the farm, where she had been occupied for two or three weeks (I have not my original paper at hand). I asked her if when she came to the farm she had any diarrhoea, but this she denied. She assured me that during these intervening weeks she had been in good health.

At this point arose a further remarkable problem. She had fallen ill at another remote farm, where she had been working; so that unless there had been fever there our difficulty was but thrown one step backward. At this farm accordingly I had full inquiries made, and with the parallel result that there, too, typhoid fever had been unknown till this woman fell ill (whether other cases had arisen there I forget, but I think it was so). Thus, as I say, our difficulties were not solved, only pushed back a step. To cut a long story short, it turned out that to this second isolated farm, then and always before free from typhoid, had come a kinsman of the occupier, who was convalescent from the disease contracted in Liverpool. He came to the farm for country air after, as was supposed, a perfect recovery had been made.

On the basis of these observations of mine I begged Dr. Dreschfeld, when engaged upon the article on Enteric Fever for my *System of Medicine*,² to go closely into this matter, urging upon him that the bacilli survived for indefinite terms in the stools—in the stools, there was my error. Dr. Dreschfeld, with unwearied kindness, examined again and again the stools of convalescents from typhoid, but with no success in this respect. With the help of Dr. Richmond the fullest investigation was made into the alleged survival, but in vain. In the stools the bacilli certainly do not survive long into convalescence, even if until convalescence. To suspect the urine never occurred to me. The victims of the arrows of Apollo fell at our feet but we failed to see whence they came.

Dr. Hugh Walsham has been so good as to demonstrate to me that tubercle bacilli are usually excreted by the kidney without the formation of "tubercle" in the organ; and Dr. Horton-Smith and others assure us that typhoid bacilli thus excreted may come through in millions, and for a period long enough to poison a county. I repeat, then, that upon all practitioners, and medical officers of health especially, it is incumbent to see that the disinfection of the urine urged by Dr. Walker is carried out systematically in every case before the patient is allowed to mix again freely with his kind.

REFERENCE.

¹ Richardson, *Journal of Experimental Medicine*, January, 1899. ² *System of Medicine*, vol. 1, 1896, pp. 799 and 800.

ON THE EVOLUTION OF MYELOPATHIC ALBUMOSURIA.

By T. R. BRADSHAW, B.A., M.D., M.R.C.P.,

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It is now more than three years since I first brought the subject of myelopathic albumosuria to the notice of the Royal Medical and Chirurgical Society. The disease was then unknown to the profession in this country, and only 8 clear cases were to be found recorded in the medical literature of Europe.

Since that time 7 more cases have been published, more or less fully, bringing the total up to 16. Of these I have personally examined 2, and in a third case I have been enabled to make a thorough investigation of the urine. There is one point in the clinical history of the disease which has never been determined—namely, the manner in which the albumose appears first in the urine. In all recorded cases it has been present in massive quantities, but it has never been ascertained whether it begins to appear in minimal quantities and gradually increases, or whether the onset is sudden, the maximal amount appearing almost from the first. The possibility of the latter mode of onset is suggested by what we know of the onset of pancreatic diabetes.

I have lately been consulted by a physician of eminence practising on the Continent of Europe, who is the subject of myelopathic albumosuria, and owing to exceptional circumstances it was possible to detect the abnormal condition of the urine at an early stage. As this is the first case of the disease which has been recognised as such in a country with a population of more than 50 millions, it is not surprising that the exact

condition of the urine was at first apparently not understood, the albumose being mistaken for albumen; but at the present time the reactions are highly characteristic, and apart from the symptoms which have recently become prominent, they leave no possibility of doubt as to the nature of the case.

The albumose was first observed in December, 1899, when the patient, who was then 53 years of age, had an attack of mild pneumonia. The quantity was at that time minute, but it has subsequently gone on continually increasing, and has reached a maximum of 10 parts per 1,000. Until the middle of May in the present year the patient felt no inconvenience and was able to follow his profession, but since then he has grown pale and lost flesh and strength. He has also found a soft tumour about the size of a hen's egg, in connection with one of his ribs.

The foregoing account of the case does not claim to be in any way complete, but it illustrates two important points, one of which is new.

1. The presence of the albumose in the urine is the earliest symptom of the disease, and may be observed for many months before there are any other indications of failing health, or any local signs of alterations in the skeleton.

2. If we may be permitted to generalise from the only case in which the point has been investigated, the albumose is first excreted in small amounts; but, owing to the fact that at this stage the subject of the disease does not suffer any inconvenience from it, he is not likely to consult a medical practitioner, and so the urine is generally not examined until the albumose has risen to a large amount. Its early discovery in the present instance was doubtless due to the fact that its onset closely coincided in time with an acute illness, and that the subject of it a member of our own profession.

ON SOME CASES OF HÆMORRHAGE INTO THE SKIN AND SUPRARENAL CAPSULES.

BY

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The following four cases are apparently of sufficient rarity to justify us in putting them on record; at all events they are the first cases that have ever been noted in this hospital.

CASE I.

G. F., a male child, aged 11 months, was admitted to the East London Hospital for Children, Shadwell, on February 4th, 1901.

History.—On the day previous to admission the child was running about and playing, apparently quite well. At 9 p.m. he went to sleep, but a quarter of an hour later he woke up and vomited slightly. He was very restless all night, and on waking in the morning he did not seem at all well, and the mother noticed one or two dark spots on the back. At 7 a.m. violent diarrhoea began, the stools being very frequent, watery, and yellow-green. He did not again vomit. From this time he got rapidly worse, and at 9 a.m. was brought to the hospital. The child was a breast-fed baby, but on the day before admission he had had in addition some potato, some bread and butter, and a little toast. He had had no previous illness of any kind, and had never been vaccinated. The family history was good, but there was a slight history of rheumatism.

Condition on Admission.—The patient, who was extremely well nourished, was moribund; his eyes were half open, respirations were slow and shallow, and the pulse rapid, but scarcely perceptible. The face and lips were cyanosed and the extremities cold, though the body felt hot. All over the surface of the body there were numerous subcutaneous hæmorrhages, irregular in shape and of various sizes from a pin's head to a threepenny piece. These were most numerous on the back, where they were of a larger size than elsewhere. On the face they were small and not very numerous, and were distributed for the most part on the forehead and behind the ears. There were a few on the scalp, and they were very numerous on the upper and lower extremities. All over the chest coarse rhonchi were heard; the heart sounds were feeble; the abdomen was natural to palpation.

Death.—The patient died at 11.15 A.M., having been ill fourteen hours altogether.

Post-mortem Examination.—The body was extremely well nourished. Subcutaneous hæmorrhages were present as above described. The lungs showed some areas of emphysema and collapse, but there were no subpleural hæmorrhages. The heart was natural. The thymus was very large and natural. The stomach was natural. The Peyer's patches throughout the whole length of the small intestines were very much swollen and prominent, as in the early stage of typhoid fever, but there was no ulceration. There were a few minute petechial spots on the peritoneal surface of the bowel. The mucous membrane of the large intestine

was rugose from muscular contraction, and also appeared thickened; there was no marked hyperplasia of the solitary follicles; at one point a minute hæmorrhagic spot was seen in the mucous membrane. The mesenteric glands were much enlarged. The liver and spleen were natural. In the kidneys the bases of the pyramids were much congested. The suprarenal capsules were both very large and markedly hæmorrhagic on the surface and on section. The brain and meninges were natural. Microscopically the suprarenal bodies proved to be evenly filled with extravasated blood.

CASE II.

W. G., male, aged 1 year, was brought to the hospital, at 9 A.M., on April 4th, 1901.

History.—The child was quite well until the previous afternoon, when he became fretful, and in the evening was "very queer," being very hot and feverish. About midnight he awoke vomiting, and for the remainder of the night was very restless. In the early morning he began breathing badly. His general condition became worse until he was brought to the hospital, before which he again vomited. He had had no diarrhoea, cough, fits, or twitching. He had had measles four or five months previously, from which he quite recovered. He was vaccinated when two or three months old, with good result. He had been hand-fed, being brought up on condensed milk, and had had nothing unusual in diet previous to the attack. The family history was very good.

Condition on Admission.—He was extremely well nourished, but was very collapsed, with an extremely feeble and rapid pulse; the breathing was rapid and shallow, and the alæ nasi were working. The face was dusky, and the lips livid, the body generally presenting a dusky, mottled appearance. Over the left side of the forehead, the upper part of the chest and back, and upper arms were a number of small subcutaneous hæmorrhages. These, according to the mother, were not present when she left home for the hospital. A few scattered rhonchi were heard over the front of the chest. The heart and abdomen were apparently natural.

Death.—The child died a few minutes after admission, having been ill about twelve hours altogether.

Post-mortem Examination.—Subcutaneous hæmorrhages as above described were present. There were two good vaccination marks on the left arm. The lungs showed scattered but not very extensive patches of collapse, with much engorgement, but no consolidation or hæmorrhagic spots. The heart and pericardium were natural except for distension of the right heart with blood clot; it showed no hæmorrhages, and the thoracic glands were natural. The liver was slightly fatty; the stomach was natural, and free from hæmorrhages. The Peyer's patches were distinctly swollen, especially in the jejunum. There was no enlargement of the solitary follicles in the colon, and no hæmorrhages. The mesenteric glands were large and very firm. The spleen was firm, mottled with dark areas on section, but not enlarged. The kidneys were congested, and presented a few hæmorrhagic points at the bases of the pyramids, but were otherwise natural. Both the suprarenal bodies were enlarged, and of a dark, purplish-red colour on the surface; on section they presented a laminated appearance, the whole dark, but some areas rather lighter than others. The suprarenal vessels appeared normal; there was no evidence of infarction. The brain and meninges were engorged, but otherwise natural.

CASE III.

M. B., female child, aged 7 months, was admitted on April 10th, 1901. *History.*—The child was a healthy and well-developed baby at birth but soon afterwards he began to waste. He had snuffles and a rash about the buttocks about two months after birth. The present illness began suddenly with diarrhoea on the day of admission. There had been no vomiting. The patient had been a breast-fed child, and for the last two months was given Ridge's food and crusts of bread. There was no history of any previous illness and he had never been vaccinated. Family history: The father and mother were healthy; there were two other children in the family, both of whom were healthy. Mother had had one child born dead and one miscarriage.

Condition on Admission.—The patient was very thin and collapsed; the face was dusky and the lips livid; the pulse was 156 and feeble, the respirations 48, and the temperature 102.6°. A few scattered rales were heard in the chest; the heart's action was rapid; the liver was slightly enlarged but nothing further abnormal was detected in the abdomen. Death took place eighteen hours after admission, the child having got rapidly worse. A few hours before death two dark purpuric spots appeared—one on the lobule of the right ear and one on the right forearm. There were no convulsions.

Post-mortem Examination.—A few subcutaneous hæmorrhages were seen on the surface. The lungs were engorged and oedematous, here and there was some patchy collapse. The heart was natural; the liver was somewhat enlarged; the spleen and the kidneys were natural; the left suprarenal capsule was large, dark, and purplish red, and hæmorrhagic on section, the medulla being of a darker colour than the cortex. The right suprarenal capsule was also hæmorrhagic, but had a pale patch of apparently healthy tissue anteriorly. The intestines were unfortunately not opened and examined. There were no other hæmorrhages anywhere.

CASE IV.

A. H., a female child, aged 1 year, was admitted to the hospital at 12.30 P.M. on April 27th, 1901.

History.—The patient when put to bed on the previous night was "well and hearty." At 9 P.M. she had some barley broth, and was sick immediately after it. She was restless all the night, and at 5 A.M. was violently sick, bringing up curdled milk. She again vomited half an hour later, the vomited matter being of a yellow colour. At 6 A.M. the patient was brought to the hospital. The temperature then was 102°, but otherwise the child did not seem to be ill, and was consequently treated as an out-patient. At midday the child was again brought to the hospital, but in a very collapsed condition, and was admitted. Half an hour before admission the patient passed a very dark stool. The child had been brought up on the breast. On the day previous to admission she had had some bread and butter, a little potato, and a small piece of sausage. There was no history of any previous illness. This child had also never been vaccinated. The family history was very good.

Condition on Admission.—The patient was extremely ill. The eyes were half open; respirations were shallow, and 72 to the minute; the pulse

was 164, and of very small volume, but regular in rhythm. The face and lips were cyanosed, and the rectal temperature was 97°. The body was generally mottled, but there were no petechiæ anywhere. Scattered throughout the chest some rhonchi were heard. The heart's action was feeble and rapid. The abdomen was natural. On admission the child was put into a mustard bath, and was stimulated with brandy and hypodermic injections of strychnine. At 2 P.M. the temperature rose to 102.6°, and twelve hours later the thermometer registered 106.4°. A boracic enema, at 7.5°, was then given, and the temperature dropped to 98.6°. A few hours later it again went up to 103.6°. The patient had altogether five stools after admission; these were yellowish-green and slimy in character. There had been no vomiting all the time the child had been in hospital. Death took place at 7.50 A.M., nineteen hours after admission.

Post-mortem Examination.—The body was that of a very well-nourished child. There was much mottling of skin generally, and over the centre of the back two small petechial spots were present. Both lungs were engorged, and there was some collapse in both lower lobes. The heart was natural. The thoracic glands were natural. In the abdomen the liver and spleen were both normal. The stomach was natural. There was marked hyperplasia of Peyer's patches and of the solitary follicles throughout the small intestines. There were also many subperitoneal punctiform hæmorrhages. The mesenteric glands were large and soft. The kidneys were natural. The left suprarenal capsule was injected on the surface, and the medulla was very dark-red on section. The right suprarenal capsule was slightly injected. The brain and meninges were engorged.

The interesting feature about these cases is the sudden onset, rapid course, and fatal termination. Not one of the patients was over a year old. The history throws absolutely no light on the causation of the disease; the question of food does not appear to bear any causative relation to it. The presence of hæmorrhages in the skin and suprarenal capsules would seem to make it more than probable that the cause of the disease is to be found in some form of toxæmia.

In two of the cases the blood from the unopened heart was examined bacteriologically with a negative result. In its extremely rapid and fatal termination the disease somewhat resembles the epidemic diarrhoea and vomiting of infants, and it is probable that the mode of infection is similar, and that the poison belongs to the same group. The similarity, however, ends here, since diarrhoea and vomiting were evidently not the main symptoms in the cases above mentioned, and in the many *post-mortem* examinations held here on cases of epidemic diarrhoea and vomiting, we have not met with a single instance in which hæmorrhage took place into the skin or suprarenal capsules.

The general condition of the patients was moreover totally different. They did not present the sunken eyes and inelastic skin so frequently met with in the epidemic diarrhoea. On the other hand the deep cyanosis present in these cases is very rarely, if ever, seen in the latter disease. Apart from the hæmorrhages that took place into the skin and suprarenal capsules, the fact that the Peyer's patches were much swollen is interesting. Macroscopically, the condition was extremely like that presented by the patches in the early stages of typhoid fever. Referring, again, to epidemic diarrhoea and vomiting, the intestinal lesion is in most cases limited to the large intestine, where the solitary follicles are very prominent. We do not remember ever having seen so distinctly an hyperplastic condition of Peyer's patches in diarrhoea and vomiting as was present in three out of the four of our cases. No history of hæmophilia was obtained.

The question now arises, What is this disease? Is it an unknown disease *sui generis*, or are the symptoms only a rare manifestation of some known specific disease? If we are disposed to place these cases under the latter heading, then we cannot put out of account the possibility that they may be cases of hæmorrhagic small-pox of the fulminating variety. One of our cases, however, had two very good vaccination scars; the other three had not been vaccinated. There was, moreover, no hæmorrhage into the conjunctivæ in any of the cases. In addition to this, there was no previous small-pox in the neighbourhood, and, as far as we know, no cases occurred subsequently. Again, it might be said that if these children had lived long enough they might have developed symptoms of some one of our well-known specific diseases. Infantile scurvy may suggest itself to some, but there was no sponginess of the gums and no tenderness in any of the limbs. Besides, the onset was so sudden and the fatal termination so rapid that both these points would strongly go against this view. On the whole it is probable that the symptoms are the manifestation of a special disease, and that the cause of this disease is a blood poisoning of some form, the nature of the poison being at present unknown.

Dr. Eustace Talbot reported two cases of hæmorrhage into the suprarenal capsules in the *St. Bartholomew's Hospital Reports* for 1900, in which he also refers to two cases reported by Drs. Andrews, Garrod, and Drysdale in the Pathological Society's *Transactions* for 1898. These four cases were all unvaccinated, were apparently healthy children, suddenly taken ill, with a rapid and fatal termination. Subcutaneous hæmorrhages occurred in the two latter cases only, and convulsions seem to have been a prominent symptom in the two cases reported by Dr. Talbot. It will be noted that in our cases no convulsions occurred. The only morbid changes found *post mortem* in all these four cases was hæmorrhage into the suprarenal capsules, all the other organs being apparently healthy.

In conclusion, we desire to express our gratitude to Drs. Eustace Smith and J. A. Coutts for permission to publish these cases, and we also have to thank Dr. Clive Riviere, Pathologist to the hospital, for the use of the *post-mortem* examination notes.

EXPERIMENTAL MALARIA: RECURRENCE AFTER NINE MONTHS.

By P. THURBURN MANSON, M.B.LOND.,
Aberdeen.

A SUCCESSFUL experiment, of which I was the subject, on the production of malarial infection by mosquito bite, is recorded in the *BRITISH MEDICAL JOURNAL* of September 29th, 1900. The sequel is of interest.

As a result of the bites of mosquitos fed in Rome on a case of benign tertian ague, I developed a double tertian fever. The first symptoms appeared on September 13th, 1900, after an incubation period of between ten and sixteen days. The illness lasted from September 13th to September 17th, when, the presence of the parasite having been fully confirmed, 10 grs. of quinine were given. This treatment was followed by 5 grs. three times a day for a week, and a subsequent after-treatment of 5 grs. of quinine three times a day on Sundays for the following three months. There was no recurrence at that time of symptoms of malarial infection after the first dose of quinine, and I kept in normal health till May 30th, 1901, a period of nine months. I changed my abode from London to Aberdeen on April 15th, 1901, having resided in London since the original attack. On May 30th, 1901, I commenced without obvious reason to have prodromal symptoms of illness; these were *malaise* and pain in the splenic region. Two days later—on June 1st—a definite malarial paroxysm occurred. The following are the notes of my case:

June 1st. For the previous two days I had been feeling out of sorts, and had experienced slight aching in the splenic region. At 2 P.M. on this date I had a sensation of chilliness, accompanied with extreme lethargy and boneache. The temperature was 99.8° F. At 5 P.M. I became extremely chilly, but no actual rigor occurred; temperature 101.2° F. I went to bed, and at 6 P.M. began to feel warmer; temperature 102.6° F. At 9 P.M. profuse sweating set in; this gave considerable relief. At 10 P.M. the temperature was 103° F., and diaphoresis became more marked. The edge of the spleen was palpable, the splenic dullness increased, and the organ was both painful and tender. During the day there had been complete anorexia. Blood films were prepared, but were not examined till June 3rd.

June 2nd. Woke at 4 A.M., having slept well and sweated profusely during the night. At 6 A.M. the temperature was 99.8° F., by 10 A.M. it had fallen to 98.6° F.; during the rest of the day there was no pyrexia. Though feeling languid I was able to get about, and ate fairly well.

June 3rd. After a good night's rest I woke at 6 A.M., feeling quite well, with a temperature of 99° F. At 10 A.M. the temperature was 98.4° F. A small crop of herpes had developed at the angle of the mouth. I examined fresh films of blood drawn at 10 A.M., and found benign tertian parasites. At 12.30 P.M. shivering set in. Dr. Duncan, Assistant to the Professor of Pathology, Aberdeen University, then stained and examined blood films, finding young and older forms of the benign tertian parasite. During the rest of the day the parasites were found on several occasions, both in fresh and stained specimens. At 3 P.M. the hot stage supervened, and at 6.30 P.M. the temperature being 103° F., pronounced diaphoresis occurred affording marked relief. By 10 P.M. the paroxysm was over, the temperature having fallen to 99° F., and no inconvenience beyond slight pain in the splenic region remained. The spleen was still enlarged and slightly tender. During the day there was little desire for food. Quinine grs. x was taken. The films prepared on June 1st were stained and examined, and were found to contain a considerable number of young and older forms of the benign tertian parasite.

June 4th. During the day the temperature remained normal or sub-normal. Spleen still palpable. The parasite was readily demonstrated in stained and fresh specimens during the whole day. A treatment of quinine grs. v three times a day was adopted, and continued during the following week.

June 5th. No recrudescence of fever. Parasites, though fewer, were still present. The spleen was no longer palpable nor tender.

June 12th. During the week there was no recurrence of fever and I am in normal health. Quinine grs. v has been taken three times daily. A further treatment of quinine grs. v three times a day on Sundays for the next three months will be adopted.

From a consideration of the facts above recorded, and from the temperature chart, it will be seen that the case was one of simple benign tertian malaria, the original infection in September, 1900, having been of the double tertian type.

I am indebted to Professor Hamilton, Professor of Pathology, Aberdeen University, for confirming the presence of the parasite both in fresh and stained blood films; to Dr. Duncan, Assistant to the Professor of Pathology, Aberdeen University, for preparing films, staining and confirming the presence of the parasite; and to Mr. A. L. N. Maclean, of Aberdeen University, for preparing films. Dr. Manson, of the London School of Tropical Medicine, to whom stained specimens were sent, also confirmed the presence of the parasite of benign tertian malaria.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

A RECTAL PROSTATIC SINUS.

THESE notes may be worth recording. A. B., aged 30, came to me over two years ago on account of a frequently recurring discharge from the rectum, and various unpleasant sensations, such as "stabbing" and throbbing about the prostatic locality, which induced a state of almost confirmed melancholia. I had treated him six years before for gonorrhœal prostatic gleet, but he never had a prostatic abscess.

On examination I found a perfectly white discharge oozing from a small sac about half an inch from the margin of the anus. Pressure on this sac caused about a drachm of the fluid to exude. The prostate seemed normal to touch. A probe passed into the sac went three-quarters of an inch in the direction of this gland. The microscope showed pure prostatic secretion, but no pus cells.

I told my patient, who was exceptionally intelligent, that I was doubtful as to the course to pursue, for if I opened up and packed the sinus, I was afraid the discharge might still continue. I told him I would first try the passage of large sounds, with the possibility of encouraging the secretion to escape through its natural channels by opening the ducts, and also to remove inflammatory relics.

This treatment did no good whatever. It was suggested that the regular performance of the function of the sexual organs, by keeping the plethoric prostatic ducts less full, might prevent the secretion appearing at the abnormal site, and such was the case. Yet the consciousness of the existence of the abnormality in itself kept my patient on the confines of insanity. Emaciation and insomnia became marked, and I feared suicide. It was remarked there was a certain periodicity about the escape of the fluid at the sinus. My patient next saw a distinguished surgeon. The latter naturally enough considered that he had an abscess, and proposed to lay it open. This was done.

This treatment was also ineffectual. I therefore tried the more radical expedient first suggested—that is, I slit up not.