, &c.—Mr. A. H. Tubby read a paper on Contraction of the Tendo Achillis as a cause of the following symptoms: pains in the calves, shambling gait, sprained ankle, everted feet, turning in of the toes, &c. He said that the contraction of the calf muscles produced a lessening of the power of dorsiflexion of the foot and shortening of the stride in consequence. It was usually due to infantile paralysis and could be remedied by lengthening the tendon, the method of doing which was described.—Dr. H. Skelding described a case of Bilharzia in a boy, aged 11 years, who came from Rhodesia. The symptoms were great drowsiness and hæmaturia. The ova were found in the urine.—Dr. G. Harvey Goldsmith described a condition occurring in children to which he gave the name of "œdema of the limbus." There were photophobia and injection of the ocular conjunctiva, on which were small grey nodules that never broke down. It was rapidly cured by the yellow oxide of mercury ointment.—Clinical cases, pathological and microscopical specimens, and skiagrams were shown by Dr. Goldsmith, Dr. Ross, Dr. R. Coates, Dr. W. Phillips, Dr. H. L. Waller, Mr. Johnson, Mr. W. A. Sharpin, Mr. W. G. Nash, and Mr. G. T. Verry.

BRADFORD MEDICO-CHIRURGICAL SOCIETY.—A meeting of this society was held on May 28th, Dr. A. Mantle, the President, being in the chair.—A paper was read by Dr. A. F. Martin entitled "A Study of Post-typhoid Pyrexia" in which an account was given of 13 cases of typhoid fever out of a total of 63 admitted to the North Staffordshire Infirmary in the year 1905 which developed post-typhoid fevers of other nature than a relapse. A review of the English and American literature of the subject was given, and a classification suggested by Delafield as a basis of study was adopted since it agreed with the findings of Dr. Martin in his cases. The important thing was to distinguish these fevers from relapses. Curschmann was quoted as insisting on the reappearance of some of the classical signs of the typhoid infection before a diagnosis of relapse could be arrived at. In the fevers in question these were absent. Of the three types which Delafield found to exist the first included all those rises of temperature which were slight and temporary. They usually occurred in those who were of a nervous introspective temperament and their only importance was to learn to disregard them. These and perhaps some others were of a purely nervous origin. In the second group the fever was of indefinite length, lasting from eight days to one month, developing early in convalescence, and was of a remitting type suggestive of chronic septicæmia. The general condition showed a progressive wasting and later cardiac weakness. Some were mild and others were very severe infections. The treatment adopted in checking the fever was as speedy a return to full diet as was consistent with safety. Salicylate of sodium was found to be of no avail. In one case fæcal accumulation had occurred before the onset of the secondary fever without any rise of temperature. The view held by Da Costa that constipation was a frequent cause of post-typhoid pyrexia did not receive support from this and other cases. The anæmic and the nervous origin of these fevers was discussed, but did not find strong support from observations made. In the third group the fever was similar in type but was accompanied by venous thrombosis. Hacker's statement was taken as evidence of their septic nature—namely, that in post-typhoid thrombosis pyogenic cocci were frequently found in the thrombus, whereas Eberth's bacillus typhosus had not been isolated. These thromboses occurred chiefly in those cases where the primary fever had been unusually lengthy, and the fever accompanying them was often severe and showed rigors. The treatment adopted was the same as in the previous group and it was found that salicylate treatment in some cases was beneficial and in others a course of antistreptococcic serum did good. There were six cases of this type, the last being a remarkable case of post-typhoid mitral endocarditis developing nine weeks after the primary fever had abated. It was argued from a similarity of the type of fever in Groups 2 and 3 that the second group could fairly be said to be of septic origin.

Reviews and Notices of Books.

George Buchanan: A Memorial 1506–1906. Contributions by various writers, compiled and edited by D. A. MILLAR. St. Andrews: W. C. Henderson and Son, University Press; London: David Nutt. 1907.

JUST a year ago Scotland celebrated the fourth centenary of one of her forgotten worthies, George Buchanan, first at St. Andrews, of which he was an alumnus and afterwards principal; and, secondly, at Glasgow, in which university, as a west country Scot, he took special interest. Both celebrations were in the highest degree successful, evoking in favour of the "Humanism," of which he was a typical exemplar, a demonstration which must have been peculiarly reassuring to those who think humanistic study doomed to