A CASE OF ACUTE PEMPHIGUS.

BY WILFRED HADLEY, M.D. DURH., F.R.C.P. LOND.,

ASSISTANT PHYSICIAN TO THE LONDON HOSPITAL;

AND

WILLIAM BULLOCH, M.D. ABERD.,

BACTERIOLOGIST TO THE LONDON HOSPITAL.

IN 1895 G. Pernet¹ described a case of rapidly fatal pemphigus in a journeyman butcher, and Pernet and Bulloch² from a study of the literature of the subject were able to bring together eight similar cases of this rare disease all occurring in butchers and following a lesion usually of the fingers or hand. The present case is submitted as a further confirmation of this remarkable coincidence.

The patient, a man, aged 23 years, was admitted into the London Hospital on June 4th, 1898. He was a powerfully developed man and a butcher by occupation. He had wounded his right thenar eminence on a piece of jagged bone while dressing the carcass of a sheep. The wound, which was over an inch in length, bled a little and afterwards discharged a small quantity of pus. On the third or fourth day he observed some blisters on his chin and anterior nares, followed on the next day by a bullous eruption on the back of the neck and later on the trunk, abdomen, and thighs. On the sixth day from the onset he was admitted into the hospital, when he presented the following condition. Around the anterior nares and on the chin the eruption had formed impetiginous crusts, as also one patch on the forehead. On the rest of the face, on the forehead, and on the scalp large numbers of bullæ were observed, some of which had burst. In size they varied from that of a pea to that of a large walnut. Between the shoulders several large bullæ had run together and had burst. Opposite the angles of the scapulæ were some raised red patches which were not accompanied by pain or itching. On the part of the chest and abdomen, especially in the neighbourhood of the pubes and also on the inside of the thighs and on the legs, the bullæ were very numerous and partly confluent. In the axillæ the bullæ were very large and tense, and they were numerous on the arms and hands, the palms being extensively affected. Ruptured bullæ were also observed on the dorsum and right side of the tongue and on the left pillar of the fauces. Two small ulcers on the inner side of the left leg were said to be caused by a kick received a month previously. Only a few of the bullæ were surrounded by an inflammatory areola. The patient did not complain of pain and his viscera were apparently normal. The temperature on admission was 101.2° F. He was ordered boric baths and boric ointment, and arsenic and quinine were administered internally. During the subsequent days the temperature fluctuated between 99.4° (morning) and 103° (evening). Fresh crops of bullæ developed and his condition became serious. Vomiting and severe diarrhoea supervened, the pulse became frequent and feeble, and the respirations shallow, and he died on June 11th, seven days after admission and 13 days from the development of the first symptoms. On the day before death—i.e., on June 10th—the temperature was 99.0° in the morning and 101.6° in the evening. On the morning of June 11th it was 101.2°.

The post-mortem examination made by Dr. Schorstein 36 hours after death showed a few adhesions of the right pleura. The spleen weighed six and a half ounces and was soft. The kidneys, which weighed 11 ounces, were in a state of cloudy swelling. The weight of the liver was 4 lb. 4 oz.; it was slightly fatty. The mesenteric glands were enlarged and softened. In one or two there were likewise a few minute hæmorrhages. The solitary follicles along the greater part of the large intestine and for three feet above the ileo-cæcal valve were enlarged and the mucous membrane was congested.

Two days before death a bacteriological examination was made of three recent bullæ (several hours old). The surface of the bulla was touched with a hot glass rod and a quantity of the clear, limpid fluid was aspirated into Pasteur's

pipettes. A number of cover-glass preparations were made. The fluid contained a diplococcus staining by Gram's method and the leucocytes present were almost exclusively eosinophile. The fluid of the blisters was inoculated into bouillon and gelatin, and agar plates were also poured. In 24 hours a pure culture was obtained of the above-mentioned diplococcus which in subsequent sub-cultures appeared to be identical with the coccus isolated from similar cases of acute pemphigus by Demme,³ Bleibtreu,⁴ Bulloch, and Russell Wells.⁵ The diplococcus, as far as could be ascertained, differed very considerably from any of the well-known pathogenic cocci. Two cubic centimetres of the fluid of a bulla were injected intravenously into a rabbit, but without result. One cubic centimetre of the first bouillon culture likewise gave negative results.

The examination of the liver, spleen, kidneys, and intestines of the patient did not yield any characteristic results. Bacterium coli commune was isolated from the spleen and from the liver (probably post-mortem invasion). No diplococci were found in the heart blood or in the sections or cultures from the viscera. The kidneys presented the appearance of severe cloudy swelling. The epithelium of the intestinal mucous membrane over the reddened area was found to be desquamated and the vessels in this coat as well as in the submucosa were widely dilated and gorged with blood. Hyperplasia of the lymphoid tissue of the intestinal wall and of the mesenteric glands was very marked. It is to be regretted that a bacteriological examination of the blood was not made during life. The condition of the patient was not considered very serious at the time and the examination was delayed.

This case resembles very closely several of the cases collected by Pernet. The disease ran an acute course and was preceded by a definite wound which probably had some relation to the symptoms. The patient was a strong young butcher who had previously enjoyed excellent health.

In regard to the occurrence of the diplococcus mentioned above it has not been proved that it is the cause of acute pemphigus. Its absence in the internal organs would seem to show that the disease is to a large extent a toxic one, the toxin being probably bacterio-genetic.

RED-WATER OR TEXAS FEVER: A FURTHER COMMUNICATION.¹

BY ALEXANDER EDINGTON, M.B., F.R.S. EDIN.,

DIRECTOR OF THE BACTERIOLOGICAL INSTITUTE, CAPE COLONY.

SINCE my communication² to the Royal Society of London by Professor Thomas R. Frazer I have been able to obtain valuable additional evidence as to the communicability of the disease by the use of blood derived from animals which have either recovered from the sickness for a very considerable

³ Beiträge zur Kenntniss des Pemphigus Acutus, Verhandlungen des Congresses für Innere Medicin, Wiesbaden, 1886.

⁴ Beitrag zur Kenntniss des Pemphigus Acutus, Berliner Klinische Wochenschrift, Band xxx., p. 671.

⁵ Two Cases of Acute Pemphigus, THE LANCET, May 2nd, 1896.

¹ Communicated to the Royal Society by Dr. Gill, F.R.S., on April 20th, 1899.

² The conclusions arrived at in that communication (June 6th, 1898) were as follows: 1. The blood of animals themselves healthy from a red-water area is dangerous if inoculated into an animal which suffers coincidentally from another disease. 2. That the blood of animals suffering from mild or modified red-water may be safely used to inoculate a healthy animal subcutaneously but is dangerous when injected into a vein. 3. That the subcutaneous inoculation of mild or modified red-water blood conveys a mild form of the disease and since the blood of such an animal is virulent when injected into a vein in another animal it is safely to be inferred that the animal suffering from the mild form becomes more or less immunised or "salted." On these grounds I would suggest a method of protective inoculation against red-water in the following manner. Having procured a healthy animal from a red-water area or one which is known to have been "salted," inoculate it by injecting five cubic centimetres of red-water blood into the jugular vein and five cubic centimetres subcutaneously. In cases where the operator is unable to attempt the vein inoculation I would recommend the subcutaneous inoculation of five cubic centimetres in four different sites. Allow at least 28 days to elapse and if any degree of illness is recognised the blood of this animal may be used, after being defibrinated, to inoculate healthy cattle. For such inoculation only five cubic centimetres should be injected into small animals and not more than 10 cubic centimetres into larger. Seeing, however, that the presence of other maladies renders such a proceeding unsafe I would recommend that it should only be practised during the autumn or winter when the veldt diseases are, as a rule, in abeyance and in no case when any epidemic disease is in the near neighbourhood.

¹ The Etiology of Acute Pemphigus, Brit. Med. Jour., Dec. 21st, 1895.

² Acute Pemphigus, a contribution to the Etiology of Acute Bullous Eruptions, British Journal of Dermatology, 1896, vol. viii., p. 157.

time or which have been inoculated many months previously to the date on which their blood has been used.

On Dec. 8th, 1898, I withdrew some blood from animal No. 18, which has been continuously under observation since it was inoculated on Dec. 22nd, 1897. After defibrinating the blood 20 cubic centimetres were used to inoculate a young ox (No. 54) by intravenous injection. On the following day a sharp rise of temperature occurred which reached to 106.6° F. On the following morning it was observed to have fallen to 99.8°. Three days later the temperature was again over 104°, but it fell previously to the next morning. From this time onward an erratic course of temperature was observed and on the twenty-fifth day subsequent to inoculation the animal was seen to be ill; it refused food, but it had no definite symptoms of "red-water." Three days later it died. The blood on examination was seen to contain the spherical forms of the parasite. On post-mortem examination the bladder and urine were found to be quite normal. The liver was not enlarged, but was somewhat discoloured in patches, and the biliary ducts were distended with bile. The bile was much altered, being stringy and of a greenish-yellow colour. The spleen was normal in size and consistence. The kidneys were enlarged and the pelvis were filled up by a yellowish gelatinous exudation. The cortex was somewhat congested, but there was no evidence of any true inflammatory change. The general muscles were pale in colour and there was slight evidence of jaundice. This experiment serves to show that an animal which has been inoculated with infected blood, while it may not develop much illness as a result of it, is really infected and, moreover, its blood, if drawn as late as a year subsequently, is yet so infective that an intravenous injection of it into susceptible animals will certainly infect and may even kill, although after a somewhat extended period of time.

Very important corroboration of this is furnished by the experience of inoculation for red-water which has lately been adopted in the Cape Colony. Four animals which were immune to red-water (three by reason of having had the disease and recovered from it and one by being born and reared on permanently infected veldt) were sent from Fort Beaufort to Queenstown to be used by the veterinary surgeon there for inoculation purposes. The animals to be used for inoculation had been "fortified"—i.e., re-inoculated with virulent blood—seven weeks previously. 20 animals were inoculated with defibrinated blood from one animal, the doses used being from 10 to 20 cubic centimetres, according to age. All had a febrile reaction and some slight symptoms of the sickness, but they easily recovered. From one of the other of the four animals blood was taken and used to inoculate seven head, giving doses of from 10 to 15 cubic centimetres. These also all had a reaction, but they made good recoveries. On Nov. 1st the four animals were re-inoculated with virulent red-water blood, and in each case five cubic centimetres were injected intravenously and 10 cubic centimetres subcutaneously. 29 days later they were bled. With this blood two lots of cattle were inoculated. One lot consisted of 107 animals which had never been exposed to red-water infection. The doses used were increased beyond those which I had recommended—namely, from 10 to 25 cubic centimetres were used, according to age. Of these animals no less than 17 died from characteristic red-water. The remainder made good recoveries. The second lot consisted of 53 head of cattle, all of which with one exception (an imported animal) had been born and reared on red-water veldt. The imported animal was the only one which showed any signs of reaction, but it made a good recovery. This experience has sufficed to show that it is not always safe to exceed the doses which I have recommended unless the animals which have been used for withdrawing blood have been untouched for at least a considerable number of months.

I have been able, with the coöperation of several farmers, to carry out experiments by which inoculated cattle have been fully exposed to infection at later dates. In May, 1898, I inoculated 10 head of old cattle with blood from an animal which had been inoculated six months previously with virulent blood. These cattle were immediately removed from the Institute and sent later to an infected area in company with 10 head of young animals which were uninoculated but, as is commonly known in this colony, are not so liable to death from this disease as are older animals. Of the young stock all have been infected by exposure in the veldt and three have died. Of the older and more susceptible animals not one has shown the slightest signs of

illness and the cows have given birth to healthy calves. Mr. J. H. Webber had 28 head of Fish River cattle inoculated on Nov. 7th, 1898, and subsequently had them removed to his farm, which is well known to be one of the worst infested areas in the Eastern Province. Previous experience has shown that if clean cattle are placed there they become very quickly affected with the disease. On Dec. 5th one died from gall-sickness, but with this exception all have done very well and are at this date in perfect health.

This method of inoculation has proved so satisfactory to the farmers themselves that it is being very generally adopted and the farmers have petitioned the Government to arrange for an inoculating station being placed at Grahamstown, so that clean cattle coming from clean Karroo areas for transmission to the coast may be inoculated previously to entering the infested belt.

A CASE OF RECURRENT APPENDICITIS; OPERATION; RECOVERY.

By W. JONES GREER, F.R.C.S., D.P.H. IREL.

THE patient in this case was a well-formed muscular youth, 18 years of age, who had had fairly good health with the exception of oft-recurring severe symptoms which could readily be connected with his present illness. The cause of his trouble was, he thought, an attack of faecal obstruction which he had had about 10 years before—at least, I judge it to be faecal obstruction from the treatment which his mother tells me was adopted. His life was despaired of, but it was saved by a "white powder" and "a large enema of castor oil." Since this time he had always had spasms of sharp pain in the right side after even slight exertion or prolonged standing which his work necessitated. During the last two years his symptoms had become of a more severe character. Hardly a day had passed without his having a sharp attack of pain. Obstinate constipation had been the rule; in fact, he believed the bowels could never have acted without the use of some aperient. If he neglected to secure a daily evacuation the pain became so severe as to require his remaining in bed for a day or more, and this neglect was, he thought, the primary cause of the four severe attacks of illness from which he suffered for two years. In July, 1898, I was asked to see him when he was then undergoing his third attack. His condition was typical of appendicitis. He was lying on his back with the right knee drawn up, his face was pale, his expression was anxious, and his tongue was thickly coated with a creamy fur. He suffered from constipation, his abdomen was distended and tender with constant pain, the point of maximum intensity of which was over McBurney's spot, where even the lightest touch with the finger caused great agony. The pulse which was small and hard varied in rate between 100 and 130, and the temperature range on more than one occasion touched 104° F. The patient was obviously dangerously ill and the prospect of recovery without operative interference was not great. I put the question of operation before the patient and his friends, but they declined it either as a deliberate procedure or, should circumstances of emergency occur, as a life-saving expedient—they would take whatever chance medical means could afford. The treatment adopted was to continue the efforts which had been made to keep the bowels unloaded and to relieve pain by means of subchloride of mercury, sulphate of magnesia, and opium, the patient being kept on a fluid diet. After over three weeks of an exhausting illness the patient began gradually to recover. When all the severe and acute symptoms had subsided I made a careful examination of the right iliac fossa. The result was not very satisfactory. No distinct tumour could be felt, but there was a sense of resistance over the region of the appendix and this, taken in conjunction with the idea which I entertained that there was probably an abscess, would readily make one feel a tumour, but I was not satisfied of its existence. Examination per rectum also gave no help. The final upshot of this attack was that he returned to business comparatively well. In January, 1899, I saw him again in his fourth severe attack. He told me that since his third severe attack he had had four or five smart spasms of pain which necessitated his lying in bed for at least one or two days at a time. This fourth attack,